Chapter 16

ENVIRONMENTAL CONDITIONS

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Multiple Lightning Strikes. (Photo contributor: Lawrence B. Stack, MD.)

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HIGH-ALTITUDE RETINAL HEMORRHAGE

Clinical Summary

Retinal hemorrhages are common above 5200 m (17,000 ft) and are not always associated with acute mountain sickness (AMS). High-altitude retinal hemorrhages (HARH) are rarely symptomatic, but if found over the macula, these hemorrhages may cause temporary blindness. The diagnosis can be established by ophthalmoscopy. Without visualization of the lesion, the differential diagnosis of unilaterally decreased vision or blindness at high altitude includes migraine equivalent, cerebrovascular accident, and dry eye (often unilateral, due to strong winds), as well as all conditions found at sea level.

Management and Disposition

HARH generally resolve spontaneously after descent to lower altitudes. No treatment is necessary for asymptomatic HARH. Patients with HARH associated with a decrease in vision should be referred to an ophthalmologist for follow-up.

Pearls

1. Patients with blurred vision and unilateral mydriasis at the high altitude should be asked about use of medications, including transdermal scopolamine patches.
2. As with almost all altitude-related problems, descent is the primary treatment. This is not emergent unless associated with severe altitude illness or progressive visual loss.
3. Although most symptomatic HARH resolve completely in 2 to 8 weeks, cases of permanent paracentral scotomata have been reported.

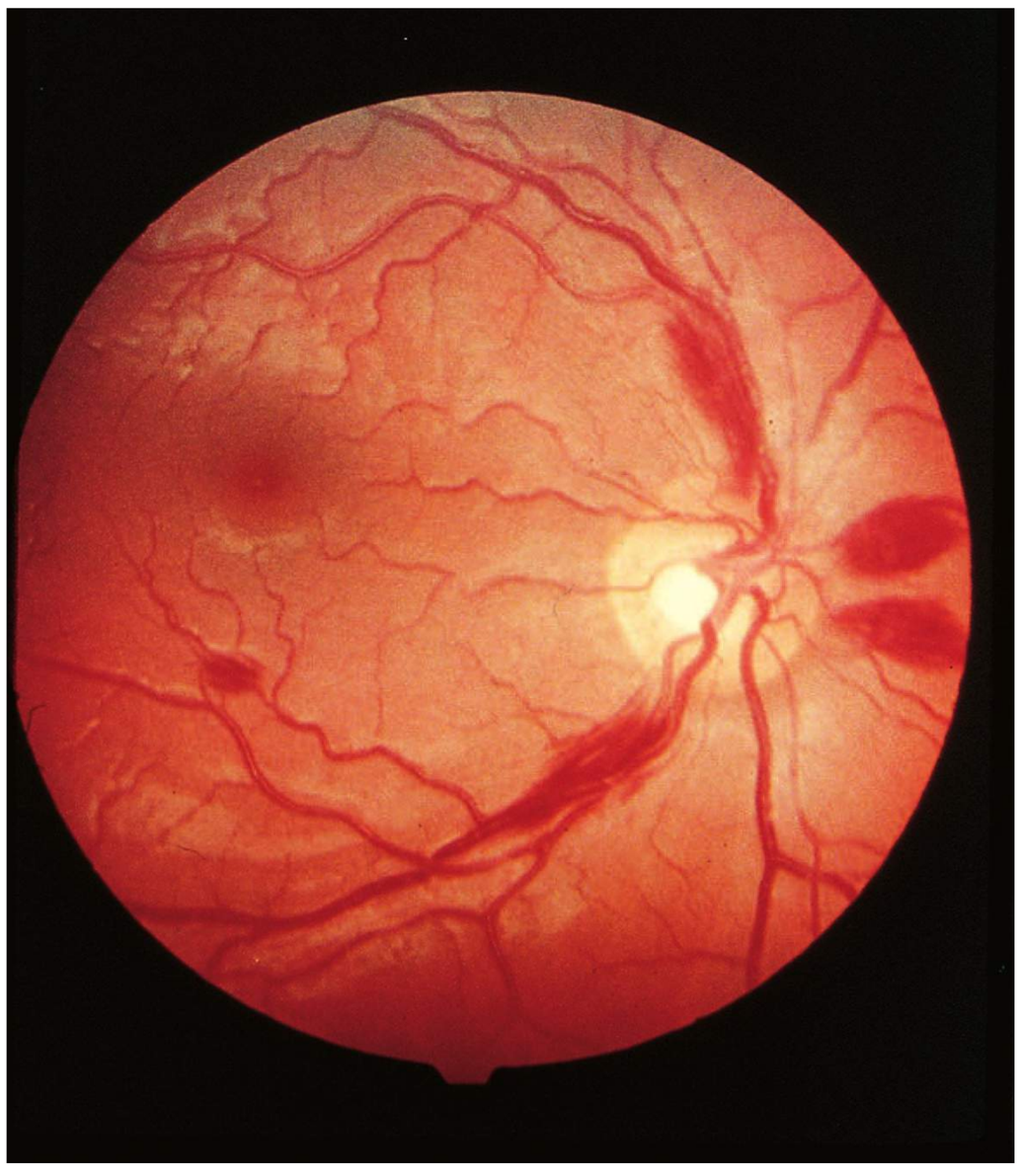

FIGURE 16.1 ■ **High-Altitude Retinal Hemorrhage.** Fundoscopic appearance of high-altitude retinal hemorrhage. (Photo contributor: Peter Hackett, MD.)

Clinical Summary

High-altitude pulmonary edema (HAPE) is a form of noncardiogenic pulmonary edema, generally beginning within the first 2 to 4 days after ascent above 2500 m (8200 ft). The earliest symptoms are fatigue, weakness, dyspnea on exertion, and decreased exercise performance. Symptoms of Acute mountain sickness (AMS) such as headache, anorexia, and lassitude may also be present, but HAPE may develop without AMS. First symptoms usually include persistent dry cough and dyspnea followed by tachycardia, tachypnea, and cyanosis at rest. Patients suffering from HAPE often experience sudden onset of symptoms upon awakening after the second night at altitude. Eventually the victim develops dyspnea at rest and orthopnea with audible crackles in the chest. Pink frothy sputum is a grave sign. Patients may experience concurrent mental status changes and ataxia due to hypoxemia or associated high-altitude cerebral edema (HACE).

Management and Disposition

Mild cases (oxygen saturation in the 90s on low-flow oxygen) at moderate altitudes (below 3500 m, 11,500 ft) may be treated at altitude with bed rest and oxygen. If supplemental oxygen and a reliable person are available, the patient may be discharged with oxygen therapy and bed rest at home or in lodgings. Patients with more severe cases should descend immediately. These patients may require admission to a hospital at a lower altitude and, in extreme cases, intubation and mechanical ventilation. Nifedipine is of benefit but is not a substitute for descent. Some experts now use PDE-5 inhibitors such as sildenafil or tadalafil instead of nifedipine. Hyperbaric therapy, especially with a portable hyperbaric chamber (Gamow bag), has an efficacy equal to that of supplemental oxygen and is mainly helpful in prehospital settings where oxygen availability is limited.

Pearls

1. Crackles may be unilateral or bilateral but usually start in the right middle lobe and are heard first in the right axilla.
2. HAPE limited to the left lung in association with a small right hemothorax without pulmonary markings is pathognomonic for unilateral absent pulmonary artery syndrome. These patients develop HAPE at relatively low altitudes, sometimes below 2500 m.
3. Patients treated for HAPE may resume normal activities once they are asymptomatic and may ascend further during the same trip.

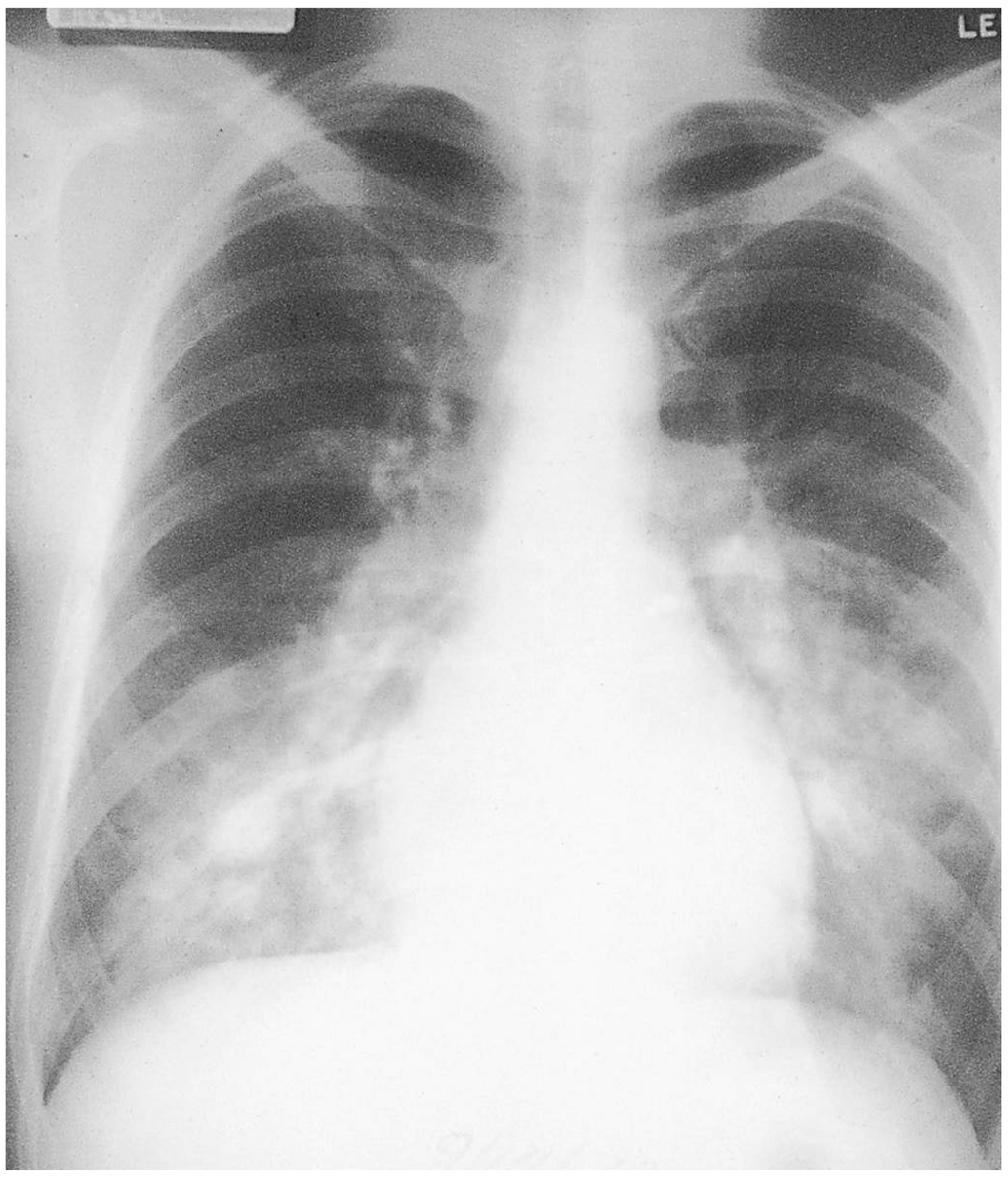

FIGURE 16.2 ■ **High-Altitude Pulmonary Edema.** Chest x-ray in patient with HAPE. Note normal heart size with bilateral "patchy" pulmonary infiltrates. (Photo contributor: Peter Hackett, MD.)

FIGURE 16.3 ■ **"Gamow Bag."** Portable hyperbaric chamber (Gamow bag). A HAPE patient is being treated at 4300 m at Pheriche, Nepal. Due to orthopnea, the patient was unable to tolerate lying flat, so the bag was propped up immediately after inflation. (Photo contributor: Ken Zafren, MD.)

HIGH-ALTITUDE CEREBRAL EDEMA

Clinical Summary

Acute mountain sickness (AMS) is a symptom complex which usually begins 12 to 24 hours after ascent to high altitude and consists of headache and one or more other symptoms, including gastrointestinal symptoms, fatigue and/or weakness, dizziness and/or lightheadedness, and difficulty sleeping. High altitude cerebral edema (HACE) is a severe form of AMS, clinically defined by the presence of acute truncal ataxia, altered mental status, or both. Usually this occurs as a progression from AMS to HACE, but HACE may occur without antecedent AMS. If not effectively treated, HACE may progress to coma or death. Focal neurologic findings other than truncal ataxia are rare and should suggest another diagnosis, such as acute stroke or venous sinus thrombosis.

Management and Disposition

Treatment of HACE consists of immediate descent or evacuation, oxygen, and high-dose dexamethasone. Simulated descent using a portable hyperbaric bag (Gamow bag) may be more effective than oxygen alone and may be used in place of oxygen in field settings. Actual descent may be complicated by the inability of the patient to walk unassisted or at all. Patients with HACE may not ascend during the same trip and probably should not reascend to altitude for several months.

Pearls

1. The first sign of HACE is usually truncal ataxia. This should be tested using tandem gait (heel-to-toe walking).
2. HACE may occur without symptoms of AMS.
3. HACE is often associated with some degree of HAPE.

FIGURE 16.4 ■ **HACE-Related Ataxia.** Ataxia due to HACE in the Nepal Himalayas. The patient (middle) developed HAPE and severe truncal ataxia at 5600 m (18,400 ft). The patient required full ambulatory assistance. Descent was started immediately. With descent, the patient's breathing improved, but he remained too ataxic to walk. (Photo contributor: Ken Zafren, MD.)

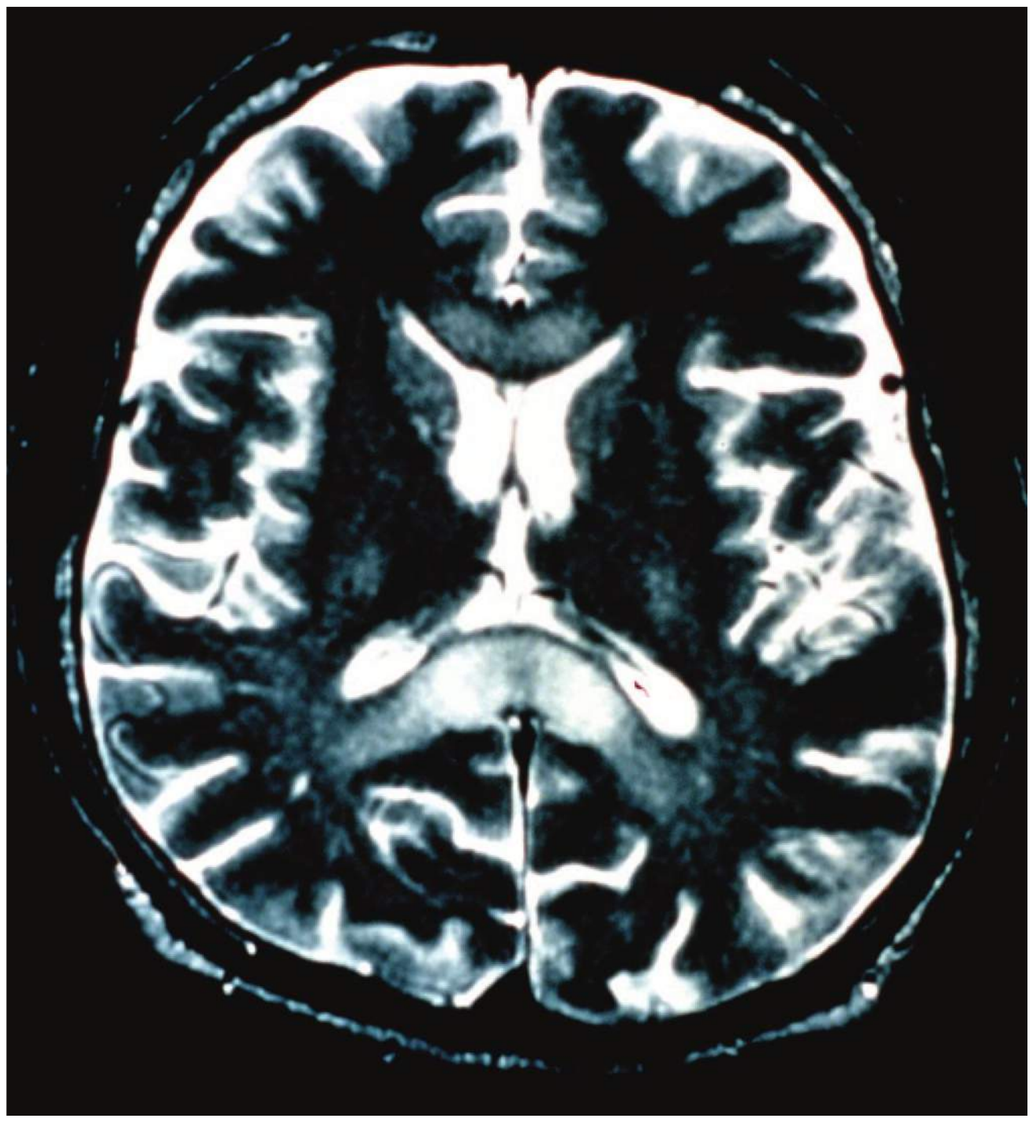

FIGURE 16.5 ■ **MRI Brain—HACE.** MRI of a patient with high-altitude cerebral edema. Note the increased white matter signal, especially in the splenium of the corpus callosum, in this T2-weighted image. (Photo contributor: Ken Zafren, MD.)

Clinical Summary

Accidental hypothermia is an unintentional decline in core temperature below 35°C (95°F). Presentation may be obvious or subtle, especially in urban settings. Symptoms vary from vague complaints to altered levels of consciousness. Physical findings include progressive abnormalities of every organ system. Following initial tachycardia, there is progressive bradycardia (50% decrease in heart rate at 28°C [82.4°F]) with decline in blood pressure and cardiac output. ECG intervals are prolonged, beginning with the PR interval followed by the QRS interval and finally the QT interval. A J wave (Osborn wave; hypothermic "hump") may be seen, but is neither pathognomonic nor prognostic. The J wave is present at the junction of the QRS complex and the ST segment. J waves may also be associated with central nervous system lesions, focal cardiac ischemia, young age, and sepsis. In mildly hypothermic patients, invisible preshivering muscle tone may obscure P waves.

Management and Disposition

Core temperature measurement is best made with an esophageal probe inserted into the lower third of the esophagus. Rectal temperature is less accurate without the use of a special low-reading thermometer. Gentle handling and appropriate warming methods are the mainstays of emergency department (ED) treatment. Cardiovascular instability often complicates rewarming; Advanced Cardiac Life Support (ACLS) guidelines for hypothermia provide guidance. If not obvious, a precipitating cause should be sought (eg, hypothyroidism, hypoglycemia, sepsis), as should associated pathology. Most patients require admission for observation or to treat associated injuries or comorbidities.

Pearls

1. The most common problem with the misdiagnosis of hypothermia in the ED stems from incomplete data on vital signs.
2. Accurate core temperatures, preferably by esophageal probe, and continuous cardiac monitoring are crucial to appropriate management.
3. Atrial arrhythmias are generally benign and should not be treated. They resolve with rewarming.
4. Most cardiovascular drugs are inactive during hypothermia and should be given only once the body temperature is above 35°C (95°F).

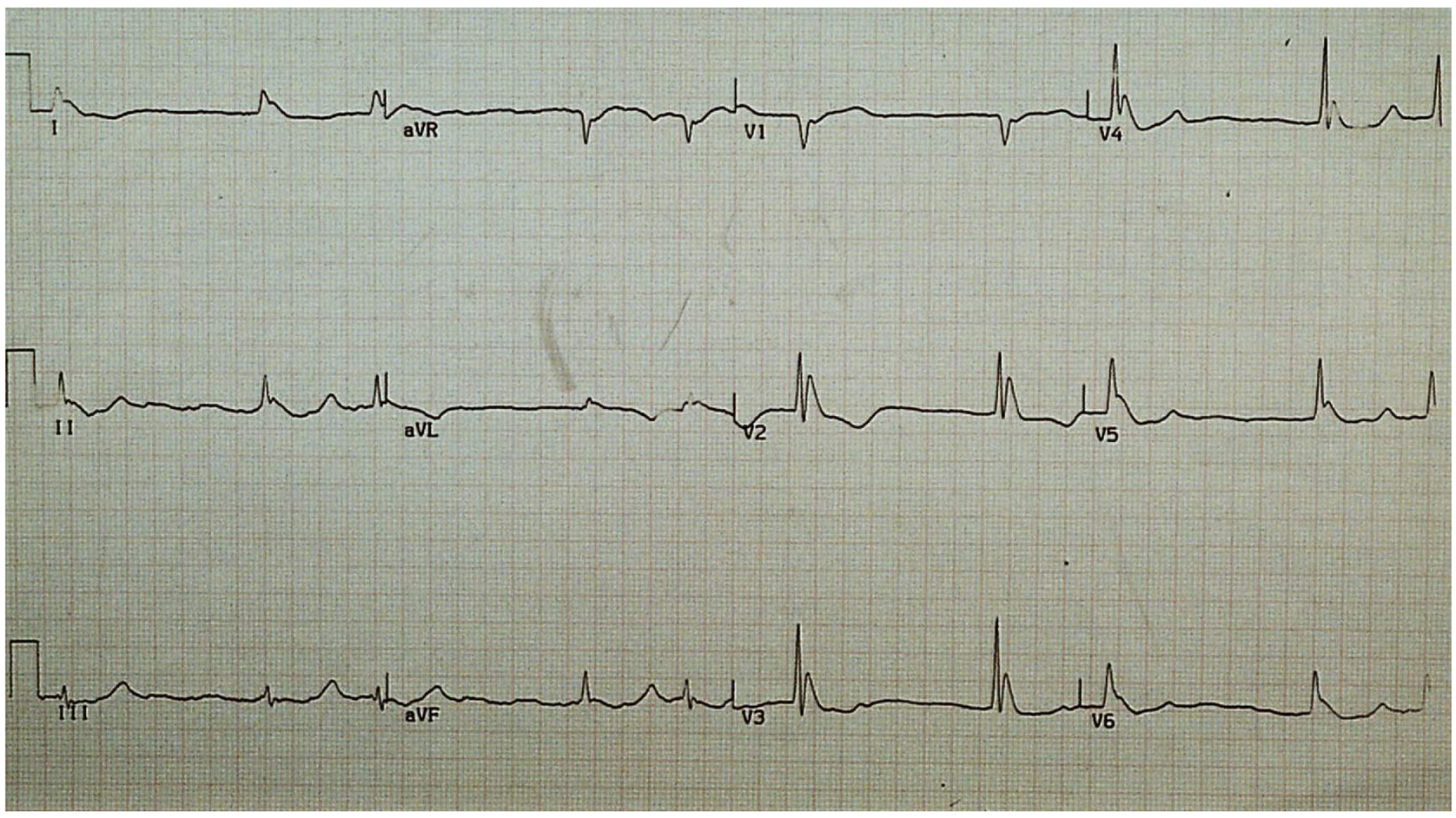

FIGURE 16.6 ■ **J Waves.** J waves in a hypothermic patient with core temperature (rectal probe) of 25.5°C. J waves may be seen at any temperature below 32.2°C, most frequently in leads II and V_6. Below a core temperature of 25°C, they are most commonly found in the precordial leads (especially V_3 and V_4) and their size increases. J waves are usually upright in aVL, aVF, and the left precordial leads (see also Fig. 23.44A "Hypothermia with Osborne Waves ("J" Waves) Present"). (Photo contributor: Alan B. Storrow, MD.)

Clinical Summary

Frostbite is tissue freezing resulting from heat loss sufficient to cause ice crystal formation in superficial or deep tissue. Frostbite usually affects the extremities, nose, or ears (and the scrotum and penis in joggers). A sensation of numbness with accompanying sensory loss is the most common initial complaint. Often, by the time the patient arrives in the ED, the frozen tissue has thawed. The initial appearance of the overlying skin may be deceptively benign. Frozen tissue may appear mottled blue, violaceous, yellowish-white, or waxy. Following rapid rewarming, there is early hyperemia even in severe cases.

Favorable signs include return of normal sensation, color, and warmth. Edema should appear within 3 hours of thawing; lack of edema is an unfavorable sign. Vesicles and bullae appear in 6 to 24 hours. Early formation of large clear blebs that extend to the tips of affected digits is a good indicator. Small dark blebs that do not extend to the tips indicate damage to subdermal plexi and are a poor prognostic sign. When seen early or after rewarming occurs, frostbite may be indistinguishable from nonfreezing cold injury such as immersion foot. Mixed injuries are common.

Management and Disposition

If other injuries are ruled out by history and physical examination, rewarm frostbitten areas in warm water bath (37°C-39°C [98.6°F-102.2°F]). If associated with severe hypothermia, active core rewarming should precede frostbite rewarming. If swelling occurs, surgical consultation is advisable along with compartment pressure measurement to determine the need for fasciotomy. Admit all patients with associated hypothermia or in whom swelling occurs. Superficial frostbite (minimal skin changes and erythema) may be treated by home care with nursing instructions. Deep superficial frostbite (clear, fluid-filled blebs, swelling, pain) may be treated by home care in a reliable patient. Deep frostbite (proximal hemorrhagic blebs, no swelling, no pulses) mandates hospital admission.

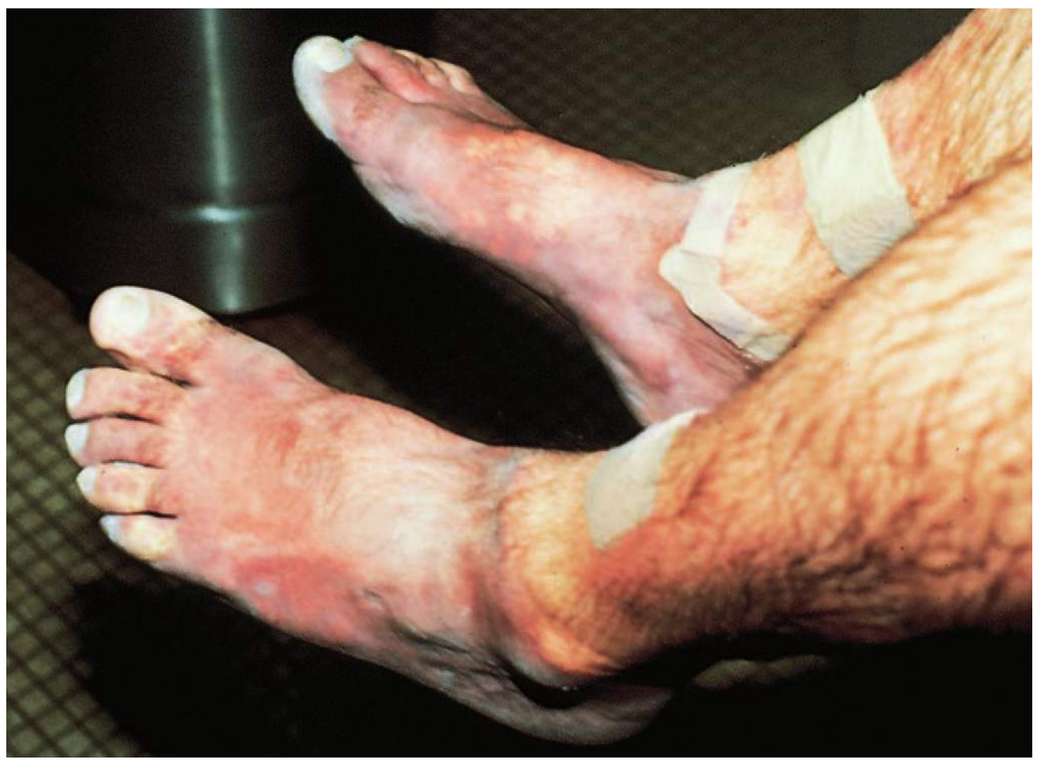

FIGURE 16.7 ■ **Thawed Frostbite.** Typical appearance of frostbite soon after rewarming. Deep frostbite was caused by wearing mountaineering boots that were too tight in extreme cold at high altitude. Note the deceptively benign appearance of this devastating injury, which ultimately resulted in bilateral below-the-knee amputations. (Photo contributor: Ken Zafren, MD.)

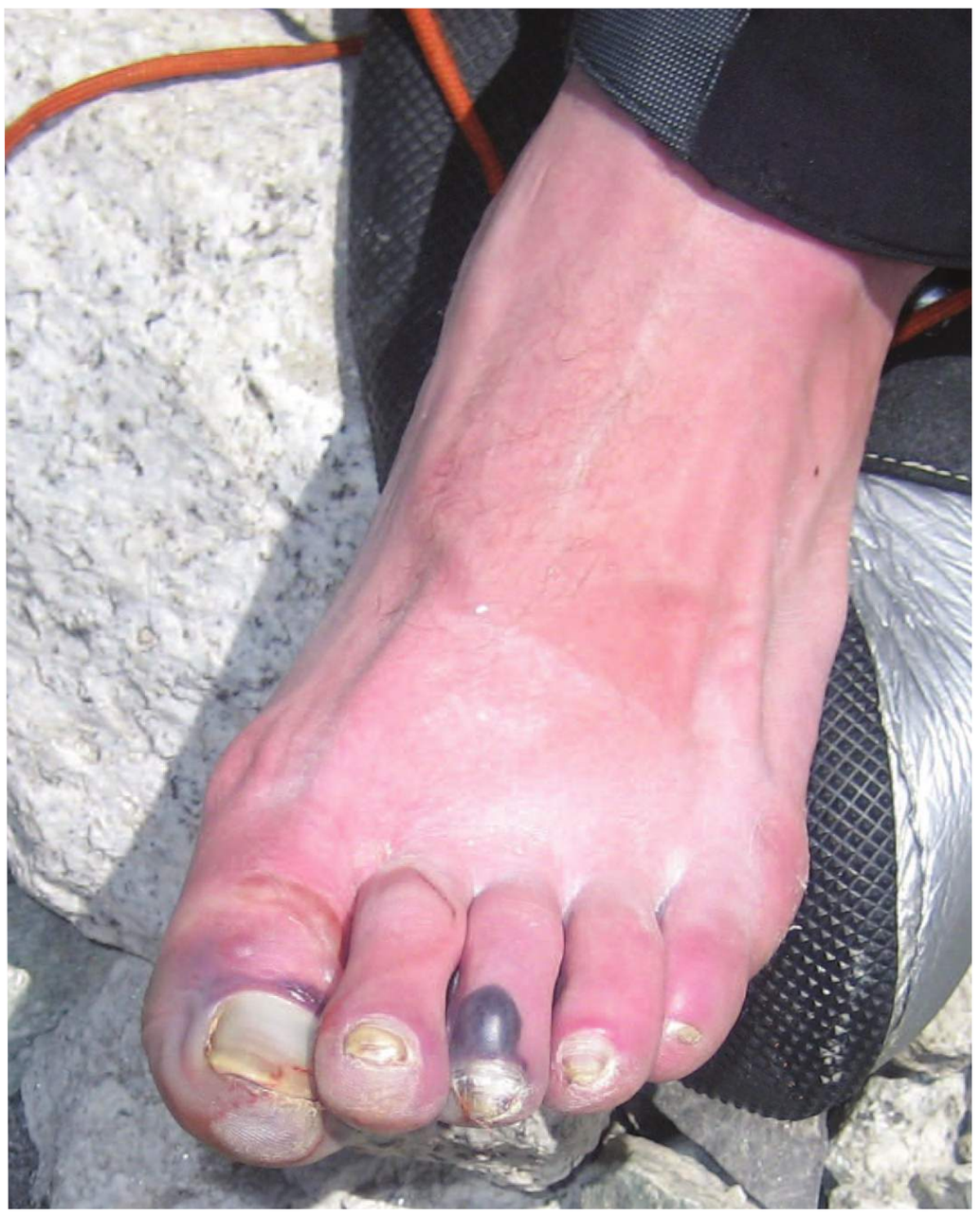

FIGURE 16.8 ■ **Deep Frostbite.** Deep frostbite at Everest Base Camp in Nepal, located at an altitude of 5360 m (17,585 ft), 3 days after exposure at 6400 m (21,000 ft). Dusky appearance and lack of distal blistering on the great toe are poor prognostic signs. The great toe eventually required partial amputation. (Photo contributor: Chris Imray, MD.)

Pearls

1. Early transfer of the patient to a center experienced in the care of frostbite injuries (even if far away) should be considered. On the other hand, transfer of the patient to a major medical center that does not generally manage frostbite is seldom in the patient's best interest.
2. Treatment of clear versus hemorrhagic blisters is controversial; one approach is to debride clear blisters and use topical aloe vera while leaving hemorrhagic blisters intact.

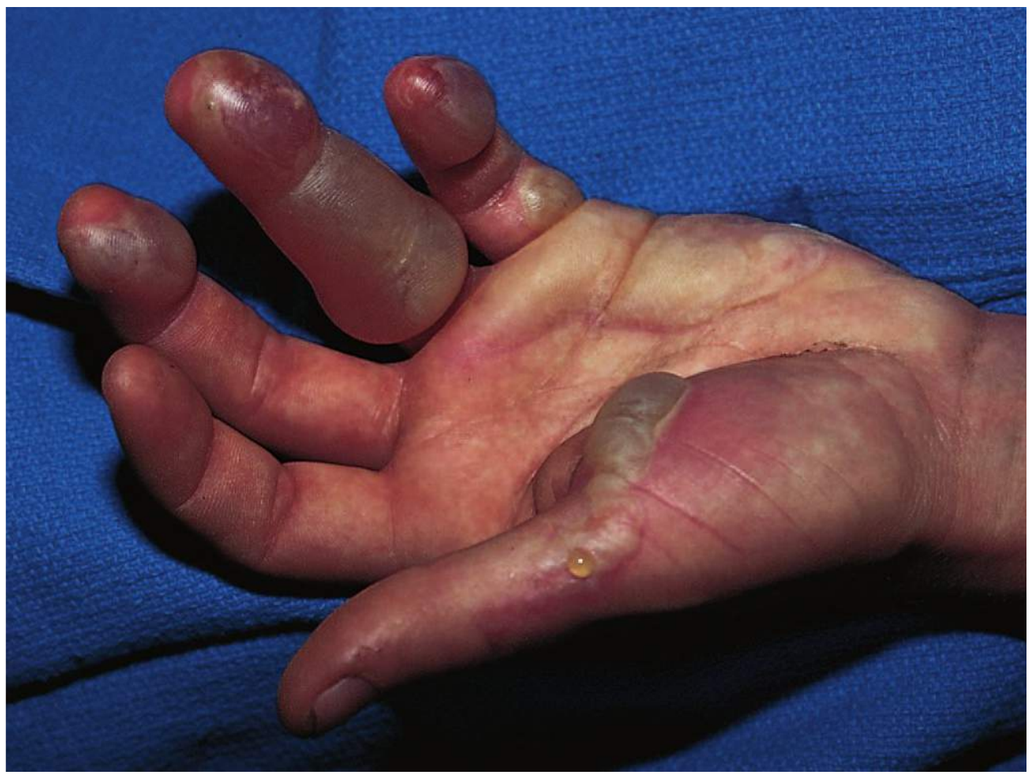

FIGURE 16.9 ■ **Frostbite Blebs.** Intact proximal blebs, both clear and hemorrhagic, indicate deep frostbite and a poor prognosis. (Photo contributor: Scott W. Zackowski, MD.)

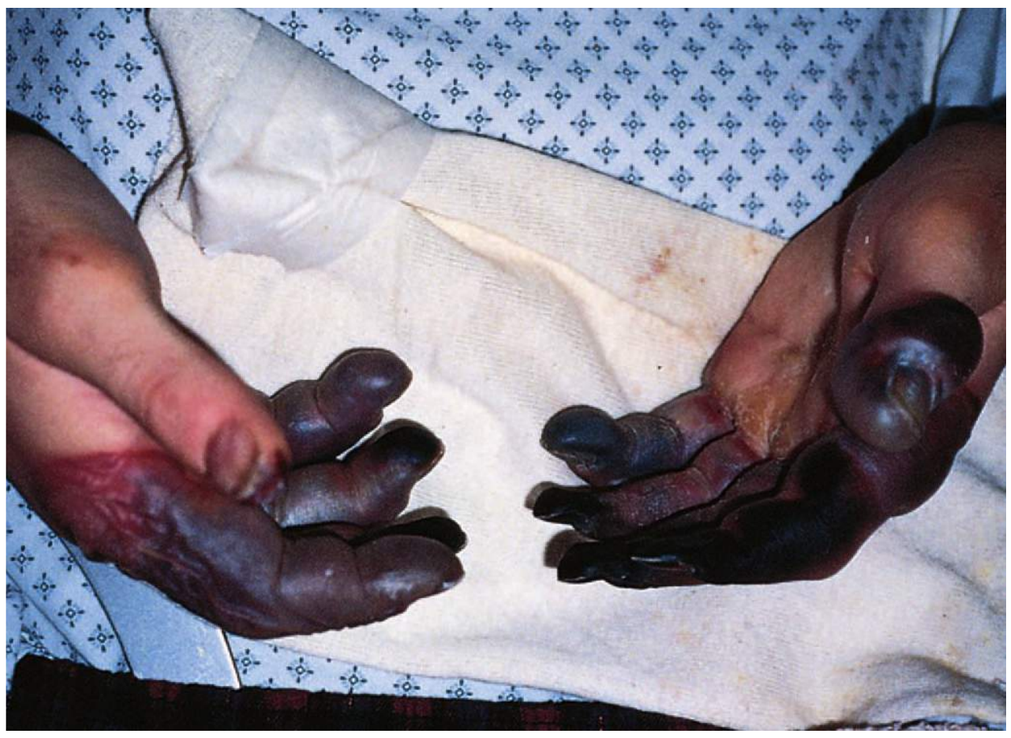

FIGURE 16.11 ■ **Late Frostbite.** Late appearance of frostbite with demarcation starting to occur. Early surgery should be avoided in favor of autoamputation unless infection supervenes. (Photo contributor: James O'Malley, MD.)

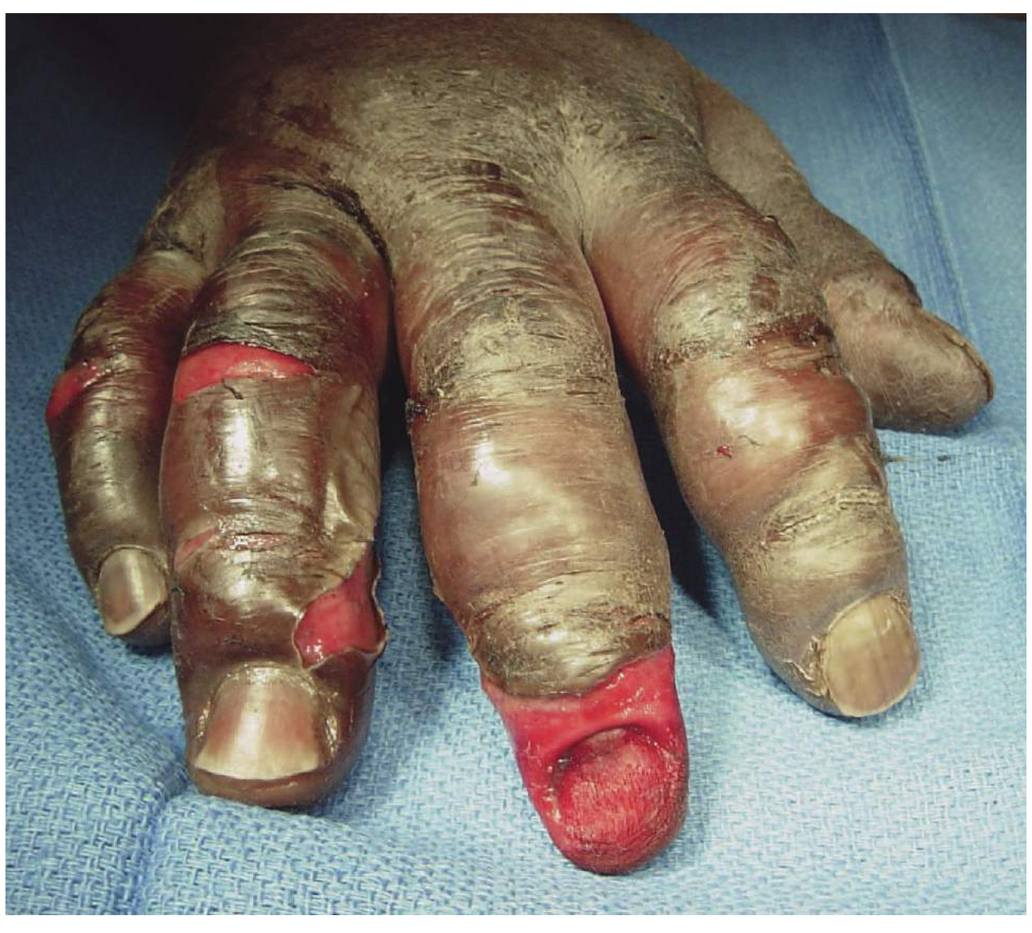

FIGURE 16.10 ■ **Frostbite-Ruptured Blebs.** Frostbite injury to the hand with ruptured blebs. (Photo contributor: R. Jason Thurman, MD.)

