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Creatures of the Deep: Marine Animal-Acquired Injuries and Envenomations

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Does the ocean fascinate you? Can you watch the surf for hours? Many will answer "yes." Perhaps it is something about the origin of life from the sea. Do you scuba dive or snorkel? I don't, but can understand the attraction. But if you do, after reading this article, you can join me in being grateful that we are land-based mammals rather than ones that reside in the ocean. We are pleased to present this review of marine animal-associated injuries and envenomations written by Drs. Gardner and Auerbach, two well-known experts.

—J. Stephan Stapczynski, MD, FACEP, Editor

Introduction

Seventy percent of the earth's surface is covered by ocean. In this marine environment lives four-fifths of the world's organisms, estimated to be about 275,000 species. About 80% of the world's human population lives near a coast. By the year 2010, it is estimated that 127 million U.S. citizens will live near an ocean.[1]

With the ease of travel and mobility, people injured in the marine environment may present for medical attention far inland. Additionally, growth of the marine aquarium industry has resulted in importation of dangerous aquatic species. As the chances for injury increase, so does the need for every emergency physician to be familiar with the basics of treating marine-acquired injuries.

Because marine-acquired wounds have not been studied to the same extent as land-acquired wounds, empirical observations and extrapolation from the traumatic wound literature are important contributors to many recommendations for clinical management.

Manner of Injury

Although treatises of marine injuries traditionally describe

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therapeutic intervention by type of animal involved, it is also useful from a practical standpoint to categorize by type of injury that presents to the emergency department (ED). In this manner, dangerous marine animals can be divided into the following groups:

- those that disrupt the skin (bite, puncture, sever, amputate, or deliver electric shock without envenomation);
- those that sting (envenomation);
- those that cause skin eruptions without envenomation; and
- those that cause allergic reactions.

For the purposes of this publication, the first two will be discussed.

General Principles

First aid. First, address the airway, breathing, and circulation. The patient should have a patent airway, spontaneous breathing, and an adequate blood pressure to assure perfusion of the tissues. Anticipate anaphylaxis.

Wound Management. Whether bite, abrasion, or puncture, any open wound to the skin should receive appropriate wound care.

Wound Irrigation. The wound should be irrigated with an appropriate diluent at a pressure of 10-20 PSI. Sterile saline is the diluent of choice, if available. Sterile water or hypotonic saline may be used if sterile normal saline is not available. Recent research indicates that tap water may be used with efficacy in some wounds. Although this has not been specifically studied in wounds acquired in a marine environment, it is likely completely effective.[2,3] Irrigation with sea water has a hypothetical risk of infection and so should be avoided.

A minimum of 150-200 cc should be used to irrigate each wound. If the wound is from a stingray spine, there may be residual organic material and likely venom in the wound. If the wound is still painful at the time of irrigation, use warm (45°C, 113°F) saline for irrigation. If a thermometer is not available to measure the water temperature, the saline should be as warm as can be tolerated by the patient (tested on uninjured and intact skin) without causing a burn injury.

Wound Anesthesia. Use local infiltration with 1% lidocaine, 0.5% bupivocaine, or a regional nerve block to achieve anesthesia of the wound. These methods have no deleterious effects on tissue healing. Topical anesthetics such as those containing epinephrine to cocaine are less desirable because they worsen wound healing as a result of local vasoconstriction.

Wound Debridement. Sharp dissection and removal of devitalized tissue results in improved healing. Remove obvious debris such as sand, vegetation, spines, or teeth. Imaging via radiograph, ultrasound, CT, or MRI may help locate foreign bodies either preoperatively or perioperatively. Proper debridement should be accomplished in a sterile field, if necessary within an operating room.

Wound Closure. The decision about whether to suture a wound balances the risk of infection with the need for an adequate cosmetic result. The presence of venom and potentially virulent microorganisms, inability to irrigate adequately and early, and delay to definitive treatment all contribute to the high potential for infection. Wounds to the hands, feet, and wrists; puncture or crush wounds; wounds to areas of poor vascularity such as fat; and wounds to persons who are immunocompromised all carry an increased risk of infection. If the decision is made to close the wound, it ideally should be closed loosely enough to allow for adequate drainage. Absorbable sutures and layered closures should be avoided in contaminated wounds to prevent a foreign body reaction.[1]

Tetanus Prophylaxis. The anaerobic bacterium *Clostridium tetani* is ubiquitous in the environment and potentially can enter any break in the skin. Proper immunization with tetanus toxoid virtually eliminates risk of disease. Administer tetanus prophylaxis according to Table 1.

Antibiotic Use. There is little scientific evidence to support the prophylactic use of antibiotics for all marine injuries. However, certain conditions dramatically increase the chances of infection, including deep or puncture wounds, large wounds, crush injuries, bite wounds, and multiple wounds.[4-6] Any person in an immunocompromised state is also at increased risk for developing infection.

The most common pathogens retrieved from marine injuries are *Staphylococcus aureus* and *Streptococcus pyogenes*, presum-

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Table 1. Tetanus Prophylaxis

	Clean Minor Wounds		Dirty Major Wounds	
HISTORY OF IMMUNIZATION (DOSES)	**TOXOID***	**TIG†**	**TOXOID**	**TIG**
Unknown	Yes	No	Yes	Yes
None to one	Yes	No	Yes	Yes
Two	Yes	No	Yes	No (unless wound older than 25 hr)
Three or more				
Last booster within 5 years	No	No	No	No
Last booster within 10 years	No	No	Yes	Yes
Last booster more than 10 years ago	Yes	No	Yes	Yes

* Toxoid: Adult: 0.5 mL dT intramuscularly (IM).
Child less than 5 years old: 0.5 mL DPT IM.
Child older than 5 years: 0.5 mL DT IM.

† Tetanus immune globulin (TIG): 250-500 units IM in limb contralateral to toxoid.

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ably from the introduction of normal flora from the patient's skin through disruption of the integumentary system.[6,7] Marine organisms implicated in skin and soft-tissue infections include *Vibrio* species, *Aeromonas hydrophila*, *Pseudomonas* species, *Plesiomonas* species, *Erysipelothrix rhusiopathiae*, *Mycobacterium marinum,* and other microbes.

Vibrio vulnificus has been implicated in serious, sometimes fatal soft-tissue infections occurring after marine-associated cutaneous wounds. Erythematous lesions may progress to hemorrhagic bullae with high fever, bacteremia, hypotension, and septic shock. The incubation period from wounding to severe systemic illness may be as short as 8-24 hours. The mortality rate from systemic *Vibrio* infections approaches 50%, with death occurring within 48 hours of admission to the hospital.[8] Patients with a wound exposure to warm seawater and concurrently existing liver disease, chronic renal disease, alcoholism, diabetes, malignancy, or other type of immunocompromise are at high risk for rapidly progressive systemic infection.[9-11] Patients with *Vibrio vulnificus* infections and septicemia require hospitalization.[12]

During warm summer months, *Vibrio* species are present in 50% of certain oyster beds. The Centers for Disease Control and Prevention received 302 *V. vulnificus* infection reports from the gulf coast states from 1988-1995. From 1981-1992, the Florida Department of Health and Rehabilitative Services reported 125 persons with *V. vulnificus* infections.

Tetracyclines are the oral antibiotics of choice for treating *V. vulnificus* infections if the drugs are administered early in the course and the victim does not require parenteral antibiotics. The recommended oral doses are doxycyline 100 mg twice daily or tetracycline 500 mg four times daily. Other recommended oral treatment regimens include ciprofloxacin 500 mg or 750 mg daily by mouth or trimethoprim-sulfamethoxazole (double strength) twice daily. Studies of U.S. coastal waters during four different seasons suggest that *Escherichia coli*, *Enterobacter cloacae*, *Klebsiella pneumoniae,* and *Bacillus* species are also important seawater pathogens, and recommend penicillin or ampicillin plus levofloxacin for empiric therapy.[13]

Monitor all marine-acquired wounds closely for signs of infection and be prepared to treat early and aggressively with parenteral antibiotics.

Injuries from Nonvenomous Marine Animals

Sharks. In April 2008, a 66-year-old man sustained a shark bite in the waters of the Pacific Ocean near San Diego, triggering numerous news reports in the media for more than 48 hours. It is a familiar story, since shark attacks hold enormous fascination for the media, scientists, water enthusiasts, and the public. About one-third of the annual number of worldwide shark attacks each year occur in U.S. coastal waters. The International Shark Attack File (ISAF), maintained by the International Elasmobranch Society and the Florida Museum of Natural History, is an authoritative collection of analyzed reports of shark attacks dating back to the mid-1500s. Each year there are 6-10 deaths reported worldwide attributed to shark attacks. Florida alone reports 20-30 "minor" attacks each year, and is the area of greatest number of attacks in the United States.[1] The most commonly reported attackers are the great white (*Carcharodon carcharias*), bull, and tiger (*Galeocerdo cuvieri*) sharks. Positive identification of an attacking species is often very difficult, since most victims do not see the attacker well enough to make an accurate identification and since even trained scientists have difficulty differentiating certain species, especially with requiem shark attacks. Retained tooth fragments can aid in identification, but this happens relatively infrequently.

Sharks are natural predators equipped with numerous finely honed senses (detecting electrical fields, vibration, chemicals in the water, etc.) intended to locate prey. There is some controversy about whether sharks can differentiate distinct colors (such as

International Orange), but it is clear that they prefer colors to black. Most human attacks are "hit-and-run" attacks in which the shark is investigating to determine if the victim is edible prey. Humans are *not* preferred prey, but certain human swimming behaviors may be similar to those of the seals, penguins, and sea turtles preferred by sharks. For large sharks, a human may just be the appropriate size to incite interest. There are known reef sharks that will attack in defense of their territory, but human bites are largely considered to be inquisitive in nature.

The odds of being attacked by a shark along the North American coastline is 1 in 11.5 million, according to ISAF data. Most attacks occur during the summer, in warm (20°C, 68°F) waters, during evening and nighttime feeding, and in recreational areas. Sharks show a predilection for bright, shiny, and reflective surfaces. They have been known to attack surfboards, buoys, and boat propellers. Sharks have attacked swimmers on surfboards (black on white), especially in migratory seal habitats.