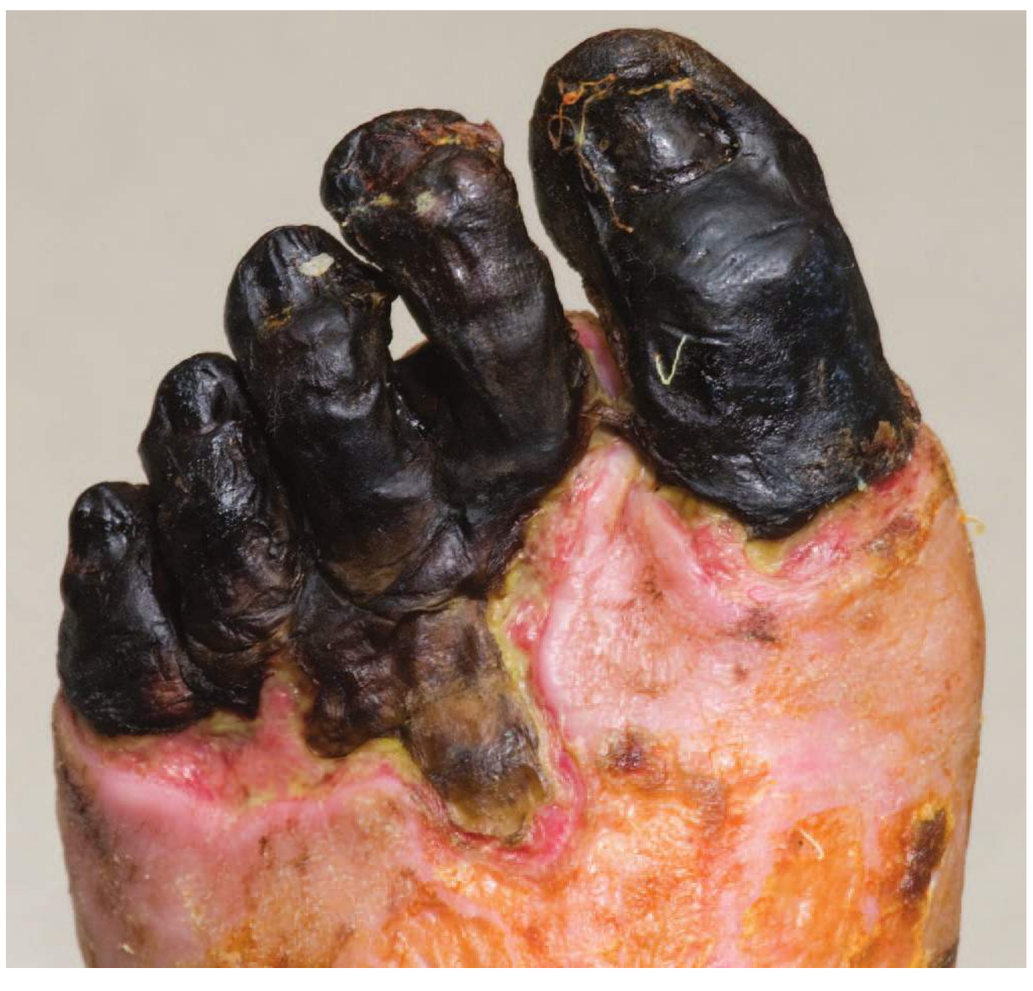

FIGURE 16.12 ■ **Late Frostbite.** Late appearance of deep frostbite with clear demarcation. (Photo contributor: Wael Alrifai, MD.)

Clinical Summary

Pernio, also known as perniosis or chilblains, is the result of nonfreezing cold exposure in susceptible individuals. Pernio appears within 24 hours of cold exposure, most frequently on the face, ears, hands, feet, and pretibial areas. A large range of lesions may be seen, with localized edema, erythema, cyanosis, plaques, and blue nodules occasionally progressing to more severe lesions including vesicles, bullae, and ulcerations. The lesions persist for up to 2 weeks and may become chronic. They are typically very pruritic and associated with burning paresthesias. Following rewarming, pernio often takes the form of blue nodules, which are quite tender. In the setting of recent cold exposure, pernio might be confused with the more severe syndrome of trench foot. If the history of cold exposure is not elicited, the differential diagnosis is potentially very broad.

Management and Disposition

Management is supportive. The skin should be warmed, washed, and dried. Affected extremities can be dressed in soft, dry, sterile dressings and elevated. Nifedipine (20-60 mg daily) may be helpful in chronic cases.

Pearls

1. Healing may be followed by hyperpigmentation.
2. Recurrences are possible following milder cold exposure.
3. Chilblains may be more frequent in young women, especially in association with Raynaud phenomenon, and may also be associated with underlying dermatologic or vascular disease.

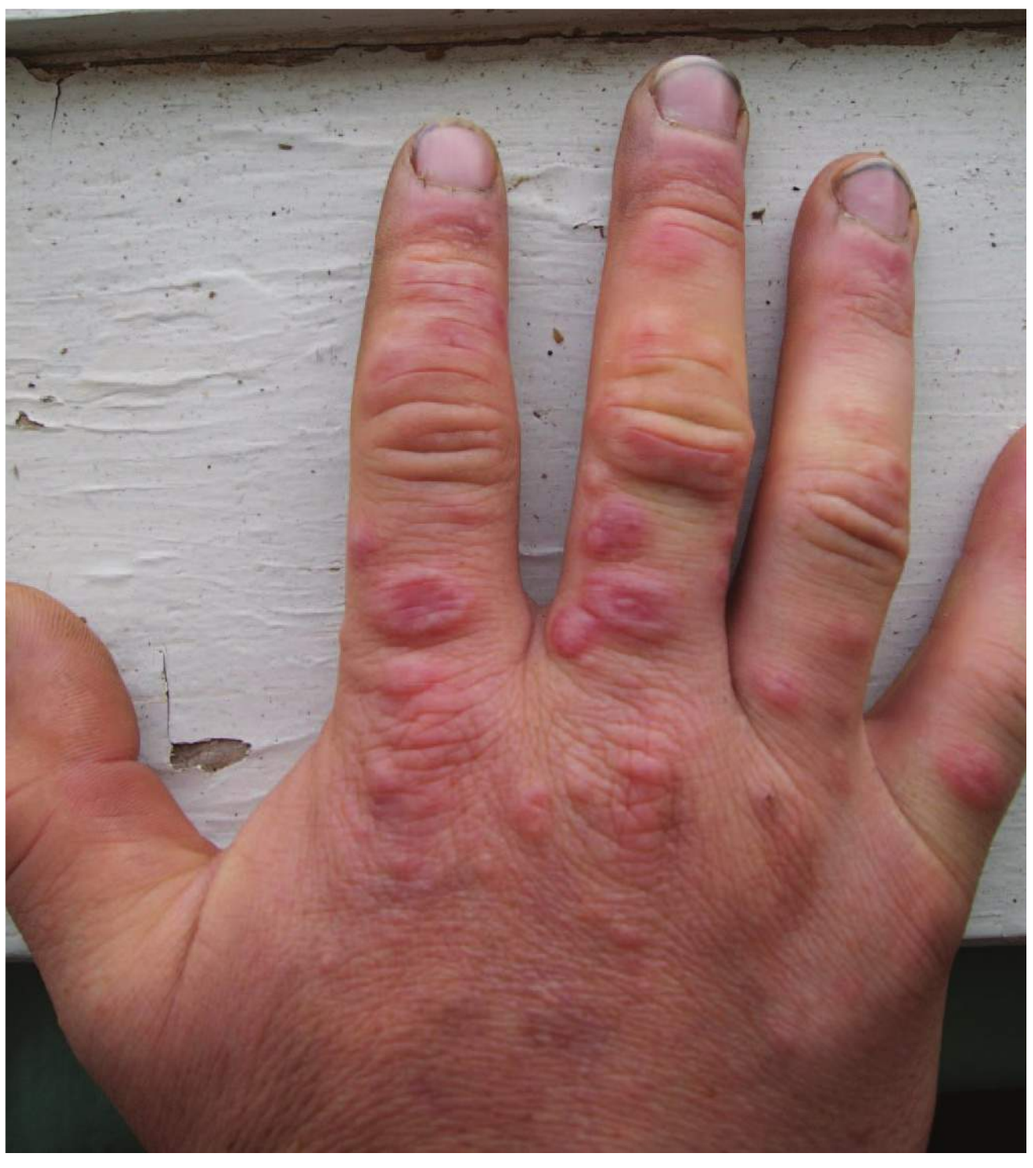

FIGURE 16.14 ■ **Pernio.** Pernio of the hand with localized erythema and painful nodules. (Photo contributor: Alison Jarman, MD.)

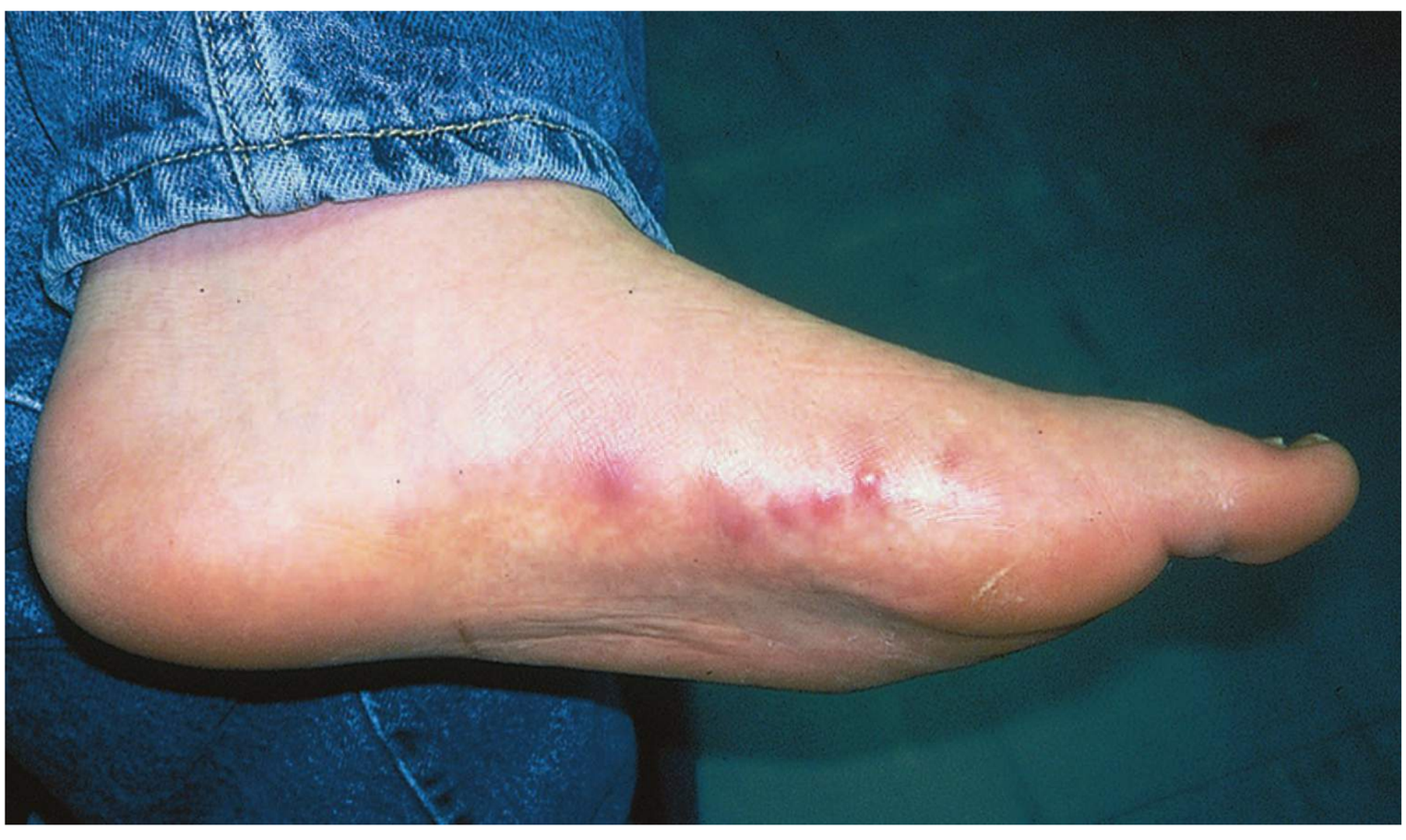

FIGURE 16.13 ■ **Pernio.** Pernio or chilblains with localized erythema, cyanosis, and nodules. (Photo contributor: Ken Zafren, MD.)

IMMERSION INJURY (TRENCH FOOT)

Clinical Summary

Immersion injury is a peripheral nonfreezing cold injury resulting from exposure to water, usually at temperatures just above freezing. However, the condition can occur during prolonged exposure to any cool wet environment. Dependency and immobility predispose to immersion injury. The degree of injury depends on exposure time and temperature. The first symptoms usually appear within hours; tissue loss may occur after many days of exposure. Prior to rewarming, the distal extremities are numb and swollen. The skin is first red, then changes to pale, mottled, or black. Cramping of the calves may occur. Immersion injury is distinct from tropical immersion foot or warm-water immersion foot as seen in the Vietnam War. Tropical immersion foot was typically seen after 3 to 7 days of exposure to water at 22°C to 32°C. Warm-water immersion foot was seen after 1 to 3 days at 15°C to 32°C. These syndromes were characterized by burning in the feet, pain on walking, pitting edema, and erythema, with wrinkling and hyperhydration of the skin. They resolved completely after rest and removal from the wet environment.

Management and Disposition

Hypovolemia, hypothermia, and associated injuries are the rule and should be treated first. General treatment of immersion foot (or hand) is similar to that for rewarmed frostbite. Swelling may produce compartment syndrome and require fasciotomy. Most patients require admission to the hospital.

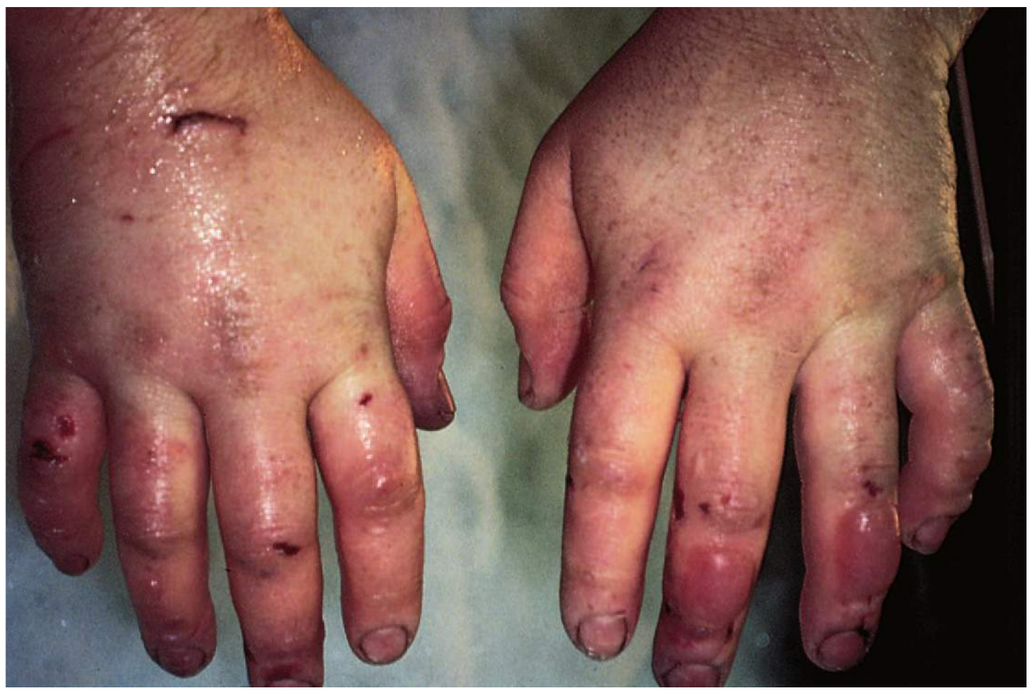

FIGURE 16.15 ■ **Immersion Injury.** Immersion injury to hands (unusual location) several hours after rewarming. The patient spent 18 hours bailing out a boat in waters just above freezing in Alaska. (Photo contributor: Ken Zafren, MD.)

Pearls

1. Doppler ultrasound may be useful to identify peripheral pulses, as pulses are often difficult to palpate in affected extremities.
2. Mixed injuries (frostbite and immersion) are common.

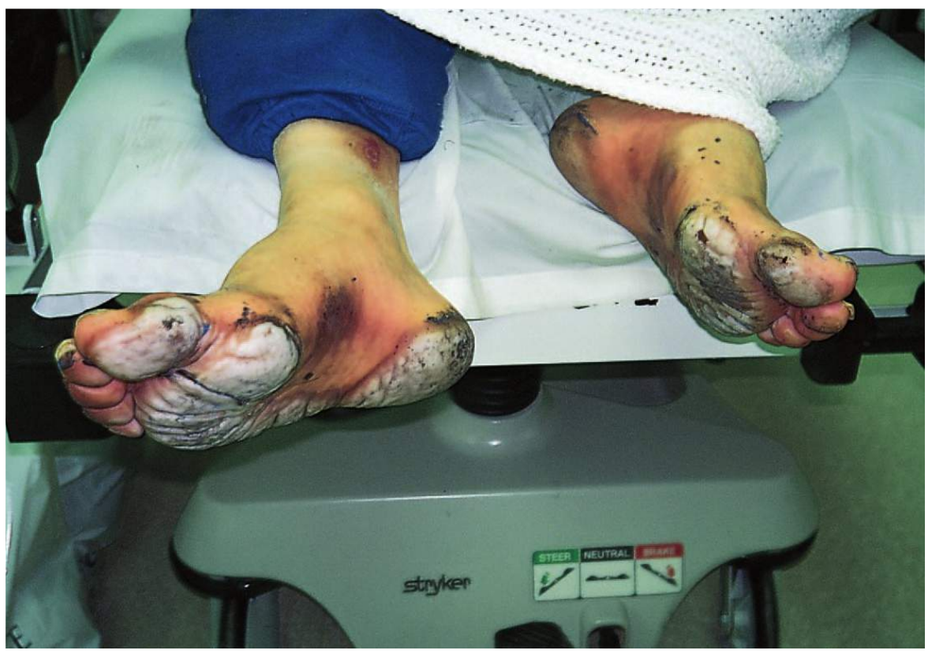

FIGURE 16.16 ■ **Immersion Foot.** Early appearance of immersion foot in a mentally ill homeless patient. (Photo contributor: Ken Zafren, MD.)

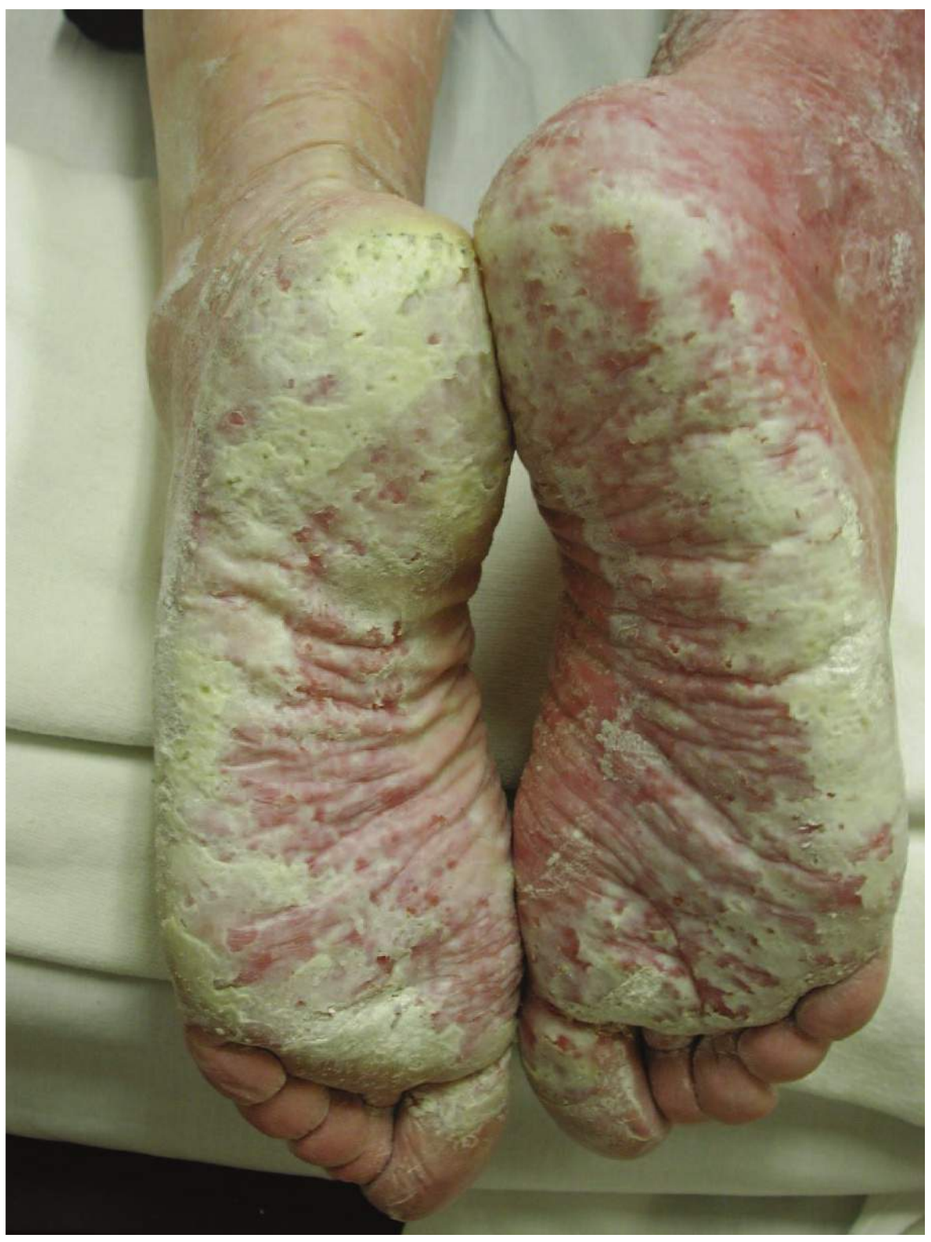

FIGURE 16.17 ■ **Severe Trench Foot.** This unfortunate homeless patient suffered prolonged exposure to a cold wet environment with resulting severe trench foot. (Photo contributor: Alan B. Storrow, MD.)

ULTRAVIOLET RADIATION EXPOSURE

Clinical Summary

Ultraviolet (UV) radiation causes both acute and chronic skin changes. Sunburn is a partial-thickness burn, which may become a full-thickness injury if infected. "Sun poisoning" is a severe systemic reaction to UV radiation. Patients may complain of nausea, vomiting, headache, fever, chills, and prostration. Excessive UV radiation may cause injury to the cornea and conjunctiva, termed UV keratitis (photokeratitis, snow blindness). This painful condition may occur in skiers, welders, or tanning salon patrons who do not wear proper eye protection.

Photosensitivity reactions (photodermatoses) are of several types. Phototoxic reactions are an abnormal response to UV radiation caused by a substance that is ingested (eg, prescription or over-the-counter medications) or applied to the skin; there is a direct relation between the amount of UV exposure and severity. Photoallergic reactions are clinically similar to contact dermatitis and, like phototoxic reactions, may be precipitated by ingested or applied drugs. Unlike phototoxic reactions, photoallergies may be precipitated by a small amount of light. Phytophotodermatitis is precipitated by skin contact with certain plants followed by exposure to UV radiation.

Phototoxicity should be suspected in any patient with severe or exaggerated sunburn. Photoallergy is easily misdiagnosed as allergic eczema or contact dermatitis, especially since onset is often delayed up to 2 days after exposure. Phytophotodermatitis may mimic severe sunburn or contact dermatitis, especially rhus dermatitis. Endogenous photosensitizers (endogenous photodermatoses) include solar urticaria, porphyria cutanea tarda, polymorphous light eruption, and systemic lupus erythrematosus. These may be provoked by visible light as well as by UV radiation.

Management and Disposition

Treatment of sunburn and sun poisoning involves standard burn therapy and supportive care. Sunburn is usually a self-limited problem. Cool compresses and nonsteroidal anti-inflammatory drugs may be beneficial. UV keratitis is treated with mydriatic-cycloplegic eye drops to decrease pain; initial examination is made easier with topical anesthetics. Severe cases may require antibiotic ointment, narcotic analgesics, and bilateral eye patches for 12 to 24 hours, though the use of eye patches remains controversial. These patients require 24- to 48-hour follow-up; ophthalmology referral is indicated to rule out retinal damage.

Treatment of photosensitivity reaction has two components: treatment of the sunburn and recognition of the sensitizing agent or endogenous medical condition. Topical steroids and oral analgesics and antipruritics may be helpful. Systemic steroids may be required. Patients with severe reactions should be referred to a dermatologist for possible photo patch testing.

Pearls

1. Para-aminobenzoic acid (PABA) in sunscreens may be a photosensitizer and can cause a photoallergic reaction.
2. The unique properties of individual skin types produce marked differences in response to UV radiation.
3. Victims of UV keratitis typically present 2 to 12 hours after exposure. As such, patients may present during the night with severe unexplained bilateral eye pain.

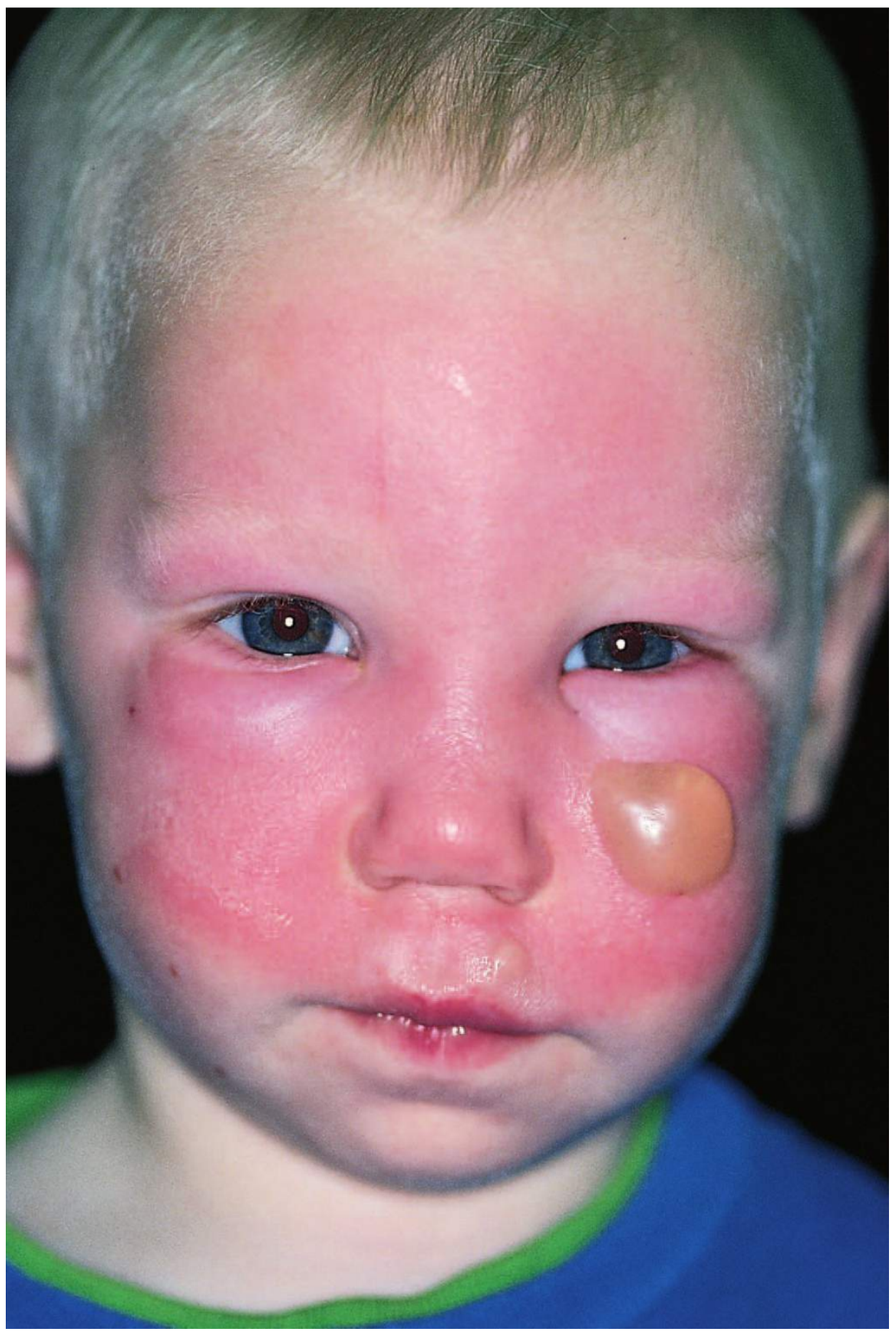

FIGURE 16.18 ■ **Sunburn.** Sunburn is characterized by erythema, edema, warmth, tenderness, and blisters. (Photo contributor: Kevin J. Knoop, MD, MS.)

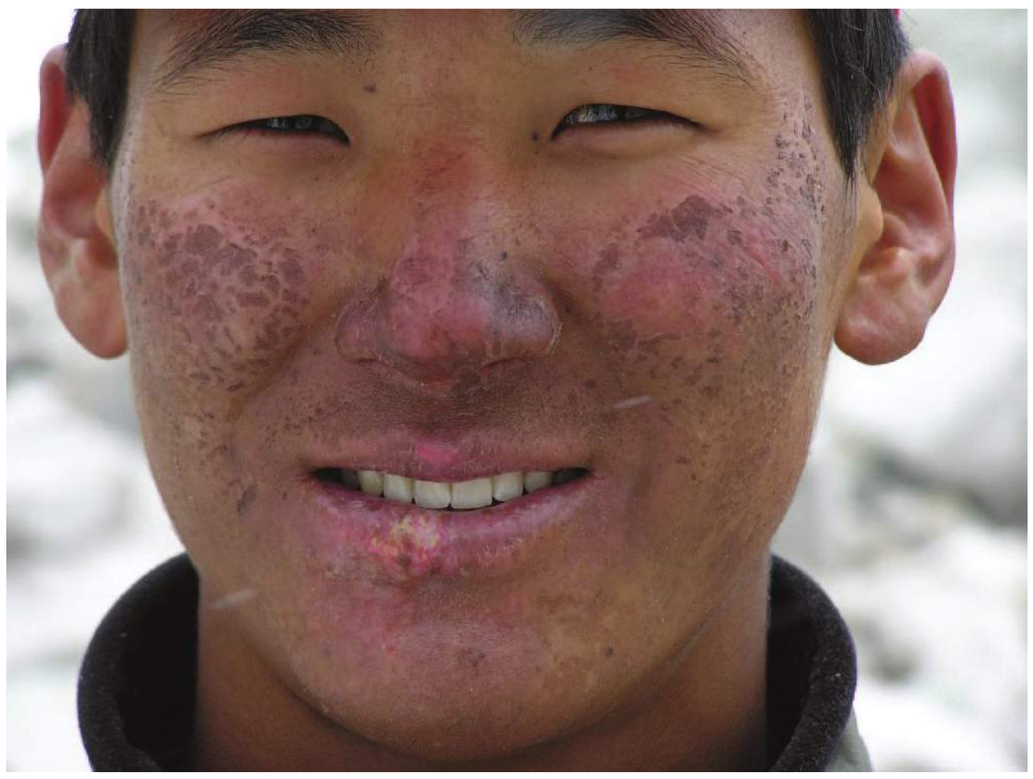

FIGURE 16.19 ■ **Healing UV Exposure.** This patient suffered significant UV exposure with resultant sunburn in the Himalayas. Darkly pigmented skin provides limited protection in high-altitude subtropical areas with very high amounts of UV exposure. (Photo contributor: Luanne Freer, MD.)

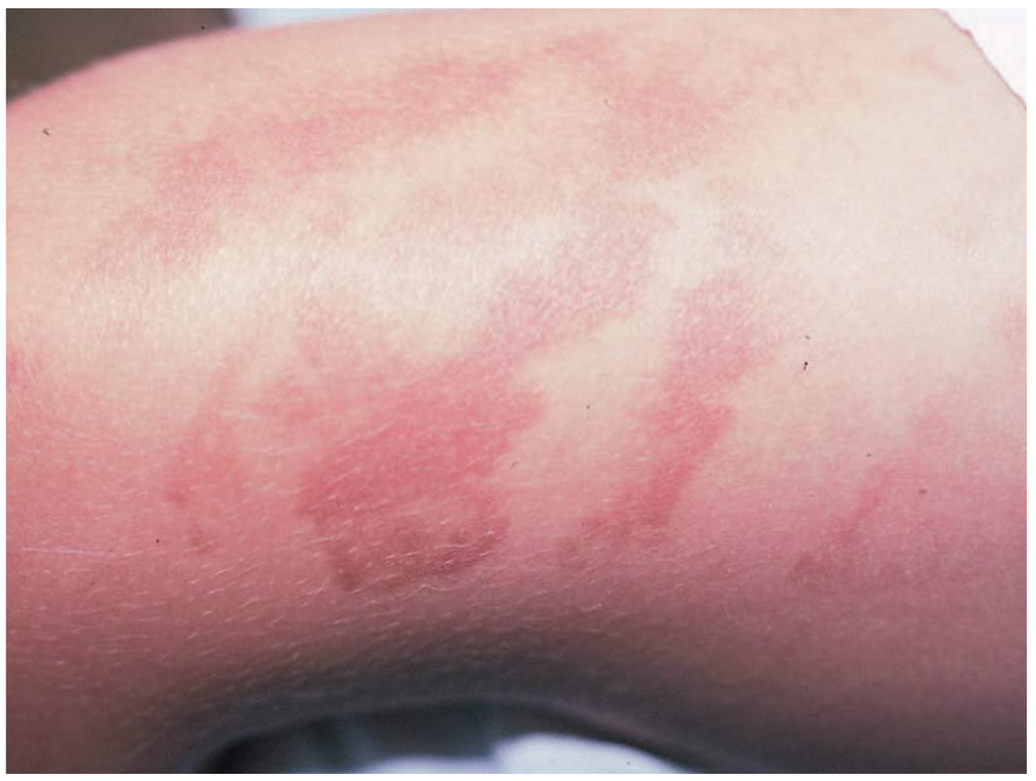

FIGURE 16.21 ■ **Phytophotodermatitis.** This reaction may require aggressive systemic steroid therapy. The case illustrated is a mild one caused by exposure to limes and UVA. A clue to the diagnosis is the patchy distribution with linear edges. More severe reactions resemble rhus dermatitis. (Photo contributor: Lee Kaplan, MD.)

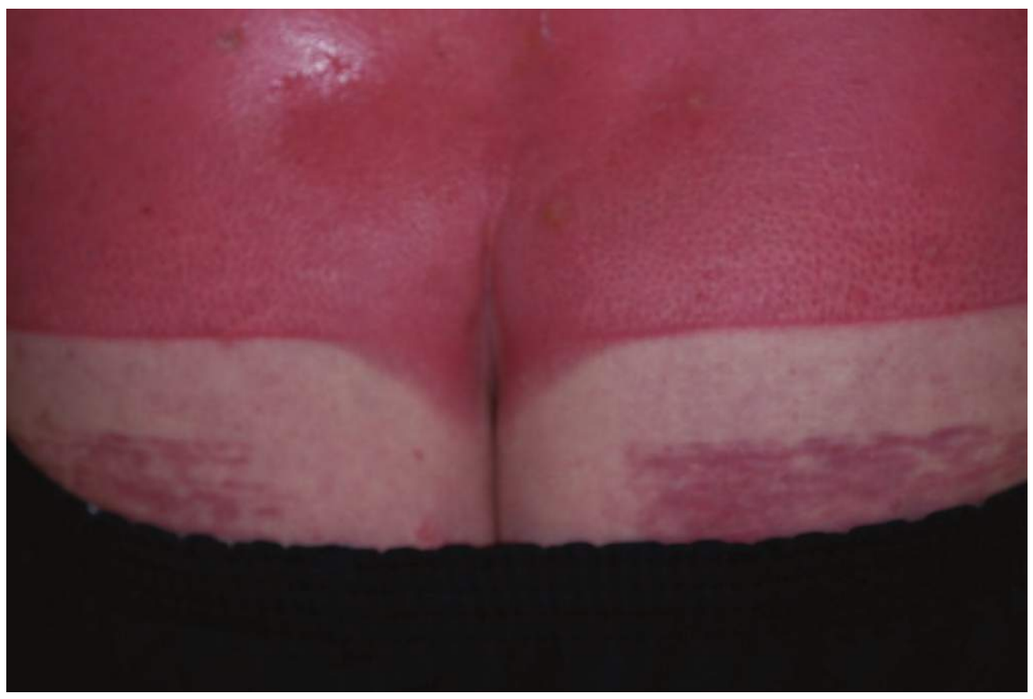

FIGURE 16.20 ■ **Tanning Bed Burn.** This patient suffered a severe diffuse partial thickness burn from prolonged UV exposure in a tanning bed. Note the sharp demarcation on the buttocks at the point where the patient was partially protected by his pants. (Photo contributor: R. Jason Thurman, MD.)

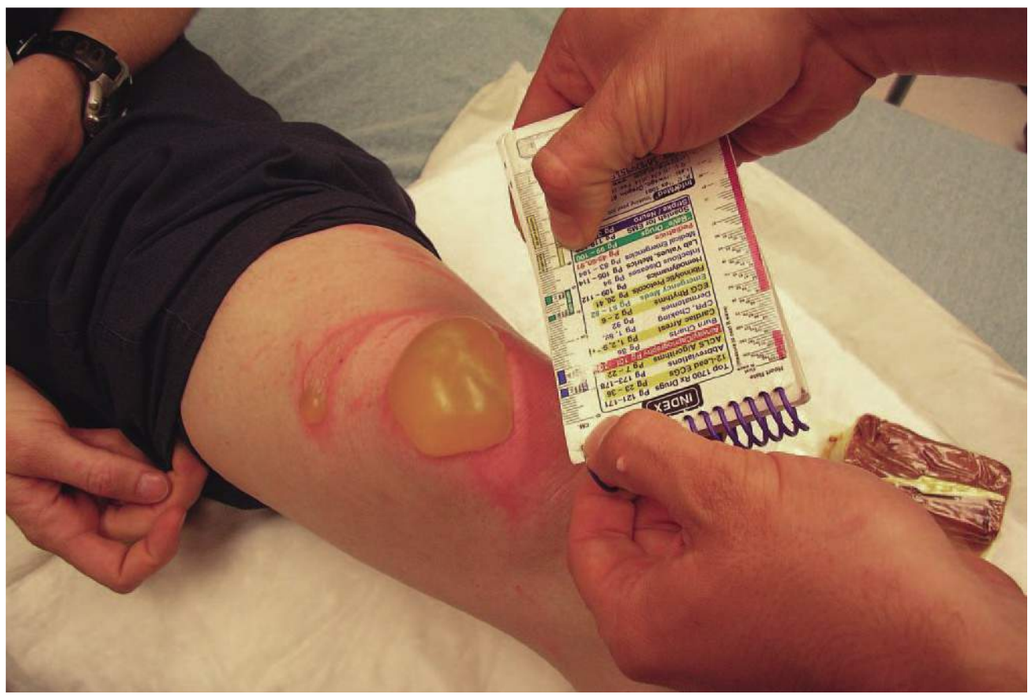

FIGURE 16.22 ■ **Phytophotodermatitis—Cow Parsnip.** Severe phytophotodermatitis caused by exposure to cow parsnip (Heracleum lanatum) in Alaska. Another name for cow parsnip is "pushkie," so this is also known locally as "pushkie burn." (Photo contributor: Kathy McCue, MD.)

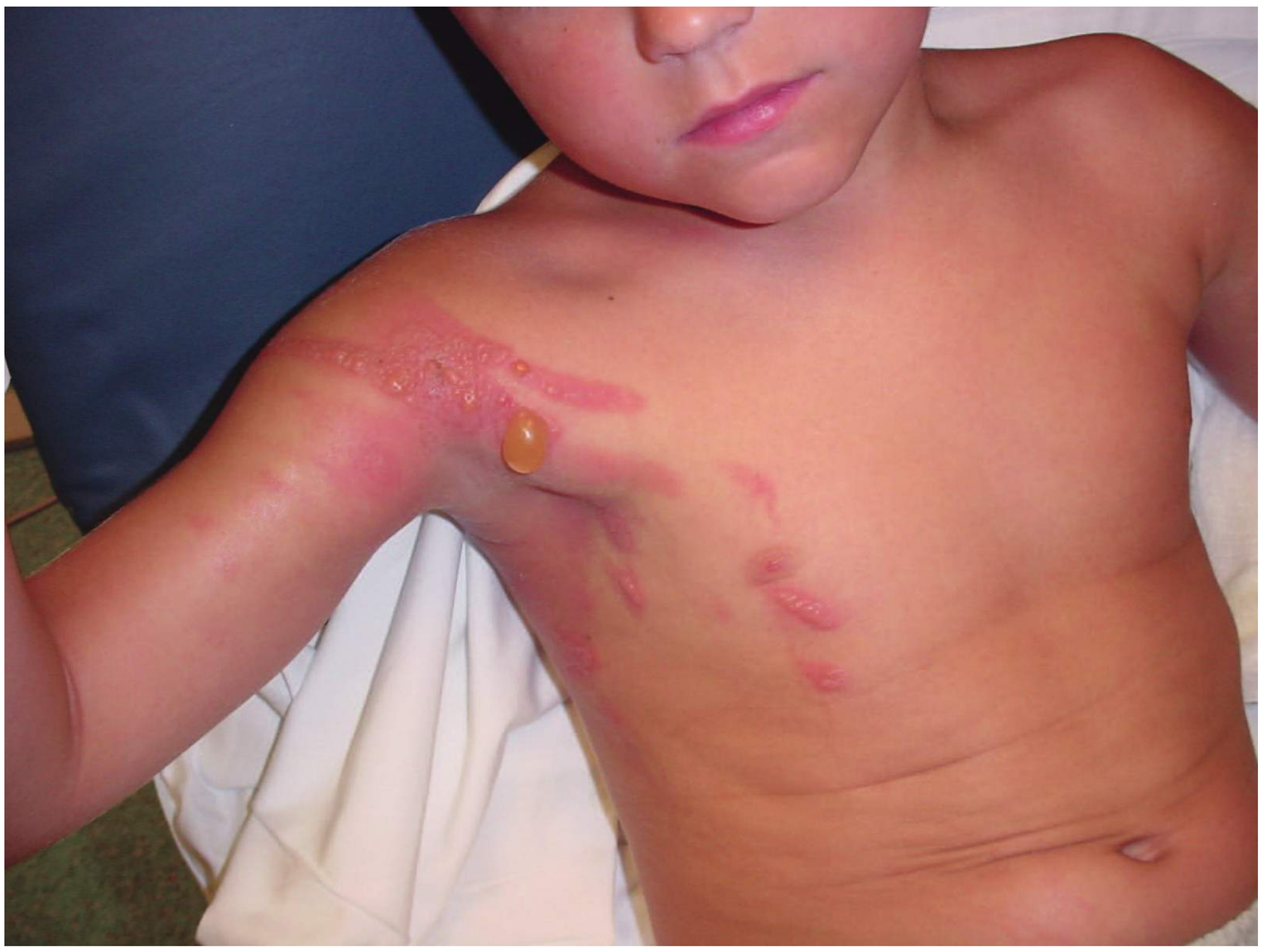

FIGURE 16.23 ■ **Phytophotodermatitis—Lime.** Phytophotodermatitis caused by exposure to limes and UV radiation in Jamaica. The patient had been exposed while riding shirtless on a horse through a lime orchard. (Photo contributor: Stephan Russ, MD.)

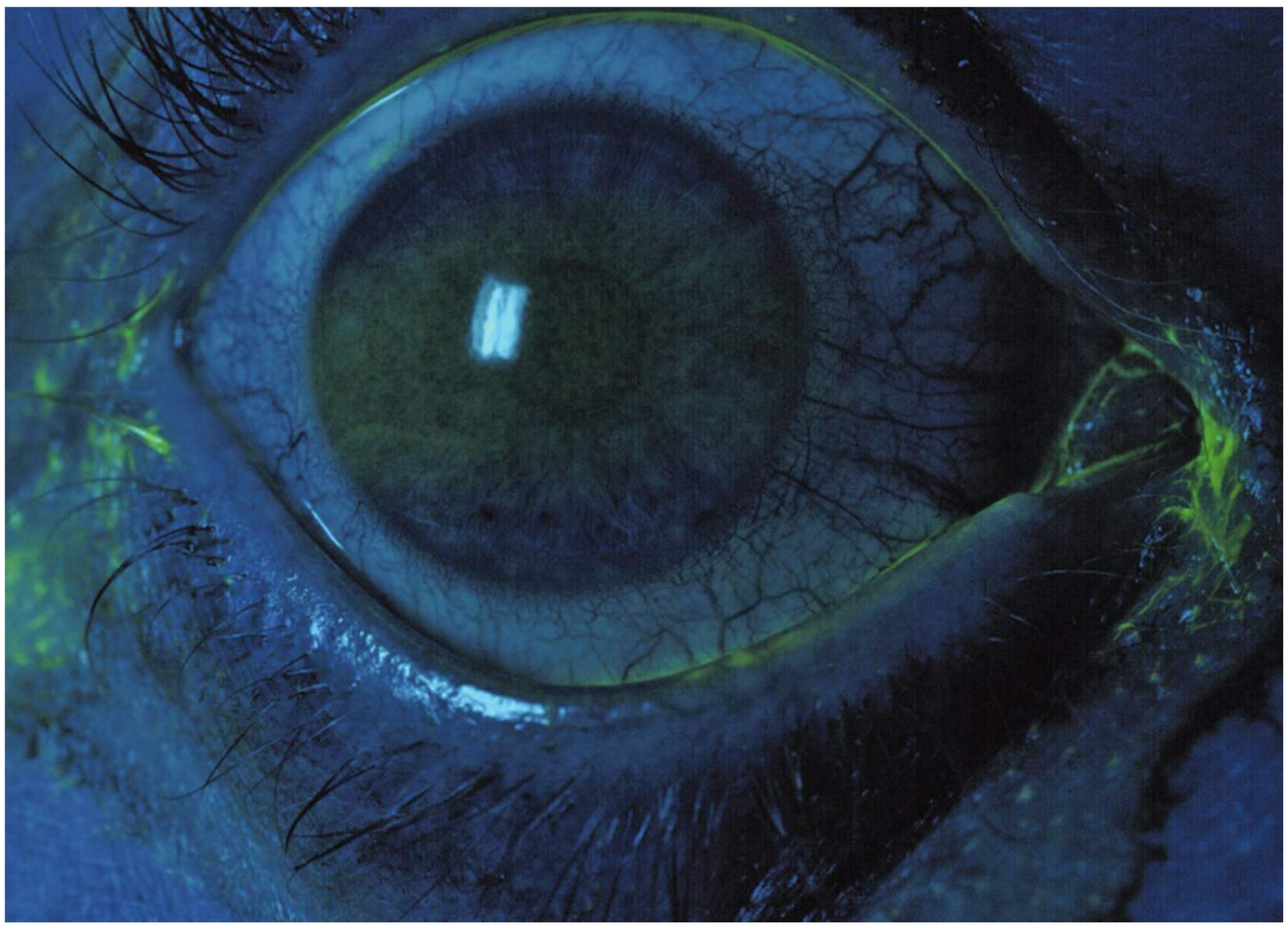

FIGURE 16.24 ■ **Ultraviolet Keratitis.** This patient presented with severe eye pain at 3 AM after a day of arc welding without proper eye protection. Notice the absence of fluorescein uptake where the eyelids were protective. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Lightning produces injury from high voltage, heat production, and explosive shock waves. Direct injuries include cardiorespiratory arrest, cardiac arrhythmias, and neurologic abnormalities such as seizures, deafness, confusion, amnesia, blindness, and paralysis. The patient may suffer contusions from the shock wave or from opisthotonic muscle contractions. Chest pain and muscle aches are common. One or both tympanic membranes (TMs) rupture in more than 50% of victims. Cataracts are usually a delayed occurrence. Hematologic abnormalities including disseminated intravascular coagulation (DIC) have been described. Fetal demise may occur.

Burns may result from vaporization of sweat or moist clothing, heating of clothing and metal objects such as climbing equipment, belt buckles or bra wiring, and direct effects of the strike. Linear burns and punctate burns are thermal burns. Feathering burns, also known as ferning or Lichtenberg figures, are not actually burns but may represent superficial bruising. They are pathognomonic of lightning injury.

Diagnosis of lightning injury is straightforward when there is a thunderstorm, when there are witnesses to the strike, or when there are typical physical findings. Lightning on relatively sunny days (without loud thunder) striking a lone victim may produce a confusing picture. The scattering of clothing and belongings may mimic an assault. Side flashes from metal objects and wiring may produce indoor victims during storms.

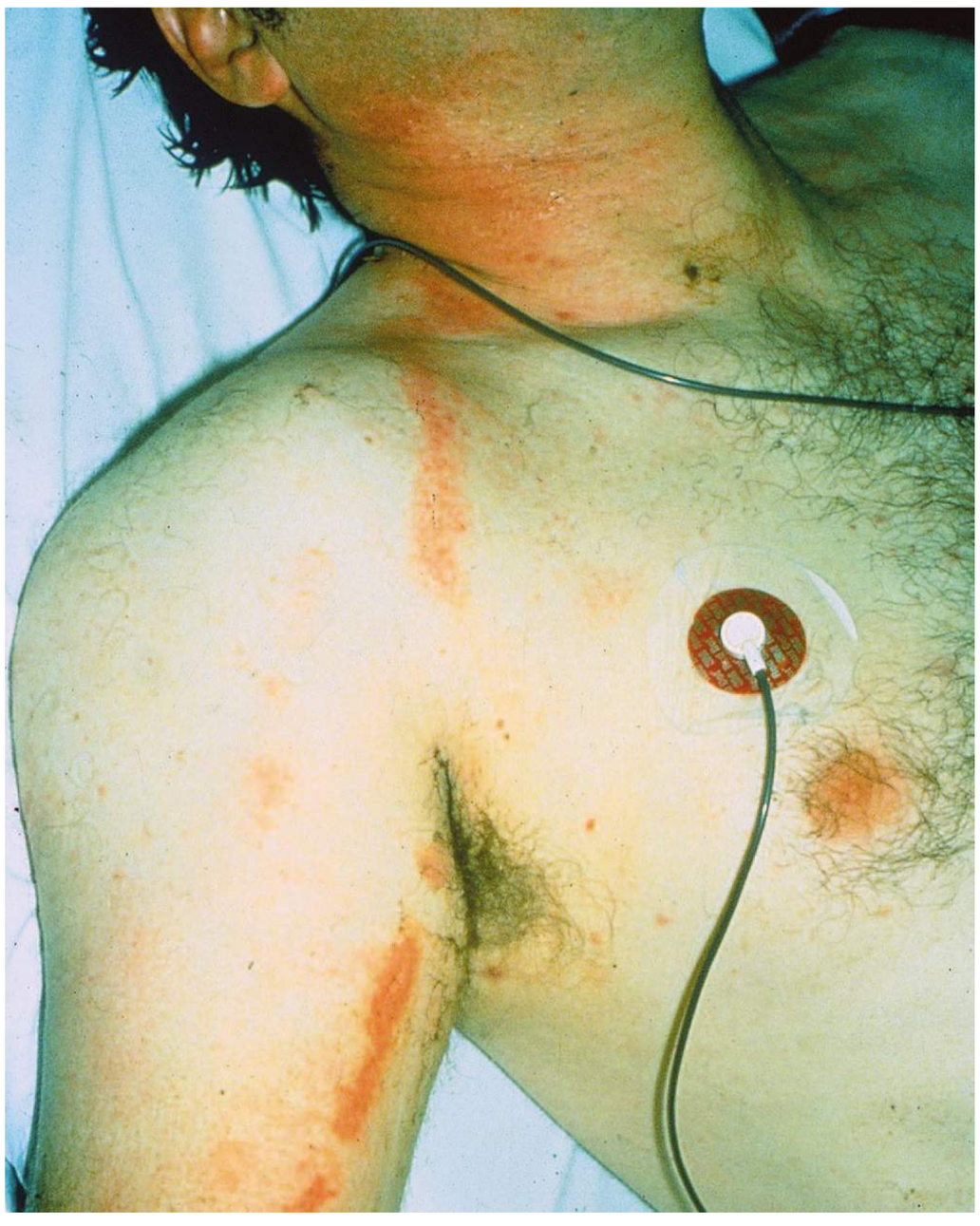

FIGURE 16.25 ■ **Linear Lightning Burns.** Linear burns from lightning are due to thermal effects. (Photo contributor: William Barsan, MD.)

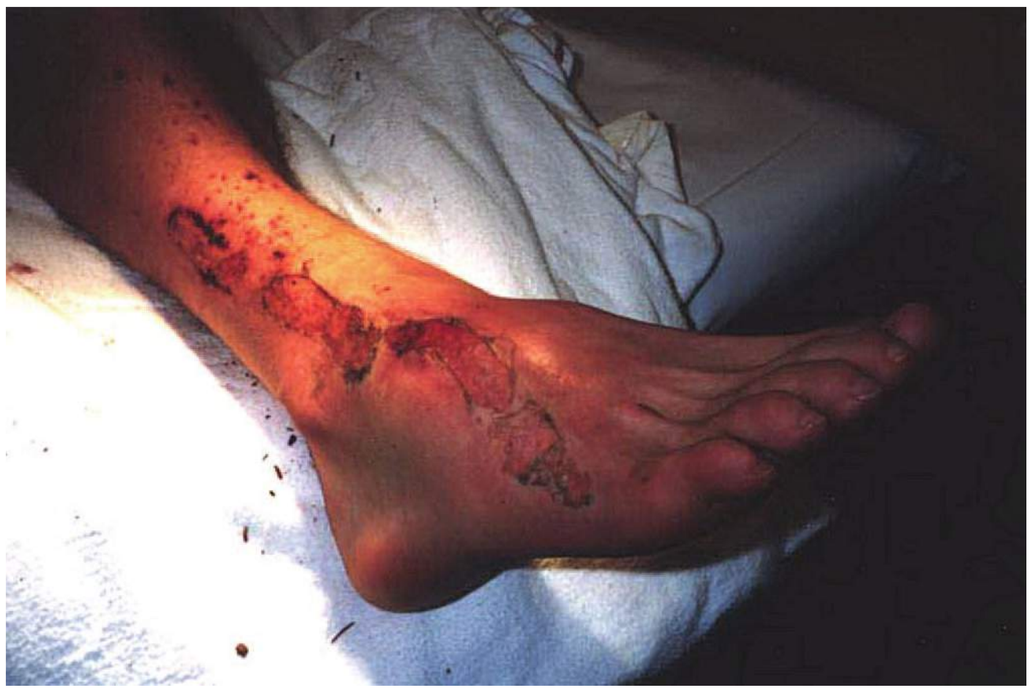

FIGURE 16.26 ■ **Linear Lightning Burns.** Linear lightning burns along the leg and foot of a strike victim. (Photo contributor: Sheryl Olson, RN, BSN, CCRN.)

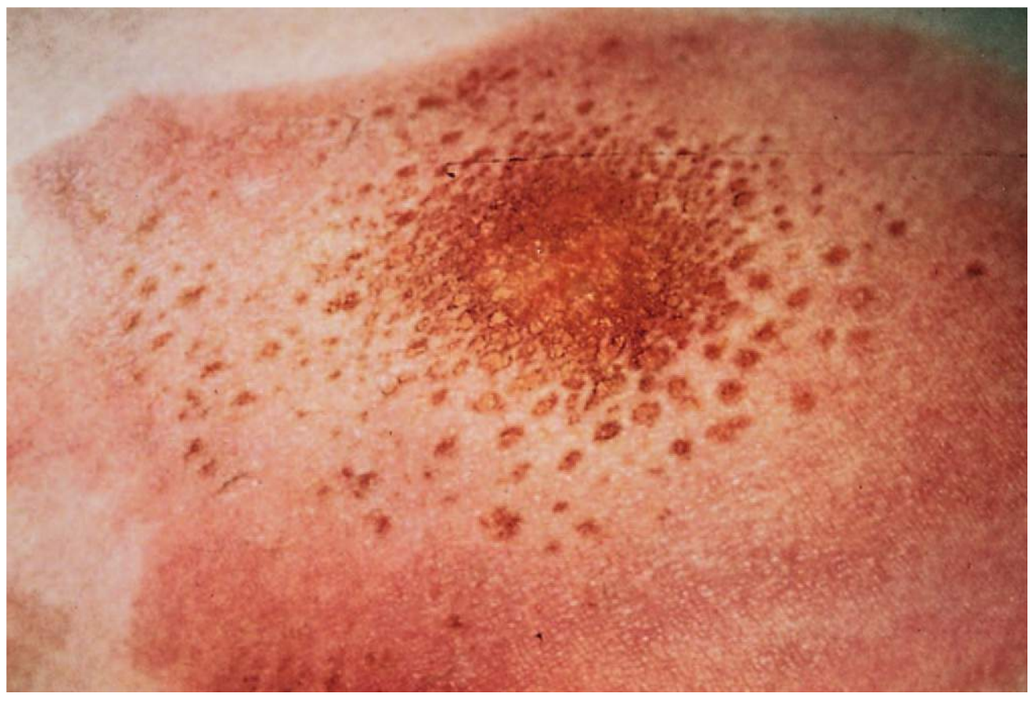

FIGURE 16.27 ■ **Punctate Lightning Burns.** Punctate burns due to lightning are partial or full-thickness thermal burns that range from a few millimeters to a centimeter in diameter. They are multiple and closely spaced. (Photo contributor: Arthur Kahn, MD.)

Management and Disposition

History and physical examination to rule out associated injuries and standard ED care and workup for critical patients—including cardiac enzymes, urinalysis, and ECG—are necessary. All patients, even those apparently well, require admission for observation since their condition may change over several hours following the lightning strike.

Pearls

1. The amount of damage to the exterior of the body does not predict the amount of internal injury.
2. Since lightning most commonly produces cardiac standstill by means of massive direct current countershock, prompt, spontaneous return of normal heart rhythm (by virtue of cardiac automaticity) is the rule. However, respiratory arrest is often more prolonged. In a triage situation, the normal rules do not apply, since victims breathing spontaneously are already recovering. The rule in lightning strikes is to resuscitate the "dead." Ventilatory support is often all that is required.
3. Keraunoparalysis (lightning paralysis) is a condition that is specific to lightning injury and is caused by extreme vasoconstriction, usually in the legs. It resolves spontaneously within a few hours.
4. Psychological sequelae following lightning injuries are underreported and may include memory loss, difficulty with concentration, and depression.

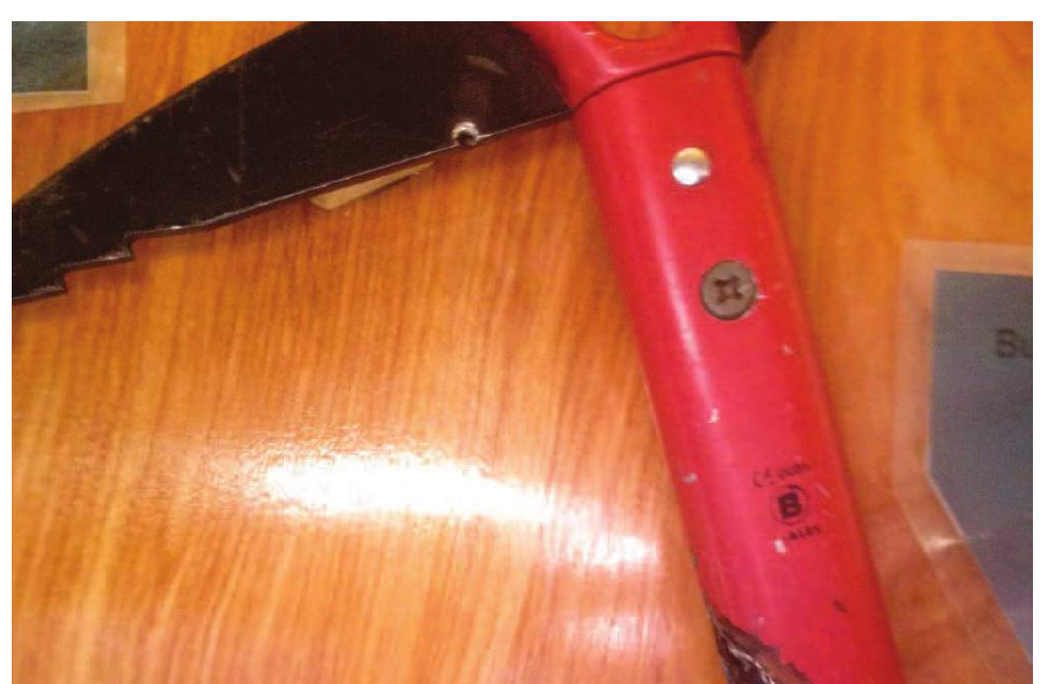

FIGURE 16.29 ■ **Feathering from Underwire Bra.** Feathering (ferning) burns are pathognomonic of lightning injury. These are not true burns but may represent superficial bruising. (Photo contributor: Sheryl Olson, RN, BSN, CCRN.)

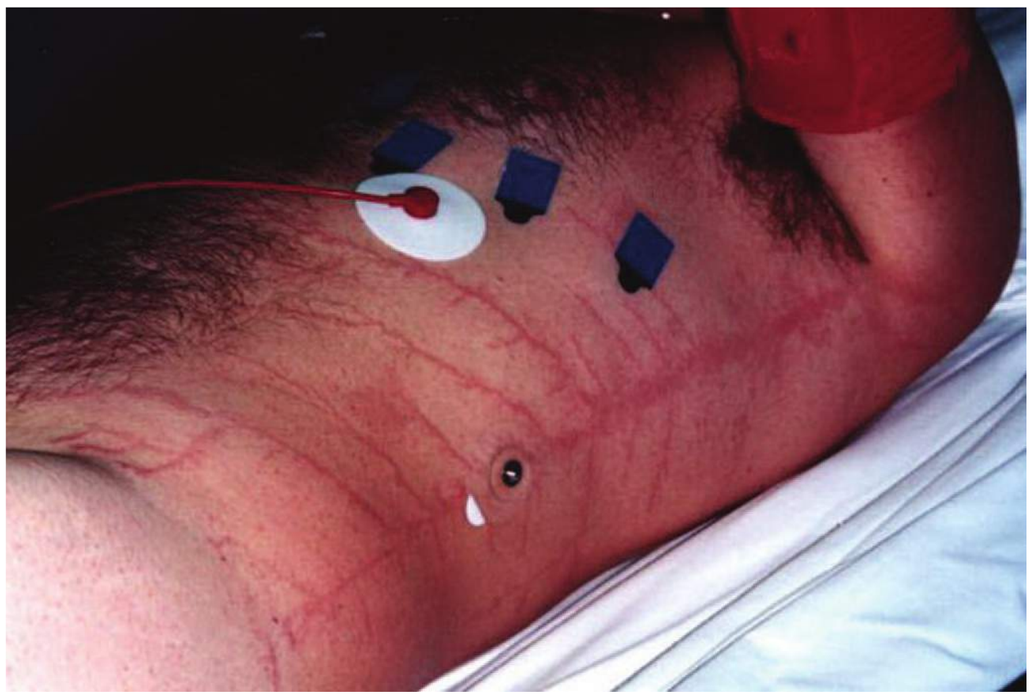

FIGURE 16.28 ■ **Feathering.** Feathering (ferning) on the left side of a lightning strike victim. (Photo contributor: Sheryl Olson, RN, BSN, CCRN.)

FIGURE 16.30 ■ **Lightning Damage.** Lightning can instantly heat metal objects, causing significant burns to skin in contact with the equipment. (Photo contributor: Nicholas Kanaan, MD.)

Clinical Summary

Both domestic and wild animals are known to attack humans. Injuries are caused by combinations of penetrating and blunt trauma. Penetrating injuries can be inflicted by teeth, claws, and horns, while severe blunt injuries may result from the victim being knocked over, trampled or otherwise crushed, being thrown into the air, or dragged. Injuries may involve massive tissue injury or avulsion often associated with neurovascular damage and long bone fractures.

Management and Disposition

Evaluation and treatment is the same as for any other multiple trauma victim with a high-energy mechanism. After attention to the ABCs, which may be complicated by injuries of the face, neck, or chest, victims should be evaluated for less obvious blunt traumatic injuries. Open fractures and injuries to internal organs may not be clinically apparent initially as with any multitrauma victim.

Pearls

1. Wounds often require operative debridement and repair by appropriate specialists. Consider tetanus and rabies prophylaxis along with antibiotic treatment for high-risk wounds.
2. Bite wounds can penetrate the skull or joint. Displaced teeth or claws may remain imbedded in wounds. Evaluation should include x-rays, ultrasound, and/or CT scanning along with appropriate surgical consultations.

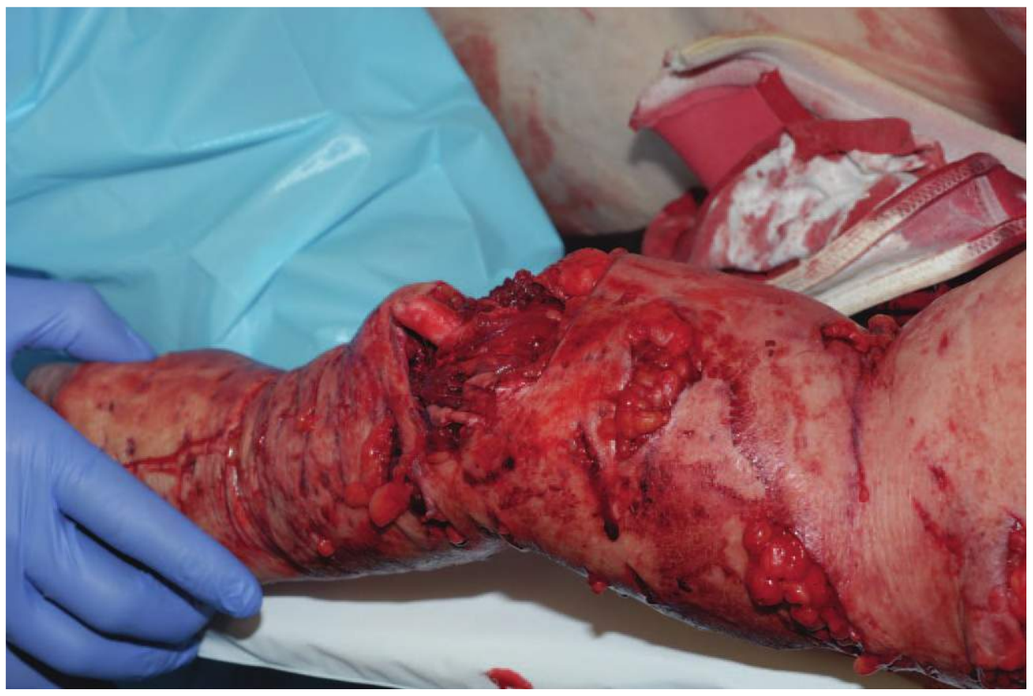

FIGURE 16.32 ■ **Pit Bull Mauling.** The left arm of a pit bull mauling victim. Note the open fracture of the radius. The patient sustained multiple severe defensive wounds to both arms resulting in extensive soft tissue and neurovascular injuries. (Photo contributor: R. Jason Thurman, MD.)

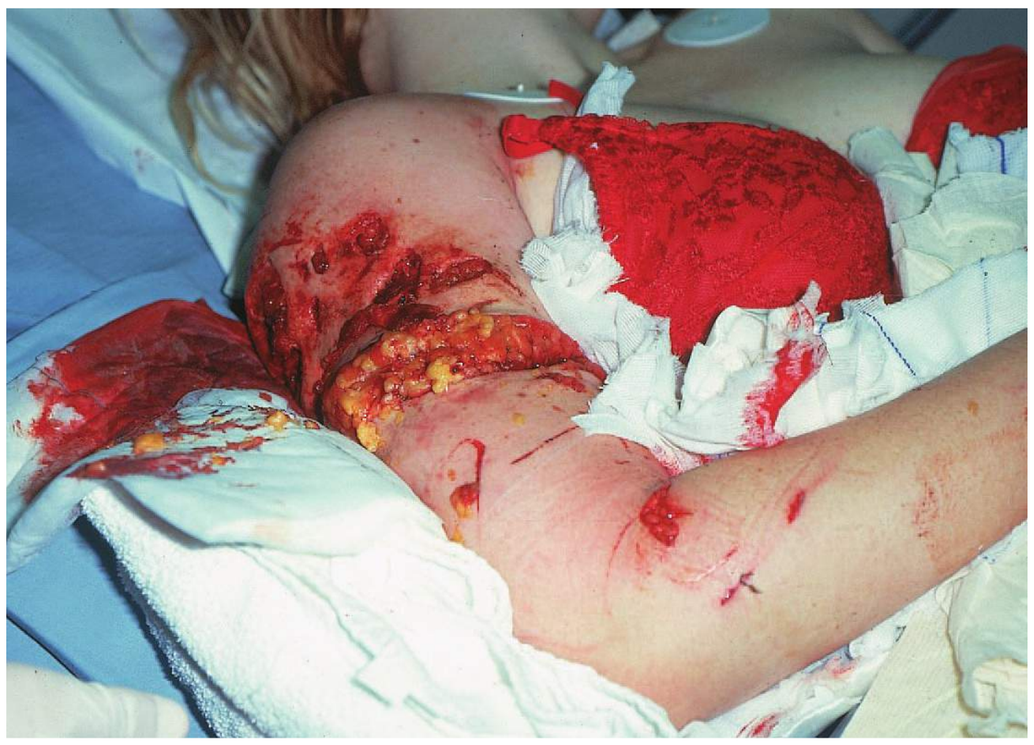

FIGURE 16.31 ■ **Cougar Mauling.** This patient sustained large wounds as a result of a cougar attack. Here, the weight and force of this large animal has resulted in severe shearing injuries. Penetrating injuries from large animals may be deeper than they appear. (Photo contributor: Alan B. Storrow, MD.)

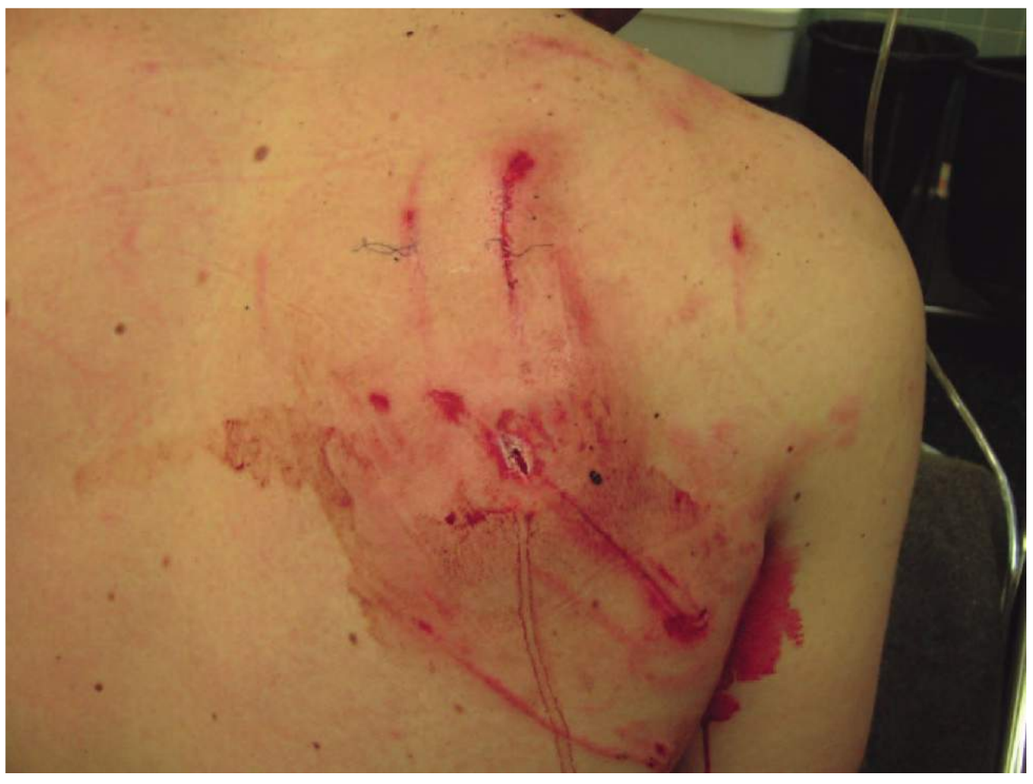

FIGURE 16.33 ■ **Bear Attack—Open Scapula Fracture.** Although grizzly claws are not very sharp, they can exert considerable force. This innocuous appearing injury was complicated by an open scapula fracture. (Photo contributor: Luanne Freer, MD.)

CORAL SNAKE ENVENOMATION

Clinical Summary

The most important species of coral snakes (Elapidae family) found in the United States are the eastern coral snake (Micrurus fulvius) and the Texas coral snake (Micrurus tener). Coral snakes have small mouths, and bites are usually limited to fingers, toes, or folds of skin. Due to a less efficient venom apparatus than their Crotalid colleagues, coral snakes generally need to hold on or chew to inflict a significant envenomation. The bite typically produces minimal local inflammation and pain. Paresthesias and muscle fasciculations are common. Systemic symptoms resulting from the powerful neurotoxic effects of the venom can include tremors, drowsiness, euphoria, marked salivation, and respiratory distress. Cranial nerve involvement, manifested by slurred speech and diplopia, may be followed by bulbar paralysis with dysphagia and dyspnea. Death may result from respiratory and cardiac arrest. Onset of severe symptoms may be delayed up to 12 hours, but may also be rapidly progressive.

Management and Disposition

In contrast to pit viper envenomation, prehospital application of a constrictive bandage may be of benefit in limiting the spread of neurotoxic coral snake venom. Severe systemic symptoms following envenomation by Elapidae may be delayed and cannot be accurately predicted by local wound reactions. It is therefore recommended that four to six vials of antivenom be administered for all suspected envenomations by eastern or Texas coral snakes. Treatment of western coral snake (Micruroides euryxanthus) bites is purely supportive. Tetanus prophylaxis should be addressed.

Pearls

1. Treatment with antivenom should be initiated early in cases of eastern coral snake bites, since symptoms are often delayed and severe.
2. The adage "red on yellow, kill a fellow; red on black, venom lack" applies to all coral snakes found in the United States but does not hold true in other parts of the world.
3. As many as 60% of North American coral snake bites do not result in envenomation of the victim.

FIGURE 16.35 ■ **Coral Snake (Close-Up).** A close-up view of the striking and distinctive markings of the highly venomous coral snake. (Photo contributor: Mike Cardwell, MS.)

FIGURE 16.34 ■ **Coral Snake.** United States eastern coral snake with typical coloring and red-on-yellow bands. (Photo contributor: Mike Cardwell, MS.)

FIGURE 16.36 ■ **Nonvenomous Milk Snake.** As opposed to the red-on-yellow rings seen in the venomous US coral snake, these red-on-black rings indicate a nonvenomous snake. Unfortunately, this applies only to animals native to the United States. (Photo contributor: Sean P. Bush, MD.)

Clinical Summary

The pit vipers (Crotalidae family) indigenous to the United States include rattlesnake species, cottonmouths, and copperheads. Physical characteristics of pit vipers include a triangular head, heat-sensing pits, elliptical pupils, and a single row of subcaudal ventral scales. Pit viper venom is complex and produces hematologic, cardiovascular, and neuromuscular effects. Clinically, pit viper envenomations are divided into four categories. Snakebites without envenomation are characterized only by the direct tissue damage caused by the strike. Minimal envenomations consist of fang marks and local swelling but no systemic symptoms. Moderate envenomations include progression of tissue changes beyond the immediate location of the bite and/or systemic symptoms and mild changes in coagulation parameters. Severe envenomations include marked local and progressive swelling and significant systemic symptoms and coagulopathy (eg, subcutaneous ecchymosis and/or signs of bleeding).

Management and Disposition

Prehospital management of pit viper bites should include immobilization and rapid transport without delay. Lymphatic constriction bands and extractor devices should not be used. These devices may worsen focal complications of envenomation. Tourniquets and local incision are ineffective and generally do more harm than good. Electric shock, cryotherapy, and mouth suction are not recommended. ED management includes resuscitation, establishing a physiologic baseline, and determining the need for antivenom. CroFab (Savage Labs), a recombinant antivenom effective in neutralizing venom toxins, is the treatment of choice. Indications for antivenom administration include any progression of swelling, erythema, or ecchymosis beyond the immediate bite area, presence of any systemic signs of envenomation, or any coagulation abnormalities and/or bleeding complications associated with envenomation. The dose of antivenom increases with the severity of envenomation. The package insert details the current recommendations for antivenom administration and allergy testing, but in general treatment is initiated with 4 to 6 vials of CroFab intravenously with further dosing regimen based on clinical course. Compartment syndrome is a possible complication of envenomation, and should initially be treated with antivenom. Fasciotomy should be performed only if compartment pressures are greater than 30 mm Hg in spite of antivenom treatment. Patients who do not develop evidence of envenomation after 8 hours of observation may be safely discharged home with close follow-up. Tetanus prophylaxis should be addressed and given when necessary.

Pearls

1. Up to half of all pit viper bites may be "dry" (without any injection of venom).
2. Intramuscular epinephrine (0.3 mg 1:1000) given prior to administration of horse serum antivenin may reduce the potential allergic response.

FIGURE 16.37 ■ **Eastern Diamondback Rattlesnake.** The eastern diamondback is the largest US rattlesnake and has a characteristic diamond-shaped pattern on its dorsal aspect. Note the triangular head, which is characteristic of pit vipers. (Photo contributor: R. Jason Thurman, MD.)

FIGURE 16.38 ■ **Western Diamondback Rattlesnake.** The western diamondback causes the most fatalities from snakebites in Mexico and the second most in the United States behind the eastern diamondback. (Photo contributor: Mike Cardwell, MS.)

FIGURE 16.39 ■ **Mojave Green Rattlesnake.** The Mojave green is found in the southwestern United States and Mexico. There are two subspecies, Types A and B, with Type A thought to have the most toxic venom of all North American snakes. Unlike most rattlesnake venoms, Mojave Type A venom contains a potent neurotoxin. (Photo contributor: Mike Cardwell, MS.)

FIGURE 16.40 ■ **Red Diamond Rattlesnake.** The elliptical pupils and heat-sensing pits in this red diamond rattlesnake are characteristic of pit vipers. (Photo contributor: Sean P. Bush, MD.)

FIGURE 16.41 ■ **Cottonmouth.** The cottonmouth is a semiaquatic venomous pit viper that may crawl or swim with its head raised at an angle of 45 degrees. When disturbed, it may open its mouth wide to reveal a white lining. (Photo contributor: Stephen J. Knoop.)

FIGURE 16.42 ■ **Copperhead Snake.** The copperhead is frequently encountered in wooded mountains, abandoned buildings, and damp, grassy areas. It is able to climb low bushes and trees in search of food. (Photo contributor: R. Jason Thurman, MD.)

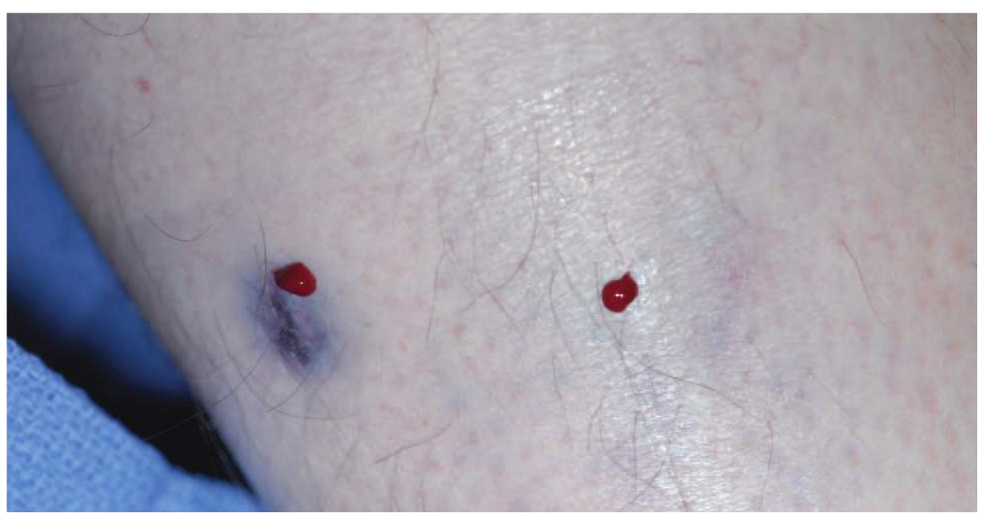

FIGURE 16.43 ■ **Rattlesnake Envenomation, Minutes Old.** Timber rattlesnake envenomation on the leg of an unsuspecting victim. The fang marks are 4 cm apart. (Photo contributor: R. Jason Thurman, MD.)

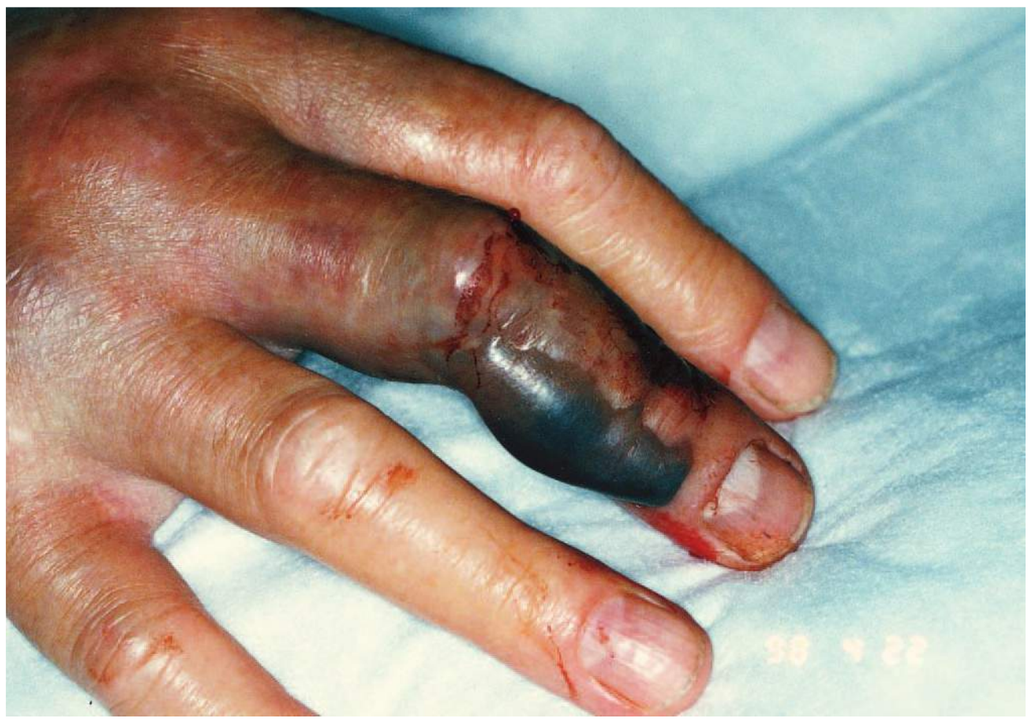

FIGURE 16.44 ■ **Rattlesnake Envenomation, Day 1.** This rattlesnake bite shows local swelling, some edema beyond the initial bite site, and a hemorrhagic bleb at 6 hours. (Photo contributor: Sean P. Bush, MD.)

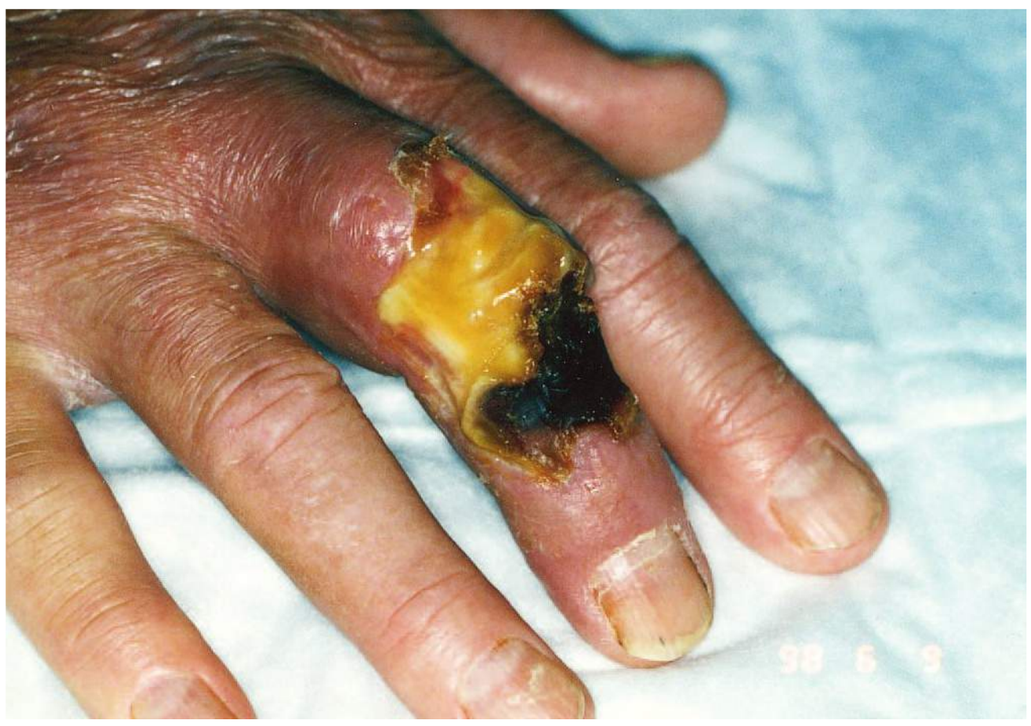

FIGURE 16.45 ■ **Progression of Rattlesnake Envenomation, 7 Weeks.** Seven weeks later, the patient shown in Fig. 16.44 has progressed to tissue loss, eschar formation, and mild changes in coagulation parameters. (Photo contributor: Sean P. Bush, MD.)