Sharks do not chew their food, but anchor it on the bottom teeth then use the serrated upper teeth to slash, aided by swinging the head from side to side. One exception to this is the cookie-cutter shark, which leaves circular 5-6 cm tissue defects where a bite has been made. For shark bites upon living victims, the legs and buttocks are the areas most commonly bitten, followed by hands and arms. Shark attacks historically have been fatal in 15-25% of cases, but advances in prehospital care and major trauma treatment have been instrumental in reducing that number to 10% in the past decade.

Treatment. The most significant risks are exsanguination and hypovolemic shock, followed by drowning. For this reason, it occasionally may be necessary to constrict arterial vessels or compress wounds while the victim is still in the water. Following this, remove the patient from the water and arrange for transport to the nearest medical center capable of treating major trauma. Selective use of tourniquets or pressure points may be necessary, with attention to preventing ischemia in the affected tissues. Keep the patient warm and well-oxygenated during transport. Start two large-bore IVs.

Examine the patient carefully for signs of cervical, intrathoracic, or intraabdominal injuries.

Replace lost blood volume with packed red blood cells. The exact ratio of crystalloid to blood products for shark injuries has not been determined, but it makes sense to follow guidelines established for acute blood loss.

Administer tetanus toxoid 0.5 cc IM and tetanus immune globulin 250-500 units IM, to cover for inoculation of *Clostridium* into the wound.

Shark bites are presumed to contain heavy contamination with seawater, plant debris, sand, shark teeth, and shark mouth flora. For this reason, administer an IV third-generation cephalosporin, trimethoprim-sulfamethoxazole, a fluoroquinolone, an aminoglycoside, or some reasonable combination.

For signs of early septicemia, particularly in an immunocompromised patient, consider meropenem or imipenem-cilastin IV.

Proper operative management of penetrating shark wounds is imperative. The operating room allows for the wide debridement of devitalized tissue and for the copious irrigation necessary to remove debris. This is difficult to accomplish in an emergency department setting.

Wounds should be closed loosely with multiple drains or packed open to await delayed primary closure.

Recovery from a shark bite is a lengthy and often complicated process, requiring attention appropriate for the multiple trauma patient.

Barracuda. The great barracuda lives in all tropical waters and is reputed to grow up to 50 kg (110 lbs) and 2.5 m (8.2 ft). Barracuda are swift, usually solitary swimmers and equally swift attackers. They seldom attack divers, but have been reported to attack in murky waters, especially when attracted to shiny objects. They have been reported to attack people dangling their legs from a boat, cleaning their hands in the water, or attempting to free fish from a net or hook. They are attracted to jewelry, fishing spears, and other metallic objects.

The barracuda has a narrow, elongated mouth with two rows of nearly parallel, razor-sharp teeth. The wounds inflicted by the barracuda are v-shaped, in contrast to the crescent-shaped wounds inflicted by sharks.

Treatment. Except for differences in the magnitude of injury, barracuda wounds are treated identically to those of the shark. The major issues are tissue loss, blood loss, and wound infection.

Moray Eels. Moray eels are found in tropical, subtropical, and some temperate waters. In U.S. waters, the most commonly observed species are the California, spotted, and green morays.

Moray eels have very poor vision and locate prey using their sense of smell. They are easily intimidated and seldom strike unless cornered or provoked. Aquarium owners who house morays must handle them with care to prevent attack. When they do attack, the moray eels are forceful and vicious. They are equipped with long, retrorse, sharp teeth in narrow, vise-like jaws. They are quite tenacious and may bite multiple times or cling to the prey. Most moray eel bites occur on the hand.

Treatment. Moray eels may cling to the victim, requiring forceful removal. Pulling the eel off without breaking the jaws or decapitating the animal may result in significant loss of tissue. After removing the head, the primary wound should be explored.

Inspect and, if necessary, explore the wounds thoroughly for debris and retained moray teeth. Obtain radiographs or other imaging studies if necessary, reevaluate for retained teeth, and assist in their removal.

Irrigate wounds copiously with normal saline.

For isolated small bites, wounds may be left open and allowed to drain. If the bite resembles a dog bite, it can be loosely closed with nonabsorbable sutures or staples. Extensive wounds require surgical evaluation and debridement.

The moray eel has a host of symbiotic organisms in the oral flora. Because of this, prophylactic antibiotics should be started on most moray eel bites. In all cases, the wounds should be re-examined at 24 and 48 hours, since infections from the bite of a moray eel may last for months or years.[1,14]

Sea Lions. Sea lions and seals are usually mild-mannered

mammals with two exceptions: during mating season and when protecting their pups. Divers, aquarium workers, and persons protecting seal habitats have been bitten.

Treatment. Treatment of the bite wounds is similar to that for shark bites. However, the risk of post-traumatic infection differs from that of sharks, both in presentation and sequelae.

Seal finger, also known as spekk finger or blubber finger, occurs after a bite wound from a seal. It also can occur if even a minor wound comes into contact with a seal mouth or pelt. The affliction has a typical incubation time of four days, but may occur between 1 and 15 days. Severe pain is the initial symptom, followed by swelling, stiffness, and a furuncle. If untreated, the digit continues to swell and develops a brown, violet hue. Tenosynovitis and articular destruction have been noted. Painless, nonsuppurative lymphandenitis may develop. The etiology is thought to be *Mycoplasma* species, possibly phocidae or phocacerebrale.[1]

Recommended therapy is oral tetracycline 1.5 g initially, followed by 500 mg four times daily for 4-6 weeks. Fluoroquinolones or macrolide antibiotics may be useful if tetracycline is not available. Beta-lactam antibiotics and cephalosporins are not thought to be useful.[1] Early antibiotics are essential for successful treatment.

Alligators and Crocodiles. Alligators and crocodiles are long-bodied loricans with ferocious reputations. The American alligator *Alligator mississippiensis* most commonly attacks swimmers, waders, and fishermen. The attacks usually occur in water but may occur on land. There were 343 alligator attacks resulting in 13 deaths between 1948 and 2003 in Florida, yielding a mortality rate of 4%; by comparison, the shark attack fatality rate during the same time period was 1.7% (ISAF data). American crocodiles (*Crocodylus acutus)* are found in the southern tip of Florida, and they attack even less frequently than alligators do.

Both species kill by dragging their prey beneath the water and drowning them. They also kill by biting the prey, then waiting for it to die and decay. The meat is easier to tear off after the prey is dead.

Crocodiles and alligators have relatively weak muscles that open the jaw but strong jaw-closing muscles that can sever an outboard boat propeller. The resulting injury is a largely contaminated crush injury, usually of an extremity.

Treatment. Cleanse and irrigate the wound thoroughly, using copious irrigation with normal saline.

Explore the wound for retained debris and possible teeth.

Surgical debridement may be required for large areas of devitalized tissue.

Provide adequate tetanus prophylaxis.

Antibiotic coverage has not been extensively studied in this population, but is assumed to be similar to that of other marine-acquired injuries. For large, contaminated wounds, consider prophylaxis against *Aeromonas hydrophila*, *Pseudomonas*, and *Vibrio*. In addition, coverage against anaerobes such as *Bacteroides* is prudent.[1]

Other Species. There are numerous other species that have been implicated in human injury, including the needlefish, giant octopus, mantis shrimp, and triggerfish. The numbers of injuries are small and often result from territorial invasion or provocation. Killer whales have been reported to "pursue" and batter a human, but one publicized incident occurred in captivity when a whale pursued its trainer. Sea lore attributes attacks to giant clams, colossal squid, and giant manta rays, but actual cases have yet to be substantiated. Divers may sustain significant abrasions when brushing against the dermal skin of the manta ray, but aggressive manta ray behavior has not been noted.

There are two groups of marine electric fish: the electric rays and the stargazers. Generally, the ventral side of an electric ray is negatively charged and the dorsal side is positively charged. An electrical discharge is produced reflexively on contact with the animal in an amount of 8-220 volts. Although the amperage is low, the electrical injury may be enough to stun an adult human and lead to drowning. The imperative is to remove the victim from the water. Anecdotally, recovery is reported to be uneventful.

Stony Corals. The true stony corals have calcerous outer skeletons with razor-sharp edges, pointed horns, or both. Nearly 1,000 species are found in colonies or as individual polyps from surface waters to depths between 20 fathoms (120 ft) and 83 fathoms (500 ft). They generally are found in waters of 20°C (68°F) or higher.

Coral cuts are probably the most common injuries sustained under water.[1] Divers, snorkelers, underwater photographers, and spear fishermen handle or brush against these living reefs, resulting in injury to themselves and to the coral. Divers without gloves commonly sustain cuts to the hands. Knees, elbows, and forearms are also frequent sites of coral cuts. The initial reaction is pain, stinging, erythema, and pruritis. The break in the skin may develop a wheal, which fades over 1-2 hours. "Coral poisoning" is the description given to the erythema and wheal when it is accompanied by fever, gastroenteritis, and/or malaise. With or without treatment, the injury may develop blisters and cellulitis. Low-grade fever may be present. The wounds heal slowly over 3-6 weeks and may develop ulceration and skin sloughing. If chronic dermal granulomatous changes develop, infection with *Mycobacterium*, such as *marinum* or *haemophilum*, should be suspected.[1]

Treatment. Initial. All new coral cuts should be scrubbed vigorously then irrigated copiously with a forceful stream of sterile saline or disinfected fresh water to remove tiny fragments of foreign material ("coral dust"). If stinging persists, the wound may be rinsed briefly with diluted acetic acid (household vinegar) or isopropyl alcohol 20%, noting that these are normally tissue toxic in open wounds and should be used solely for decontamination of suspected coelenterate envenomation. Follow this topical decontamination with a freshwater or saline rinse.