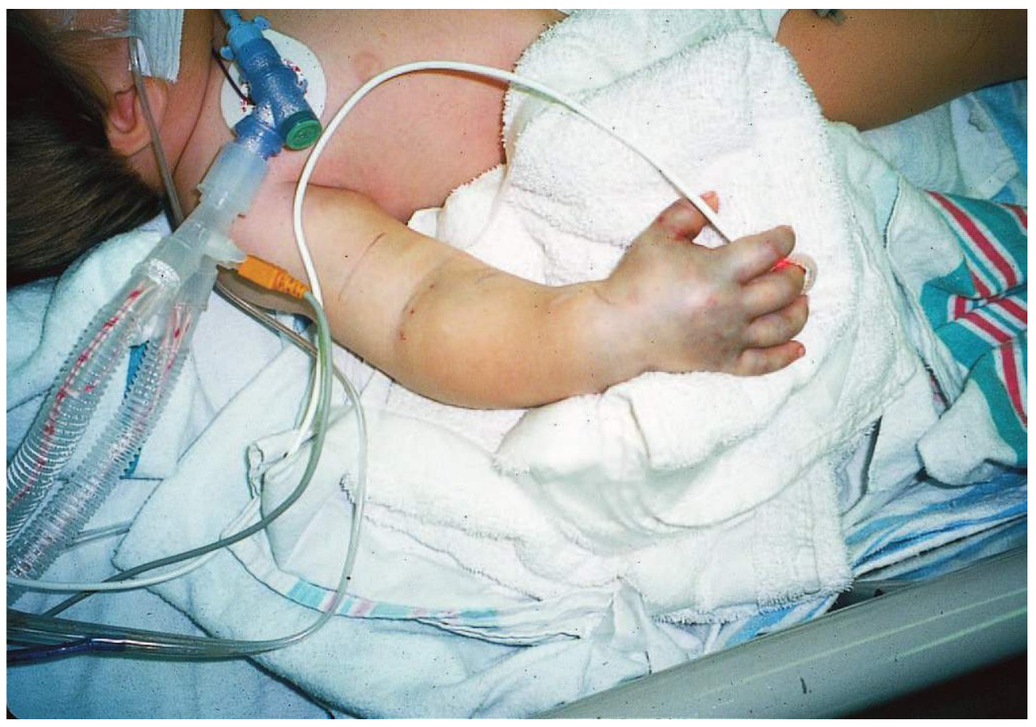

FIGURE 16.47 ■ **Severe Rattlesnake Envenomation.** This patient sustained a rattlesnake bite to his hand and presented with marked and progressive swelling, subcutaneous ecchymosis, and a significant coagulopathy. (Photo contributor: Sean P. Bush, MD.)

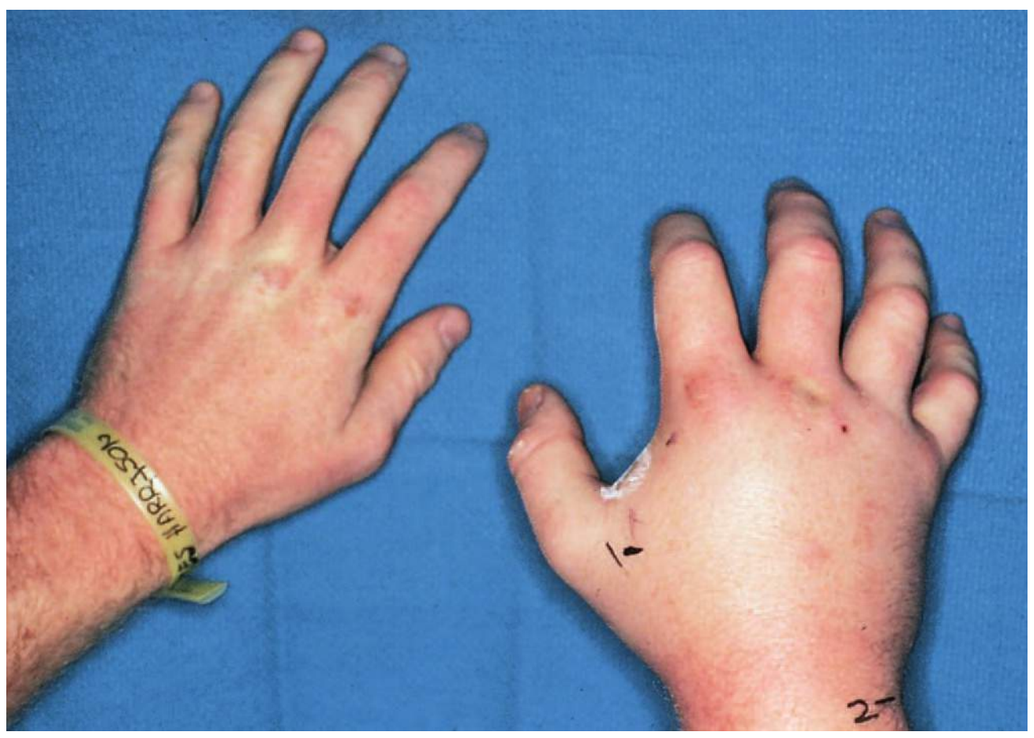

FIGURE 16.46 ■ **Moderate Crotalid Envenomation.** This patient was bitten by a rattlesnake on the dorsal aspect of the right hand and presented with edema extending to the wrist as well as nausea and vomiting. (Photo contributor: Edward J. Otten, MD.)

FIGURE 16.48 ■ **Nonvenomous Snake.** In contrast to the elliptical pupils and triangular head characteristic of pit vipers, this nonvenomous rat snake has a rounded head and circular pupils. (Photo contributor: Sean P. Bush, MD.)

Clinical Summary

The epidemiology of snakebites in tropical regions differs considerably from that seen in more temperate climates. In general, the absolute number of venomous snakes is higher in the tropics and snakes are often located in areas of high population density. The prevalence of bites is also higher due to differences in agricultural and hunting practices, frequent flooding, lack of adequate footwear in many locations, and housing that allows access of snakes into living areas. The annual mortality of snakebite in India possibly exceeds 20,000 and has been stated to be the fifth most common cause of death in Burma (Myanmar). In some native populations in South America, up to 20% of adult deaths are from snakebite. Places where snakebites are common are often in remote locations where medical care may not be immediately available. Signs and symptoms will depend upon the type of envenomation and the amount of toxin injected. Local pain, swelling, and blistering are seen with many snakebites. Clotting disturbances, frank hemorrhage, and shock are seen with many viper bites, while neurotoxicity can be seen with elapid and sea snake bites.

Management and Disposition

Patients should be reassured and kept as immobile as possible. Any involved extremity should be splinted. Tourniquets are generally discouraged, but may be useful in some neurotoxic snakebites. Many modalities such as suction, incision, pumping apparatus, cryotherapy, and electrical shocks have been advocated, but are of no proven benefit and may be harmful. Specific antivenin is used as indicated, but is often either not available or prohibitively expensive.

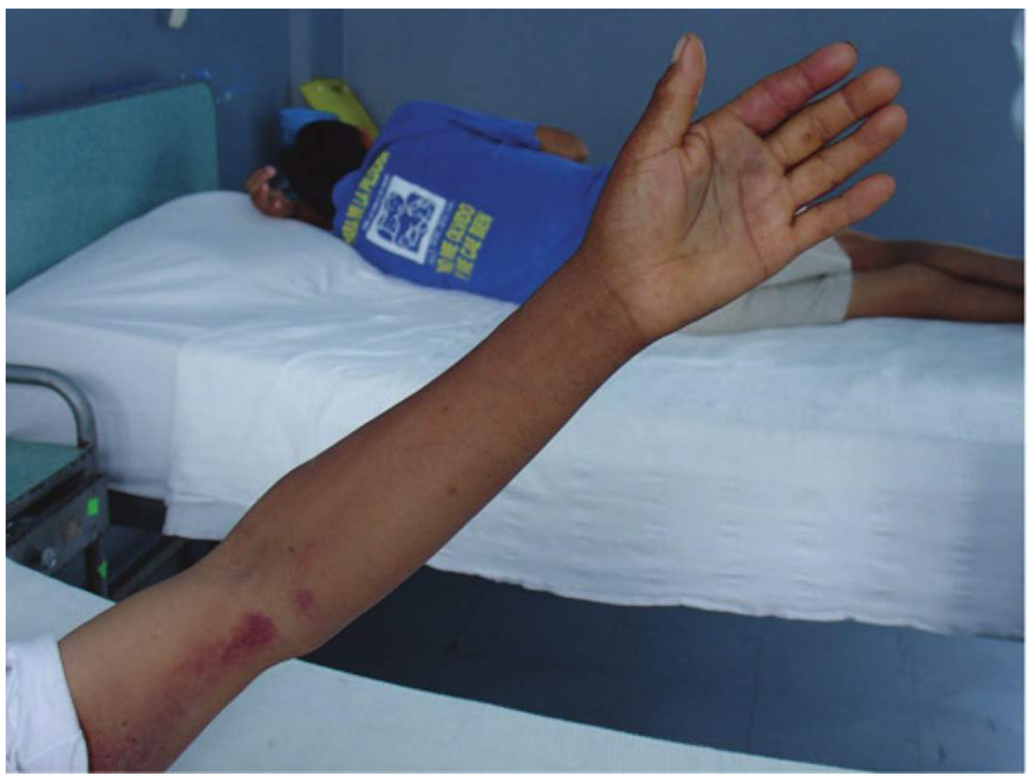

FIGURE 16.50 ■ **Fer-de-Lance Bite.** This patient was bitten on the index finger by a Fer-de-Lance snake while clearing bush in rural Peru. Note the ecchymosis proximal to the bite. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 16.49 ■ **Fer-de-Lance (Bothrops jararaca).** Fer-de-Lance, French for "lance head," refers to a number of venomous pit vipers of the genus Bothrops in Central and South America (B atrox, B asper, B jararaca, B lanceolatus). This family of snakes is responsible for more deaths than any other New World snake. (Photo contributor: Cybele Sabino Lisboa, BSc.)

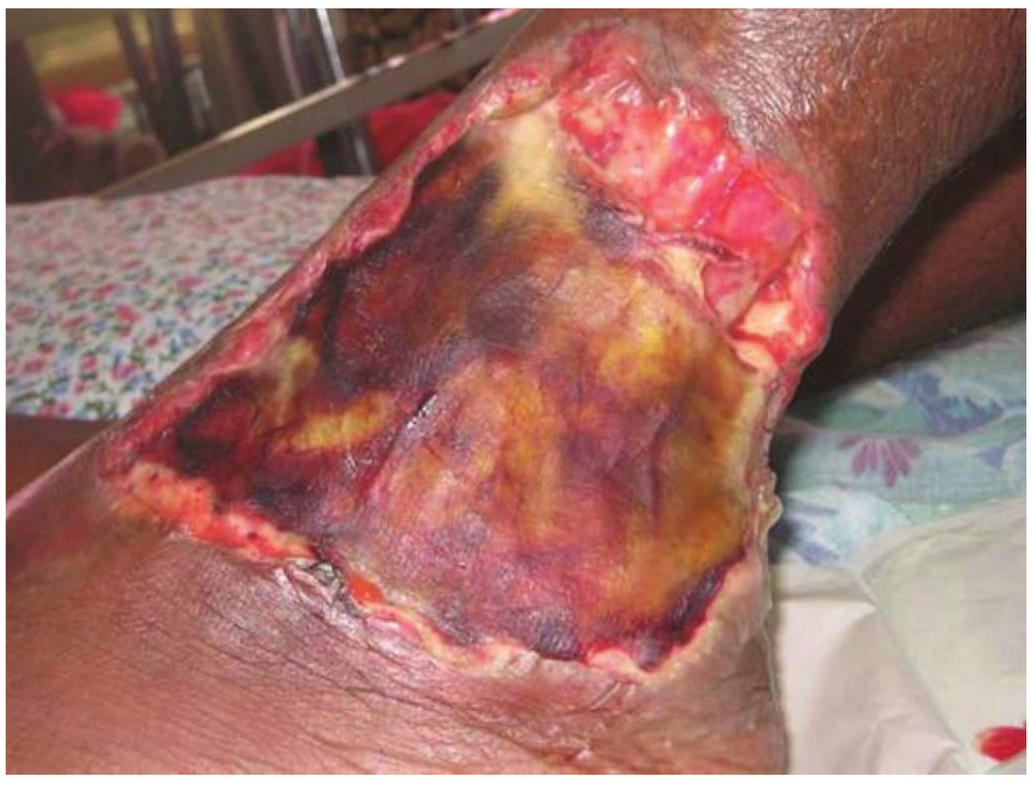

FIGURE 16.51 ■ **Fer-de-Lance Bite (Late).** Local tissue destruction at the site of a Bothrops atrox bite in a Guyanese teenager. (Photo contributor: Ian Jones, MD.)

Pearls

1. There are approximately 3000 species of snakes in the world, with 600 being venomous, and over 200 considered to be medically important. Venomous snakes can be found at altitudes as high as 4000 m in equatorial tropical regions.
2. Neurotoxicity is seen in bites of elapids such as kraits, coral snakes, mambas, and cobras, but is not a feature of the African spitting cobra. The most common cause of death in neurotoxic envenomation is respiratory paralysis.
3. Snake venom ophthalmia is the syndrome caused by the spitting cobras. Severe pain, swelling, and corneal ulceration are seen, and blindness can ensue as a secondary complication.
4. A simple bedside 20-minute blood clotting test can be used to determine the presence of significant coagulopathy in areas without laboratory coagulation capability.
5. The WHO has an online database with all antivenom products currently under production.

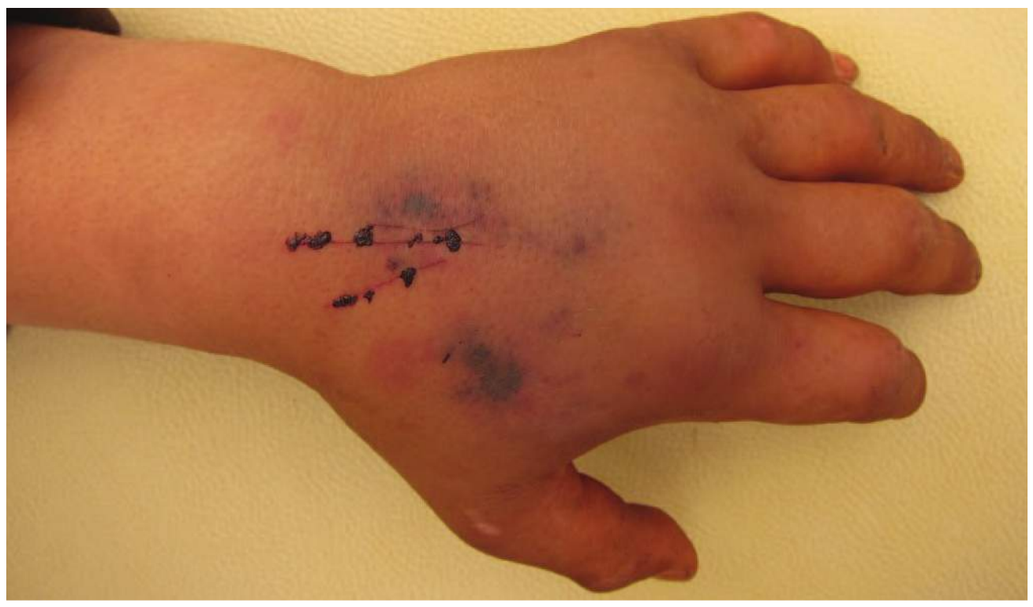

FIGURE 16.52 ■ **Old World Viper Bite.** Local hemorrhagic manifestations at the site of a snake bite in a Syrian man. Old World vipers lack the heat sensing pits of the pit vipers. Bites can cause significant hemorrhagic, coagulopathy, and local tissue complications. (Photo contributor: Seth W. Wright, MD.)

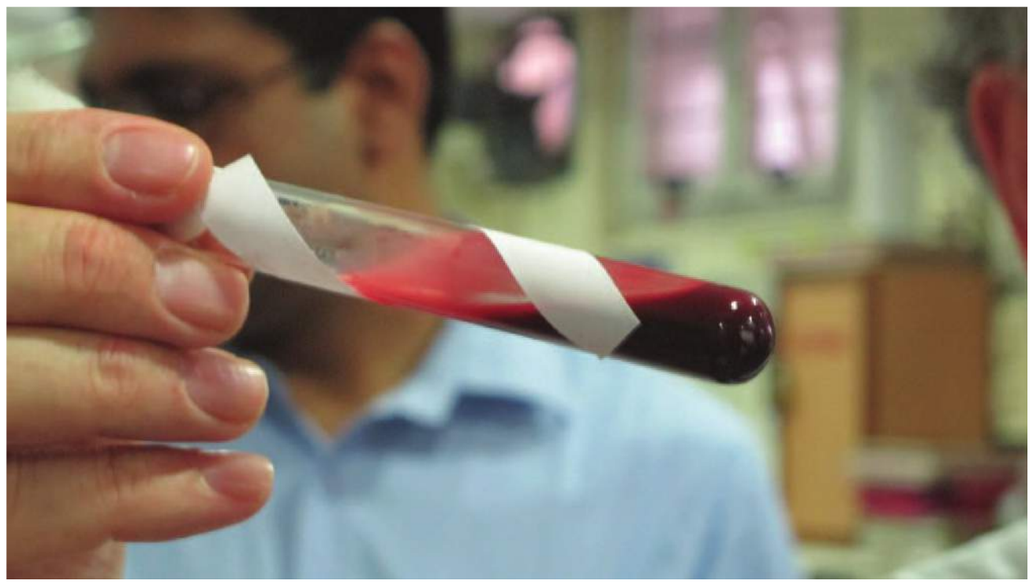

FIGURE 16.53 ■ **Clotting Test.** Positive results of a 20-minute whole blood clotting test are seen with the blood from a man bitten by a Russell's viper. Lack of clotting at 20 minutes correlates closely with other clotting studies. (Photo contributor: Christian Medical College, Vellore, India.)

GILA MONSTER ENVENOMATION

Clinical Summary

The Gila monster (Heloderma suspectum) is a venomous lizard found in the southwestern United States and northwestern Mexico. The only other venomous lizard in the Americas is the Mexican beaded lizard, found from Mexico to Guatemala. Both species are unlikely to bite unless provoked. They secrete venom into their saliva and increase saliva production when they are agitated. When biting their victim, the Gila monster chews rather than injecting, and is known to hang on vigorously and often must be forced to release its powerful grasp. Teeth may break off and contaminate wounds. Like snake venoms, Gila monster and beaded lizard venoms are complex mixtures of proteolytic enzymes and vasoactive substances. The bite may cause pain with local edema and proximal radiation. Tachycardia and hypotension may occur, along with anaphylactic reactions. Systemic complaints may include generalized weakness, nausea and vomiting, diaphoresis, and paresthesias.

Management and Disposition

If not already separated from the victim in the prehospital phase, the lizard must be removed. Putting the lizard under running hot water or prying the jaws apart using an appropriate tool are two options. Caregivers should take precautions against being bitten themselves and should also take care to insure the victim is not bitten again in the removal process. Hypotension can be treated with volume resuscitation and anaphylaxis should be treated with epinephrine. There is no antivenom. Wound care should include copious irrigation and debridement as needed, and imaging should be employed to rule out retained teeth. Tetanus prophylaxis is indicated, but prophylactic antibiotics are generally unnecessary. If the victim has no systemic symptoms several hours after observation, discharge with return precautions is appropriate. Patients with systemic symptoms should be admitted.

Pearls

1. Gila monsters and Mexican beaded lizards have very powerful jaws. Forcible removal is often necessary and must be carefully undertaken to avoid further bites.
2. Patients with systemic symptoms should be admitted, as progression to anaphylactic reactions is possible.

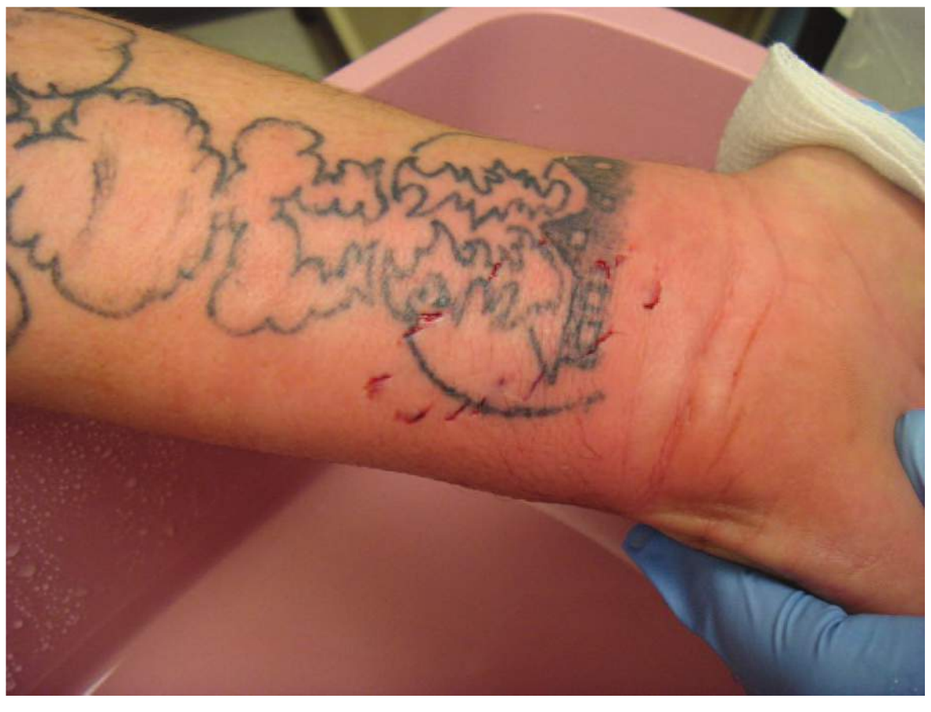

FIGURE 16.55 ■ **Gila Monster Bite.** Gila monster bites may be extremely painful. They usually do not cause severe tissue damage. Retained teeth are a hazard. (Photo contributor: John Sakles, MD.)

FIGURE 16.54 ■ **Reticulate Gila Monster.** The Gila monster (Heloderma suspectum) is one of two venomous lizards found in the Americas. Its range is the southwestern United States and northwestern Mexico. (Photo contributor: Jeff Servoss, US Fish and Wildlife Service.)

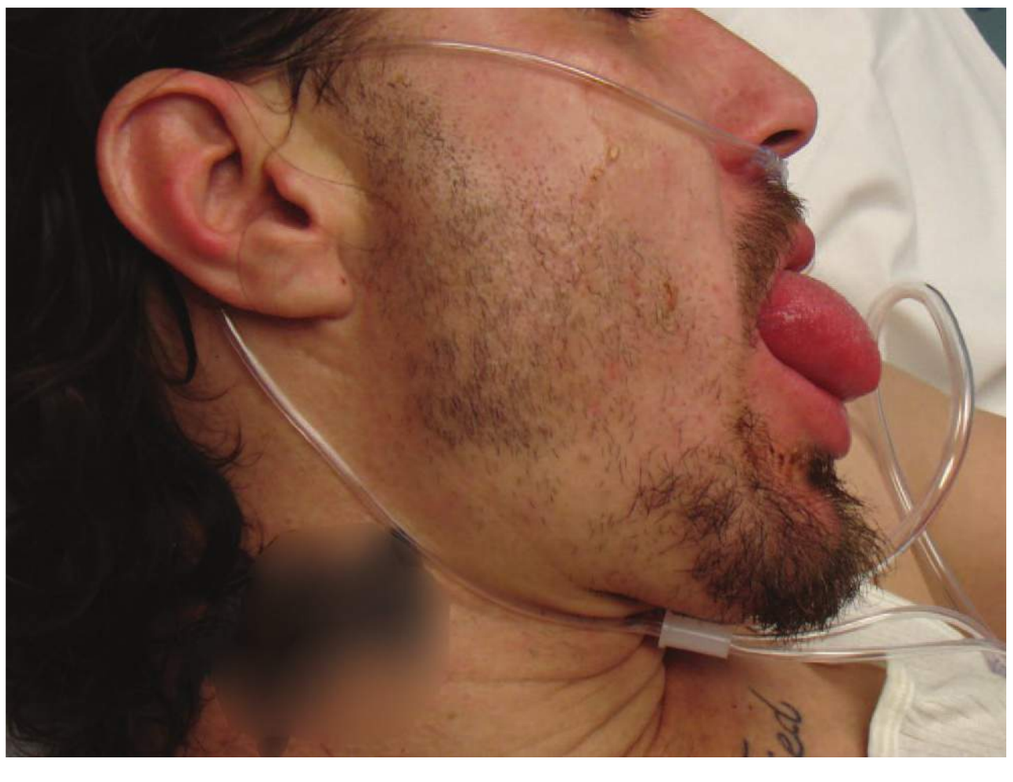

FIGURE 16.56 ■ **Gila Monster Bite, Tongue Swelling.** Gila monster bites may cause anaphylactic reactions. (Photo contributor: John Sakles, MD.)

BLACK WIDOW SPIDER ENVENOMATION

Clinical Summary

The black widow spider (Latrodectus mactans) is the prototype for the genus Latrodectus, several members of which cause human disease. The black widow spider is not particularly aggressive but will defend her web, which is often found in woodpiles, basements, and garages. Most envenomations occur between April and October, with bites most commonly located on the hand and forearm. The clinical presentation of severe and sustained muscle spasm is produced by a neurotoxic protein, which causes the release of acetylcholine and norepinephrine at the presynaptic neuromuscular junction. The initial bite may be mild to moderately painful, but is often missed. Within approximately 1 hour, local erythema and muscle cramping begin, followed by generalized cramping involving large muscle groups such as the thighs, shoulders, abdomen, and back. Associated clinical features can include fasciculations, weakness, fever, salivation, vomiting, diaphoresis, localized sweating at the envenomation site, and a characteristic pattern of facial swelling called Latrodectus facies. Rare cases of seizures, uncontrolled hypertension, and respiratory arrest have occurred.

Management and Disposition

Treatment of the local wound should include cleansing and tetanus prophylaxis. Severe pain and spasm may require intravenous benzodiazepines and opiates. Calcium gluconate infusion has long been recommended to reduce symptoms, although evidence for its efficacy is controversial. Antivenom exists, but carries the same risk as all horse serum products. Antivenom should be considered only in cases of respiratory arrest, seizures, uncontrolled hypertension, and pregnancy.

Pearls

1. Of the five Latrodectus species indigenous to the United States, only three are black and only one has the orange-red hourglass marking.
2. Envenomations by L mactans can mimic an acute abdomen and should be considered in the differential diagnosis of patients presenting with severe acute abdominal pain.

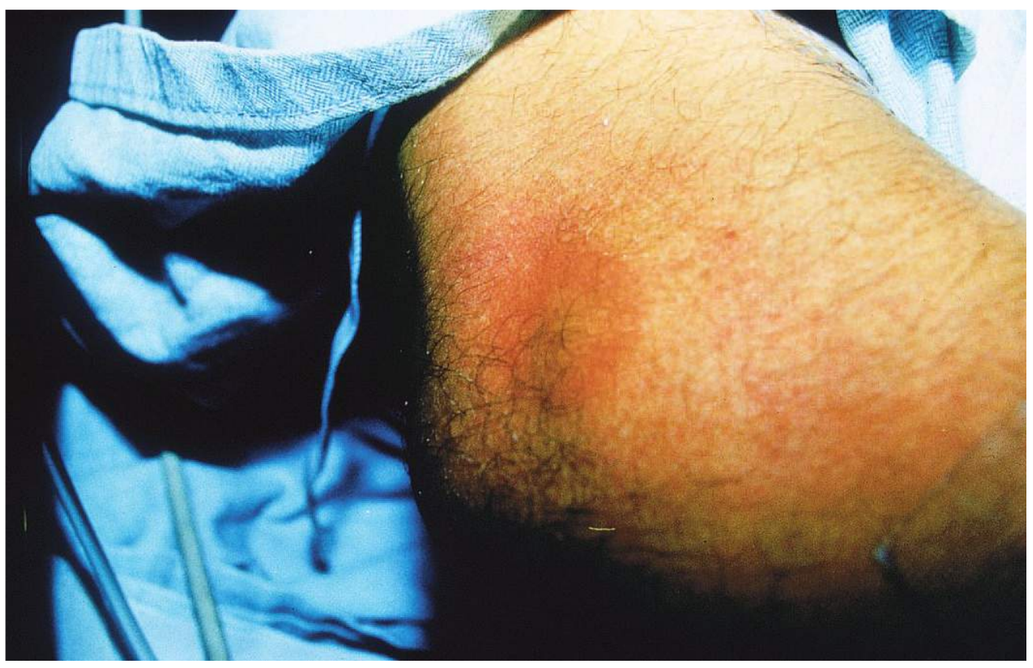

FIGURE 16.58 ■ **Black Widow Spider Bite.** The bite of the black widow spider is clinically subtle. Local reaction is usually trivial, as in this confirmed bite with a small patch of mild erythema. (Photo contributor: Gerald O'Malley, DO.)

FIGURE 16.57 ■ **Black Widow Spider (With Offspring).** Latrodectus mactans, with characteristic hourglass marking on its abdomen. (Photo contributor: Lawrence B. Stack, MD.)

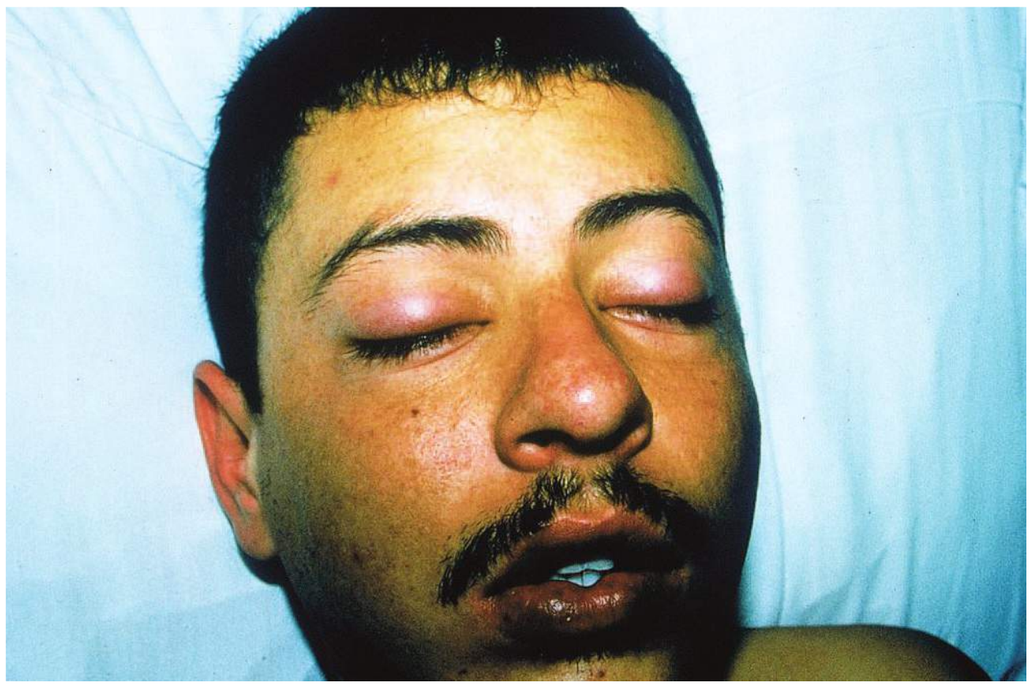

FIGURE 16.59 ■ **Black Widow Facies.** A pattern of facial swelling, known as Latrodectus facies, may occur several hours after envenomation. (Photo contributor: Gerald O'Malley, DO.)

BROWN RECLUSE SPIDER ENVENOMATION

Clinical Summary

The brown recluse spider (Loxosceles reclusa) is the prototypical member of the genus Loxosceles, which as a group can produce necrotic arachnidism following envenomation. These small spiders (approximately 1 cm in body length and 3 cm in leg length) have a worldwide distribution and are identified by fiddle-shaped markings on their anterodorsal cephalothorax. Initial envenomation may be painful, although patients often report no recollection of being bitten. Initial stinging gives way to aching and pruritus. The wound then may become edematous, with an erythematous halo surrounding a violaceous center. The erythematous margin often spreads in a pattern influenced by gravity, leaving the necrotic center near the superior aspect of the lesion. Bullae may erupt, and—over a period of 2 to 5 weeks—the eschar sloughs, leaving a deep, poorly healing ulcer. In approximately 10% of cases, systemic symptoms (loxoscelism) are present. Systemic features of brown recluse envenomation may include fever, nausea, vomiting, headache, morbilliform rash, arthralgias, and, in severe cases, hemolytic anemia, coagulopathy, renal failure, and even death. Children are at higher risk of systemic disease.

Management and Disposition

Most cutaneous lesions secondary to brown recluse spider bites can be managed with cold compresses, elevation, loose immobilization, and attention to tetanus immunization. Severe lesions may require reconstructive plastic surgery several weeks after wound stabilization. The use of dapsone to prevent lesion progression is controversial. Any systemic reaction with evidence of hemolysis, hemoglobinuria, or coagulopathy should prompt admission. Hyperbaric oxygen (HBO) therapy and antivenom have been suggested as possible adjuncts, but no clear consensus of preferred treatment has been established.

FIGURE 16.60 ■ **Brown Recluse Spider.** Brown recluse spider with characteristic "fiddle" marking on the anterodorsal aspect of the cephalothorax. (Photo contributor: R. Jason Thurman, MD.)

FIGURE 16.61 ■ **Fiddle Back Marking.** A close-up look at the characteristic fiddle back marking of the brown recluse spider. (Photo contributor: R. Jason Thurman, MD.)

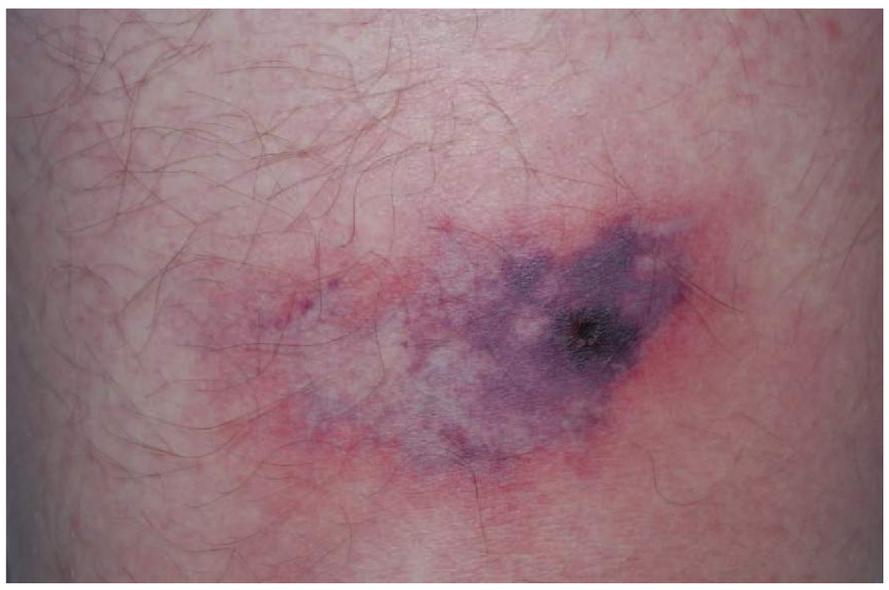

FIGURE 16.62 ■ **Early Brown Recluse Spider Bite.** Early brown recluse spider bite (approximately 8 hours) with a violaceous center surrounded by faint-spreading erythema. Note the "red, white, and blue" appearance. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. The asymmetric spread of erythema, due to the local effects of gravity on the toxin, may help to distinguish a brown recluse spider bite from other arthropod envenomations.
2. If dapsone therapy is to be administered, screening for glucose-6-phosphate dehydrogenase (G6PD) deficiency should be considered.
3. Urinalysis may be helpful in identifying hemolysis early in the clinical course of system reactions.

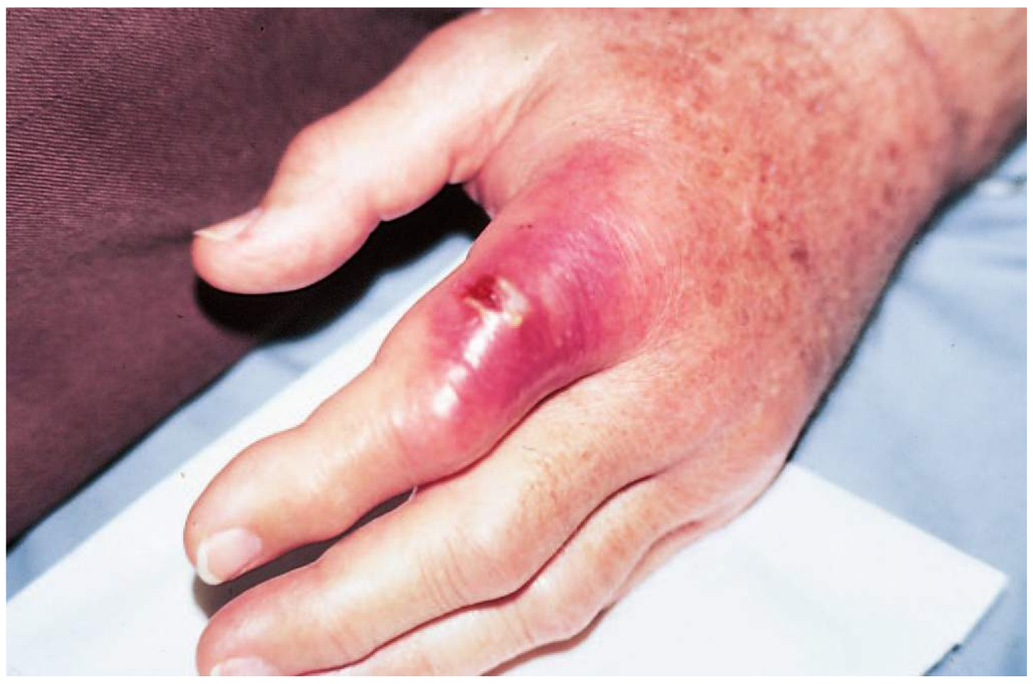

FIGURE 16.63 ■ **Later Recluse Spider Bite (24 hours).** Brown recluse spider bite at approximately 24 hours. Note asymmetric spread of erythema and early central ulcer formation. (Photo contributor: Edward Eitzen, MD, MPH.)

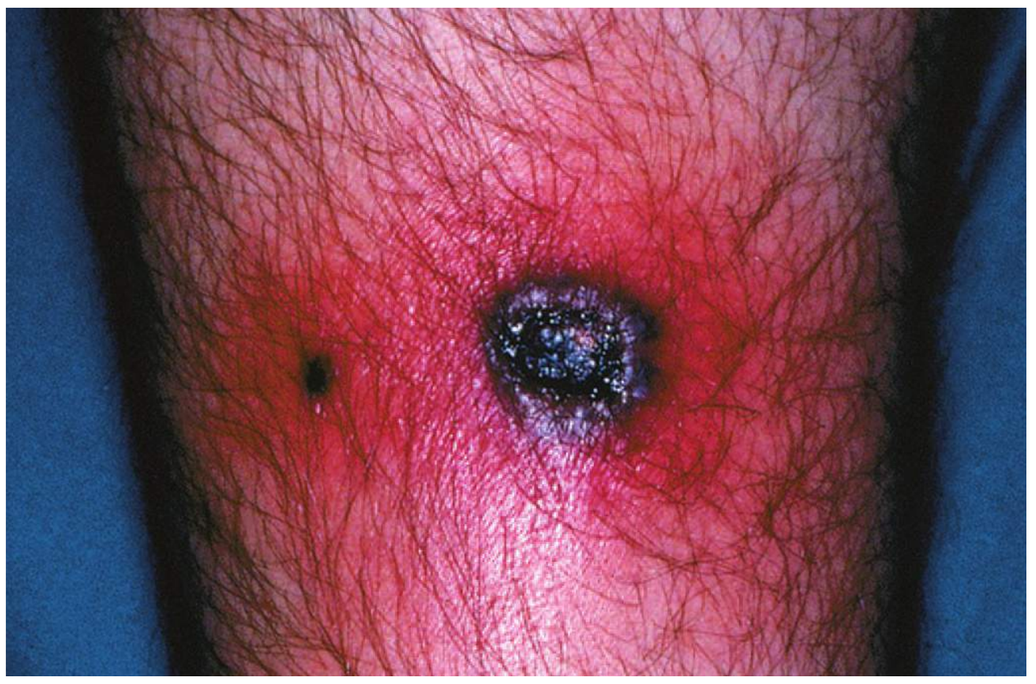

FIGURE 16.64 ■ **Recluse Necrosis (Weeks).** Within about 2 to 5 weeks, significant brown recluse envenomations may produce a deep, poorly healing ulcer with necrosis. (Photo contributor: Kevin J. Knoop, MD.)