Subsequent Wound Care. There are a number of approaches to subsequent wound care; none have been evaluated by prospective trial. They are:

• twice-daily wet to dry dressings using sterile saline and sterile dressings;

• application of a nontoxic antibiotic (antiseptic) ointment

(e.g., mupirocin, bacitracin) and a nonadherent dressing (e.g., Telfa);

• application of a half- or full-strength antiseptic solution, followed by a powdered antibiotic such as tetracycline. This has largely fallen out of favor.

If the laceration is severe, close loosely with adhesive strips. In all cases, debride the wound daily for 3-4 days, removing devitalized tissue.

These wounds typically heal in a 4- to 15-week period, requiring close observation for wound infection, lichenoid papule formation, or systemic symptoms.

Envenomation by Aquatic Invertebrates

Sponges (Phylum Porifera). There are approximately 5000 known species of sponges. Sponges are acellular animals that attach to the sea floor or to coral beds. Sponges have internal collagenous skeletons of "spongin." Embedded in the connective tissue matrices are spicules of calcium carbonate (calcite) and silicon dioxide (silica). Sponges may be colonized by other sponges, algae, hydrozoans, mollusks, coelenterates, annelids, crustaceans, echinoderms, and fish. These secondary inhabitants may be responsible for the local necrotic skin reaction and dermatitis known as *maladie des plongeurs*, or sponge diver's disease.

There are two general syndromes produced by contact with sponges. The first is a pruritic dermatitis similar to that seen with plant-induced allergic dermatidities and the second is an irritant dermatitis secondary to penetration of the skin by the small calcium or silicone spicules of the sponge.

The most common offender is the Hawaiian or West Indian fire sponge (*Tedania ignis*), found off the Hawaiian islands and the Florida keys. Other culprits include *Fibula* (or *Neofibularia*) *nolitangere*, the "poison bun sponge," and *Microciona prolifera*, the red moss sponge. *F. nolitangere* is found in deep water and grows in clusters, with oscula (openings) wide enough to accommodate a diver's fingers. *M. prolifera* is found in the waters off the coast of the northeastern United States.

Divers without proper gloves sometimes handle sponges, which are friable and may crumble, injuring the hand. Any exposed skin may be abraded by contact with the sponge. Ten minutes to a few hours later, the affected area begins to itch and burn. This progresses to joint swelling, soft-tissue edema, vesiculation, and stiffness. Without treatment, mild reactions usually subside in 3-7 days. Rarely, erythema multiforme or anaphylactoid reactions may occur, developing 7-14 days after the exposure.

The second type of dermatitis is caused by spicules from the sponge breaking off in the skin. Most sponges have spicules and some have crinotoxins that enter the tiny wounds caused by the spicules. In severe cases of sponge dermatitis, the skin may subsequently desquamate 10 days to 2 months after the initial injury. Rarely, recurrent eczema and persistent arthralgias follow. There is no proven medical intervention to retard this process, although administration of potent topical or systemic steroids may be helpful for the eczema.

Treatment. It is virtually impossible to distinguish the two types of dermatitis on initial clinical examination. Therefore, it is prudent to treat both types. It is important to perform the initial decontamination as soon as possible, since delay and/or incomplete initial decontamination may lead to the formation of bullae. If these bullae become purulent, healing may extend for months.

Gently dry the skin.

Remove the spicules using adhesive tape, a thin layer of rubber cement, or a facial peel.[1]

Apply 5% acetic acid (vinegar) soaks for 10-30 minutes every 6-8 hours. Isopropyl alcohol is a reasonable second choice.

After completion of the initial decontamination, apply a mild emollient cream or steroid preparation.

If the allergic or eczematous component is severe, use a systemic glucocorticoid, beginning with a moderately high dose (prednisone 60-100 mg) tapered over 2-3 weeks.

Severe itching may be relieved by an antihistamine.

Anecdotal remedies for management of sponge envenomation include antiseptic dressings, broad-spectrum antibiotics, methdilazine, pyribenzamine, phenobarbital, diphenhydramine, promethazine, topical carbolic oil, or zinc oxide. None of these have been shown to have any proven efficacy in the treatment of sponge dermatitis.

Coelenterates (Phylum Cnidaria). There are at least 100 members of the coelenterates that are dangerous to humans, a small portion of the approximately 10,000 species. Coelenterates ("hollow gut") organisms are one of the lowest forms of life. Medically significant coelenterates with the venom-charged stinging apparatus, known as a nematocysts, are called Cnidaria. Those without nematocysts are called acnidaria.

Classification. The Cnidaria can be divided into three main groups:

• Hydrozoans, e.g., Portuguese man-of-war;

• Scyphozoans, e.g., true jellyfish; and

• Anthozoans, e.g., soft corals (alcyonarians), stony corals, and anemones.

An alternative categorization by Fenner[15] divides jellyfish into three main classes:

• Schyphozoans—true jellyfish, which have a transparent bell and tentacles arranged at regular intervals around the bell;

• Cubozoans—box jellyfish, which have a bell with tentacles only at the four corners. (These are further subdivided into: Carybdeids with only one tentacle at each corner, e.g., Irukandji; and Chirodropids with more than one tentacle at each corner); and

• Other jellyfish, which includes the hydrozoans. (This includes *Physalia* [Portugese man-of-war] species.)

It is important to distinguish this difference in classification when reviewing the pertinent literature. Although the identification and treatment of deadly cnidarians found only in Indo-Pacific waters if fascinating, this publication will emphasize those that could be seen by emergency physicians in the United States.

Venom Apparatus. For clinical purposes, all stinging species in this phylum are somewhat related, so the clinical features of coelenterate syndromes are fairly constant, with a spectrum of severity. The basis of the venom apparatus is the cnidocyte. The

cnidae (nematocysts, spirocysts, and ptychocysts) are nonliving intracytoplasmic stinging organoids that are encapsulated in this living cell called the cnidocyte. Cnidocytes line the epithelial surfaces of the tentacles and/or the area near the mouth and are triggered by contact with the victim. The nematocyst is contained within a cnidoblast (outer capsule) and is attached to a cnidocil (single, pointing trigger). The nematocyst is filled with fluid and a coiled tubule (nema). When the cnidocil is triggered by physical contact, the operculum opens and the venom-bearing tubule is everted in less than 3 microseconds. The thread penetrates the epidermis and upper dermis and delivers venom into the circulation of the victim. Theoretically, a human encounter with a Portugese man-of-war could trigger several million stinging cells.[16]

Factors affecting the severity of a coelenterate envenomation include season, species (venom potency), number of nematocysts triggered, size of the animal, size of the victim, age of the victim, location and surface area of the sting, and health of the victim. Brief envenomation may result in only mild dermatitis, while severe envenomation can result in mortality. Clinicians should suspect coelenterate envenomation in all near-drownings, diving accidents, and collapses that occur in a marine setting.

Treatment – Mild envenomation/dermatitis. Rinse the wound immediately with sea water. Do not use fresh water. It is believed that nonforceful fresh water rinsing or rubbing will cause nematocysts to discharge, worsening the injury.

Recent studies indicate that a hot fresh water shower with a forceful stream applied by lifesavers at the beach can reduce the pain of envenomation.[17,18] It may be that the mechanical effect of the water dislodges the nematocysts and/or tentacle fragments, preventing worsening of the injury.

Remove any visible tentacle fragments with forceps. If absolutely necessary, a gloved (double thickness of surgical gloves) hand or the relatively keratinized surface of the palm of the hand can be used to remove tentacle fragments.

Decontaminate using the appropriate solution. There is a great deal of variety in recommendations for the "appropriate" solution for immediate decontamination. Acetic acid 5% (vinegar) is the solution of choice for treating *Chironex fleckeri*. It may not be as useful against Chrysaora or Cyanea. Sodium bicarbonate is reported to be effective against the sea nettles (*Chrysaoara quinquecirrha*) found in the Chesapeake Bay and the coastal Atlantic waters of the United States. Isopropyl alcohol and dilute ammonium hydroxide also have been used with some clinical success. A slurry of papain (meat tenderizer) has been recommended by some for sting of thimble jellyfishes that cause seabather's eruption. Lime or lemon juice may be effective. The following are *not* effective: perfume, aftershave lotion, high-proof liquor, formalin, ether, or gasoline.

Flood the area with the decontaminant for at least a few minutes. More time may be required for most decontaminants, but if using papain, remove it after 5 minutes and do not use it on delicate or thin skin.

Cold Pack/Hot Pack. Commercial cold packs traditionally have been recommended, applied over a thin cloth or plastic membrane. Recent studies indicate that hot water immersion may be equally or more effective for certain species in the Indo-Pacific, but this has not yet been confirmed for U.S. coastal species.[17,19,20]

Remove the remaining (perhaps "invisible") nematocysts using shaving cream and a razor. In a primitive setting, make a paste of sand and sea water and "shave" the area using a sharp shell or piece of wood.

Anesthetize the area using a topical anesthetic ointment (lidocaine, 2.5%) or spray (benzocaine, 14%). Antihistaminic cream (diphenhydramine or tripelennamine) or a mild topical steroid (hydrocortisone, 1%) also may be soothing. Administer pain medication as needed.

Tetanus prophylaxis is required.

No prophylactic antibiotics are required. Wounds should be checked at 3 days and 7 days for signs of infection. If a lesion develops ulceration, apply a thin layer of mupirocin daily.

NOTE: *A jellyfish sting involving the cornea should be referred immediately to an ophthalmologist. Do not use standard decontaminating solutions in the eye.*

Treatment – Moderate to Severe Envenomation. The prime offenders in this group are the anemones, *Physalia* species, and scyphozoans. The targets of treatment are to provide supportive care for systemic symptoms, provide adequate pain relief, and treat the dermatitis. Treatment of the dermatitis is identical to that for a mild envenomation.

Provide supportive treatment based on the signs and symptoms.

Treat hypotension with prompt IV administration of crystalloid. However, do not discontinue the decontamination while starting fluids because it is important to continue to interrupt further venom release into the victim.

Hypertension is an occasional side effect of a cubomedusan envenomation, such as that from the Irukandji. Although this is more typically reported from the Indo-Pacific, there is a reported Irukandji-type death from the Gulf of Mexico. The hypertension is felt to be secondary to the release of circulating catecholamines. Treat with phentolamine 5 mg IV initial dose followed by an infusion of up to 10 mg/hr.

Manage bronchospasm and wheezing as an allergic component. Arterial blood gases may be useful to guide oxygen administration by mask.

Seizures are usually self-limited and can be managed with IV diazepam. They rarely recur after the first 48 hours.

Monitor older patients for arrhythmias and obtain an electrocardiogram.

Obtain a urinalysis to check for hemoglobinuria, which occurs when the venom of certain *Physalia* species attaches to red blood cell glycoprotein sites, causing red cell lysis. If hemoglobinuria is present, alkalinize the urine with sodium bicarbonate and administer a loop diuretic (furosemide) or mannitol to maintain a moderate diuresis of 30-50 cc/hr.

To date, the only coelenterate antivenom available is for the box-jellyfish *Chironex fleckeri*.

Phylum Echinodermata. This phylum includes starfish, sea cucumbers, sea urchins, sea lilies, and brittle stars. Only the first

three are of significant medical interest.

Starfish are stellate, simple, free-living organisms covered with calcium carbonate spines. The crown-of-thorns starfish (*Acanthaster planci*) is injurious to humans and is found throughout the Pacific and the Gulf of California. The venom of *A. planci* covers the sharp, thorny spines. The venom has hepatotoxic, myonecrotic, and anticoagulant effects.

The thorns can penetrate a thick diver's glove or reef slipper. There is immediate severe pain, initial blanching followed by erythema, mild edema, and copious bleeding. Multiple puncture wounds may result in systemic symptoms including nausea, vomiting, paresthesias, lymphadenopathy, and muscular paralysis.

Treatment. Immediately immerse the wound in hot water to tolerance (45°C, 113°F) for 90 minutes. If a thermometer is not available, first place a noninjured extremity into water of the same temperature to determine the patient's tolerance and avoid burning the envenomed tissue, which may have an altered sensory perception of heat.[21]

Irrigate the wound copiously with normal saline.

Explore the wound for retained fragments. The thorns of *A. planci* are very sturdy and usually do not leave fragments. However, if a smaller starfish is encountered, the tips may break and become embedded foreign bodies. If there is a suspicion of retained thorns, obtain a radiograph of the affected extremity.

Allow the wound to heal without sutures.

Granulomas from retained starfish spines may require surgical excision after the wounds have healed.

Sea Urchins. There are approximately 600 species of sea urchins, roughly 80 of which are venomous to humans. The venom is contained in the hollow spines and in the triple-jawed pedicellariae. Most people are injured when they step on, handle, or brush up against a sea urchin. The long, needle-like spines of the sea urchin break off easily and may remain in the wound.

The puncture-type wound is immediately intensely painful. The burning pain rapidly becomes local muscle aching and the skin develops erythema at the site. The external surfaces of the spines of some species (*Diadema setosum* or *Strongylocentrotus purpuratus*) carry a purplish-black dye that may be deposited in the wound(s) and leave the false impression that there is a retained spine in the wound.

If a spine invades a joint space, tenosynovitis may develop rapidly. If spines are deeply embedded or if there are multiple spine fragments, systemic symptoms may develop. These include nausea, vomiting, paresthesias, numbness, muscular paralysis, abdominal pain, hypotension, and respiratory distress. Frank neuropathy suggests direct contact with a peripheral nerve. Spines that break off near the finger- or toenail may cause a subungual or periungual granulomatous nodular lesion.

Treatment. Immediately immerse the affected extremity in hot water to tolerance (45°C, 113°F) for 30-90 minutes.

Remove any pedicellariae still attached to the skin. This is accomplished with shaving cream and a razor.

Remove any of the long, needle-like spines that are easily retrieved. Very thin spines may be absorbed in 1-21 days but thick, calcified spines must be removed from the tissues.

Obtain soft-tissue-technique radiographs, ultrasound, or an MRI to localize retained spines.

Retained spines in the hands or feet potentially could intrude upon a joint or blood vessel or could be in a location to cause chronic inflammation if they remain. If it is determined that the retained spines should be removed, they should be removed by a qualified surgeon using an operating microscope as soon as possible after the injury.

Injuries that involve interphalangeal joints should be splinted to prevent further fragmentation of the retained spines. Retained spines become a nidus for granuloma formation. These granulomas may be excised at a later date.

Sea Cucumbers. Sea cucumbers are free-living bottom-dwellers found in both shallow and deep waters. The animals extrude tentacles that may contain a cantharidin-like liquid toxin known as holothurin. On occasion, the body walls of the animals contain the toxin. Normally, holothurin is diluted by the surrounding sea water. Injury may occur when sea cucumbers are handled for food preparation.

Holothurin produces contact dermatitis similar to that caused by starfish. If ingested, holothurin is a potent cardiac glycoside and may cause death. Holothurin in the water from recent handling of sea cucumbers can contact the cornea when a diver clears his mask. The resulting inflammation may be severe.

Treatment. Treat the contact dermatitis in the same fashion as dermatitis from starfish. Because sea cucumbers feed on coelenterates and excrete the venom, contact may cause a dermal reaction. Flood the wound with 5% acetic acid (vinegar) for decontamination.

For eye exposures, anesthetize the eye with 1% proparacaine, then irrigate the affected eye with 100-250 cc of normal saline to remove debris. Perform a slit lamp examination to determine the presence of corneal injury. Examine the anterior chamber and iris for involvement. Prompt referral to an ophthalmologist is imperative.

Annelid Worms (Phylum Annelida). Most marine worms are carnivorous free-moving organisms that live from the tidal zone to depths of 16,000 feet. The bristleworm (*Hermodice carunculata*), which releases chitinous, irritating bristles upon contact, is improperly handled by divers and home aquarists.

The bite or sting of an annelid worm may result in intense pain and inflammation, accompanied by an urticarial, erythematous rash. Some spines are too small to be visualized with the naked eye, and most, regardless of size, are easily broken off into the skin.

Symptoms are self-limited, but the inflammation may last 7-10 days.

Treatment. Dry the skin without abrading or rubbing.

Remove the spines using adhesive tape, a facial peel, or a thin layer of rubber cement.

After spine removal, douse the area with 5% acetic acid (vinegar), dilute ammonia, or 70% isopropyl alcohol.

Severe inflammation can be treated with a topical corticosteroid.

Mollusks (Phylum Mollusca). The phylum Mollusca contains more than 45,000 members. Of the 5 main classes, three

pose a danger to humans:

- Pelecypods—scallops, oysters, clams, and mussels;
- Gastropods—snails and slugs; and
- Cephalopods—squids, octopuses, and cuttlefish.

Mollusks are also implicated in poisonous ingestions.

Cone Snails (Cone Shells). There are 500 species of marine snails that are thought to be venomous, but fewer than two dozen species have been reported to cause human envenomations.[22] The brightly colored geometric patterns on the shells are a lure for shell collectors.

Venomous cone snails hunt prey and protect themselves using a harpoon-like radula located at the tapered end of the shell within a proboscis that may extend more than 3 times the length of the shell.[23,24] The snail contracts a muscular pharynx and launches the radula. Unlike the passive discharge of a nematocyst in a coelenterate, the extension of the proboscis and launching of the radula is under the voluntary control of the cone snail.

Through a combination of alpha and mu conotoxins, which act both presynaptically and postsynaptically, effective neuromuscular paralysis may result from only minute inoculations of toxin. The wound may or may not produce initial pain, but paresthesias at the site rapidly affect the limb, the perioral area, and then the entire body. Death is attributed to diaphragmatic paralysis or cardiac failure.

Treatment. There is no specific antivenom. The pressure-immobilization technique for venom sequestration has been suggested but has not been proven effective as a first-aid measure.

Cardiovascular and respiratory support are life-saving in this envenomation.

Inspect the wound for a foreign body (the radula) and remove it if present.

Numerous topical therapies have been suggested, but none has proven beneficial by scientific standards.

One reasonable approach suggested is the use of edrophonium (10 mg IV in an adult) as empiric therapy for paralysis. Choose a relatively weak group of muscles for which strength can be objectively tested and inject 2 mg IV. If there is improvement, inject 8 mg IV.

Adverse reactions to edrophonium, such as salivation, nausea, diarrhea, and muscle fasciculations, can be treated with atropine 0.6 mg IV.

Octopuses. Octopuses and cuttlefish are cephalopods that are usually harmless and shy. The media has created the image of a giant octopus surrounding its prey with its tentacles, but the truth is that the most dangerous of the octopuses are less than 10-20 cm (4-8 inches) in length.

Octopus bites are rare, but can result in severe envenomation. The most venomous of these is the blue-ringed octopus found only in Australasian waters. The common octopus *Octopus vulgaris* is nonvenomous. The *Octopus joubini* sometimes frequents empty containers or submerged bottles in the waters of the Caribbean.

The venom apparatus of the octopus is composed of the salivary ducts, buccal mass, beak, and salivary glands. It is located on the central ventral surface surrounded by tentacles. Maculotoxin from the blue-ringed octopus has at least one component identical to tetrodotoxin.

Most octopus bites occur on the hand, acquired while handling the animal. The bite itself consists of one or two puncture marks and may initially go unnoticed. Sometimes local urticarial reactions occur, and profuse bleeding at the site is caused by a local anticoagualant effect. Local reactions are minimal compared with the systemic effects.

For a blue-ringed octopus bite, within 5-10 minutes of the bite, paresthesias develop and ascend the affected limb. Perioral numbness develops between 10 and 30 minutes, leading rapidly to total flaccid paralysis and respiratory collapse.

Treatment. The mainstay of therapy is respiratory support.

Pressure-immobilization has been proposed for the local injury and should be used.

Provide immediate ventilatory support, including endotracheal intubation and mechanical ventilation.

The effects of the venom last between 4 and 10 hours. If no anoxia occurs during that time, recovery should occur within 2-4 days.

Summary. Treatment of marine envenomations by invertebrates can be difficult, especially since the exact culprit is not always reliably identified by the victim. Figure 1 provides a useful algorithm for the treatment of envenomations.