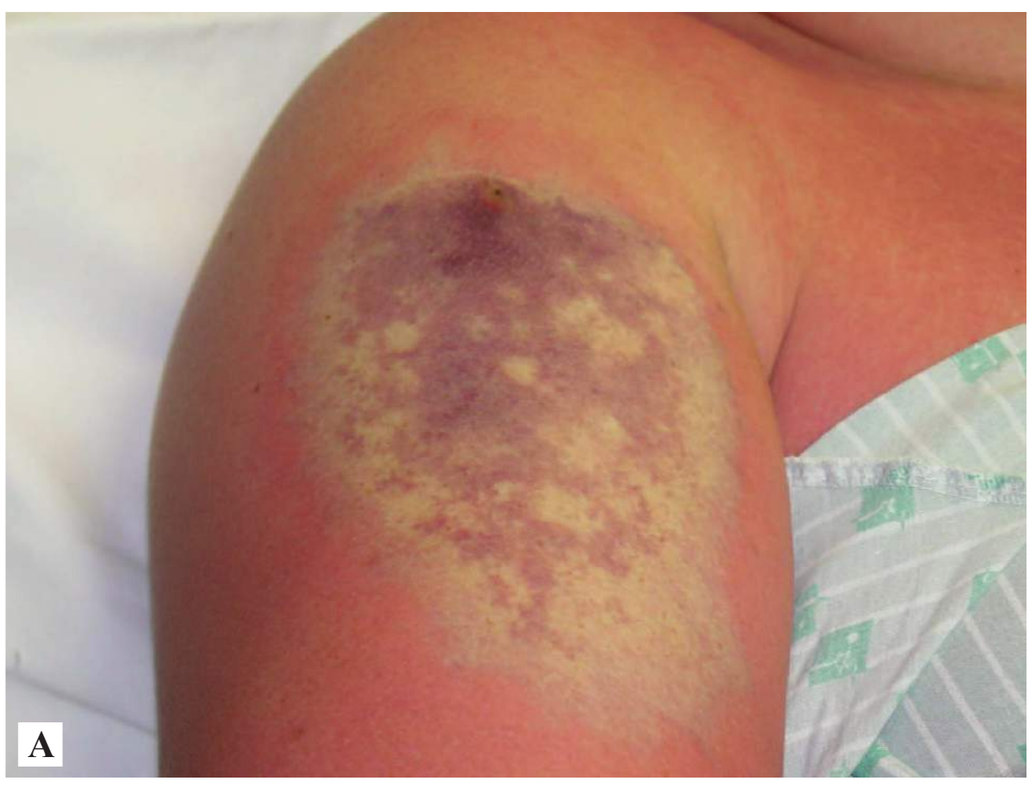

FIGURE 16.65A ■ **Brown Recluse Spider Bite.** Red, white, and blue appearance with significant asymmetric spread of erythema inferiorly down the arm beyond the envenomation site due to effect of gravity. (Photo contributor: Shannon B. Snyder, MD.)

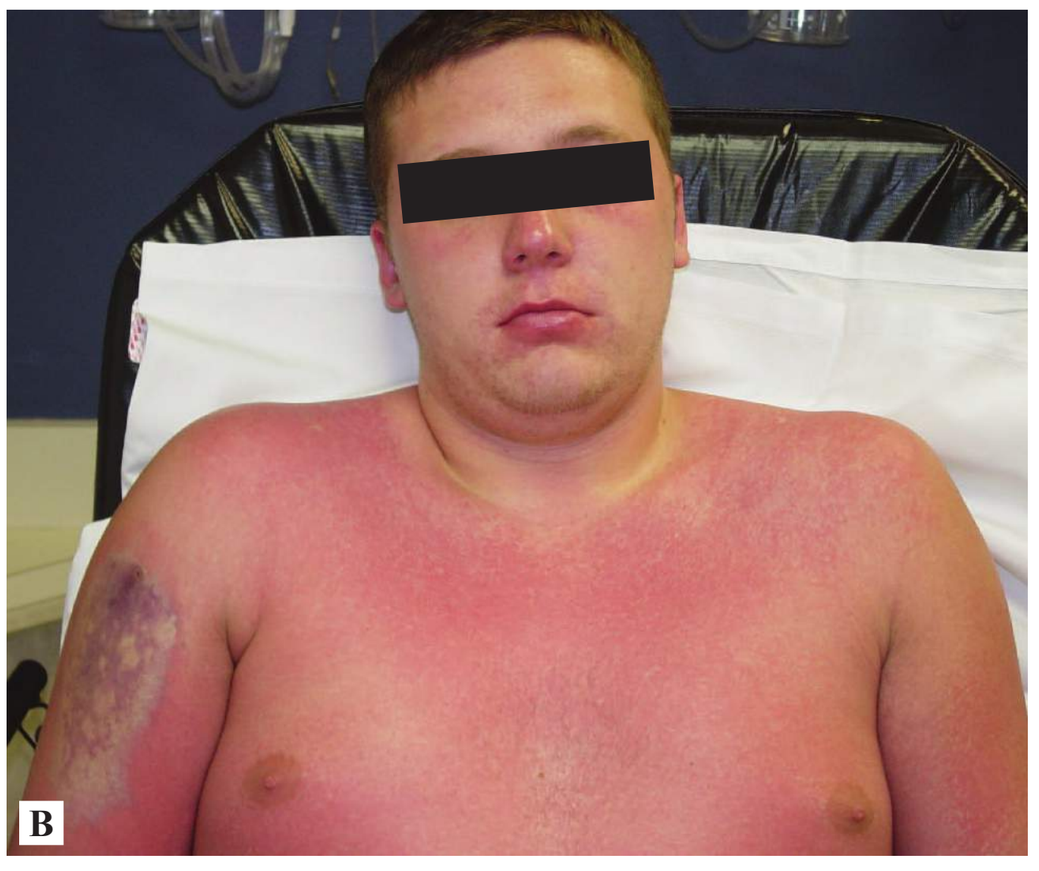

FIGURE 16.65B ■ **Systemic Loxoscelism.** This patient suffered fever, headache, and a diffuse erythematous rash as a result of systemic loxoscelism. Note the brown recluse envenomation on the patient's right arm, seen also in Fig.16.65A. (Photo contributor: Shannon B. Snyder, MD.)

Clinical Summary

The most significant morbidity and mortality from scorpion stings is from the Buthidae family, characterized by a triangular central sternal plate. This family includes the venomous Androctonus genus in northern Africa, Leiurus in the Middle East, Tityus in South America, and Centruroides in North America.

Centruroides are found primarily in the southwestern United States and northern Mexico and are characterized by a variable subaculear tooth beneath the stinger. They may be striped, and are yellow to brown in color. These scorpions tend to hide in crevices, woodpiles, bedding, clothing, and shoes. Envenomation produces a fairly mild local reaction of pain, swelling, burning, and ecchymosis.

Centruroides exilicauda (the bark scorpion) envenomation can lead to progressive symptoms and, very rarely, death. The venom of C exilicauda initially produces local paresthesias and pain (grade 1), which may be accentuated by tapping the involved area. More severe envenomations may produce remote paresthesias (grade II) and either somatic or autonomic nervous system dysfunction (grade III). Systemic symptoms may include tachycardia, nausea, wandering eye

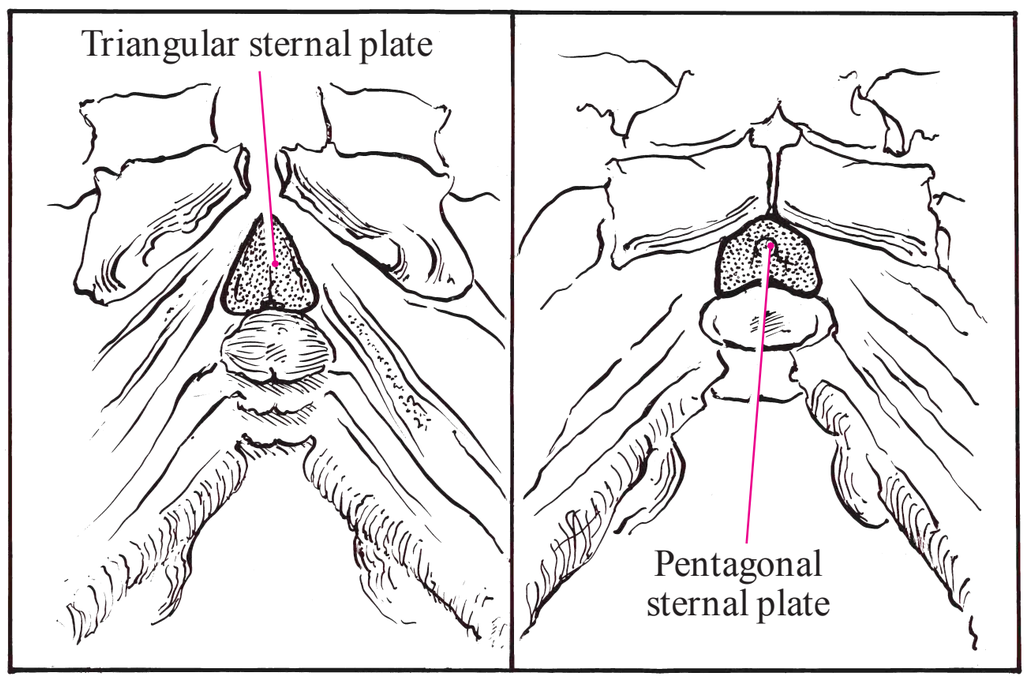

FIGURE 16.66 ■ **Buthidae Sternal Plate.** The Buthidae family is associated with the most significant envenomations and is characterized by triangular sternal plates (left). Members of the other scorpion families have pentagonal sternal plates (right).

FIGURE 16.68 ■ Centruroides exilicauda. Members of this species are yellow to brown and usually less than 5 cm long. Below the stinger is the telson, within which are two glands containing venom. (Photo contributor: Sean P. Bush, MD.)

FIGURE 16.67 ■ **Buthidae Sternal Plate.** The triangular appearance of the sternal plate is well seen in this scorpion, a member of the Buthidae family. (Photo contributor: Sean P. Bush, MD.)

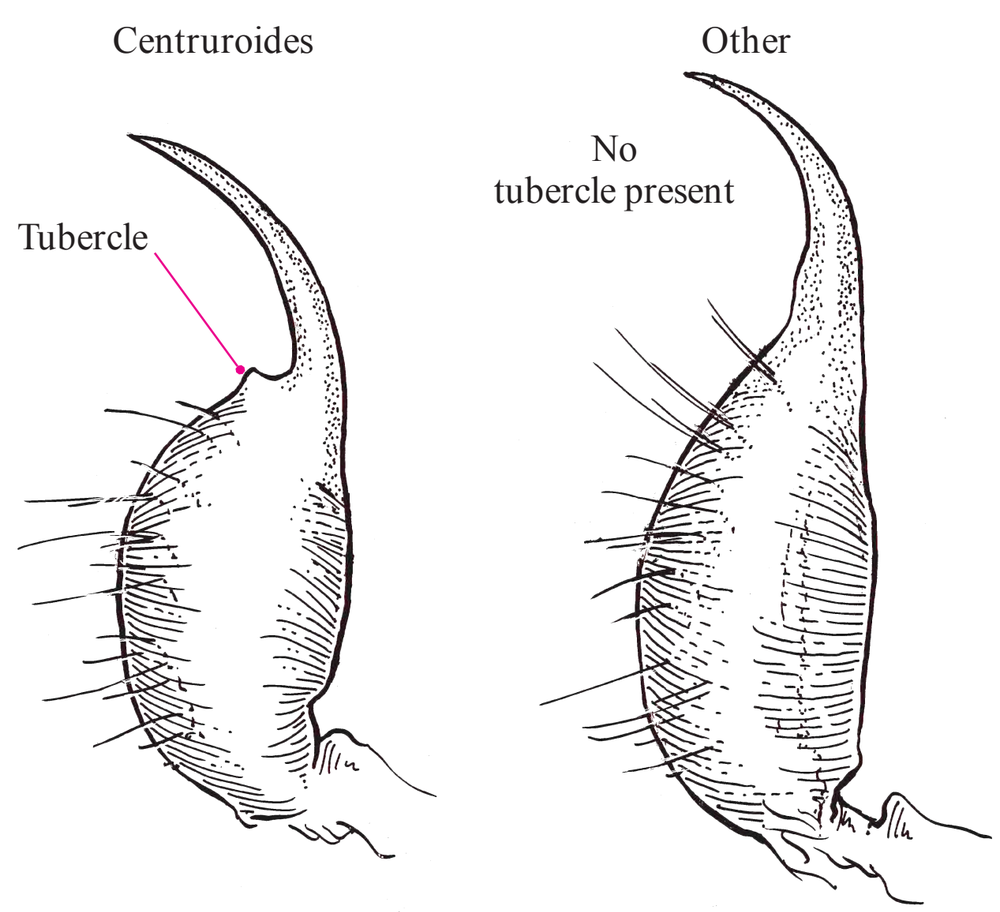

FIGURE 16.69 ■ **Subaculear Tooth.** The barb noted at the base of the stinger is variably present in Centruroides (left) and absent in other species (right).

movements, blurred vision, difficulty breathing, trouble swallowing, restlessness, and involuntary shaking. Both somatic and autonomic dysfunction may be present (grade IV). Systemic reactions tend to be more severe in younger patients and may result in death, usually from respiratory arrest.

Management and Disposition

Treatment depends on the severity of envenomation. Grade I or II envenomations are treated with supportive care (ice, oral analgesia) and tetanus immunization. Envenomations that progress to grade III or IV must be treated aggressively and may require paralysis and intubation for severe spasms. Centuroides antivenom (Anascorp) is available, but is horse-serum based and carries a risk of hypersensitivity reactions. Pain and paresthesias may persist for up to 2 weeks; most systemic symptoms improve within 9 to 30 hours without antivenom treatment and usually peak at about 5 hours.

Pearls

1. Exercise caution when treating pain of C exilicauda envenomations with opioids, as synergistic respiratory depressive effects between venom and opiates may occur.
2. If the scorpion is brought in, it should be examined for the presence of a triangular plate and subaculear tooth.
3. Almost all scorpions, including C exilicauda, fluoresce with intense brightness under cobalt light.

FIGURE 16.70 ■ Centruroides limbatus. Subaculear tooth. A variable subaculear tooth is characteristic of Centruroides. This is an example of a large subaculear tooth on the telson from C limbatus. Centruroides exilicauda typically has a smaller, sometimes subtle "tooth." (Photo contributor: Sean P. Bush, MD.)

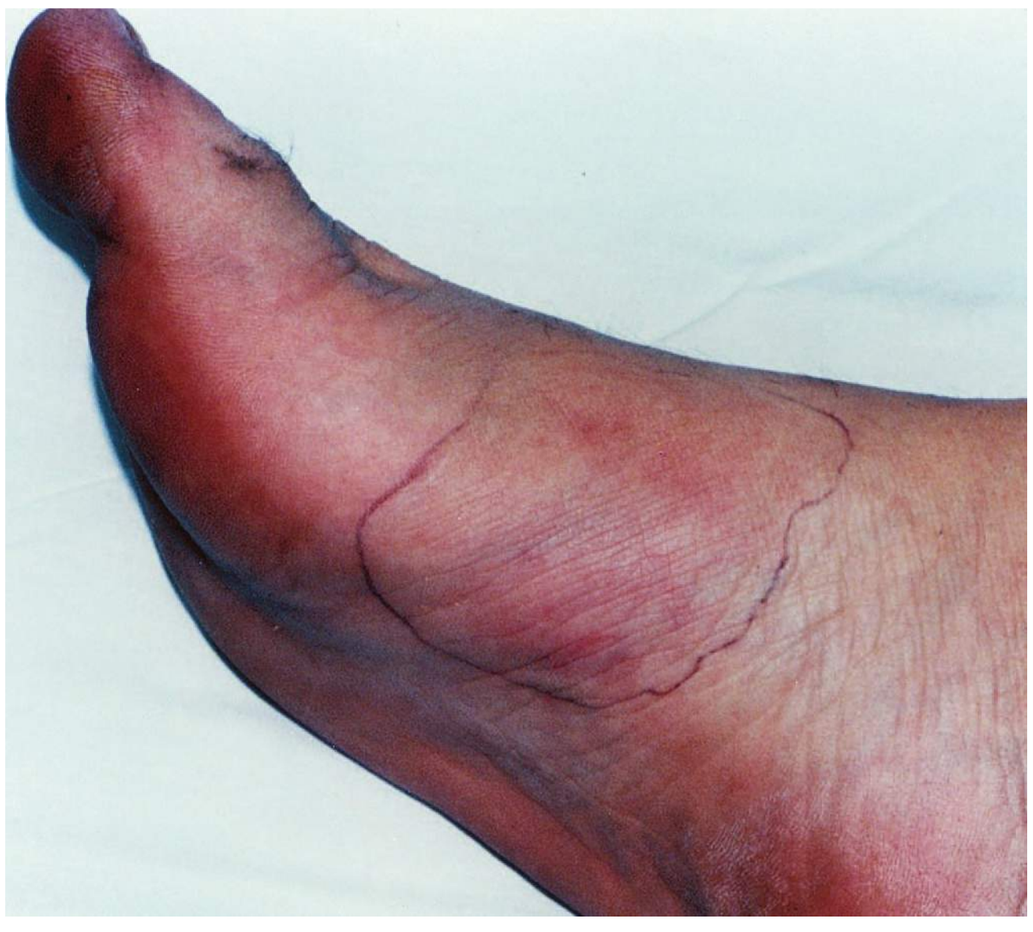

FIGURE 16.71 ■ **Scorpion Sting.** Most scorpion envenomations are mild and produce local pain, swelling, paresthesias, and mild ecchymosis. (Photo contributor: Stephen W. Corbett, MD.)

Clinical Summary

Ticks are blood-sucking parasites of people and animals. Ticks cause illness by acting as vectors for pathogens, or by secreting toxins or venoms. Ticks carry more types of infectious pathogens than any other arthropods except mosquitoes. The most important of these include Borrelia (responsible for Lyme disease and relapsing fever), Rickettsia (eg, Rocky Mountain spotted fever [RMSF]), Ehrlichia (ehrlichiosis), viral pathogens (eg, Colorado tick fever), and babesiosis. Rashes are prominent in Lyme disease, RMSF, and southern tick associated rash illness (STARI), sometimes present in relapsing fever, uncommon in Colorado tick fever, and absent in babesiosis.

Clinically important ticks in North America include Ixodes dammini, the deer tick (Lyme disease and babesiosis), Dermacentor andersoni, the wood tick (RMSF and Colorado tick fever), Dermacentor variabilis, the dog tick (RMSF, ehrlichiosis), and Amblyomma americanum, the lone star tick (a very widespread tick implicated in the transmission of Lyme disease outside of the range of I dammini as well as STARI and ehrlichiosis). More than 40 species of ticks can cause tick paralysis. In North America, the most common cause is D andersoni, but A americanum and Ixodes species have also been associated with tick paralysis.

Tick paralysis develops 5 to 6 days after an adult female tick attaches. Over the next 24 to 48 hours, an ascending, symmetric, flaccid paralysis develops. Alternative presentations include ataxia and associated cerebellar findings without muscle weakness or isolated facial paralysis. Resolution of the paralysis after removal of the tick establishes the diagnosis.

Management and Disposition

If still embedded, the tick should be removed promptly by grasping it as close to the skin surface as possible, using blunt curved forceps or tweezers. The tick should be pulled out with slow, gentle traction, taking care not to crush or squeeze the body, which may result in injection of contaminated tick fluids. Other methods of tick removal—such as application of fingernail polish, isopropyl alcohol, or a hot match head—have not been proven to effect detachment and may induce regurgitation of tick contents into the wound.

Patients with tick paralysis may require supportive care, including mechanical ventilation. Patients with tick-borne illnesses may require admission for supportive care or intensive antibiotic treatment, but when clinically appropriate may be treated as an outpatient with appropriate antibiotic therapy.

Pearls

1. Prevention of tick bites includes the use of protective clothing containing N, N-diethylmetatoluamide (DEET).
2. A clear history of a tick bite is present in less than one-third of Lyme disease cases.
3. Unusual neurologic presentations, particularly bilateral peripheral seventh-nerve palsies, should prompt consideration of Lyme disease.
4. Patients with paralysis in endemic areas should be thoroughly searched for embedded ticks.

FIGURE 16.72 ■ **Deer Tick.** Ixodes dammini, the deer tick, is a vector of Lyme disease and babesiosis. (Photo contributor: Centers for Disease Control and Prevention, Atlanta, GA.)

FIGURE 16.73 ■ **Wood Tick.** Dermacentor andersoni, the wood tick, is a vector of Rocky Mountain spotted fever and Colorado tick fever. (Photo contributor: Centers for Disease Control and Prevention, Atlanta, GA.)

FIGURE 16.74 ■ **Lone Star Tick.** Amblyomma americanum, the lone star tick, has been implicated as a vector in STARI. (Photo contributor: Sherman Minton, MD.)

FIGURE 16.76 ■ **American Dog Tick.** Dermacentor variabilis, the dog tick, is a vector of Rocky Mountain spotted fever and ehrlichiosis. (Photo contributor: R. Jason Thurman, MD.)

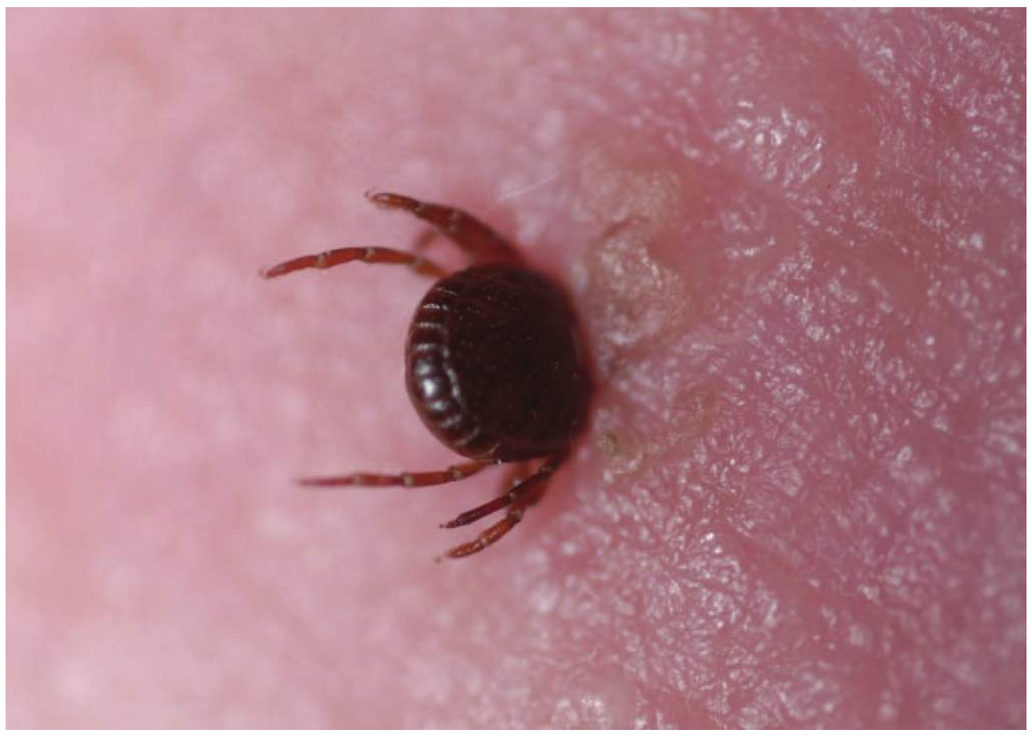

FIGURE 16.75 ■ **Imbedded Tick.** This lone star tick was found imbedded into the patient's shoulder. It was easily removed intact with tweezers. (Photo contributor: R. Jason Thurman, MD.)

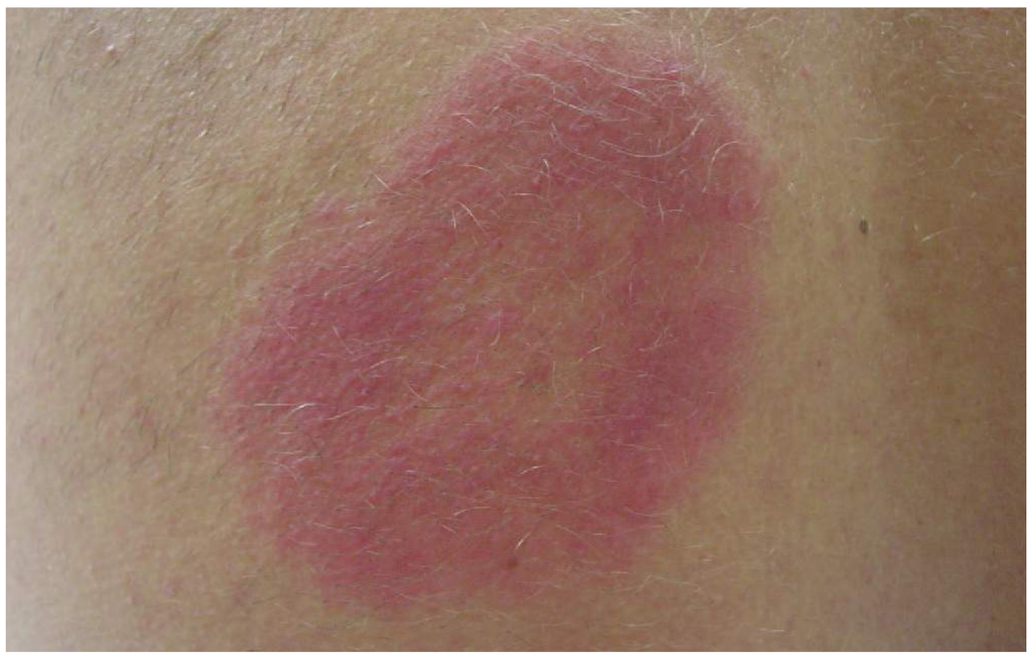

FIGURE 16.77 ■ **Erythema Migrans Rash.** This erythema migrans rash was located at the site of a tick bite in a patient who developed southern tick associated rash illness (STARI). Erythema migrans rash is also associated with Lyme disease. (Photo contributor: Shannon B. Snyder, MD.)

HYMENOPTERA ENVENOMATION

Clinical Summary

The order Hymenoptera includes wasps, hornets, yellow jackets, bees, and ants. Envenomation usually results in local pain, mild erythema, swelling, and pruritus. Severe systemic or toxic reactions may occur from one or multiple stings, manifesting as gastrointestinal symptoms, headache, pyrexia, muscle spasms, or seizures. Anaphylaxis may occur within minutes from a single sting, and may cause death from airway obstruction and/or cardiovascular collapse. A serum sickness–type reaction may occur 7 to 14 days after envenomation.

Solenopsis invicta was imported from South America and is the most prominent fire ant in the United States. These ants are primarily found in the South and build mound nests in open grass settings, commonly in urban yards. Disturbing the nests may result in severe swarming attacks, a common occurrence in the unsuspecting barefoot victim. Bites are painful and produce sterile pustules that crust over in a few days.

Management and Disposition

Anaphylaxis is treated with conventional therapy with careful attention to airway management. Local reactions may be treated with ice packs, steroid cream, and oral antihistamines. Opiates may be needed for severe pain.

Pearls

1. Honeybee stings are usually apparent since the stinger apparatus, including barb and venom sac, is often detached and present on the patient's skin. These should be removed by scraping them off, as grasping with tweezers may result in the release of more venom.
2. "Brazilian killer" or "Africanized" bees are present primarily in the southwestern United States. Their venom is not known to be more toxic than that of other bees, but their aggressiveness, tendency to swarm in large numbers, and ability to travel long distances make them potentially more dangerous to humans.
3. Immediately remove any rings when envenomations are located on the hands or feet as marked local swelling can occur, putting the distal digits at risk for ischemia.

FIGURE 16.79 ■ **Paper Wasp Nest.** A typical paper wasp nest in a roof corner. Disturbance of a nest may result in swarming attacks. (Photo contributor: Clay B. Smith, MD.)

FIGURE 16.78 ■ **Paper Wasp.** Paper wasps are found throughout the world and often establish nests close to or within human dwellings. (Photo contributor: R. Jason Thurman, MD.)

FIGURE 16.80 ■ **Fire Ant Mound.** This typical fire ant mound is a raised area of dirt in an urban yard. (Photo contributor: Alan B. Storrow, MD.)

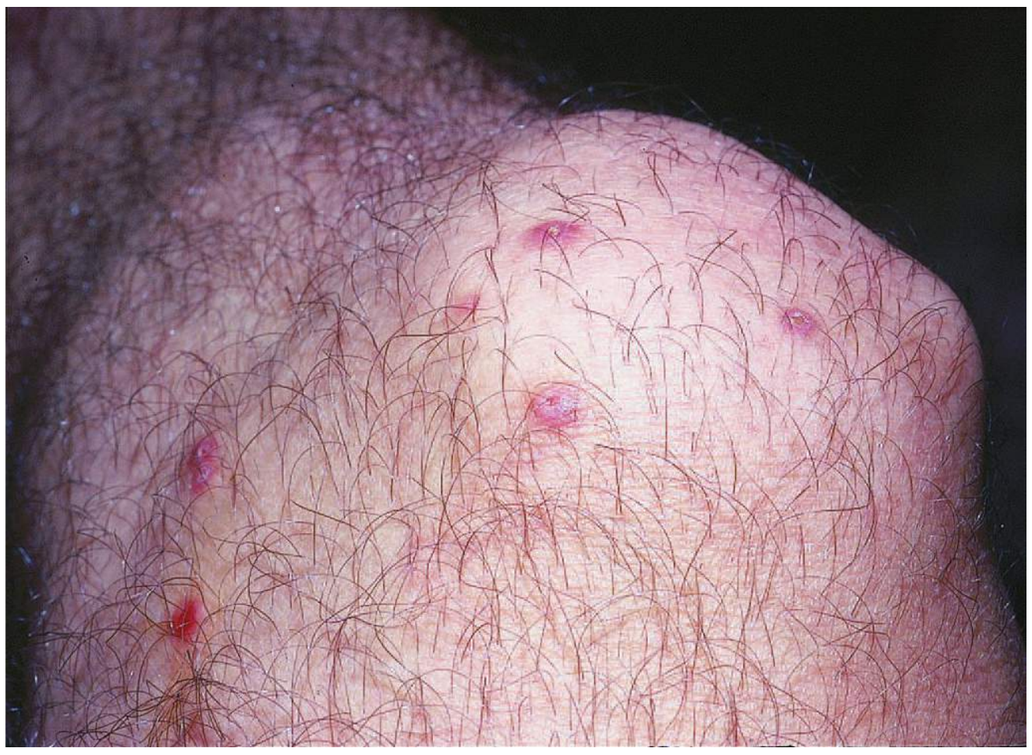

FIGURE 16.81 ■ **Fire Ant Bites.** These fire ant bites on the anterior knee occurred after this patient knelt on a mound. These bites are 3 days old; the initial sterile pustules have begun to crust over. (Photo contributor: Alan B. Storrow, MD.)

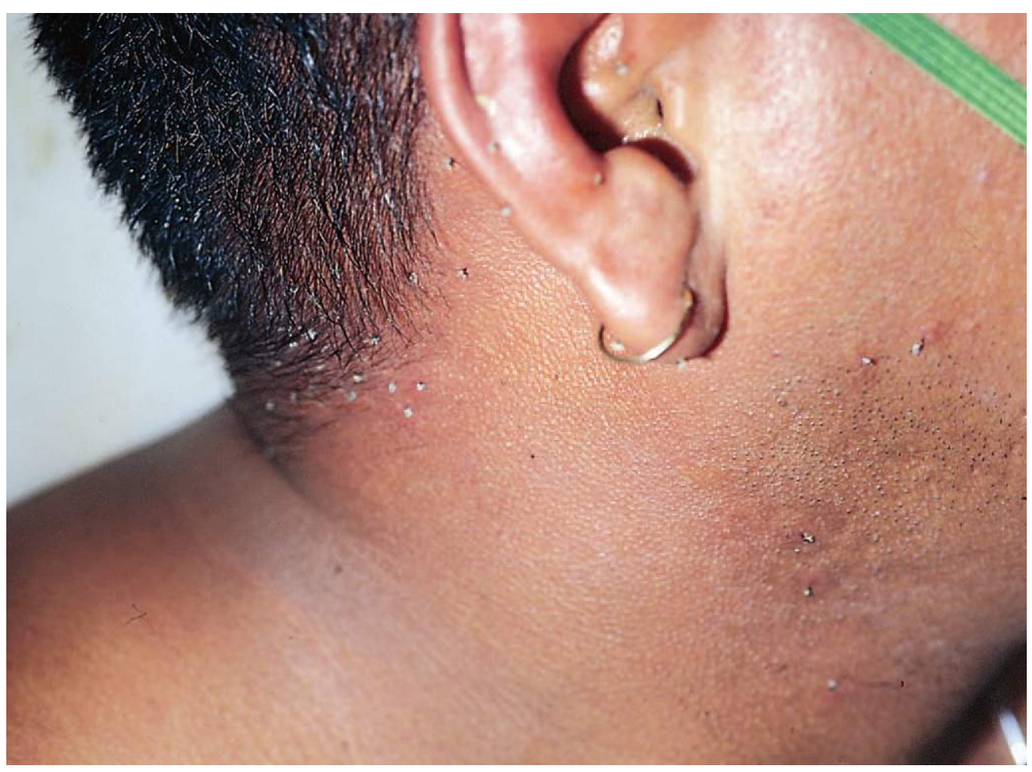

FIGURE 16.82 ■ **Honeybee Envenomation.** Many honeybee stingers (barbs and venom sacs) are seen on this patient's cheek and ear and along the hairline. (Photo contributor: Alan B. Storrow, MD.)

FIGURE 16.83 ■ **Honeybee Stingers.** The barbs and attached venom sacs (stinger apparatus) after removal from the patient. (Photo contributor: Alan B. Storrow, MD.)

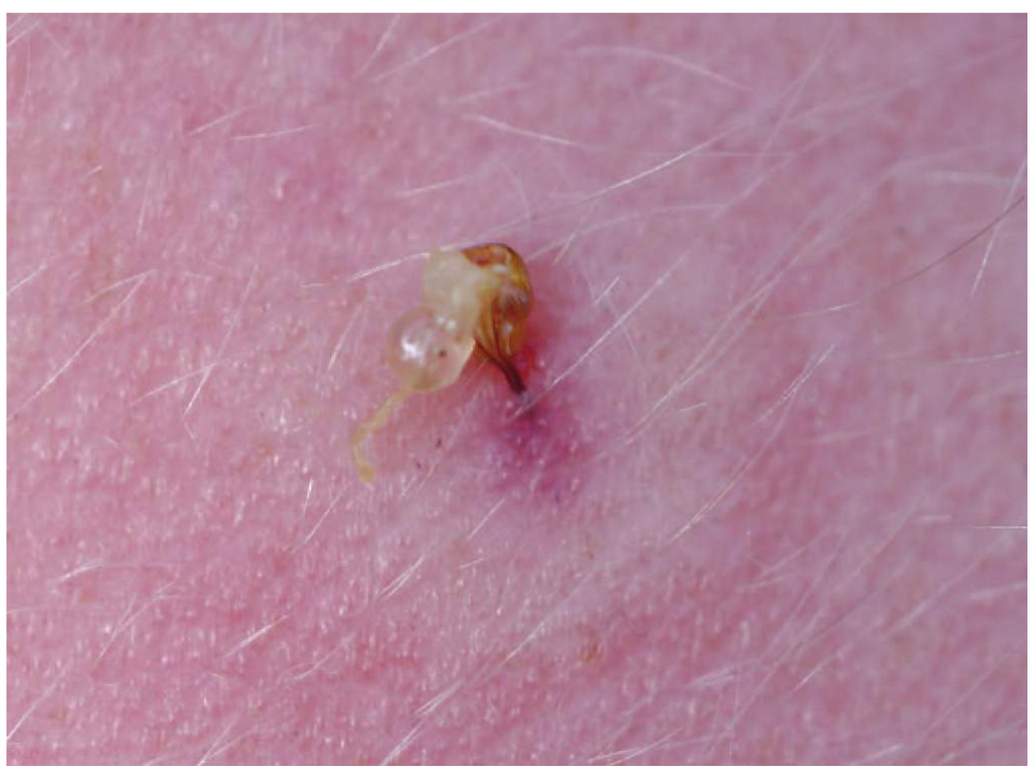

FIGURE 16.84 ■ **Imbedded Honeybee Stinger.** Note the venom sac, still pulsating and attached to the honeybee stinger imbedded in the victim. (Photo contributor: Lawrence B. Stack, MD.)

CATERPILLAR, MITE, AND CENTIPEDE ENVENOMATION

Clinical Summary

Caterpillar venom apparatus typically consists of barbed spines arranged in clumps or scattered about the dorsal surface of the insect. These are purely defensive in nature. Patients who are stung commonly have intentionally handled the insect or have had accidental skin contact while gardening. Envenomated patients typically present with acute pain followed by focal erythema and swelling. Caterpillars with a less sophisticated venom apparatus or low-potency venom may cause simple focal pruritus or urticaria, though some caterpillars are capable of producing a very painful sting requiring aggressive pharmacological pain control. Systemic symptoms are rare. The puss caterpillar, or wooly slug (Megalopyge opercularis) is perhaps the most well-known and important venomous caterpillar in the United States. Wooly slug caterpillars have a widespread distribution, appear hairy and flat, and may reach a length of 4 cm.

Chiggers are the larvae of trombiculid mites and may inflict multiple intensely pruritic bites on their victims. They are parasitic only as larvae and infest humans by crawling onto them and latching on. Proper clothing precautions and repellents are usually effective in reducing unpleasant chigger infestations.

Centipedes are venomous arthropods that have one pair of legs per body segment. The first segment contains hollow curved "fangs" (really modified legs) bearing venom glands at the bases, which are capable of penetrating human skin. Centipedes generally use venom to kill prey, but, when provoked, may envenomate humans and produce local intense burning pain, erythema, and swelling. Systemic reactions may occur but are uncommon.

Management and Disposition

Treatment of caterpillar, mite, and centipede envenomations is purely supportive and consists of pain control, antihistamines, topical antipruritic creams, and basic wound care.

Pearls

1. Attached caterpillar spines may be removed easily with adhesive tape.
2. Infiltration with local anesthetics may be useful in markedly painful centipede envenomations or for the removal of retained "fang" fragments.
3. The caudal appendages of centipedes are not associated with a venom apparatus.

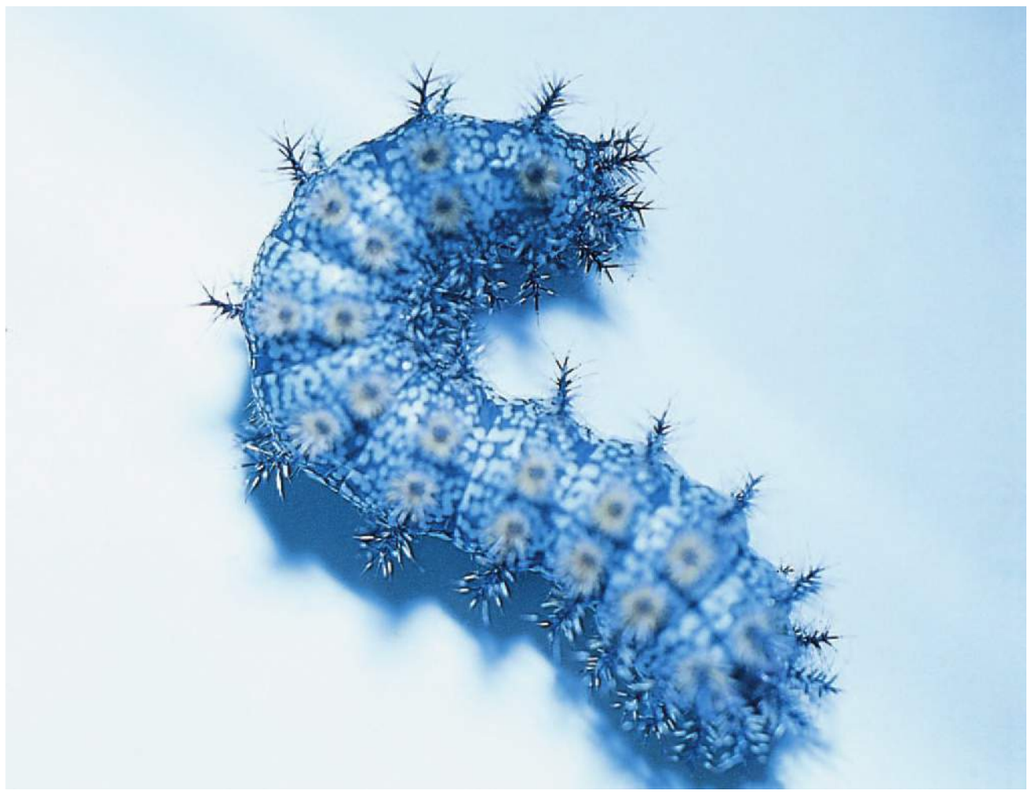

FIGURE 16.85 ■ **Caterpillar with Barbed Spines.** Typical garden caterpillar with barbed spines arranged in clumps. (Photo contributor: Alan B. Storrow, MD.)