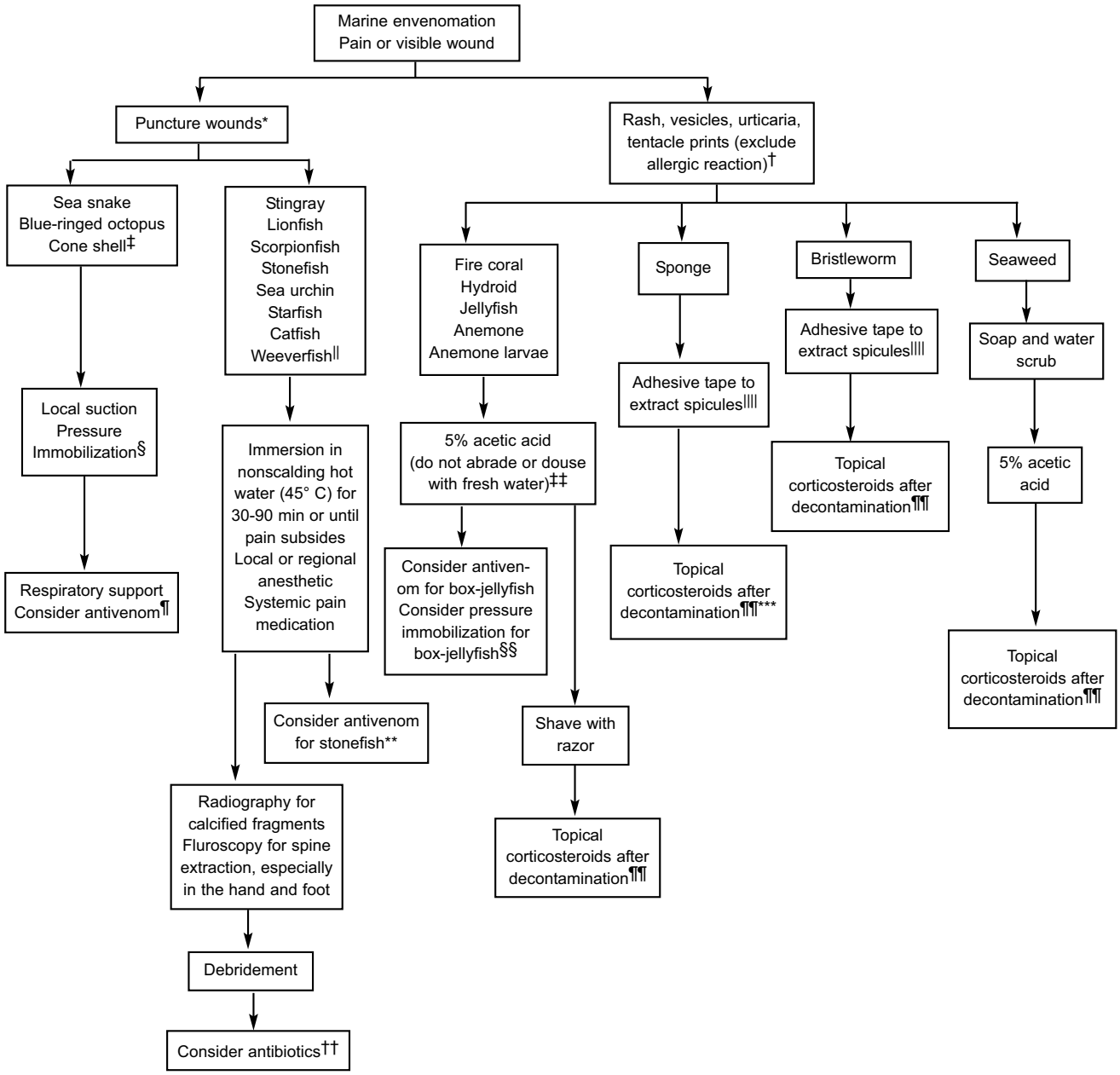
Aquatic Vertebrate Envenomation

Stingrays. The "devil ray" or the "demon of the deep" has been recognized as venomous since ancient times. It is the most commonly incriminated group of fish involved in human envenomations. Twenty-two species of rays are found along the U.S. coast, eight in the Pacific, and 14 in the Atlantic.

About 2000 stingray injuries occur each year in the United States, most caused by members of the family Dasyatidae. Most injuries occur in the summer and autumn months when vacationers take advantage of the surf. Coincidentally, that is the time that rays congregate to spawn. In the southeastern United States, the southern stingray (*Dasyatis americana*) is a frequent stinger. On the west coast, the round stingray (*Urolophus halleri*) has that distinction. There are no known freshwater species in the United States.

Rays vary from small (several inches) to large (12 ft x 6 ft) and have a flattened, wide body with large pectoral fins. They are bottom-feeders and tend to stay on the bottom of the sea, partially submerged in the sand. For this reason, a frequent cause of injury is stepping on the body of the animal, causing the whip-like tail to reflexively snap up and thrust a spine into the unwary human, causing a puncture wound or laceration. (*See Figure 2.*) There are occasional abrasions reported by divers who "hitch a ride" on large manta rays, but these are caused by the rough dermis of the creature, not by envenomation. In certain parts of the world, stingrays are hand-fed by tourists. Since the ray feeds by suctioning water and prey through the ventral opening, a "hickey" can be caused by its attempt to "vacuum" the food from the hand of the unsuspecting tourist, or a direct bite by the animal.

Figure 1. Algorithmic Approach to Marine Envenomation



***** A gaping laceration, particularly of the lower extremity, with cyanotic edges suggests a stingray wound. Multiple punctures in an erratic pattern with or without purple discoloration or retained fragments are typical of a sea urchin sting. One to eight (usually two) fang marks are usually present after a sea snake bite. A single puncture wound with an erythematous halo and rapid swelling suggests scorpionfish envenomation. Blisters often accompany a lionfish sting. Painless punctures with paralysis suggest the bite of a blue-ringed octopus; the site of a cone shell sting is punctate, painful, and ischemic in appearance.

† Wheal and flare reactions are nonspecific. Rapid (within 24 hours) onset of skin necrosis suggests an anemone sting. "Tentacle prints" with cross-hatching or a frosted appearance after application of aluminum-based salts suggest a box-jellyfish (*Chronex fleckeri*) envenomation. Ocular or intraoral lesions may be caused by fragmented hydroids or coelenterate tentacles. An allergic reaction must be treated promptly.

‡ Sea snake venom causes weakness, respiratory paralysis, myoglobinuria, myalgias, blurred vision, vomiting, and dysphagia. The blue-ringed octopus injects tetrodotoxin, which causes rapid neuromuscular paralysis.

§ As soon as possible, venom should be sequestered locally with a proximal venous-lymphatic occlusive band of constriction, or (preferably) by the pressure-immobilization technique, in which a cloth pad is compressed directly over the wound by an elastic wrap that should encompass the entire extremity at a pressure of 9.33 kPa (70 mmHg) or less. Incision and suction are not recommended.

¶ Early ventilatory support has the greatest influence on outcome. The minimal initial dose of sea snake antivenom is one to three vials; up to 10 vials may be required.

(***Continued on next page.***)

Figure 1. Algorithmic Approach to Marine Envenomation (*continued*)

‖ The wounds range from large lacerations (stingrays) to minute punctures (stonefish). Persistent pain after immersion in hot water suggests a scorpionfish or stonefish sting or a retained fragment of spine. The puncture site can be identified by forcefully injecting 1% to 2% lidocaine or another local anesthetic agent without epinephrine near the wound and observing the egres of fluid. Do not attempt to crush the spines of sea urchins if they are present in the wound. Spine dye from already-extracted sea urchin spines will disappear (be absorbed) in 24 to 36 hours.

** The initial dose of stonefish antivenom is one vial per two puncture wounds.

†† The antibiotics chosen should cover *Staphylococcus*, *Streptococcus*, and microbes of marine origin, such as *Vibrio*.

‡‡ Acetic acid (5%; i.e., vinegar) is a good all-purpose decontaminant and is mandated for the sting from a box-jellyfish. Alternatives, depending on the geographic region and indigenous jellyfish species, include isopropyl alcohol, bicarbonate (baking soda), ammonia, papain, and preparations containing these agents.

§§ The initial dose of box-jellyfish antivenom is one ampule intravenously or three ampules intramuscularly.

¶¶ If inflammation is severe, steroids should be given systematically (beginning with at least 60 to 100 mg of prednisone or its equivalent), and the dose should be tapered over a period of 10 to 14 days.

‖‖ An alternative is to apply and remove commercial facial peel materials.

*** An alternative is to apply and remove commerical facial peel materials followed by topical soaks of 30 mL of 5% acetic acid (vinegar) diluted in 1L of water for 15 to 30 minutes several times a day until the lesions begin to resolve. Anticipate surface desquamation in 3 to 6 weeks.

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The venom apparatus is a bilaterally retroserrated spine and the enveloping integumentary sheath. There may be up to 4 spines on the tail of a single ray. The spines are attached to the dorsum of the tail. Along the edges of the spines are hollow grooves that contain soft venom glands.

A stingray wound is both a mechanical wound and an envenomation. The cartilagenous spine and strength of the strike combine to cause significant tissue damage. Most injuries involve the lower extremity. There are also reported injuries to the upper extremity, abdomen, and thorax. In very rare cases, the heart may be injured directly. In addition, a detached stingray spine may be used as a combat weapon.

Envenomation is accompanied by immediate intense local pain with variable edema and bleeding. The pain radiates centrally, peaks at 30-60 minutes, and may last for 48 hours. Systemic symptoms include nausea, vomiting, diarrhea, diaphoresis, vertigo, tachycardia, headache, syncope, seizures, muscle cramps, fasciculations, generalized edema, paralysis, hypotension, arrhythmias, and death.

Treatment. Treatment is aimed at combating the effects of the venom, relieving pain, and preventing subsequent infection.

First, as in all marine injuries, remove the victim from the water. Muscle contractions and fasciculations may be caused by the venom and/or pain, causing problems for a swimmer.

Immediately soak the injury in nonscalding hot water (45°C, 113°F) for 30-90 minutes. If hot water is not available, irrigate the wound with nonheated water or saline. If sterile water or saline is not available, use tap water. The goal is to remove as much of the venom and organic material as quickly as possible. In addition, the heat may attenuate some of the thermolabile components of the venom or interrupt nerve impulse transmission. It has no proven effect on the ultimate degree of tissue necrosis.

There is no indication for additives for the soaking solution.

During the hot water soak, explore the wound and remove any visible portions of the sting or its integumentary sheath. *Note: The exception is a penetrating injury of the chest or other body location* where the presence of the spine may be occluding a blood vessel with the propensity to bleed torrentially. These should be removed in the operating room under sterile conditions with adequate preparation for uncontrolled bleeding or other catastrophic events.

There is no role for cryotherapy, steroids, or antihistamines.

Initiate pain control as soon as possible during the first soaking period. Parenteral narcotics may be required. Local infiltration with 1% or 2 % lidocaine or bupivicaine 0.25-0.50% without epinephrine may be needed. A regional nerve block can be used.

Upon completion of the soaking procedure, explore the wound in a sterile fashion.

Excise hemorrhagic fat and obviously devitalized tissue.

Pack the wound for a delayed primary closure or close loosely around adequate drainage. An alternative includes excising the wound and packing it with an alginate dressing.[25]

Provide tetanus prophylaxis.

Prescribe prophylactic antibiotics to prevent secondary infection. To cover *Vibrio* species implicated in cases of stingray infection,[26] administer oral doxycycline 100 mg twice daily, tetracycline 500 mg four times daily, or trimethoprim-sulfamethoxazole double-strength twice daily. Ciprofloxacin 500 mg or 750 mg daily is an alternative. Topical becaplermin gel was used with some success in one patient who suffered prolonged healing.[27]

Obtain an MRI if a retained foreign body is expected and none is seen on plain radiographs. Stingrays have cartilaginous endoskeletons, so spine fragments may not be visible without advanced imaging.

Obtain surgical consultation for repair of vascular injuries, lacerated nerves and tendons, and for all stab wounds of the abdomen, thorax, or neck.[28,29]

Observe patients who are not being admitted for 3-6 hours to make sure that systemic symptoms do not develop.

Venomous Fish. The family Scorpaenidae is divided into three groups:

- Pterois—the zebrafish, lionfish, and butterfly cod;
- Scorpaena—scorpionfish, bullrout, and sculpin; and
- Synanceja—stonefish.

These fish dwell in tropical and, less often, in more temperate waters. Two fish that sting in a similar manner are the Pacific ratfish and the Atlantic toadfish.

Zebrafish (lionfish, turkeyfish) have been imported to the United States to dwell in home aquariums. They have now been spotted in the Atlantic Ocean from North Carolina to Florida.

The scorpionfish proper (Scorpaena) are found on the tip of southern Florida, in the Gulf of Mexico, off the coast of southern California, and in Hawaii.

Stonefish are not indigenous to U.S. waters.

Venom is associated with venom glands in the 12 dorsal, 2 pelvic, and 3 anal spines. The ornate, plumelike, and beautiful pectoral fins of these fish are nonvenomous. Each venomous spine is covered with an integumentary sheath that covers a hollow groove on the anterolateral portion of the spine. When the fish is handled, or more often, stepped on, it erects the dorsal spine and flares the pectoral and anal fins. If provoked in the water, the stonefish may attack.[30,31]

As with stingray injuries, the majority of injuries occur when a person steps on the fish. Most reported envenomations in the United States result in painful local wounds. Severe systemic effects and fatalities are rare.

The patient experiences immediate excruciating pain at the site, which quickly spreads to the entire limb and regional lymph nodes. The pain peaks at 90 minutes and may last up to 12 hours. Mild subsequent pain can persist for weeks. Occasionally, vesicles form in the region of the original puncture wound. This may be followed by skin sloughing, cellulitis, and surrounding hypesthesia.

Systemic symptoms include nausea, muscular weakness, respiratory distress, and hypotension.

Treatment. Remove the victim from the water.

Remove any visible spines immediately to prevent further penetration and breakage.

Immerse the affected limb into nonscalding water (45°C, 113°F) for 30-90 minutes.

Provide pain relief with local anesthesia using lidocaine 1-2% or bupivicaine 0.25-0.5% without epinephrine. Regional anesthesia also may be effective. Systemic narcotics may be needed for pain control, especially if the envenomation is in an area that is difficult to immerse in hot water.

After immersion, explore the wound in a sterile environment. Spine fragments in critical areas such as joints, weight-bearing surfaces, and in proximity to nerves and vessels, usually should be removed.

Remove any foreign material directly with forceps then irrigate copiously with warm normal saline.

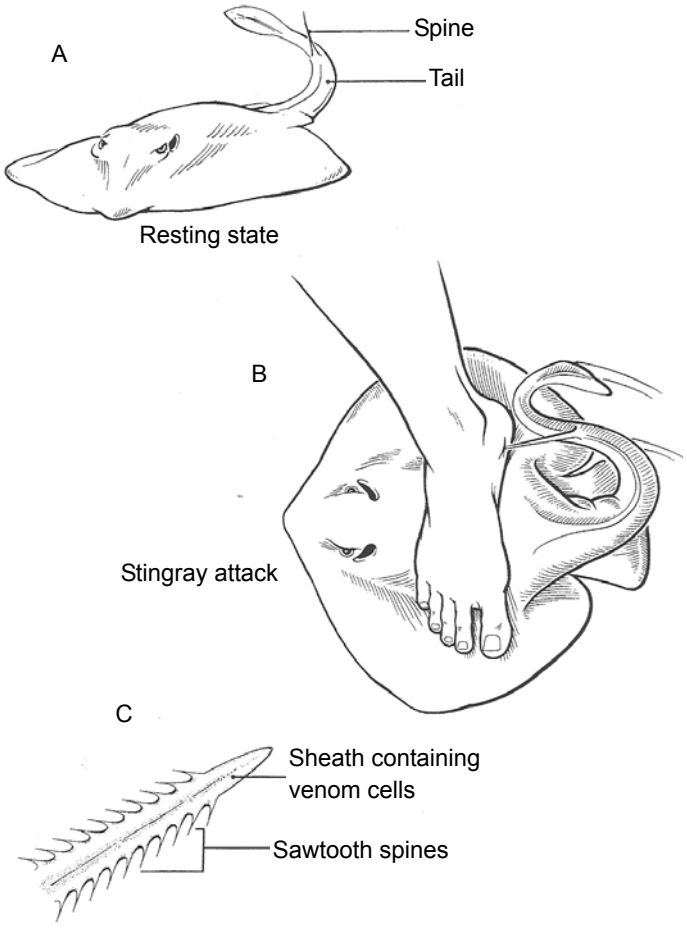
Wide excision usually is not necessary. It has been required in cases of severe extensive necrosis associated with stonefish envenomation.

Provide tetanus prophylaxis.

Prophylactic antibiotics are needed if the wound is very deep or involves the hand or foot. Appropriate antibiotics include oral doxycycline 100 mg twice daily, tetracycline 500 mg four times daily, trimethoprim-sulfamethoxazole double-strength twice daily, or ciprofloxacin 500-750 mg twice daily.

Stonefish Antivenom. There is antivenom available from the Australian Venom Research Unit, Department of Pharmacology, University of Melbourne, Parkville, Vic 3052, Australia. Telephone: 61-3-934447753. The antivenom is no longer available at Sea World in San Diego. Regional poison centers may be of

Figure 2. Stingray Injuries



A, Stingray resting on the bottom of the ocean, usually covered by a layer of sand. An unsuspecting victim steps on the stingray, and the whiplike tail impales the foot (even through a heavy boot) with one or more spines (B). The spine has backward-facing barbs covered by a sheath with venom-containing cells (C), causing a toxic envenomation and the potential for multiple foreign bodies.

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value in locating the closest supply of antivenom.

The antivenom is hyperimmunized equine-derived antisera, so there are risks of allergic reaction and serum sickness in the recipient. Skin testing has traditionally been recommended prior to administration. Australian sources recommend pretreatment rather than skin testing to save time.[32] Subcutaneous epinephrine and an intramuscular antihistamine are recommended, followed by an intramuscular corticosteroid for known hypersensitivity. If the patient has a history of hypersensitiviy to equine products, the risk of the antivenom must be weighed carefully against the risk of the envenomation.

Following premedication, administer the antivenom as follows: 1 ampule (2000 U) IV for every 1-2 punctures, up to 3 ampules for more than 4 punctures.

Slow IV administration requires dilution in 50-100 cc of normal saline and administration over at least 20 minutes.

The Commonwealth Serum Laboratories (CSL) stonefish antivenom is recommended only for prediluted IM use. This route may not be ideal in serious envenomations because of erratic and unpredictable absorption.[33,34]

Sea Snakes. Sea snakes are probably the most abundant reptiles on earth, and all of the 52 species are venomous. Fortunately, Hawaii is the only state in the United States that has sea snakes (*Pelamis platurus*).

Sea snakes have two to four hollow maxillary fangs and a pair of associated venom glands. The proteinaceous venom is highly toxic and contains stable neurotoxins that are often more potent than those of terrestrial snakes.

The patient is usually in the water or handling nets when the snake strikes. The bite site may exhibit the traditional fang marks, but these are often overlooked initially since there is little or no local pain on envenomation. Symptoms can develop in as little as 5 minutes, but typically evolve over 8 hours. Symptoms include euphoria, malaise, anxiety, muscle aching, sialorrhea, and a "thick tongue." Ascending flaccid or spastic paralysis ensues, followed by nausea, vomiting, myoclonus, muscle spasm, ophthalmoplegia, trismus, and bilateral painless swelling of the parotid glands.

Severe systemic envenomations develop symptoms rapidly over 2 hours. Victims complain of loss of vision, then become cool and cyanotic. Acute renal failure, respiratory failure, and coma ensue. The mortality rate is 25% untreated and 3% overall.

Treatment. Remove the victim from the water.