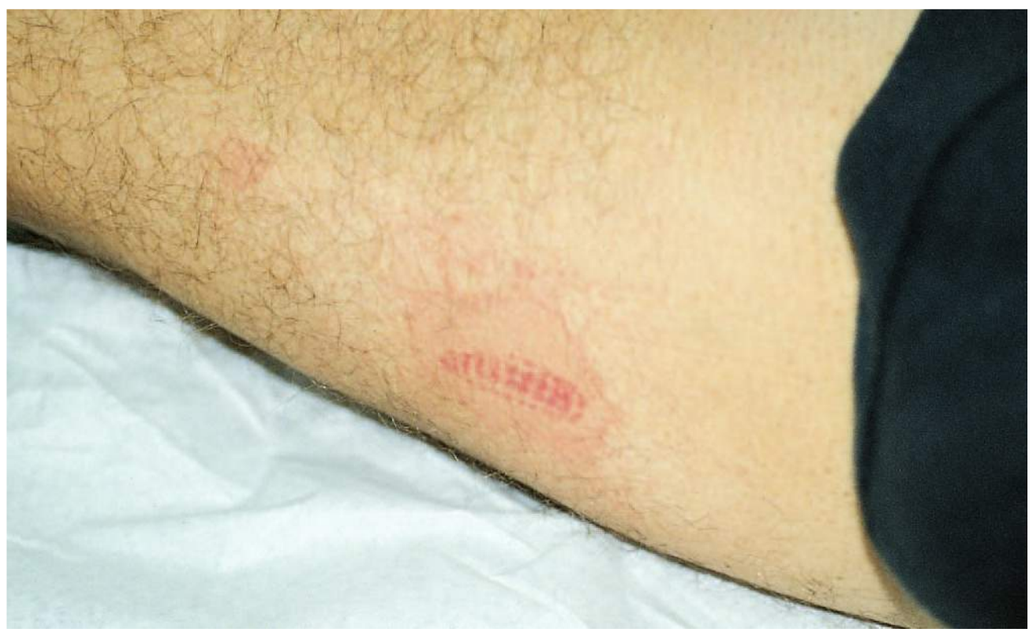

FIGURE 16.86 ■ **Caterpillar Sting.** Appearance of a caterpillar sting at 2 hours. The patient presented with moderate pain and severe itching. Note how the erythema follows the pattern of the caterpillar. (Photo contributor: Alan B. Storrow, MD.)

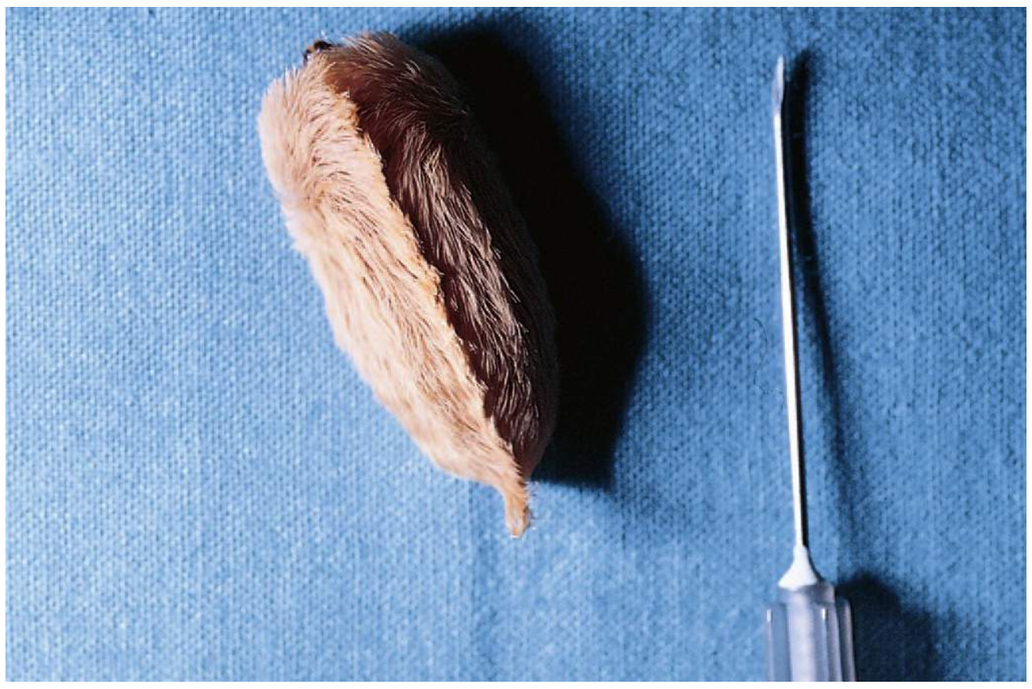

FIGURE 16.87 ■ **Puss Caterpillar.** The "puss caterpillar" or "woolly slug" is likely the most important venomous caterpillar in the United States. The hairy appearance and small hair tail is characteristic. (Photo contributor: Alan B. Storrow, MD.)

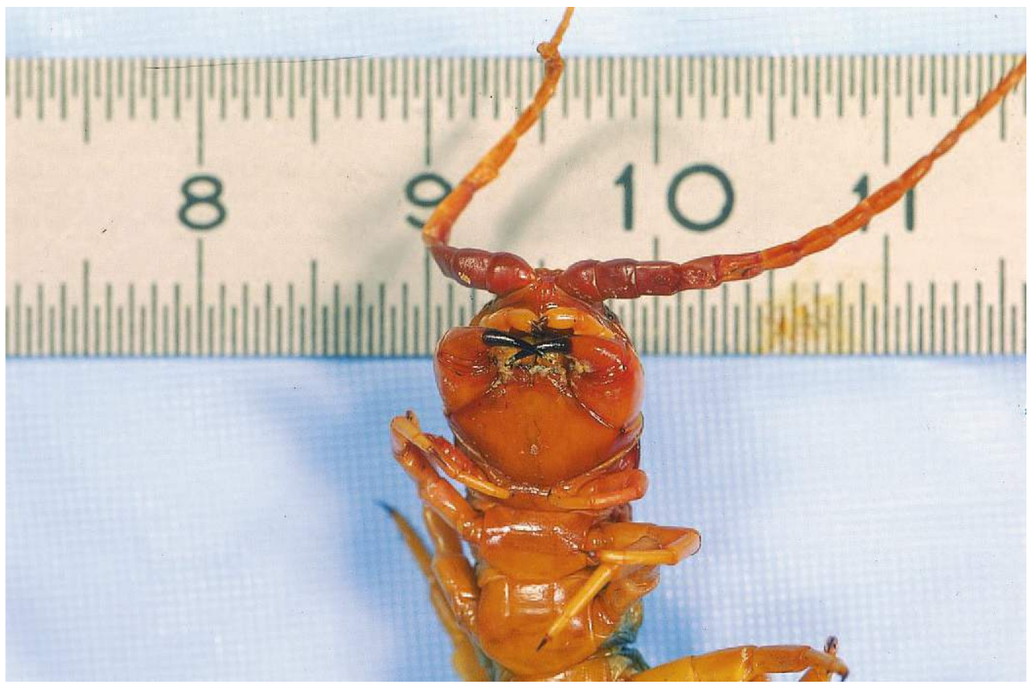

FIGURE 16.88 ■ **Centipede.** Note the curved "fangs" (actually modified legs) on the first segment of this centipede from Texas. (Photo contributor: Alan B. Storrow, MD.)

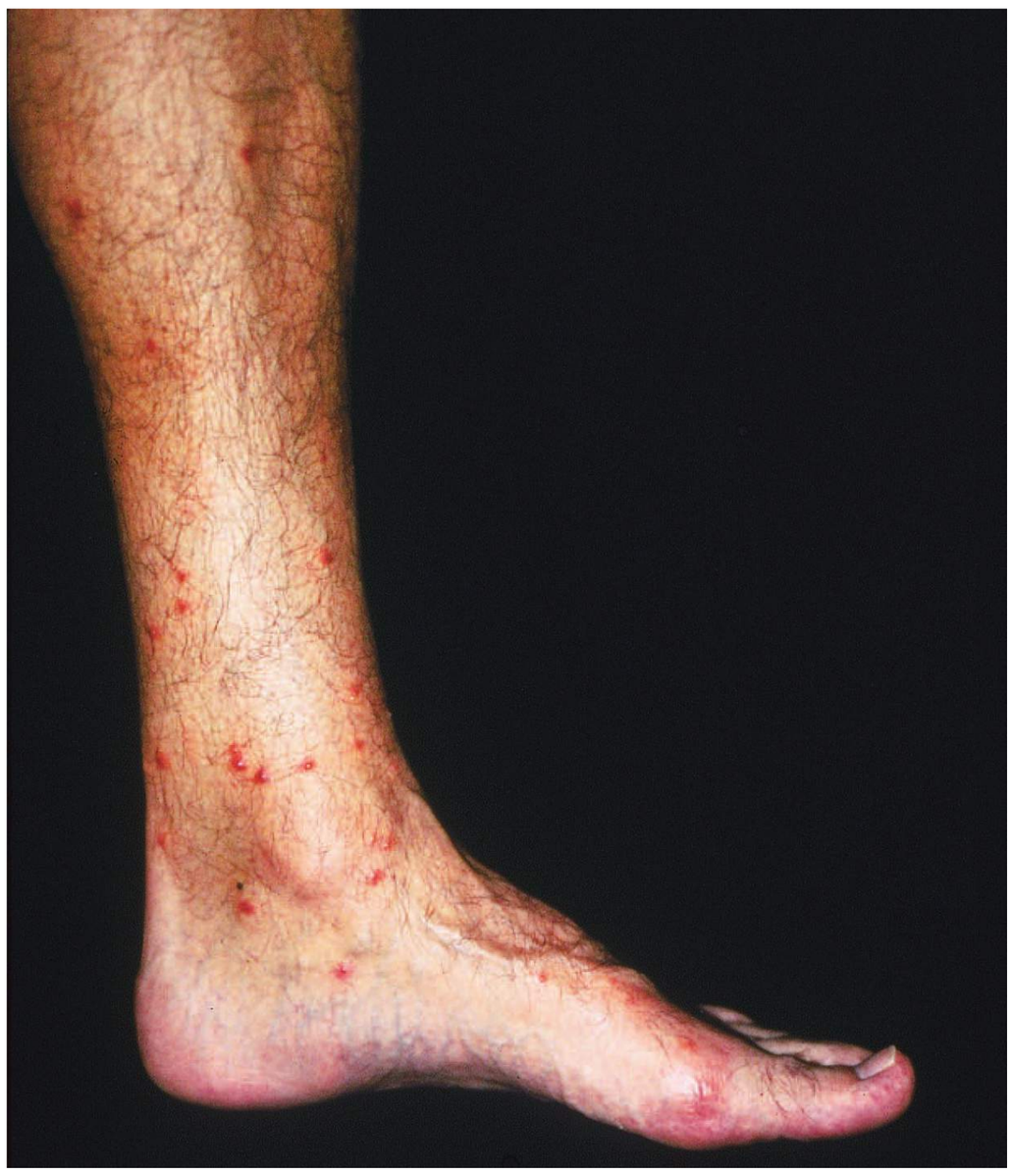

FIGURE 16.89 ■ **Chigger Bites.** Chigger bites on a leg appear as puncta surrounded by erythema. (Photo contributor: Kevin J. Knoop, MD, MS.)

MIDDLE EAR SQUEEZE

Clinical Summary

A middle ear squeeze (barotitis media) results from a decrease in pressure within the middle ear as an individual descends through water or is exposed to an increase in atmospheric pressure that can be seen in descending aircraft or while driving in mountainous terrain. According to Boyle's law, as pressure increases, volume must decrease proportionately. At a depth of approximately 4 ft, the pressure difference is great enough to collapse the eustachian tube and cause obstruction. If attempts to equalize the pressure (eg, Valsalva or Frenzel maneuver) fail, ascent is necessary or injury may ensue. If a diver continues to descend, hemorrhage and edema occur within the middle ear and rupture of the TM may occur. The influx of water into the middle ear may cause extreme vertigo and lead to a diving disaster.

Barotitis media may present with pain only (grade 0), TM erythema (grade 1), erythema and mild TM hemorrhage (grade 2), gross TM hemorrhage (grade 3), free middle ear blood (grade 4), or free blood with TM perforation (grade 5).

Management and Disposition

Treatment includes decongestants and appropriate analgesia. Antihistamines may be of use for allergy-related eustachian tube dysfunction. Antibiotics are recommended for preexisting infections or for TM rupture. Most cases resolve spontaneously within hours to days. The patient should not resume diving until the condition has resolved or the TM is completely healed.

Pearls

1. Barotitis media is the most common medical problem associated with diving.
2. Associated barotraumatic injuries should be considered when the diagnosis of barotitis media is made.

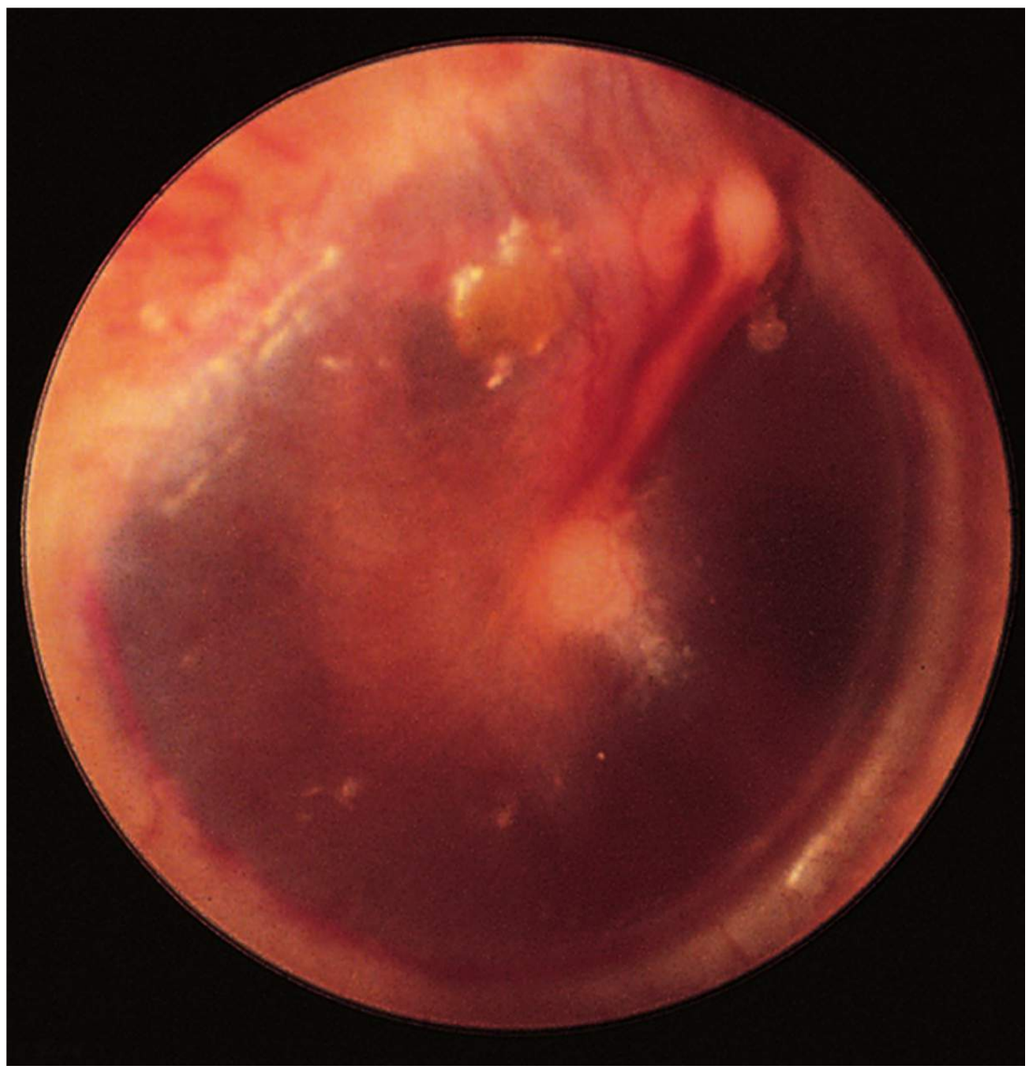

FIGURE 16.90 ■ **Barotitis Media.** Tympanic membrane erythema and mild hemorrhage consistent with barotitis media. (Photo contributor: Richard A. Chole, MD, PhD.)

Clinical Summary

Mask squeeze results when a diver fails to maintain the balance between the air pressure within his or her mask and the external water pressure during descent. If a diver descends without equalizing pressure by exhaling through the nose, significant negative air pressure will exist inside the mask. The net result may include rupture of capillary beds, leading to conjunctival hemorrhage and skin ecchymosis.

Management and Disposition

Treatment consists of ascent and essentially supportive care. A history of recent eye surgery or use of anticoagulant medication should be sought. A thorough eye examination should be performed and ophthalmologic consultation considered if warranted.

Pearls

1. Diver education and proper diving technique minimize the risk of mask squeeze.
2. Special consideration should be given to patients with anticoagulant use or recent keratotomy, as corneal incisions heal relatively slowly.

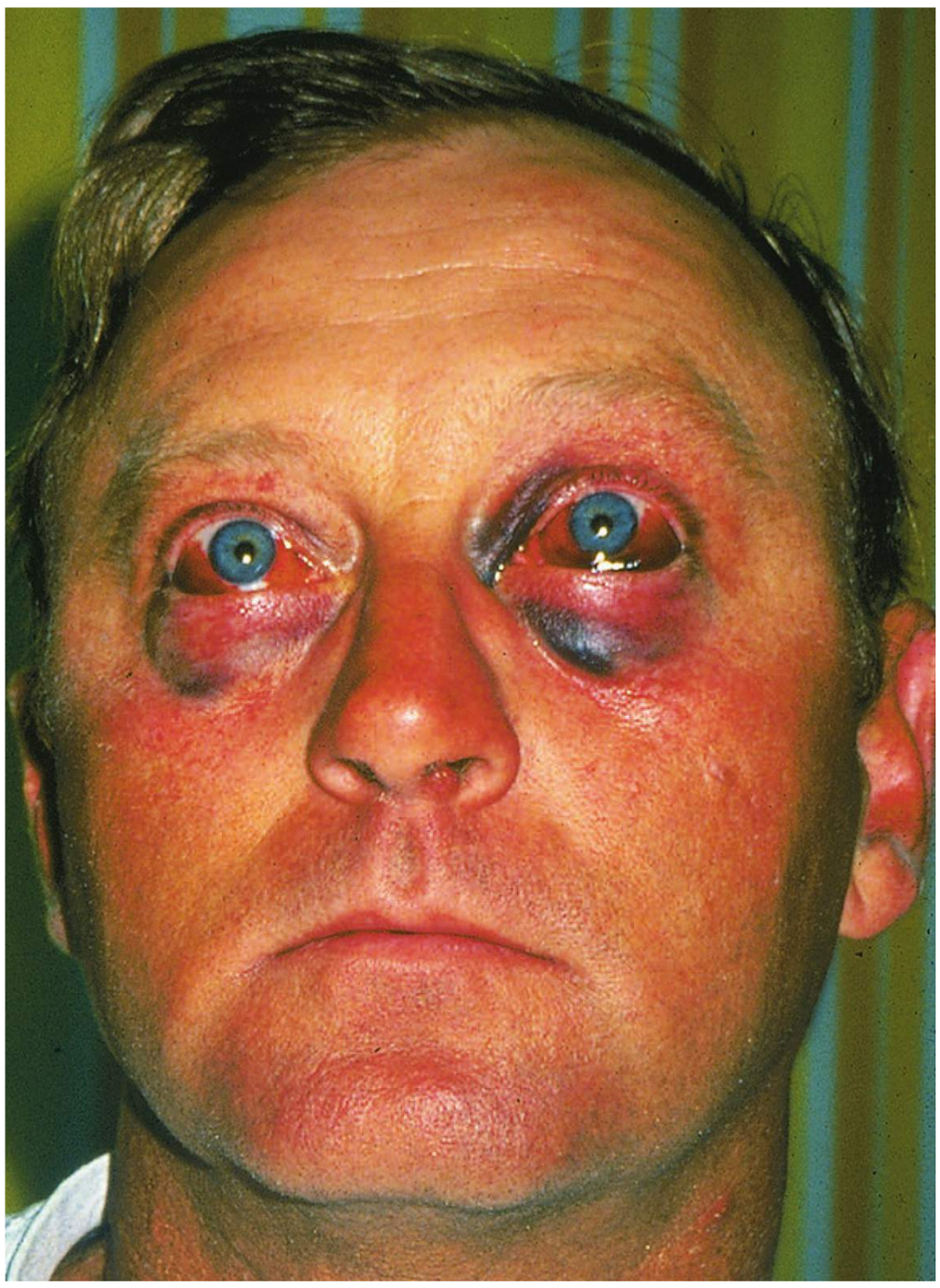

FIGURE 16.91 ■ **Mask Squeeze.** Mask squeeze in a diver who descended to 45 FSW without exhaling into his mask. (Photo contributor: Kenneth W. Kizer, MD; reprinted with permission from Auerbach PS (ed). Wilderness Medicine: Management of Wilderness and Environmental Emergencies. 3rd ed. St. Louis, MO: Mosby-Year Book; 1995. Copyright © Elsevier.)

STINGRAY ENVENOMATION

Clinical Summary

Stingrays are found throughout the oceans of the world. Stingrays are not typically aggressive and the majority of envenomations are defensive in nature. Injuries typically involve the lower extremity if the animal is stepped upon or upper extremity if the animal is handled. Fatal injuries have been reported from chest trauma, which may result in perforation of the myocardium. Stingray envenomation occurs when a reflexive and forceful forward thrust of the caudal spine or spines of the animal impacts the victim, producing a puncture wound or laceration. The force of injection causes the integumentary sheath covering the spine to be driven into the wound, fragmenting and potentially releasing venom, mucus, pieces of the sheath, and spine fragments deep within the wound. Envenomation typically produces immediate and intense pain, edema, and bleeding. The initially dusky or cyanotic wound may progress to erythema, with rapid fat and muscle hemorrhage. Systemic symptoms may include nausea, vomiting, diarrhea, diaphoresis, muscle cramps, fasciculations, weakness, headache, vertigo, paralysis, seizures, hypotension, and syncope.

Management and Disposition

The wound should be irrigated immediately and primary exploration accomplished to remove any visible debris. Pain relief should be initiated early, and opiates may be needed. Stingray venom is made up of heat-labile polypeptides that may be inactivated by immersion in hot water (43°C-46°C [110°F-115°F]) for 30 to 90 minutes. After soaking, wounds should be formally explored, debrided, and dressed for delayed primary closure or primary closure with drainage.

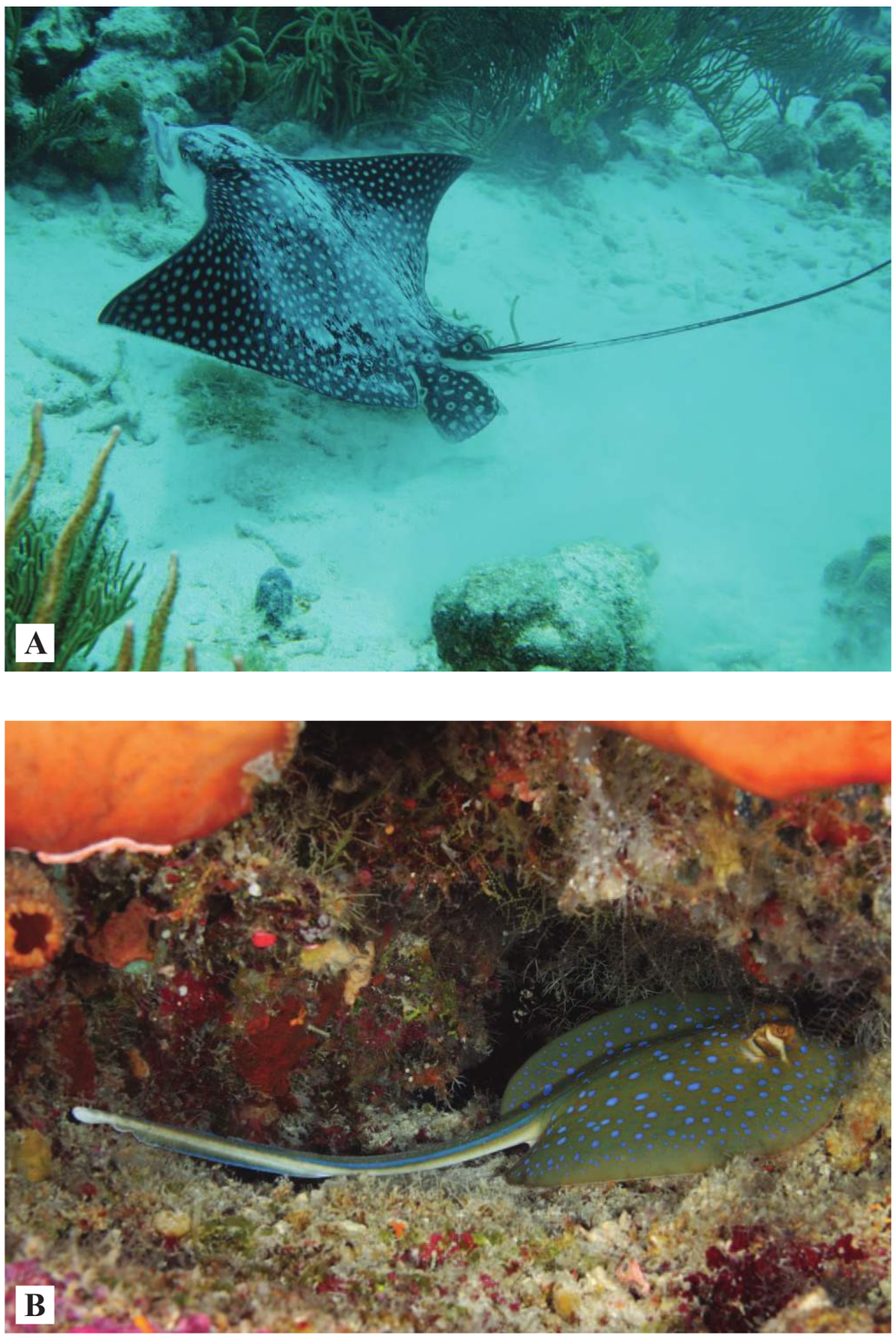

FIGURE 16.92 ■ **Stingrays.** (**A**) Spotted eagle stingray. This graceful stingray was photographed in waters off the coast of Bonaire. Note the three venomous spines at the base of the tail. (Courtesy of Lynne Bentsen, RN.); (**B**) Blue spotted stingray. This stingray was photographed in waters off Indonesia. Stingrays often dwell on the ocean floor and may burrow into the sand, leading to envenomation by accidentally stepping on the animal. (Photo contributor: Ian D. Jones, MD); (**C**) Southern stingray. This stingray was photographed in the Caribbean. The coloring of the stingray tends to blend with the ocean floor, leading to injury from inadvertent stepping on the stingray. (Photo contributor: Kevin J. Knoop, MD.)

Surgical consultation may be warranted in certain injury locations. A radiograph should be obtained after debridment to rule out retained foreign body. Broad-spectrum antibiotics covering marine organisms are recommended. Patients can usually be discharged home after a 3- to 4-hour observation period if no systemic symptoms arise. Tetanus prophylaxis should be given if indicated.

Pearls

1. Aggressive debridement is of primary importance in managing stingray wounds, as retained foreign bodies are a common problem.
2. Bacteria cultured from marine envenomations are extremely diverse. Antibiotics chosen should include coverage of staphylococci, streptococci, and Vibrio species.

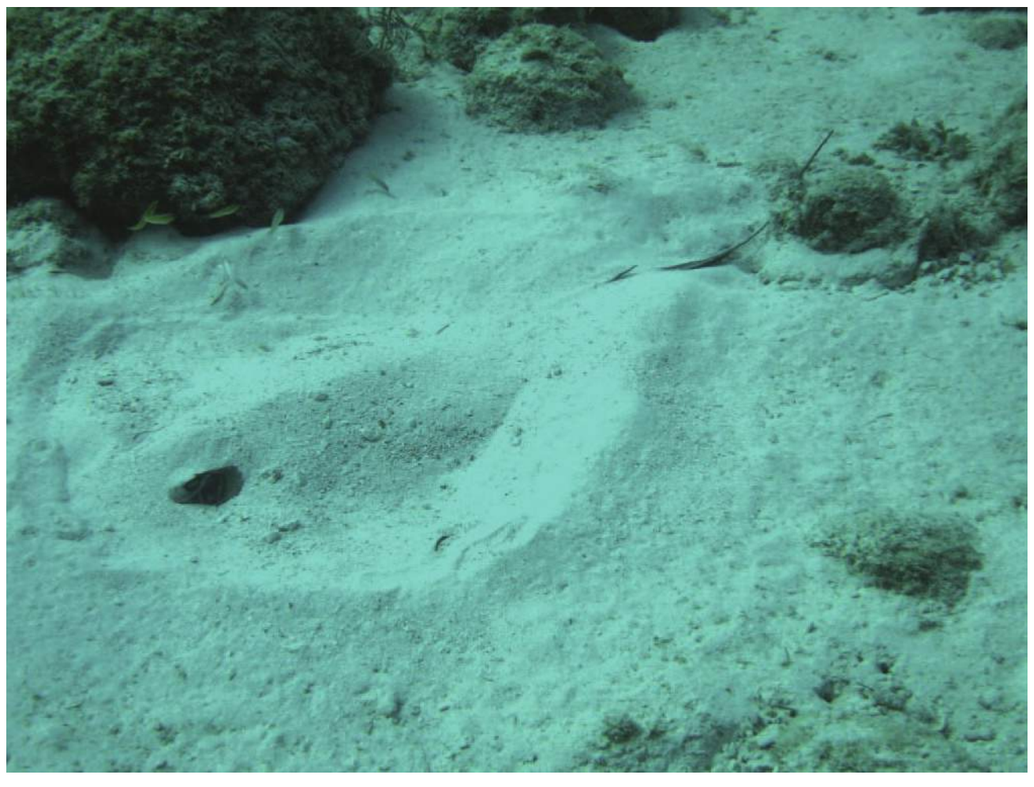

FIGURE 16.93 ■ **Buried Stingray.** Stingrays like to burrow into the sand on the ocean floor, making them very difficult to see and easy to inadvertently step on. (Photo contributor: Keven Reed, OD.)

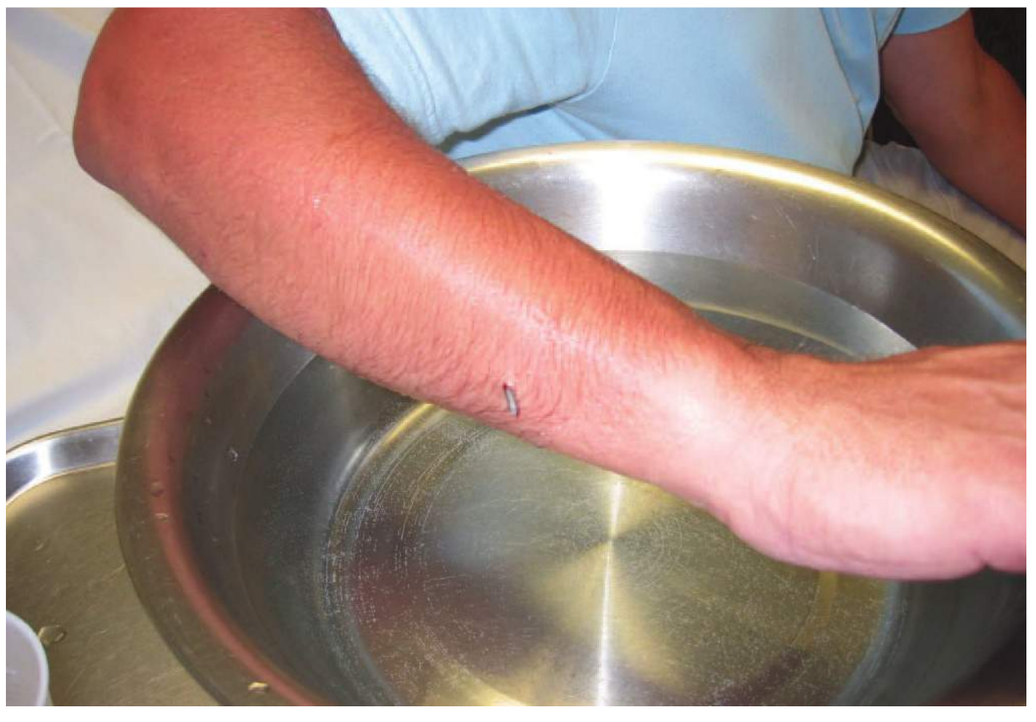

FIGURE 16.94 ■ **Stingray Barb in Forearm.** This envenomation occurred after the fisherman inadvertently caught a stingray and was envenonmated taking the animal off the hook. The barb is imbedded in the patients forearm. (Photo contributor: John Meade, MD.)

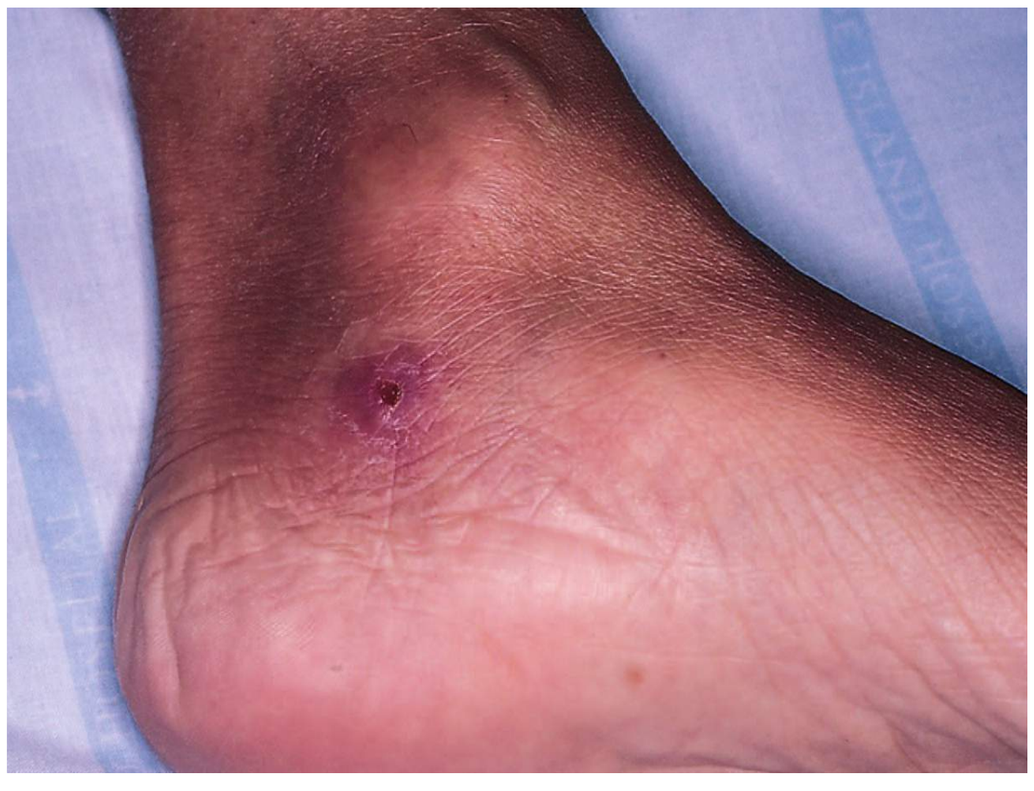

FIGURE 16.95 ■ **Stingray Envenomation.** Puncture wound from stingray envenomation in a lower extremity. (Photo contributor: Daniel L. Savitt, MD.)

SEA URCHIN ENVENOMATION

Clinical Summary

Sea urchins belong to the phylum Echinodermata and are nonaggressive, slow-moving creatures. Envenomation usually occurs after accidental contact with the organism. Long, brittle, venom-filled spines or specialized jaw-like appendages (pedicellariae) are responsible for the injury. Other echinoderms, notably the crown of thorns starfish, may also cause injury via similar mechanisms. The spines frequently break and pedicellariae can remain attached and active for several hours. They may advance into muscle or joint spaces and cause infection or injury from the venom itself. The usual presentation is burning pain progressing to localized muscle aches. Erythema and edema may be present. Multiple envenomations may produce systemic symptoms including nausea, vomiting, abdominal pain, paresthesias, numbness, paralysis, hypotension, syncope, or respiratory distress. While the envenomation causes a reaction that may be quite painful, deaths, though reported, are exceedingly rare.

Management and Disposition

Following envenomation, the affected area should be submersed in hot water (43°C-46°C [110°F-115°F]) for 30 to 90 minutes. Pedicellariae may be removed by applying shaving cream and gently scraping with a razor. Obvious embedded spines should be removed. An x-ray should be performed to rule out retained foreign body. In certain cases, CT, ultrasound, or MRI may be considered. Hand wounds often require surgical debridement. Retained spines often dissolve spontaneously; however, granulomas may form, producing locally destructive inflammation. Antibiotics may be useful in certain cases, and tetanus prophylaxis should be addressed.

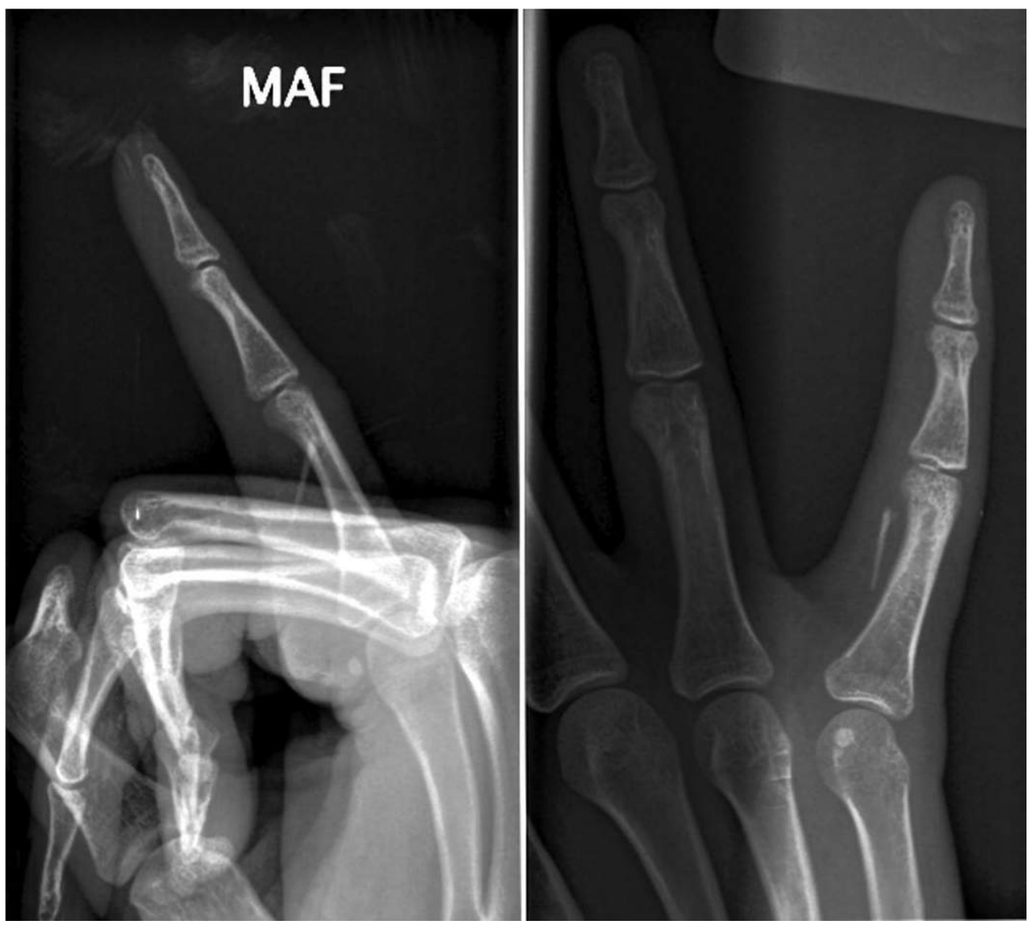

FIGURE 16.96 ■ **Sea Urchin Spine on X-Ray.** This x-ray is of a patient who was reaching into a rock crevasse in the ocean and experienced sudden sharp pain. Note the retained sea urchin spine in the fifth digit adjacent to the proximal phalanx. (Photo contributor: Marion Berg, MD.)

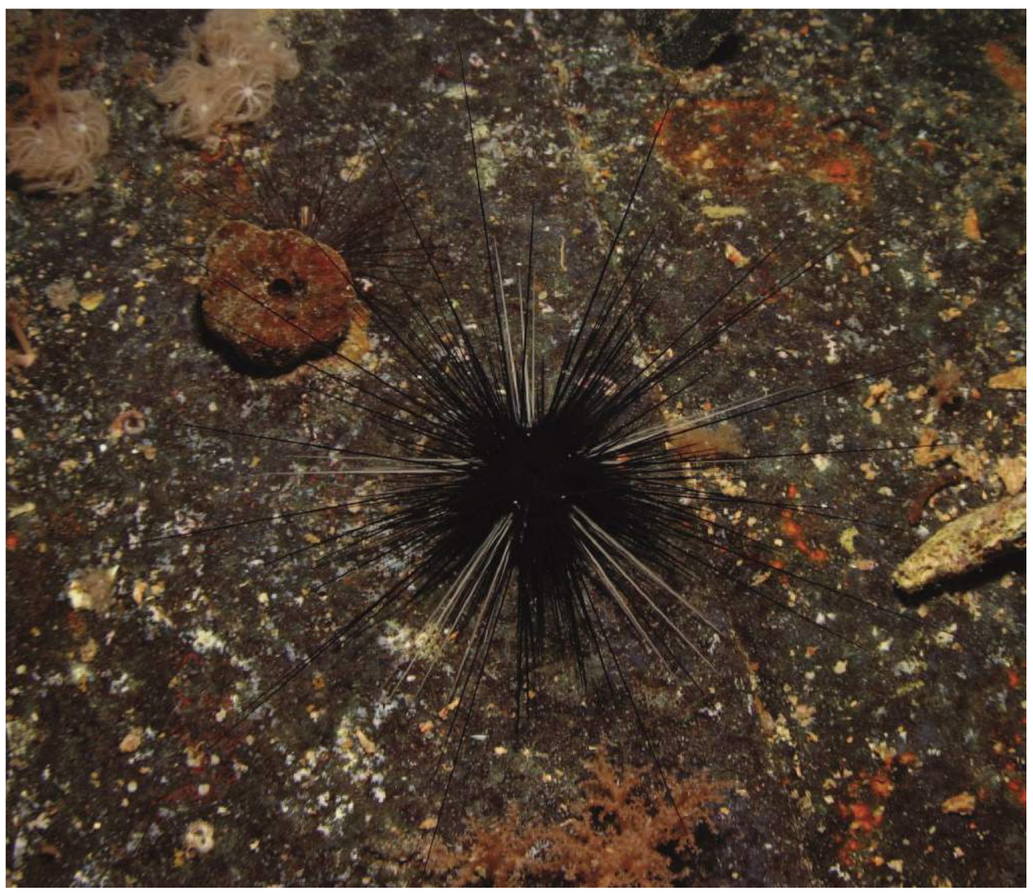

FIGURE 16.97 ■ **Long-Spined Sea Urchin.** The majority of urchins are nonvenomous and only cause injury from puncture wounds. Their spines may break off in the victim, resulting in a high likelihood of infection. (Photo contributor: Ian D. Jones, MD.)