Immobilize the wound and keep the victim as calm as possible. The pressure-immobilization technique should be used.[35]

Sea snake antivenom is required if envenomation is present or highly suspected.

Intensive care management is necessary for sea snake envenomations. Respiratory collapse secondary to paralysis is anticipated, and endotracheal intubation and mechanical ventilation will likely be needed.

Treat myoglobinuria by alkalinizing the urine with sodium bicarbonate and a loop diuretic. Progressive nephropathy may require hemodialysis.

Repeat electrolyte monitoring will guide the administration of fluids and electrolyte supplements. Hyperkalemia secondary to rhabdomyolysis and renal failure should be anticipated and treated aggressively.

If the patient has no symptoms of systemic envenomation during the emergency department visit, observe for at least 8-12 hours before release.

Conclusion

Evaluation and management of the serious bite and envenomation injuries from marine animals can be summarized by some general key principles. For bite wounds, the basic principle is appropriate wound care and antibiotics directed against expected pathogens. For most envenomations, immersion in warm water will provide local relief. Many envenomations may have residual venom-containing objects in the wound that require appropriate removal. Finally, some envenomations may have serious systemic effects that necessitate a period of observation and cardiopulmonary monitoring.

References

1. Auerbach PS ed. *Wilderness Medicine*, 5th edition. Mosby-Elsevier 2007:1654-1772.
2. Moscati RM, Mayrose J, Reardon RF, et al. A multicenter comparison of tap water versus sterile saline for wound irrigation. *Acad Emerg Med* 2007;14:404-409.
3. Fernandez R, Griffiths R. Water for wound cleansing. *Cochrane Database Syst Rev* 2002;(4):CD003861.
4. Reed KC. Skin and soft-tissue infections after injury in the ocean: Culture methods and antibiotic therapy for marine bacteria. *Military Medicine* Mar 1999.
5. Myers JP. Skin and soft tissue infections and envenomations acquired at the beach. *Current Infectious Disease Reports* 2006,8:394-398.
6. Dept of Health and Human Services, Centers for Disease Control and Prevention. Guidance for management of wound infections. Issued Sept 6, 2005. Retrieved from www.bt.cdc.gov/disasters.
7. Auerbach PS. *A Medical Guide to Hazardous Marine Life*, ed. 3. Flagstaff, AZ; Best Publishing: 1997.
8. Johnson RA. Bacterial skin and soft tissue infections. *Curr Opin Infect Dis* 1992.
9. Shapiro RL, Altekruse S, Hutwagner L, et al. The role of Gulf Coast oysters harvested in warmer months in *Vibrio Vulnificus* infections in the United States, 1988-1996. *J Infect Dis* 1998;178:752-759.
10. Centers for Disease Control. Vibrio vulnificus infections associated with eating raw oysters. Los Angeles 1996. *MMWR* 1996:45:621-624.
11. Centers for Disease Control. Vibrio vulnificus infections associated with raw oyster consumption: Florida, 1981-1992. *MMWR* 1993;42:405-407.
12. Klontz KC, Lieb S, Schreiber M, et al. Syndromes of *Vibrio vulnificus* infections. Clinical and epidemiologic features in Florida cases, 1981-1987. *Ann Intern Med* 1988;109: 318-323.

13. Kargel JS, Dimas VM, Kao DS, et al. Empiric antibiotic therapy for seawater injuries: A four-seasonal analysis. *Plastic & Reconstructive Surgery* 2008;121:1249-1255.
14. Brown TP. Diagnosis and management of injuries from dangerous marine life. *Med Gen Med* 2005;7:5.
15. Fenner P. Marine envenomation: An update–A presentation on the current status of marine envenomation first aid and medical treatments. *Emergency Medicine, Tropical Emergency Medicine Series* 2000;12:295-302.
16. Nimorakiotakis B, Winkel KD. Marine envenomations. Part 1–Jellyfish. *Aust Fam Physician* 2003;32:969-974.
17. Nomura JT, Sato RL, Ahern RM, et al. A randomized paired comparison trial of cutaneous treatments for acute jellyfish (*Carybdea alata*) stings. *Am J Emerg Med* 2002:20:624-626.
18. Bowra J, Gillet M, Morgan J, et al. A trial comparing hot showers and icepack in the treatment of physalia envenomation [abstract]. *Emerg Med* 2002;14:A22.
19. Thomas CS, Scott SA, Galanis DJ, et al. Box jellyfish (*Carybdea alata*) in Waikiki: Their influx cycle plus the analgesic effect of hot and cold packs on their stings to swimmers at the beach: A randomized, placebo-controlled, clinical trial. *Hawaii Med J* 2001;60:100-107.
20. Loten C, Scokes B, Worsley D, et al. A randomized controlled trial of hot water (45º C) immersion versus ice packs for pain relief in blue bottle stings. *Med J Aust* 2006;184: 329-333.
21. Atkinson PRT, Boyle A, Hartin D, et al. Is hot water immersion an effective treatment for marine envenomation? *Emerg Med J* 2006;23:503-508.
22. Cruz LJ. Conotoxins. In: Spencer PS, Schaumburg HH, eds. *Experimental and Clinical Neurotoxicology*, 2nd ed. Oxford: Oxford University Press; 2000:417-419.
23. Watters MR, Cannard KR. Marine neurotoxins. In: Chopra JS, Sawhney IMS, eds. *Neurology in Tropics*. New Delhi: Churchill Livingstone; 1999:45-68.
24. Watters MR, Yanagihara AA. Marine neurotoxins: envenomations and contact toxins. In: Watters MR, ed. *Marine Toxins*, AAN Syllabus 5BS.003, St Paul, MN: American Academy of Neurology; 2003:1-27.
25. Fenner P. Dangers in the ocean: The traveler and marine envenomation. II. Marine vertebrates. *J Travel Med* 1998; 5:213-216.
26. Diaz JH. The evaluation, management, and prevention of stingray injuries in travelers. *J Travel Med* 2008;15:102-109.
27. Baldinger P. Treatment of stingray injury with topical becaplermin gel. *J Am Podiatr Med Assoc* 1999;89:531-533.
28. Fenner PJ, Williamson JA, Skinner RA. Fatal and non-fatal stingray envenomation. *Med J Aust* 1989;151: 621-625.
29. Weiss BF, Wolfender HD. Survivor of a stingray injury to the heart. *Med J Aust* 2001;175:33-34.
30. Brenneke F, Hatz C. Stonefish envenomation–a lucky outcome. *Travel Med Infect Dis* 2006;4:281-285.
31. Bedry R, de Haro L. Venomous and poisonous animals. IV. Envenomations by venomous aquatic vertebrates. *Med Trop* (Mars) 2007;67:111-116.
32. Winkel KD, Mirtschin P, Pearn J. Twentieth century toxinology and antivenom development in Australia. *Toxicon* 2006; 48:738-754.
33. Lyon RM. Stonefish poisoning. *Wilderness Environ Med* 2004;15:284-288.
34. Sutherland SK. Antivenom use in Australia. Premedication, adverse reactions and the use of venom detection kits. *Med J Aust* 1992;157:734-739.
35. Singletary EH, Adam SR, Bodmer JCA. Envenomations. *Med Clin North Am* 2005;89:1195-1224.

Physician CME Questions

101. The first task to accomplish when treating a patient with a marine injury is:
    A. irrigate the wound.
    B. apply a sterile dressing.
    C. remove the patient from the water.
    D. start an IV.

102. The antibiotic of choice for seal finger (also known as spekk finger or blubber finger) is:
    A. tetracycline 1.5 g initially, followed by 500 mg four times daily for 4-6 weeks.
    B. keflex 500 mg four times daily for one week.
    C. trimethoprim/sulfamethoxazole twice daily for 2 weeks.
    D. clindamycin 300 mg twice daily for 10 days.

103. In Florida, mortality from alligator attacks is about twice that of shark attacks.
    A. True
    B. False

104. The most common lacerations occurring under water are due to:
    A. giant manta rays.
    B. true stony corals.
    C. giant clams.
    D. colossal squid.
    E. killer whales.

105. A 47-year-old man returns from a diving trip to Cozumel with an injury to the right index finger. The wound is a puncture wound, with a small area of erythema and a purplish discoloration of the surrounding skin. The emergency physician obtains an x-ray to rule out the presence of a foreign body, and it is negative. What is the most likely source of this wound?
    A. A blue ink pen
    B. A shark
    C. An octopus
    D. A sea urchin

106. Which of the following contains holothurin and may cause dermatitis, conjunctivitis, or cardiac complications?

Emergency Medicine Reports
CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

A. An octopus
B. A giant manta ray
C. A sea cucumber
D. A sea urchin

107. A 21-year-old female presents with an erythematous, intensely painful rash of the dorsum of the right hand. She states that this started after she cleaned her home aquarium. What is the most likely cause of the rash?
A. The bristle worm
B. A piranha
C. Charcoal from the filter
D. A goldfish

108. Which of the following has a specific antivenom?
A. The cone shell
B. The box jellyfish
C. The octopus
D. The stingray

109. There are no venomous fish in the coastal waters of the United States.
A. True
B. False

110. There is extensive scientific evidence about the use of prophylactic antibiotics to treat all marine injuries.
A. True
B. False