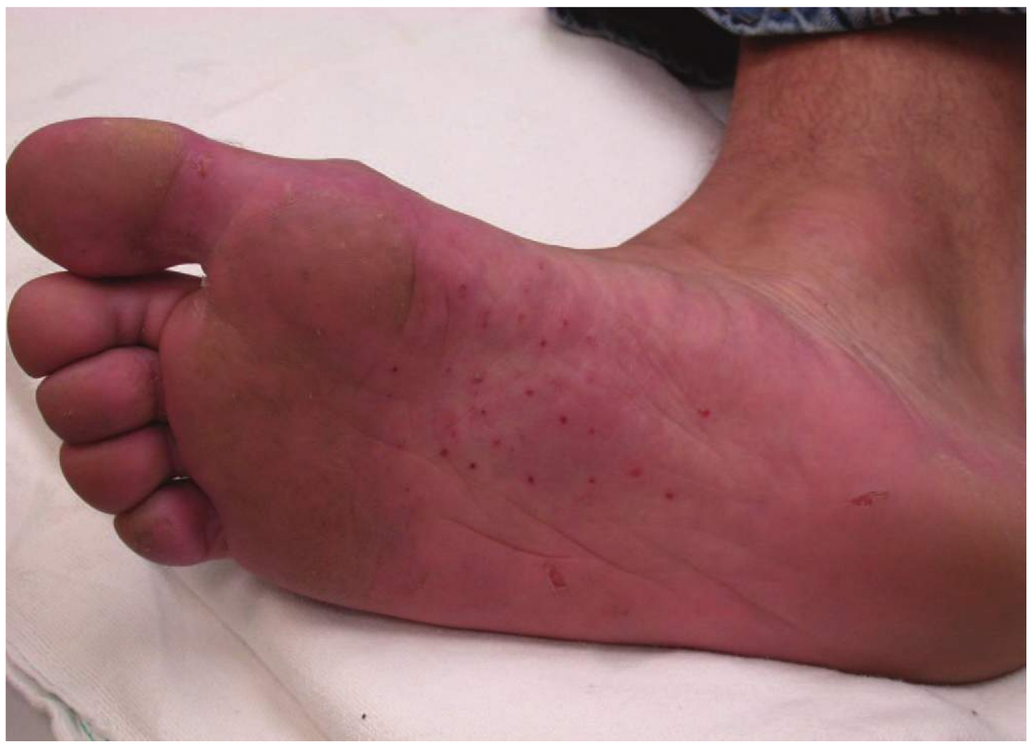

FIGURE 16.98 ■ **Sea Urchin Envenomation.** Note the multiple puncture wounds on the foot of a patient who accidentally stepped on a sea urchin in shallow water. (Photo contributor: Saralyn R. Williams, MD.)

Pearls

1. Sea urchin envenomation involving a joint may produce severe synovitis.
2. Some species of sea urchin contain dye, which may give the false impression of a retained spine.
3. Sea urchins known to be hazardous to humans are generally found in the Indian Ocean, Pacific Ocean, and Red Sea.

FIGURE 16.99 ■ **Fire Urchin.** A beautiful fire urchin, found in the Indo-Pacific. (Photo contributor: Ian D. Jones, MD.)

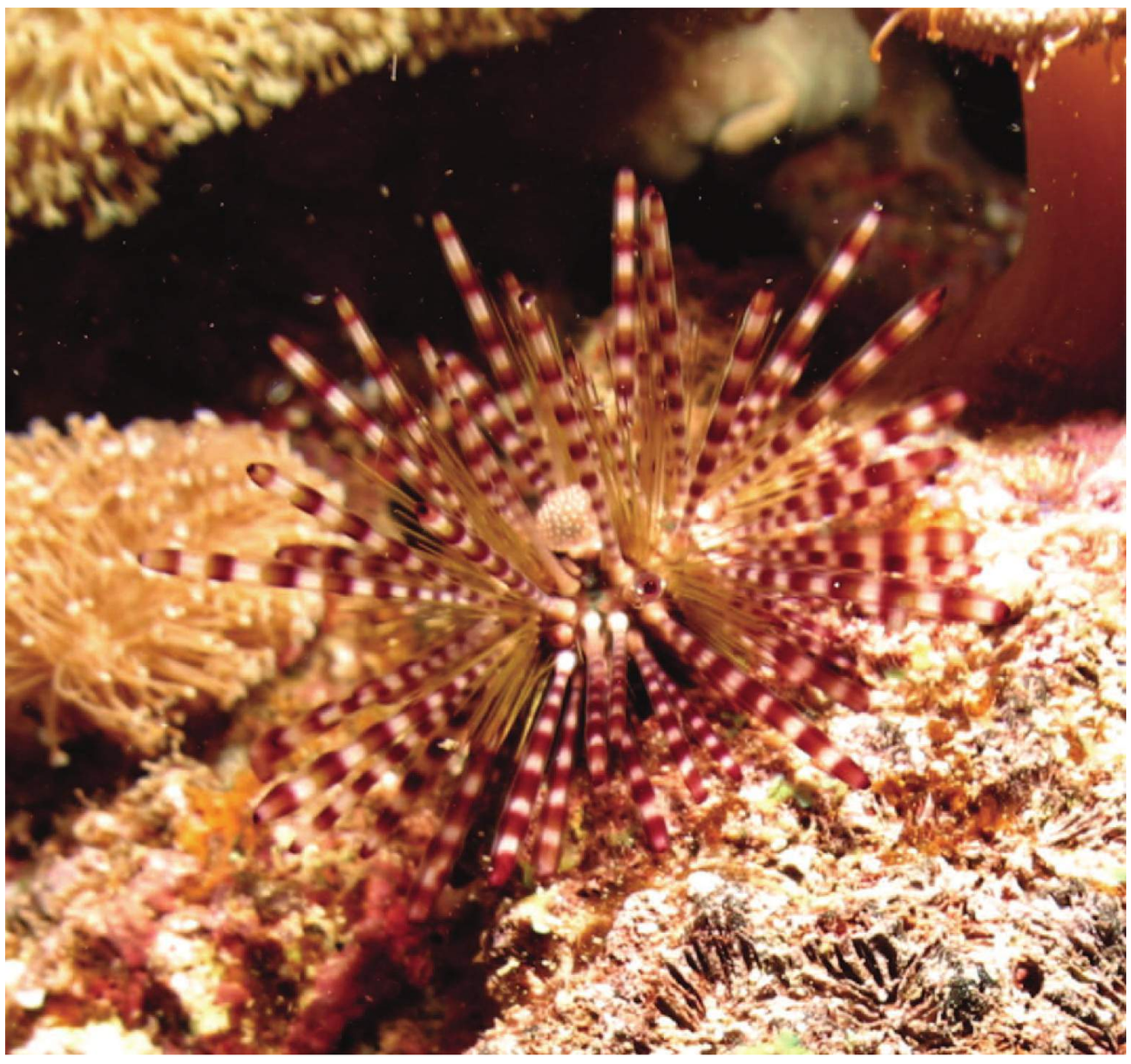

FIGURE 16.100 ■ **Fire Urchin.** Fire urchins are venomous sea urchins. Envenomations from fire urchins, while extremely painful, are seldom if ever fatal. (Photo contributor: Ian D. Jones, MD.)

Clinical Summary

The phylum Coelenterata contains approximately 10,000 different species, of which several hundred are a danger to humans. This diverse group includes hydrozoans (eg, Portuguese man-of-war, stinging hydrozoans, and fire coral), scyphozoans (ie, "true" jellyfish), and anthozoans (ie, soft corals, stony corals, and anemones). They account for more marine envenomations than any other phylum. The important species involved in human injuries have stinging cells called nematocysts. Nematocysts are enclosed in venom sacs and are present in tentacles that hang from air-filled structures. After external contact, the nematocysts are discharged from their sacs, often penetrating the skin, and release their venom. Nematocyst venom is an extremely complex substance containing numerous proteins and enzymes. Clinical presentation following envenomation ranges from the mild dermatitis to cardiovascular and pulmonary collapse. Mild envenomations usually result in a self-limited papular inflammatory eruption associated with burning and limited to areas of contact. Moderate to severe envenomations produce a spectrum of neurologic, cardiovascular, respiratory, and gastrointestinal symptoms. Anaphylactoid reactions—including hypotension, dysrhythmias, bronchospasm, and cardiovascular collapse—may occur, resulting in unexplained drownings.

Management and Disposition

Concurrently with primary resuscitation, nematocyst decontamination should be accomplished beginning with

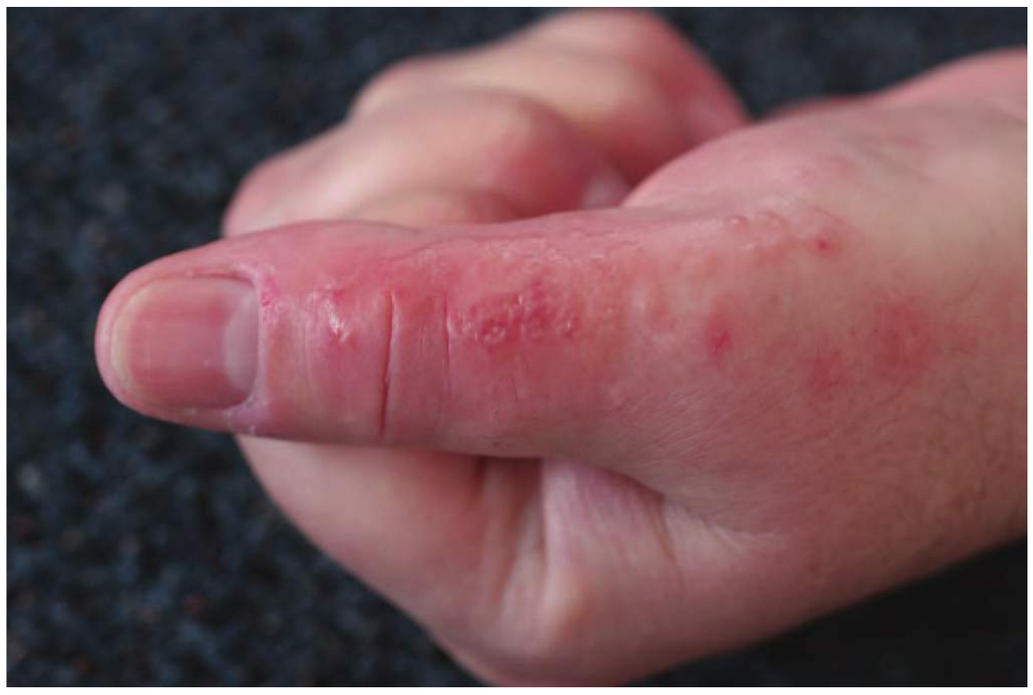

FIGURE 16.102 ■ **Hydrozoan Envenomation.** This hydrozoan sting resulted from accidental direct contact with the diver's finger while taking an underwater photograph. (Photo contributor: Ian D. Jones, MD.)

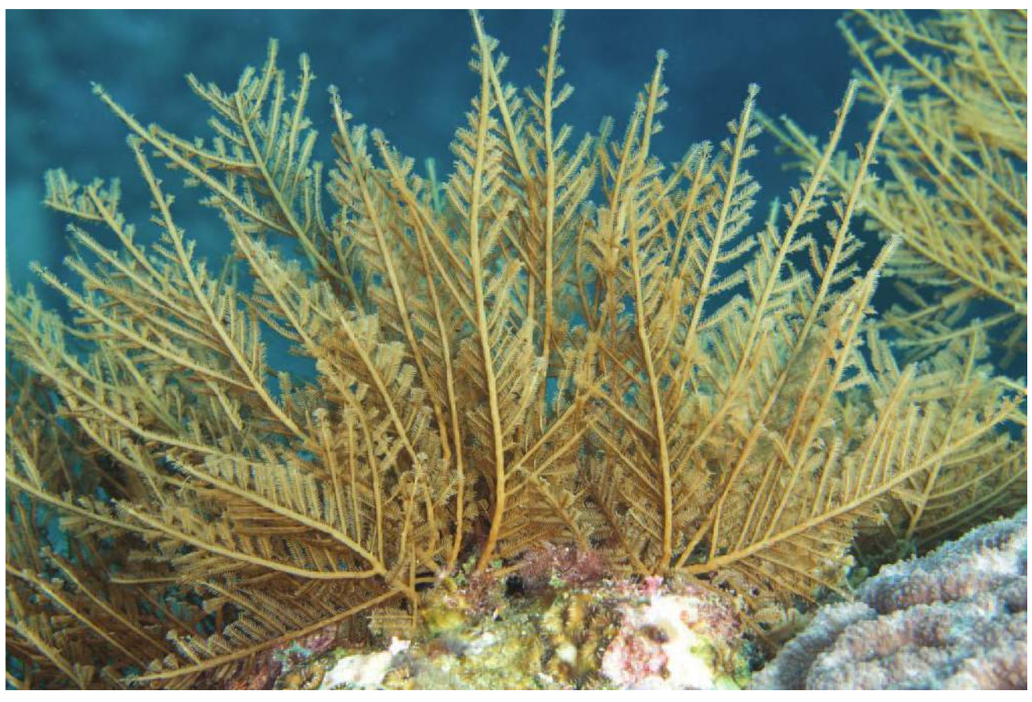

FIGURE 16.101 ■ **Stinging Hydrozoan.** Similar to fire coral, contact with stinging hydrozoan results in local burning pain followed by erythematous papules or urticarial eruptions and blisters. (Photo contributor: Shawn Miller.)

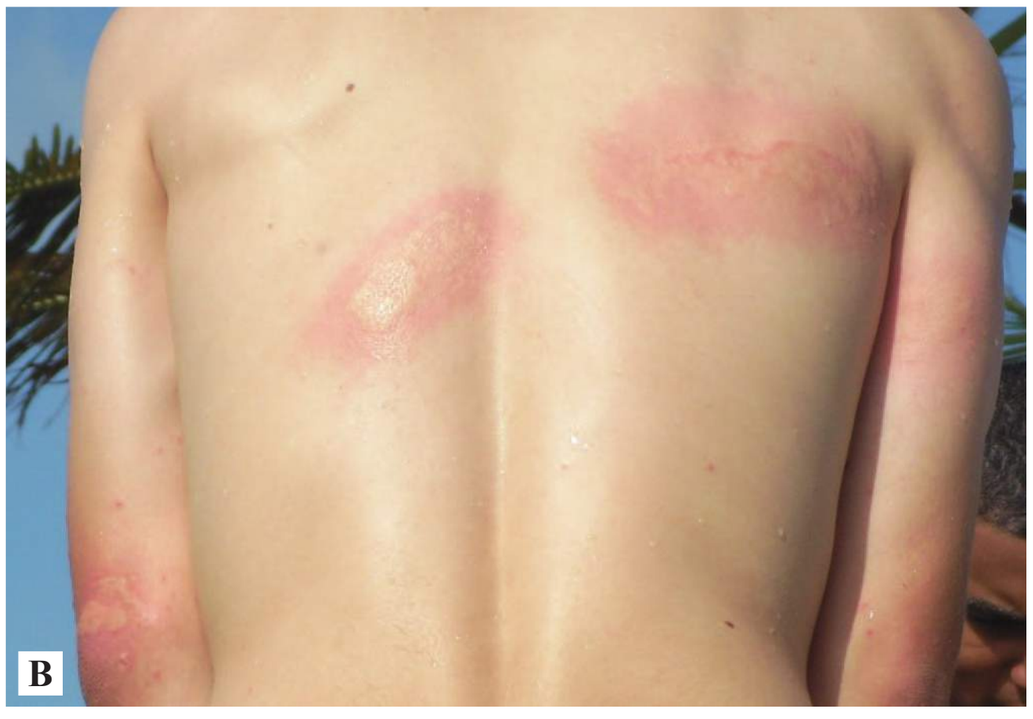

FIGURE 16.103 ■ **Fire Coral.** (**A**) Fire coral. (Photo contributor: Shawn Miller); (**B**) Fire coral envenomation. After contact, fire coral most commonly causes immediate local burning pain, followed by erythematous papules or urticarial eruptions. Pruritus may last for several days. (Photo contributor: Emily R. Stack.)

seawater flushing. The hypotonic nature of fresh water, as well as isopropyl alcohol, may cause additional nematocysts to fire and should be avoided. A 5% solution of acetic acid (vinegar) applied for at least 30 minutes is the most widely accepted mechanism for inactivating nematocysts. It has been suggested to remove tentacles with the application of shaving cream, followed in 5 minutes by a careful scraping with a firm, dull object (eg, tongue blade, credit card). Pruritus may be treated with antihistamines. Pain may be addressed with immersion in hot water or with systemic analgesics. Any victim with systemic symptoms requires at least 6 to 8 hours of observation because rebound phenomena are common.

Pearls

1. The box jellyfish (Chironex fleckeri) is generally considered the most deadly of marine animals and is most predominant in Australian and Southeast Asian waters.
2. The detached tentacles of some species may contain active nematocysts for months, even when fragmented on the beach or floating in water.
3. The Portuguese man-of-war is present in the Floridian Atlantic coast and the Gulf of Mexico. It is known to have a neurotoxin that may cause severe pain. Death, while reported, is extremely rare.

FIGURE 16.104 ■ **Portuguese Man-of-War.** The beautiful Portuguese man-of-war with multiple tentacles dangling in the water. The tentacles, filled with venomous nematocysts, can extend several meters in length. (Photo contributor: Adam Laverty.)

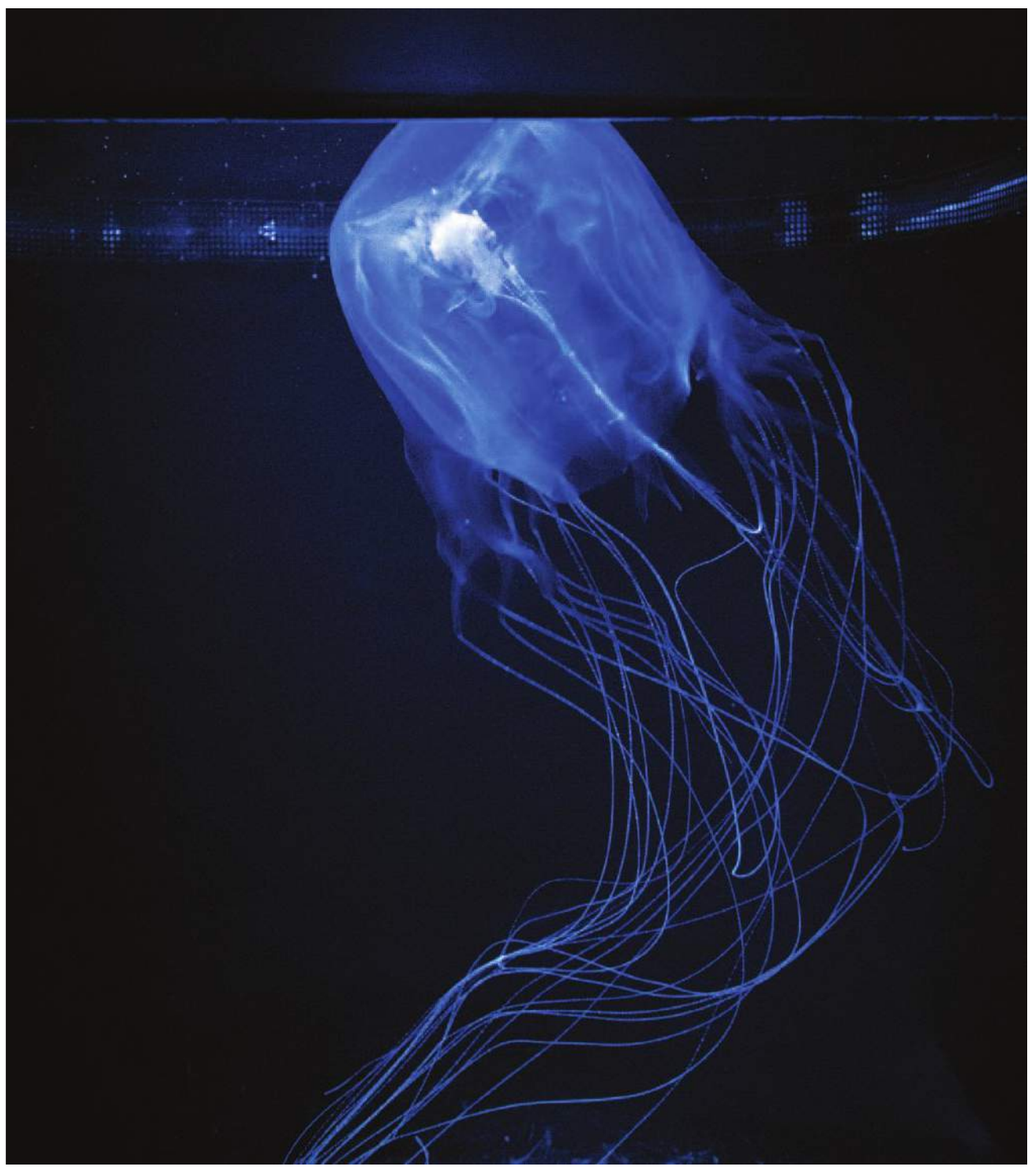

FIGURE 16.105 ■ **Box Jellyfish (Under Blue Light).** The box jellyfish delivers an unbearably painful sting. Envenomations can be life-threatening and require immediate treatment. (Photo contributor: Shawn Miller.)

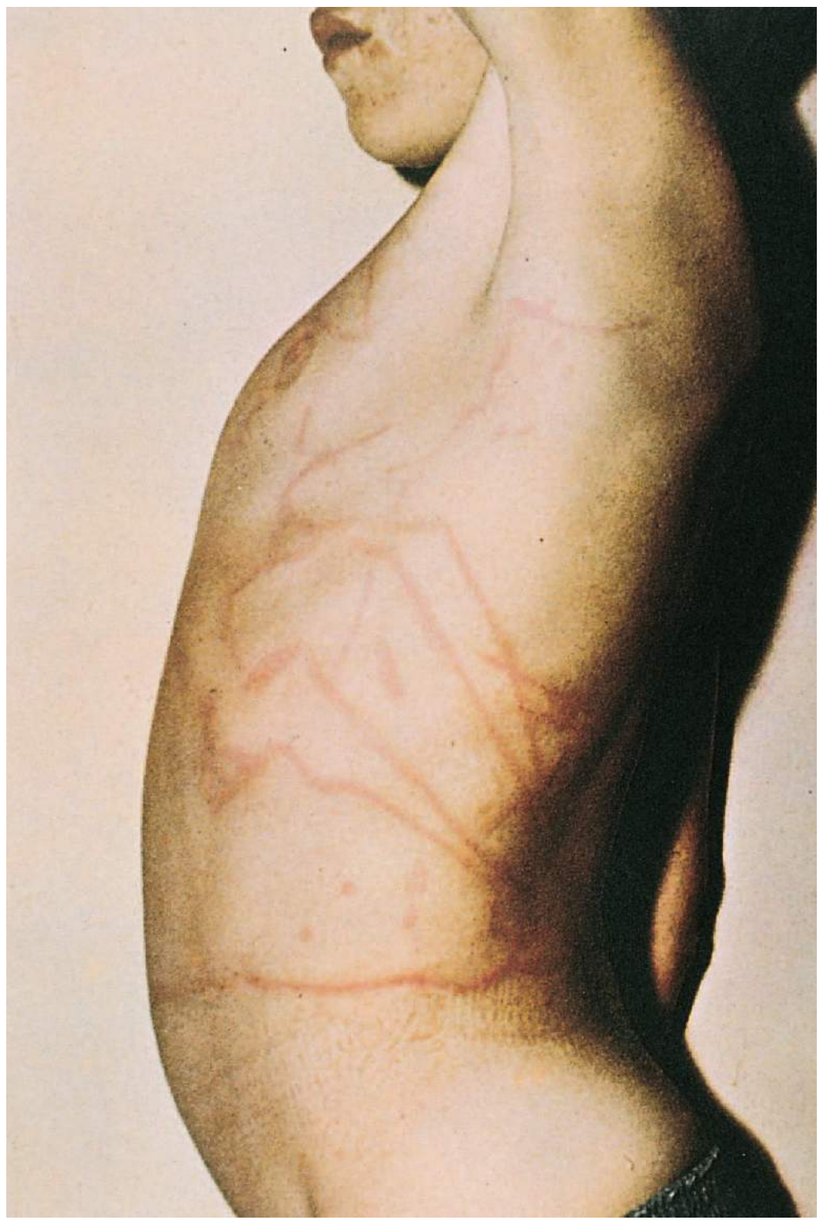

FIGURE 16.106 ■ **Coelenterate Envenomation.** The sharp angulations and undulations characteristic of jellyfish envenomation. (From Halstead BH. Venomous Marine Animals of the World. Washington, DC: US Government Printing Office; 1965.)

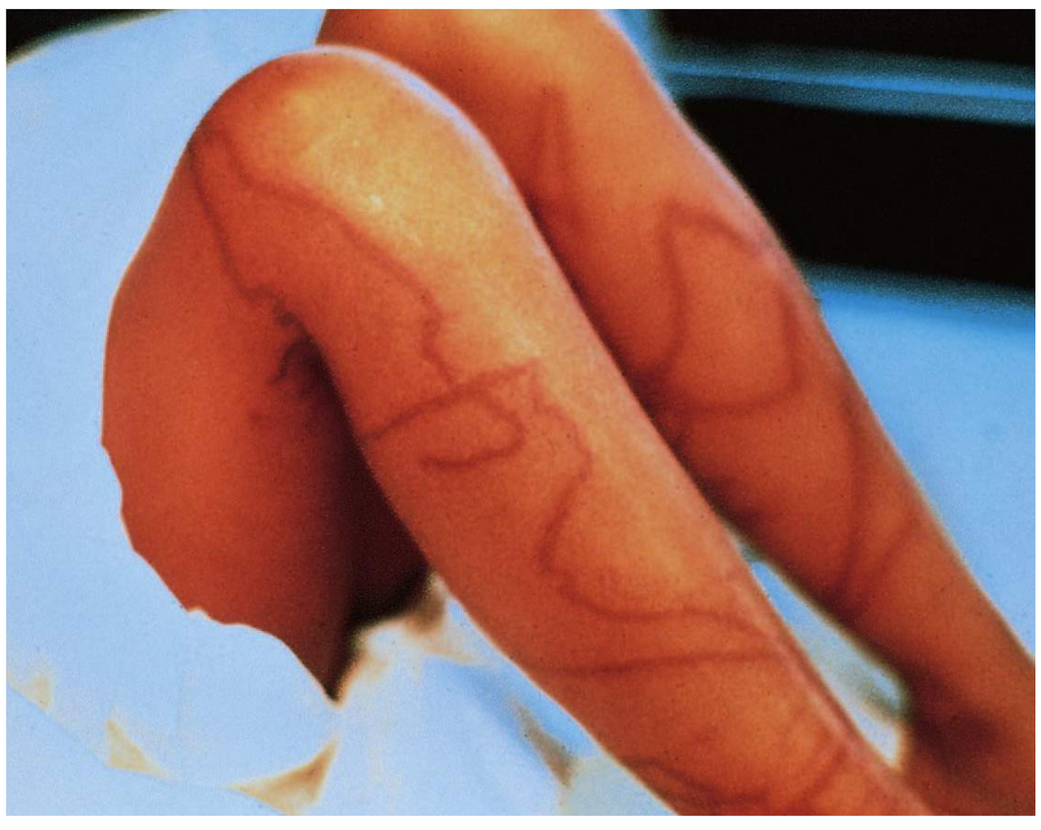

FIGURE 16.107 ■ **Coelenterate Envenomation.** Jellyfish envenomation on the lower extremities. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)

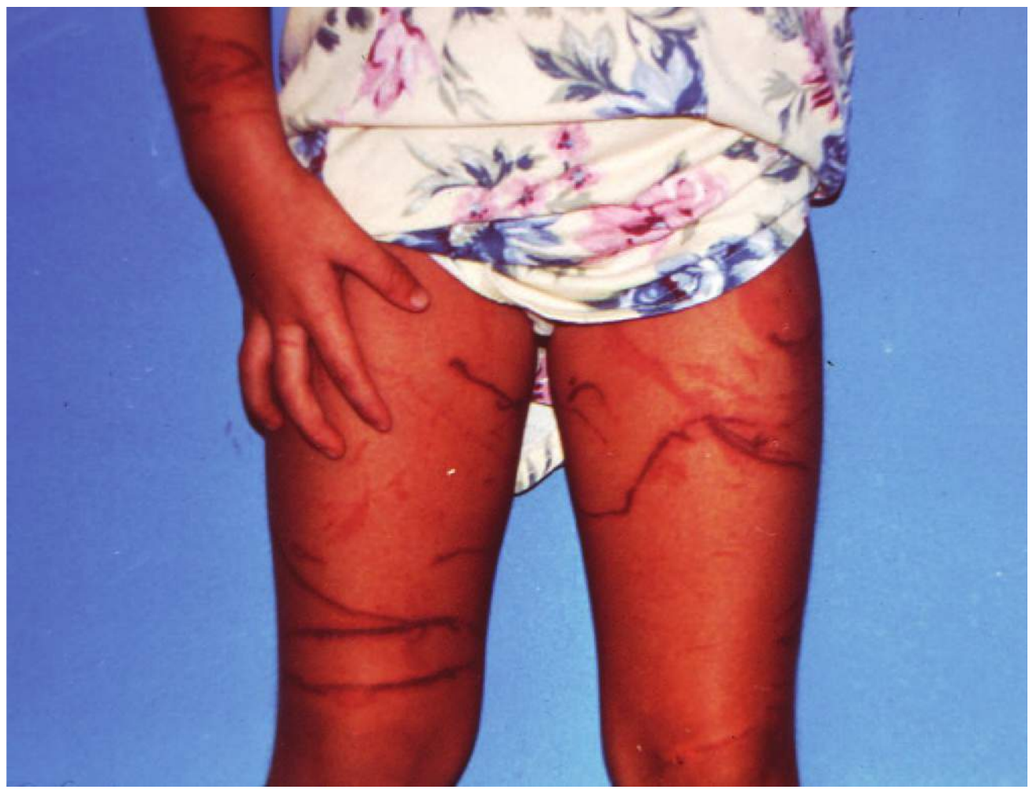

FIGURE 16.108 ■ **Box Jellyfish Sting.** This 5-year-old child was stung by a box jellyfish (Chiropsalmus quadrigatus) off the coast of Japan. Severe envenomations may be fatal. (Photo contributor: Keven Reed, OD.)

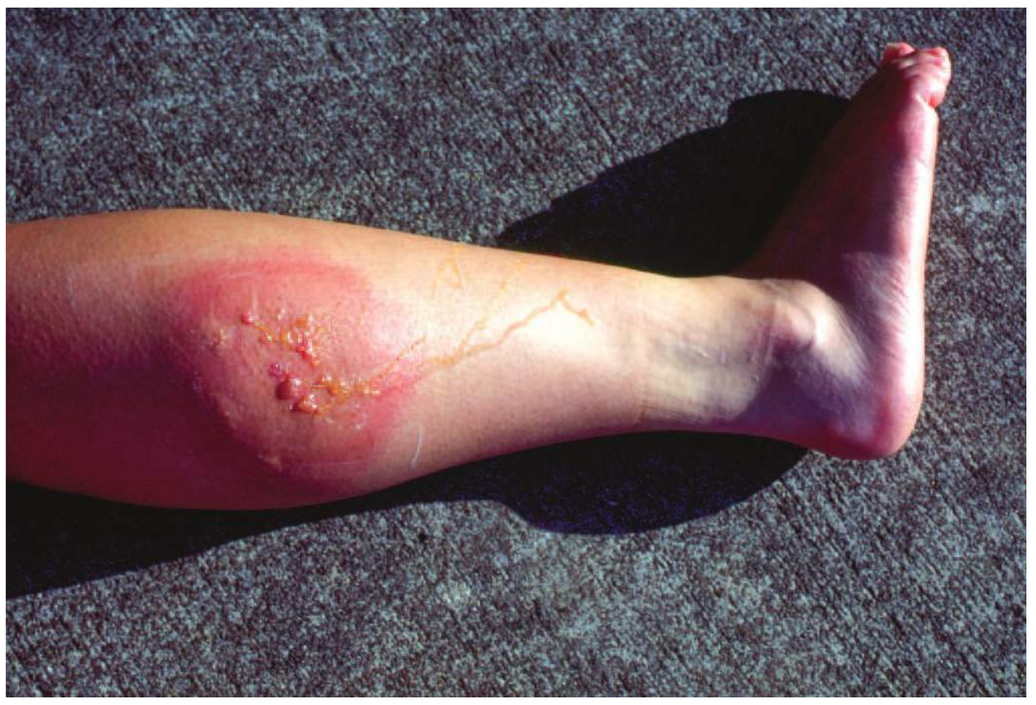

FIGURE 16.109 ■ **Sea Anemone Envenomation.** Contact with sea anemones results mainly in local skin irritation, initially manifest as pruritus, burning, throbbing, and, sometimes, radiation of pain to other areas. The area involved may reveal blistering, local edema, and violaceous petechial hemorrhages. Skin lesions are confined to the areas of contact. (Photo contributor: Susan Scott.)

Clinical Summary

Marine dermatitis, also known as "sea bather's eruption," is a pruritic condition commonly mislabelled sea lice. Symptoms usually occur a few minutes to 12 hours after exposure. The offending organisms are probably numerous and include the larval form of the thimble jellyfish and the planula form of the sea anemone Edwardsiella lineata. The rash consists of erythematous wheals and papules, which may be extremely pruritic. Systemic manifestations include fever, malaise, headache, conjunctivitis, and urethritis. Unlike cercarial dermatitis, marine dermatitis primarily affects areas of the body covered by caps, fins, and bathing suits.

Cercarial dermatitis, or "swimmer's itch," occurs when humans become accidental hosts of nonhuman schistosomes. This causes an immune response that results in itching, erythema, and mild edema. After 60 minutes, the classic signs are red macules that become pruritic papules 3 to 5 cm in diameter and surrounded by erythema.

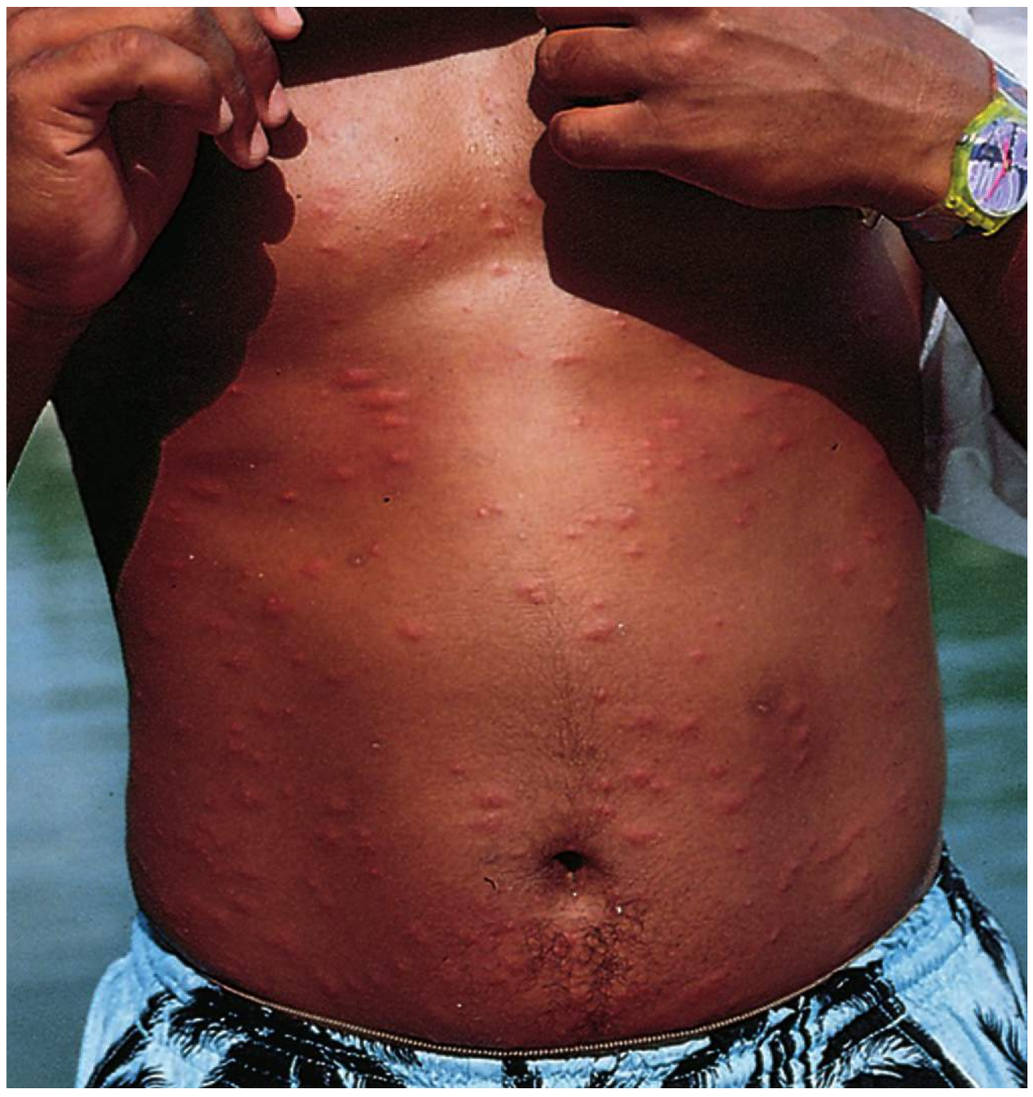

FIGURE 16.110 ■ **Marine Dermatitis.** Typical appearance of marine dermatitis. (Photo contributor: Richard A. Clinchy III, PhD.)

Management and Disposition

Marine dermatitis is self-limited, rarely persisting beyond 2 weeks. The dermatitis may be partially prevented by a vigorous soap-and-water scrub after saltwater bathing. Treatment is symptomatic, and calamine lotion with 1% menthol may bring relief. Topical steroids may provide additional relief. In severe cases, oral antihistamines and corticosteroids may be necessary.

Cercarial dermatitis is treated with isopropyl alcohol or calamine lotion. Severe cases may require systemic corticosteroids, whereas bacterial infection may require topical or oral antibiotics.

Pearls

1. Marine dermatitis primarily affects areas covered by caps, fins, and bathing suits.
2. During late spring and summer, incidence increases along the US east coast. In one reported outbreak, 25% of individuals entering the water were affected.

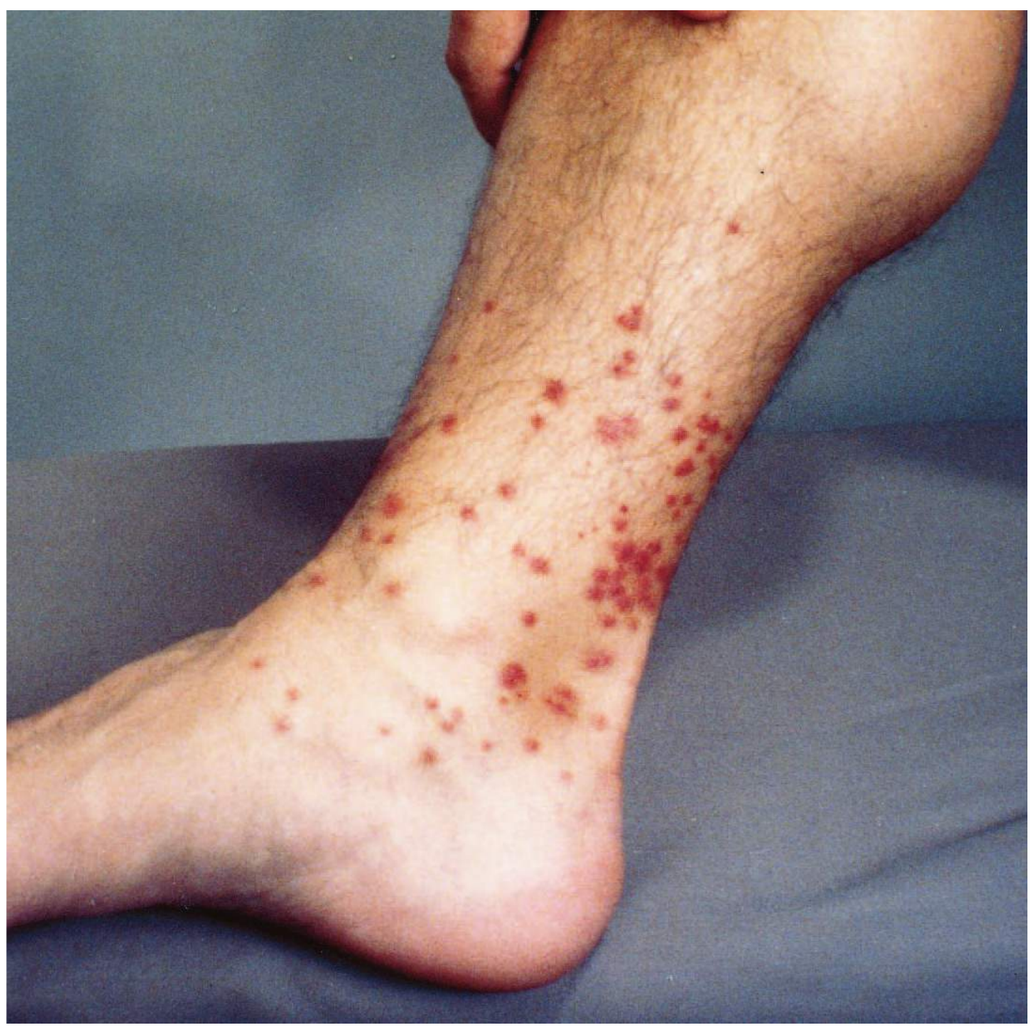

FIGURE 16.111 ■ **Cercarial Dermatitis.** The discrete and highly pruritic papules of cercarial dermatitis commonly occur on exposed body areas. (Photo contributor: David O. Parrish, MD. Reproduced with permission from Parrish DO. Seabather's eruption or diver's dermatitis. JAMA. 1993;270:2300-2301. Copyright © 1993 American Medical Association. All rights reserved.)

SCORPION FISH STING

Clinical Summary

Scorpion fish are colorful venomous marine animals found primarily in tropical waters. Their exotic beautiful appearance has made them increasingly popular among marine aquarists in the United States. Many envenomations have resulted from mishandling. They are well camouflaged in the wild and stings are usually caused by accidentally stepping on them. Scorpion fish are grouped into the genera Pterois (lion fish), Scorpaena (scorpion fish proper), and Synanceja (stone fish), with the severity of envenomation progressing, respectively. All have multiple spines associated with venom glands; envenomation results from skin puncture followed by venom release. Immediately following a sting, the victim experiences intense pain that, if untreated, lasts for hours. The site may become warm, erythematous, and edematous and vesicles may arise. Lion fish stings are painful but relatively mild, usually limited to localized pain and tissue responses. Severe systemic effects are more common with stone fish stings and may produce a constellation of cardiovascular, pulmonary, neurologic, and gastrointestinal sequelae. Death has been reported from stone fish stings.

Management and Disposition

Hot water immersion (43°C-46°C [110°F-115°F]) for 30 to 90 minutes is initiated as soon as possible. Rebound pain is common and is treated with repeated hot water immersion. The wound should be inspected for pieces of spine and sheath. Thorough warm saline irrigation should be performed along with wound exploration. Severe pain is treated with local injection of lidocaine without epinephrine and with narcotic analgesia. Antibiotic prophylaxis should be considered in high-risk wounds and tetanus status addressed.

Pearls

1. Stone fish stings are the most dangerous. Severe systemic reactions may occur. Antivenin (Commonwealth Serum Labs, Australia) is available.
2. Scorpion fish venom is heat-labile. Hot water immersion is effective in treating pain and inactivating venom.
3. Since 2002, Indo-Pacific lionfish have been reported in increasing numbers from New York to the Bahamas.

FIGURE 16.113 ■ **Scorpion Fish.** Note the well-camouflaged scorpion fish, leading to accidental contact and envenomation in unsuspecting divers. (Photo contributor: Kevin J. Knoop, MD.)

FIGURE 16.112 ■ **Lion Fish** (Pterois volitans). Envenomations occur by contact with the erectile spines on the dorsal, pelvic, and anal fins of the fish. (Photo contributor: Kevin J. Knoop, MD.)

FIGURE 16.114 ■ **Stonefish.** The extremely venomous stonefish is spectacularly camouflaged. Envenomation usually results when the unsuspecting victim steps on the fish. (Photo contributor: Ian D. Jones, MD.)

Clinical Summary

Cone snails, also referred to as cone shells or cone fish, are venomous predatory marine gastropod molluscs capable of inflicting a painful and even dangerous sting to humans. They may be found in wide distributions across the world's oceans and seas. Cone snails prey on marine worms and fish, using their venom apparatus to inject the victim with paralytic toxins. The cone snail uses a dart like tooth which fires out from the shell. Many have beautiful patterns on their shell, making them attractive to collect for unsuspecting divers. When disturbed, the snail may deploy its harpoon like tooth and envenomate the handler. Smaller species inflict a sting similar to that of a wasp, but envenomations from larger species may cause intense pain, swelling, paresthesias, and vomiting. Rarely, severe envenomations may progress to muscle paralysis, respiratory failure, and death.

Management and Disposition

Cone snail venom is heat-labile, so initial management consists of hot water immersion similar to the treatment of scorpion fish stings. Rebound pain is common and is treated with repeated hot water immersion. Inspect the wound for foreign material, and perform thorough warm saline irrigation. Severe pain may be treated with local injection of lidocaine without epinephrine and with narcotic analgesia. Consider antibiotic prophylaxis in high-risk wounds and address tetanus status. Intravenous edrophonium has been reported in the treatment of cone snail–associated paralysis.

Pearls

1. Cone snail venom is heat-labile. Hot water immersion is effective in treating pain and inactivating venom.
2. Cone snail venom may have potential therapeutic uses, including the management of severe pain and intractable epilepsy.
3. Although fatal cone snail envenomations are rare, severe stings may cause death secondary to the rapid onset of respiratory paralysis.

FIGURE 16.116 ■ **Geographic Cone Snail.** This cone snail is seen in full view with tentacles, water siphon, and short pharynx (proboscis) visible. The geographic cone snail is highly venomous with a potentially fatal sting. (Photo contributor: Shawn Miller.)

FIGURE 16.115 ■ **Cone Snail.** The beautiful shells of the cone snail make it attractive to collect for divers. Envenomations commonly occur on the hand and fingers of unsuspecting victims due to handling. (Photo contributor: Kevin J. Knoop, MD.)

FIGURE 16.117 ■ **Cone Snail (old).** As cone snails age, the shells may become covered in adherent materials, making them difficult to see and increasing the risk of inadvertent contact. (Photo contributor: Kevin J. Knoop, MD.)

SEA SNAKE ENVENOMATION

Clinical Summary

Sea snakes are members of the elapid family of snakes that have evolved to survive in a variety of ocean habitats. There are over 70 species of sea snakes with the majority found in the tropical portions of the Indian and Pacific Oceans. Sea snakes are not found in the Atlantic or Carribean. Most species of sea snakes live close to the coast although there is a pelagic species, Pelamis platurus, that is found across a large swath of the Pacific Ocean. Reports vary regarding the docile nature of sea snakes, although it is well reported that some species have a much higher propensity to bite. Bites occur when the animal is disturbed or inadvertently handled. There are many reports of bites occurring as fishermen are removing the snake from their nets. Because of the small size of the organism's fangs, bites may be inconspicuous and are often painless. The majority of bites do not result in envenomation.

The most potent toxin in sea snake venoms, similar to other elapids, is a neurotoxin that competes for acetylcholine at the neuromuscular junction. Severe envenomations may ultimately lead to paralysis and respiratory failure. Additional components of the venom are myotoxic and may result in rhabdomyolysis and renal failure.

The symptoms of sea snake envenomation generally occur within 30 minutes of envenomation and may be quite variable. Common symptoms include confusion, headache, myalgias, weakness of the facial muscles, followed by an ascending flaccid paralysis, and ultimately respiratory arrest.

Management and Disposition

Treatment for a sea snake bite includes a combination of supportive care and the immediate administration of polyvalent sea snake antivenom. Supportive care should include intubation if indicated. The patient should also be carefully observed for signs of rhabdomyolysis and hyperkalemia. The patient should be aggressively hydrated to avoid further complications such as renal failure. Consultation with a toxicologist or individual experienced in managing sea snake envenomations should be considered.

Pearls

1. An effective polyvalent antivenom exists and is effective against envenomation from all species of sea snakes.
2. In addition to respiratory support, victims of sea snake envenomation should be observed for the development of rhabdomyolysis and hyperkalemia.

FIGURE 16.118 ■ **Sea Snake.** The sea krait, seen here swimming free in the ocean, is very docile and rarely bites humans unless provoked. (Photo contributor: Ian D. Jones, MD.)

FIGURE 16.119 ■ **Sea Snake.** A close-up of a sea snake found feeding on a reef. (Photo contributor: Kevin J. Knoop, MD.)

Clinical Summary

There are over 750 species of cephalopods found throughout the world's oceans. Cephalopods are members of the phylum mollusca and include cuttlefish, squid, octopuses, and the nautilus. Several species of cephalopods are known or thought to be venomous but only the blue-ringed octopus, genus Hapalochlaena, poses a significant threat to humans.

Four separate species of blue-ringed octopus are known to exist. All are small and none exceed a few inches in size. The animal is found in tide pools and shallow reefs across a wide area of the Indo-Pacific ranging from Japan to Australia. The blue-ringed octopus is generally not aggressive and is relatively nondescript when left in peace. When threatened, however, the animal will flash or pulsate with striking iridescent blue rings. Bites occur when the animal is disturbed or inadvertently handled. It may be found hiding in old bottles or shells in tidal pools and in this way poses a hazard to collectors. Like other cephalopods, the blue-ringed octopus has a beak that can be powerful enough to penetrate a wet suit. Some bites may be relatively painless.

Blue-ringed octopus venom has several identified components. The most powerful component of the venom, tetrodotoxin, is a potent sodium channel blocker that is identical to the toxin found in pufferfish. Once bitten, symptoms may develop in as few as 10 minutes and include perioral paresthesias, facial weakness, and nausea and vomiting. In some patients, especially children, hypotension may be present. As symptoms progress, flaccid paralysis and respiratory arrest may result.

Management and Disposition

Treatment for a blue-ringed octopus bite is entirely supportive, as no antivenin currently exists. The patient almost always has a normal sensorium unless profoundly hypoxic or hypercarbic. Primary management of severe envenomations includes management of hypotension and prolonged ventilatory support until the toxin can be degraded and excreted.

Pearls

1. No antivenin exists for a blue-ringed octopus envenomation.
2. The patient's mental status is usually normal and a victim may be fully conscious despite being paralyzed and apneic.

FIGURE 16.120 ■ **Blue-Ringed Octopus.** The beautiful but highly venomous blue-ringed octopus. (Photo contributor: Russell C. Gilbert, MD.)

FIGURE 16.121 ■ **Blue-Ringed Octopus.** The blue-ringed octopus demonstrates its iridescent blue colorations, seen when the animal is threatened. (Photo contributor: Kevin J. Knoop, MD.)

ERYSIPELOID

Clinical Summary

Erysipeloid, also known as "fish handler's disease," is a bacterial skin infection caused by Erysipelothrix rhusiopathiae. This condition is frequently seen in those who handle raw meat, fish, and shellfish. The offending organism enters the body through a break in the skin and causes a local infection within 2 to 7 days. Lesions are characterized by an edematous central purplish-red area, surrounded first by central clearing and then circumscribed by an advancing raised, erythematous ring. The area is usually pruritic and painful and may be associated with fever, malaise, and regional lymphadenopathy.

Management and Disposition

If left untreated, erysipeloid will usually resolve spontaneously in about 3 weeks. Skin infections may be treated with penicillin VK, cephalexin, or erythromycin. If severe infection occurs, it should be treated with appropriate broad-spectrum antibiotics.

Pearls

1. A history of occupational or recreational exposure to fish or shellfish is the key to diagnosis.
2. E rhusiopathiae is usually resistant to aminoglycoside antibiotics, thus these should be avoided.

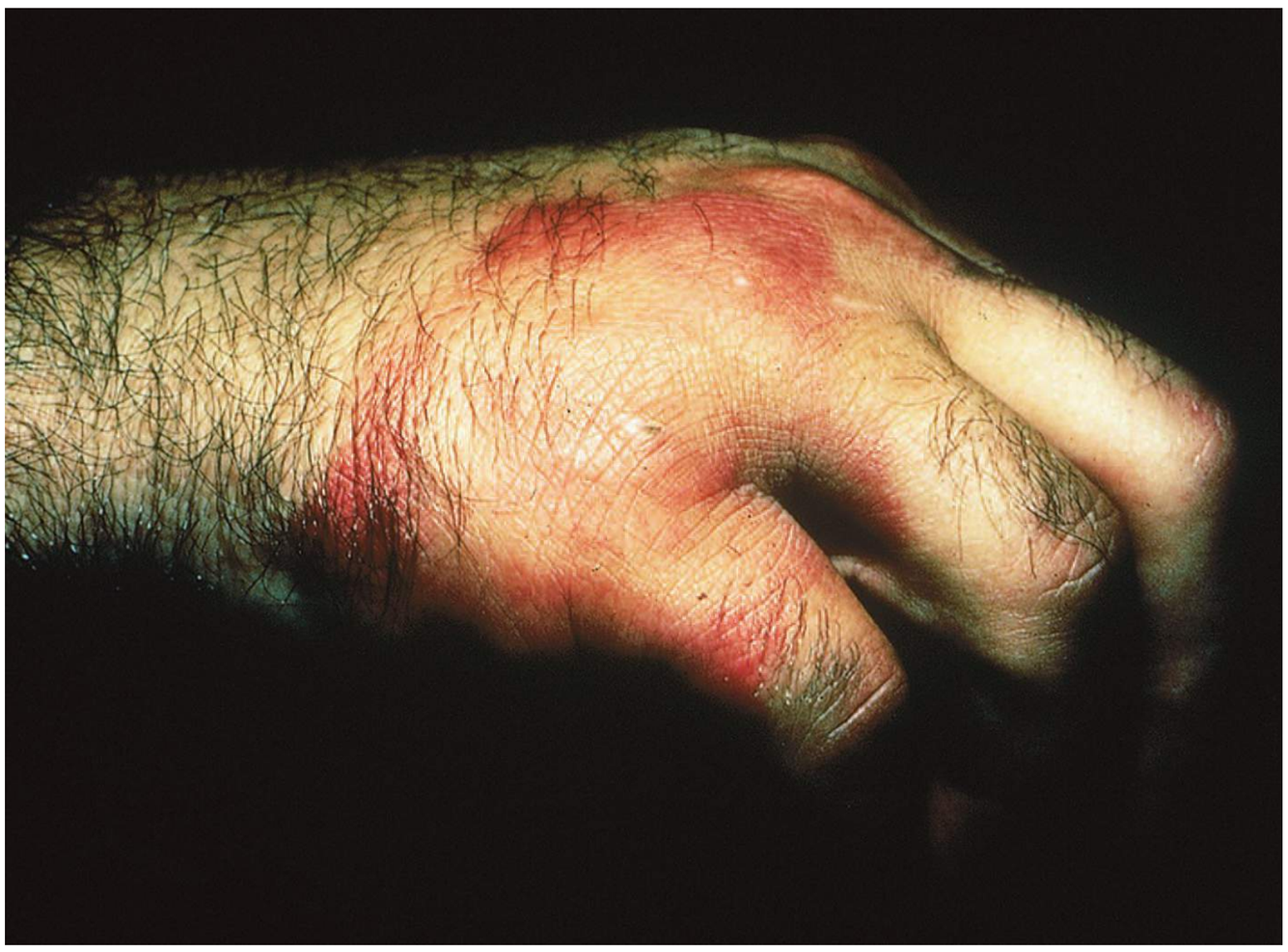

FIGURE 16.122 ■ **Erysipeloid Envenomation.** Typical appearance of Erysipelothrix rhusiopathiae skin infection. (Photo contributor: Paul S. Auerbach, MD; reprinted with permission from Auerbach PS (ed). Wilderness Medicine: Management of Wilderness and Environmental Emergencies. 3rd ed. St. Louis, MO: Mosby-Year Book; 1995. Copyright © Elsevier.)

Clinical Summary

Poison ivy, oak, and sumac cause more cases of allergic contact dermatitis in the United States than all other allergens combined. At least 70% of the US population is sensitive to these Toxicodendron species. The allergen urushiol is responsible for Toxicodendron dermatitis, also known as rhus dermatitis. Urushiol is found in many other plants, including in the skin of mangos. Mangos are a common cause of plant dermatitis in subtropical and tropical areas, including Hawaii.

The dermatitis begins as pruritus and redness usually within 2 days of exposure in susceptible persons. The degree of dermatitis depends on the patient's degree of sensitivity, amount of allergen exposure, and the reactivity of the skin at the exposed body location. The dermatitis may vary from erythema alone or may include papules, vesicles, and bullous eruptions. A linear distribution of the cutaneous lesions is strongly suggestive of Toxicodendron dermatitis. This distribution occurs after plant parts have rubbed against the skin or when contaminated fingernails have scratched it.

Management and Disposition

An immediate rinse or shower with warm water and soap may minimize the reaction. If symptoms are limited to erythema and papules and a small surface area, calamine lotion or topical steroid sprays may provide adequate symptomatic relief. Pruritus may be decreased with oral antihistamines (eg, diphenhydramine or cyproheptadine) and oatmeal baths. Vesicles and bullae may benefit from Domeboro compresses (60 minutes three times daily) to help dry these lesions and relieve pruritus. Systemic corticosteroids tapered over 3 weeks are used in severe reactions. Secondary infection should be treated with systemic antibiotics against staphylococcal and streptococcal species.

Pearls

1. Fluid from the vesicles or bullae does not contain any allergen.
2. Removal of the allergen from the skin within 30 minutes of exposure may prevent dermatitis.
3. Deliberate removal of allergen from under the fingernails may prevent spreading.
4. Treatment with systemic steroids for less than 3 weeks may result in rebound exacerbations of the dermatitis.

FIGURE 16.123 ■ **Poison Ivy.** Toxicodendron radicans (poison ivy—shrub or climbing vine). Note that the leaves of poison ivy have three leaflets and the stems are commonly reddish orange. Poison ivy occurs throughout the United States. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 16.124 ■ **Poison Oak.** Toxicodendron diversiloba (poison oak). Like poison ivy, the terminal part of the branch has a cluster of three shiny leaves. It grows as a tree or woody shrub and occurs west of the Rocky Mountains. (Photo contributor: Ken Zafren, MD.)

FIGURE 16.125 ■ **Poison Sumac.** Toxicodendron vernix (poison sumac). Note that the leaves of poison sumac have 7 to 13 leaflets. It grows as a tree or woody coarse shrub. Only one species of poison sumac is found in the United States. (Photo contributor: Lawrence B. Stack, MD.)

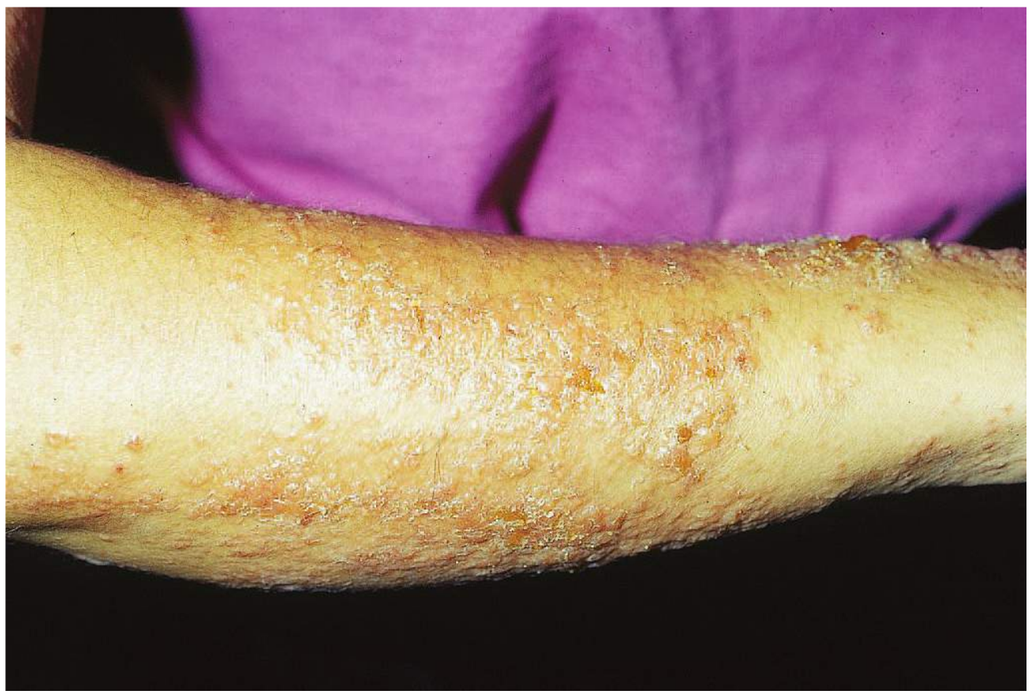

FIGURE 16.126 ■ **Poison Sumac Dermatitis.** A moderately severe local reaction to poison sumac. Note the vesicles, bullae, and exudates characteristic of a contact dermatitis. (Photo contributor: Alan B. Storrow, MD.)

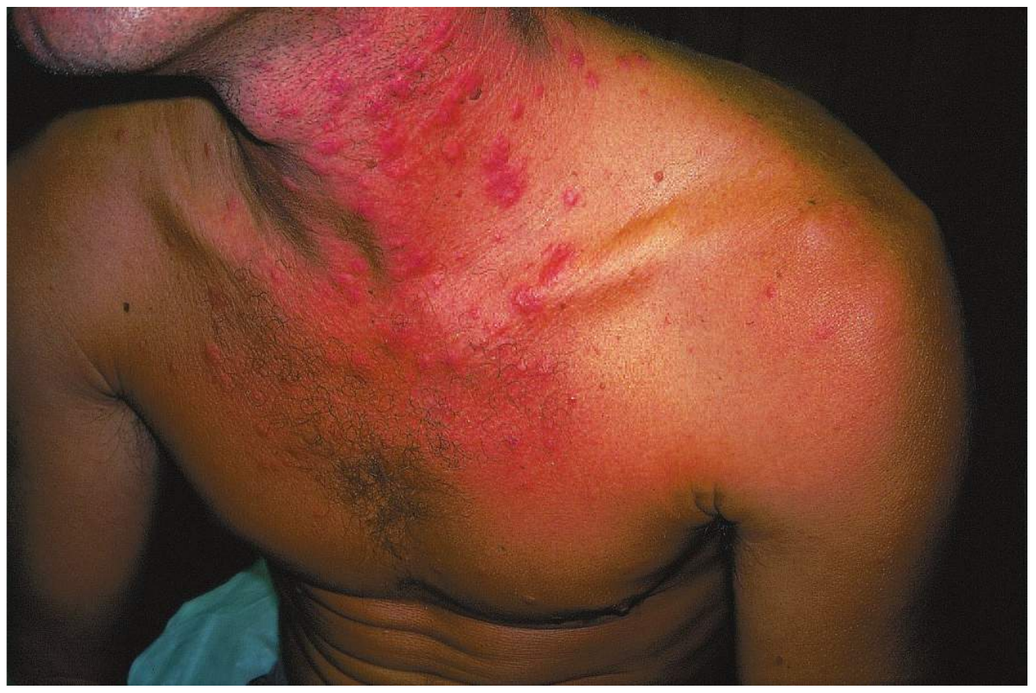

FIGURE 16.127 ■ **Poison Oak Dermatitis.** Erythematous papules and vesicles and vesicles. This fire-fighter was exposed to urushiol, the allergen of poison oak, ivy, and sumac, in smoke from burning poison oak. (Photo contributor: Ken Zafren, MD.)

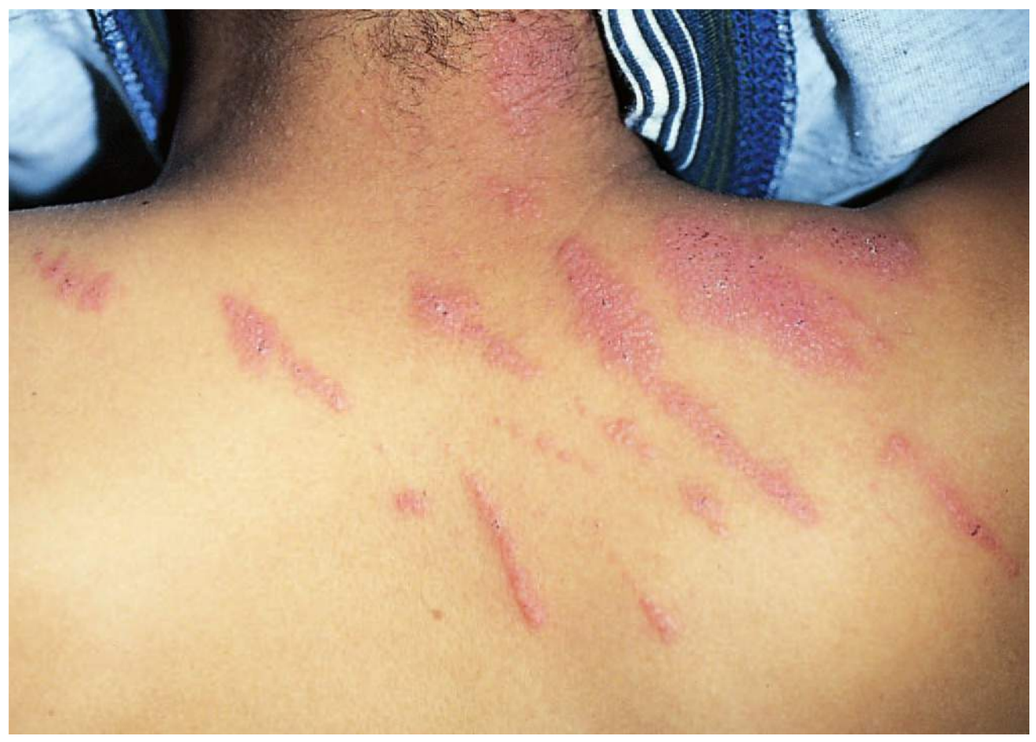

FIGURE 16.128 ■ **Poison Ivy.** Erythematous papules and vesicles in a linear distribution consistent with Toxicodendron dermatitis. (Photo contributor: Alan B. Storrow, MD.)

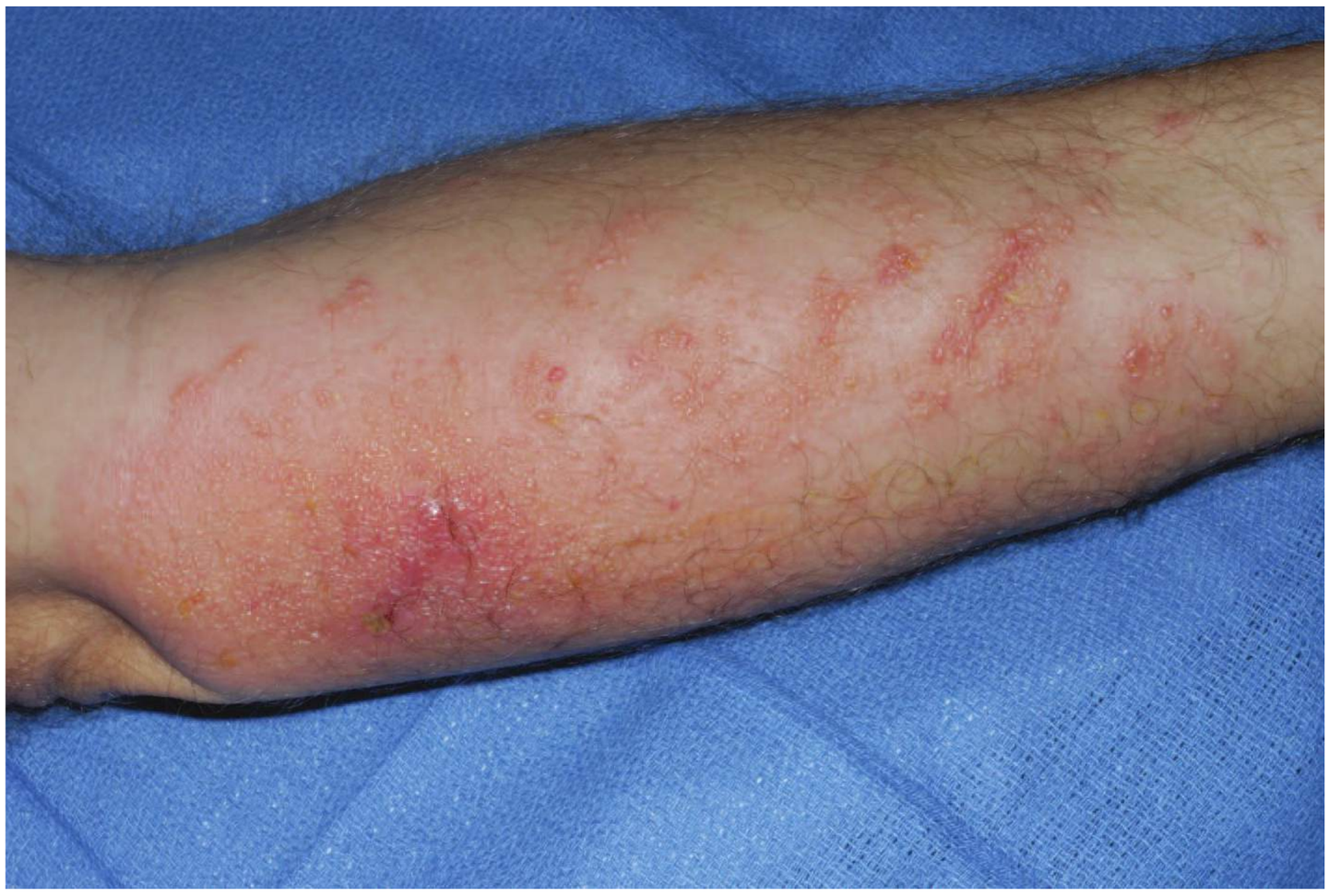

FIGURE 16.129 ■ **Poison Ivy.** A highly pruritic vesicular eruption of Toxicodendron dermatitis on the patient's forearm. Note the linear groupings of vesicles, likely from scratching with contaminated fingernails. (Photo contributor: R. Jason Thurman, MD.)

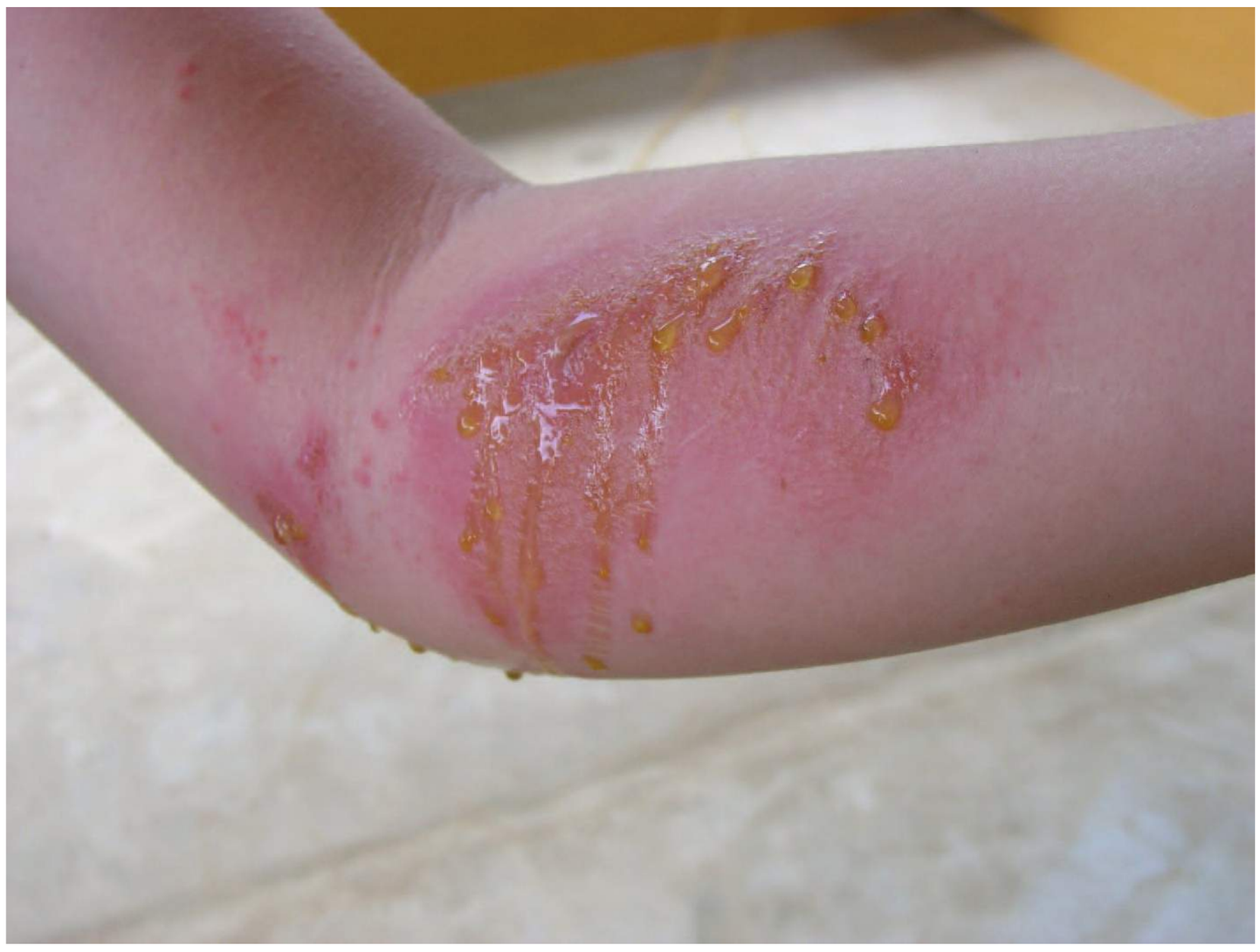

FIGURE 16.130 ■ **Mango Dermatitis.** This patient was catching mangos as they dropped from a tree in Mexico. The rash progressed rapidly over a 48-hour period. This patient can eat mangos, but is sensitive to the skin of the fruit. (Photo contributor: Melissa Gray, WEMT.)

SPOROTRICHOSIS

Clinical Summary

Sporotrichosis is a fungal skin infection caused by Sporothrix schenckii, an organism primarily found on plants and flowers and in soil; the problem is common among gardeners and florists. It also affects those who handle animals, since the fungus may inhabit animals' claws. Infection arises when contaminated thorns, spines, or claws penetrate the victim's skin. After an average incubation period of 3 weeks, localized infections become apparent. "Fixed" cutaneous infections are localized to the inoculation site and are manifest as 2- to 4-mm papules or nodules. They may ulcerate, become surrounded by raised erythema, and are typically painless. Progression to lymphocutaneous infections occurs in about 70% of cases. Patients present with a nodule at the site of penetration, with appearance of subcutaneous nodules and skip areas along lymphatic tracks later. The lesions may wax and wane over months to years. Patients with cutaneous sporotrichosis typically lack systemic symptoms.

Management and Disposition

Sporotrichosis may be successfully treated with oral potassium iodide for 1 month after clinical manifestations have resolved. Alternative therapy includes oral itraconazole, ketoconazole, or terbinafine, while disseminated infections may require intravenous amphotericin B. Outpatient therapy is appropriate for nondisseminated infections. Tetanus status should be addressed.

Pearls

1. Fungal cultures and tissue biopsy cultures are somewhat useful to confirm the diagnosis.
2. Treatment must be continued for 1 month following clinical resolution to eradicate S schenckii.
3. Although rare, a pulmonary form of sporotrichosis after inhalation exposure has been reported.

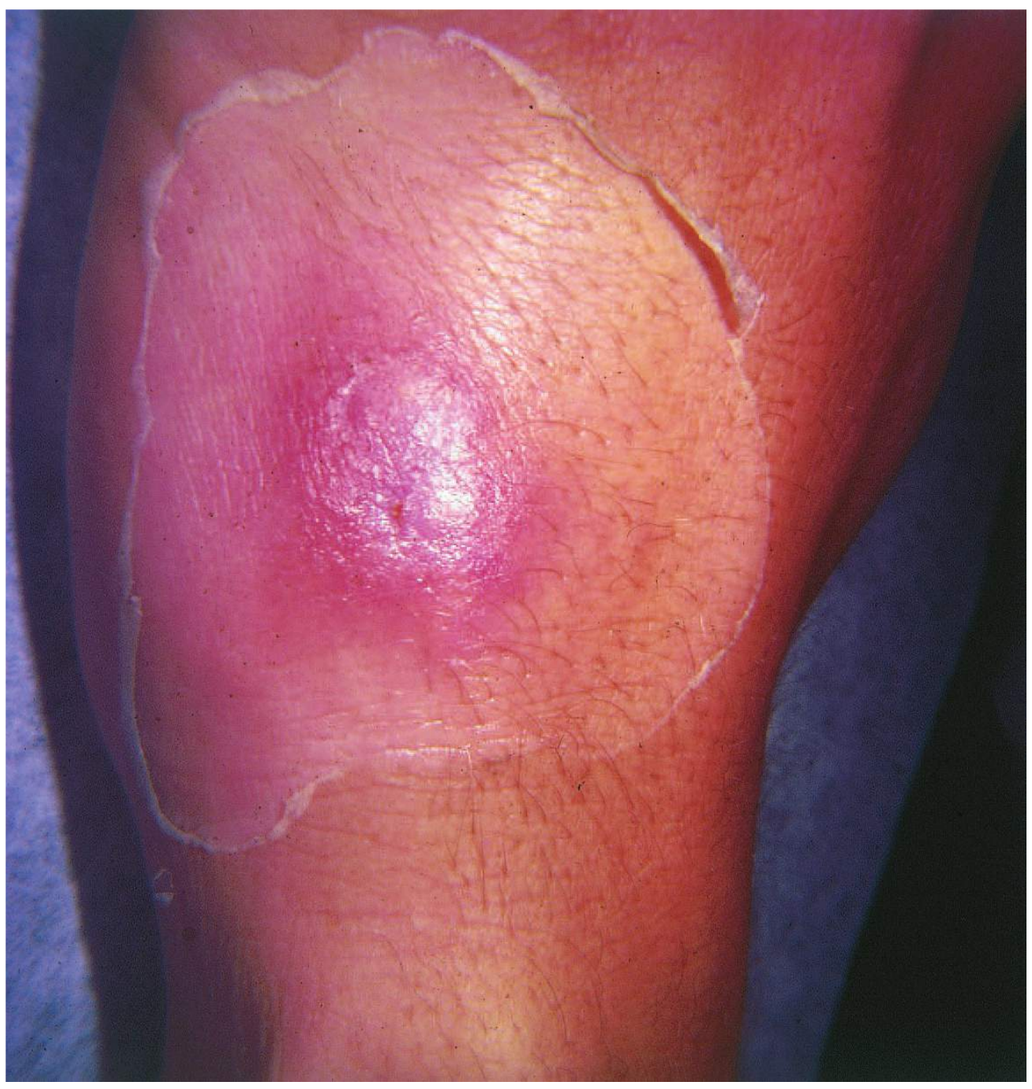

FIGURE 16.131 ■ **Fixed Sporotrichosis.** The ulcer and surrounding erythema of fixed cutaneous sporotrichosis could be confused with a brown recluse spider bite. (Photo contributor: Edward J. Otten, MD.)

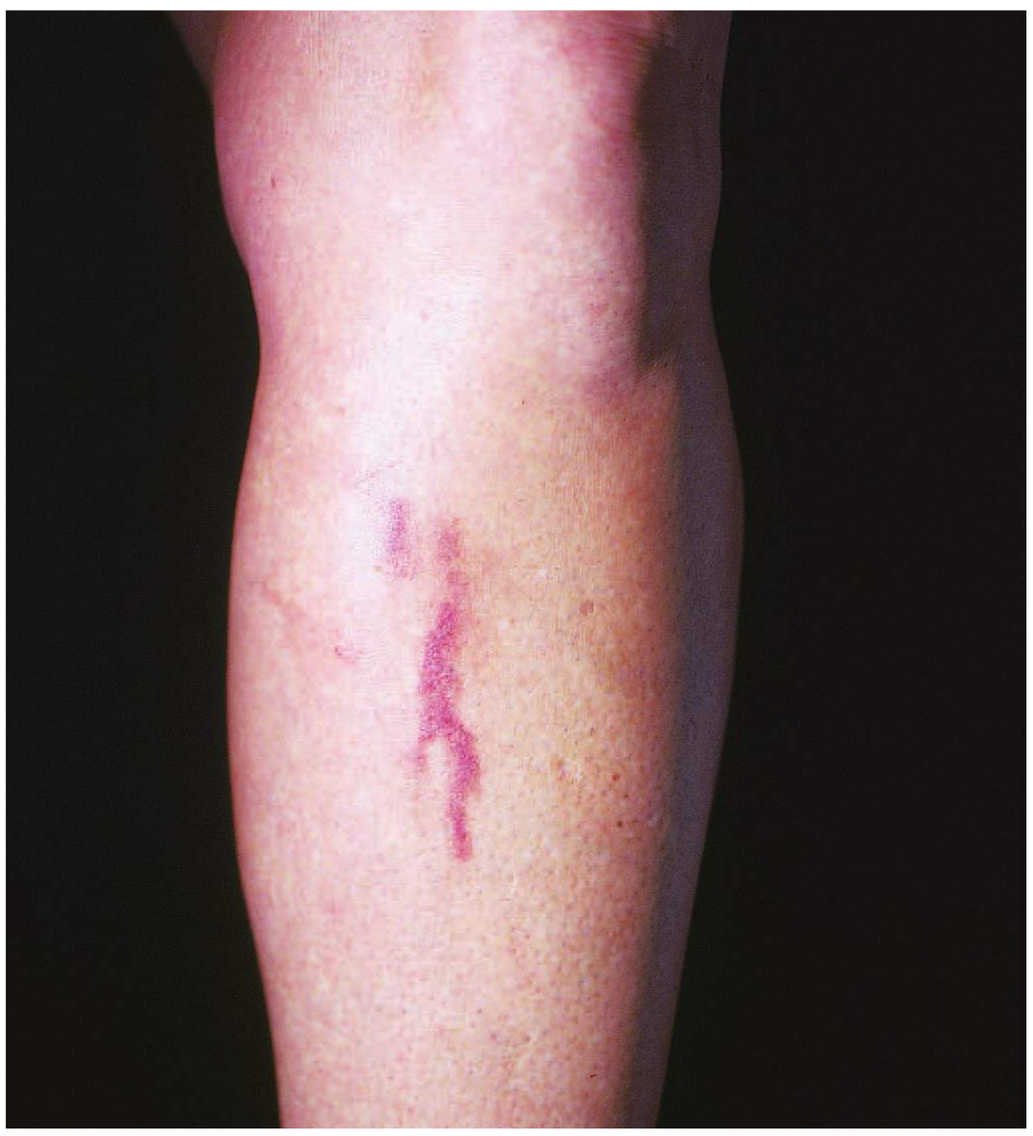

FIGURE 16.132 ■ **Lymphocutaneous Sporotrichosis.** Lymphatic spread is common in cutaneous sporotrichosis. (Photo contributor: Kevin J. Knoop, MD, MS.)