CME Answer Key

101. C
102. A
103. A
104. B
105. D
106. C
107. A
108. B
109. B
110. B

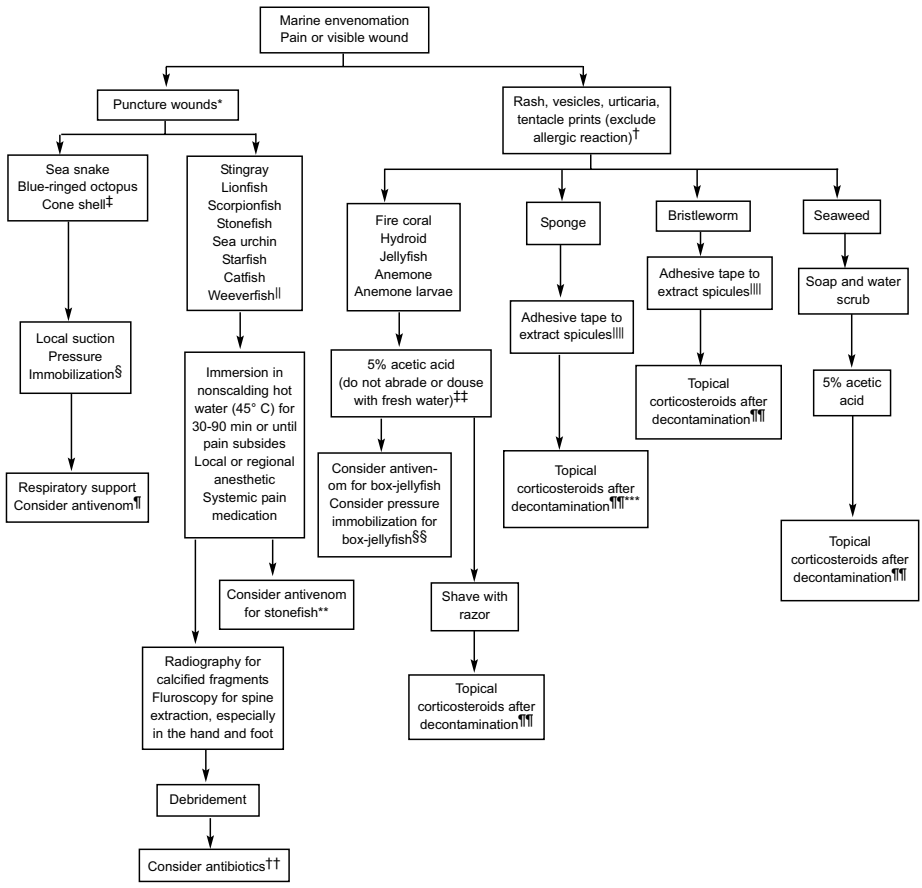
In Future Issues:

Head Injury

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Marine Animal-Acquired Injuries

Algorithmic Approach to Marine Envenomation



* A gaping laceration, particularly of the lower extremity, with cyanotic edges suggests a stingray wound. Multiple punctures in an erratic pattern with or without purple discoloration or retained fragments are typical of a sea urchin sting. One to eight (usually two) fang marks are usually present after a sea snake bite. A single puncture wound with an erythematous halo and rapid swelling suggests scorpionfish envenomation. Blisters often accompany a lionfish sting. Painless punctures with paralysis suggest the bite of a blue-ringed octopus; the site of a cone shell sting is punctate, painful, and ischemic in appearance.

† Wheal and flare reactions are nonspecific. Rapid (within 24 hours) onset of skin necrosis suggests an anemone sting. "Tentacle prints" with cross-hatching or a frosted appearance after application of aluminum-based salts suggest a box-jellyfish (*Chronex fleckeri*) envenomation. Ocular or intraoral lesions may be caused by fragmented hydroids or coelenterate tentacles. An allergic reaction must be treated promptly.

‡ Sea snake venom causes weakness, respiratory paralysis, myoglobinuria, myalgias, blurred vision, vomiting, and dysphagia. The blue-ringed octopus injects tetrodotoxin, which causes rapid neuromuscular paralysis.

§ As soon as possible, venom should be sequestered locally with a proximal venous-lymphatic occlusive band of constriction, or (preferably) by the pressure-immobilization technique, in which a cloth pad is compressed directly over the wound by an elastic wrap that should encompass the entire extremity at a pressure of 9.33 kPa (70 mmHg) or less. Incision and suction are not recommended.

¶ Early ventilatory support has the greatest influence on outcome. The minimal initial dose of sea snake antivenom is one to three vials; up to 10 vials may be required.

‖ The wounds range from large lacerations (stingrays) to minute punctures (stonefish). Persistent pain after immersion in hot water suggests a scorpionfish or stonefish sting or a retained fragment of spine. The puncture site can be identified by forcefully injecting 1% to 2% lidocaine or another local anesthetic agent without epinephrine near the wound and observing the egres of fluid. Do not attempt to crush the spines of sea urchins if they are present in the wound. Spine dye from already-extracted sea urchin spines will disappear (be absorbed) in 24 to 36 hours.

** The initial dose of stonefish antivenom is one vial per two puncture wounds.

†† The antibiotics chosen should cover *Staphylococcus*, *Streptococcus*, and microbes of marine origin, such as *Vibrio*.

‡‡ Acetic acid (5%; i.e., vinegar) is a good all-purpose decontaminant and is mandated for the sting from a box-jellyfish. Alternatives, depending on the geographic region and indigenous jellyfish species, include isopropyl alcohol, bicarbonate (baking soda), ammonia, papain, and preparations containing these agents.

§§ The initial dose of box-jellyfish antivenom is one ampule intravenously or three ampules intramuscularly.

¶¶ If inflammation is severe, steroids should be given systematically (beginning with at least 60 to 100 mg of prednisone or its equivalent), and the dose should be tapered over a period of 10 to 14 days.

‖‖ An alternative is to apply and remove commercial facial peel materials.

*** An alternative is to apply and remove commerical facial peel materials followed by topical soaks of 30 mL of 5% acetic acid (vinegar) diluted in 1L of water for 15 to 30 minutes several times a day until the lesions begin to resolve. Anticipate surface desquamation in 3 to 6 weeks.

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Tetanus Prophylaxis

	Clean Minor Wounds		Dirty Major Wounds	
HISTORY OF IMMUNIZATION (DOSES)	**TOXOID***	**TIG†**	**TOXOID**	**TIG**
Unknown	Yes	No	Yes	Yes
None to one	Yes	No	Yes	Yes
Two	Yes	No	Yes	No (unless wound older than 25 hr)
Three or more				
Last booster within 5 years	No	No	No	No
Last booster within 10 years	No	No	Yes	Yes
Last booster more than 10 years ago	Yes	No	Yes	Yes

* Toxoid: Adult: 0.5 mL dT intramuscularly (IM).
Child less than 5 years old: 0.5 mL DPT IM.
Child older than 5 years: 0.5 mL DT IM.

† Tetanus immune globulin (TIG): 250-500 units IM in limb contralateral to toxoid.

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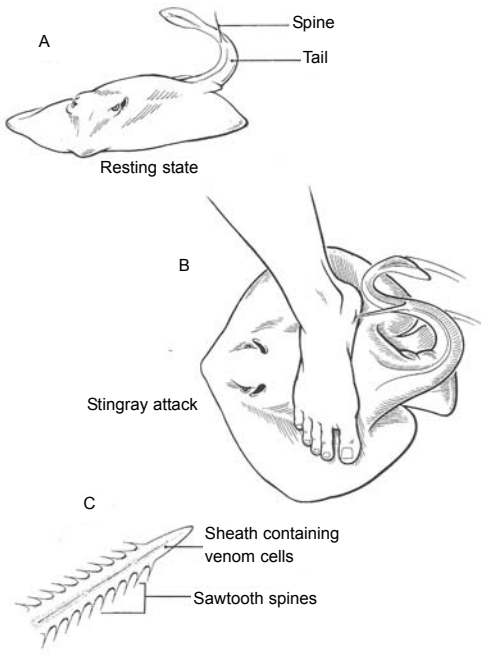
Stingray Injuries



A, Stingray resting on the bottom of the ocean, usually covered by a layer of sand. An unsuspecting victim steps on the stingray, and the whiplike tail impales the foot (even through a heavy boot) with one or more spines (B). The spine has backward-facing barbs covered by a sheath with venom-containing cells (C), causing a toxic envenomation and the potential for multiple foreign bodies.
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Supplement to *Emergency Medicine Reports*, November 10, 2008: "Creatures of the Deep: Marine Animal-Acquired Injuries and Envenomations." *Authors:* **Angela F. Gardner, MD, FACEP**, Assistant Professor, Department of Surgery/Emergency Medicine, University of Texas Medical Branch, Galveston; and **Paul Auerbach, MD, MS, FACEP, FAWM**, Professor of Surgery, Division of Emergency Medicine, Stanford University School of Medicine, Palo Alto, CA.

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Current Management of Splenic Injury

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The spleen is the most frequently injured organ following blunt abdominal trauma.[1] The high incidence of splenic injury has demanded continuous reappraisal of our understanding of splenic function, as well as our approach to its management. Habitual splenectomy following trauma initially gave way to operative salvage via splenorrhaphy. The practice of splenorrhaphy was largely influenced by the recognition of the risk for post-splenectomy sepsis following splenectomy.[2-4] Techniques of surgical splenorrhaphy evolved as surgeons learned how to handle the injured splenic tissue more effectively.[5] Nonoperative management evolved in hemodynamically stable children and adults who had CT imaging studies to determine the presence of organ injury.[6-8] Guidelines evolved for the management of these organ injuries that utilized a standard scoring system first articulated by the American Association for the Surgery of Trauma.[9] The addition of angiography plus embolization completed the repertoire[10] that today can yield splenic salvage success rates of 80% or greater in both adults and children, depending on grade of injury.[6,10]

The spectrum of current splenic management, stretching from nonoperative care to splenectomy, has developed along with the concept that splenic function should be preserved whenever possible. Recognition of the existence of certain conditions that continue to require splenectomy compel medical personnel to evaluate the entire clinical presentation of each patient, balancing the desire to save splenic function with the need to avoid the potential disaster of splenic hemorrhage. The decision-making process of care following splenic injury ultimately begins with an individualized approach to each patient based upon presentation, stability, concurrent injuries, and practitioner experience. Education of those who treat patients with splenic trauma is imperative, and studies designed to improve the outcomes of splenic management

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in trauma centers should continue to be employed to further our progress in this area.

— The Editor

Introduction

As noted previously, the spleen is the most frequently injured organ following blunt abdominal trauma.[1,11] Prompt recognition and appropriate scoring of splenic injury is vital for preventing the potentially disastrous complication of hemorrhage and hypovolemic shock. However, due to the well-recognized risks for post-splenectomy sepsis,[2-4] routine splenectomy for injury has given way to the practice of splenic salvage, utilizing techniques of splenorrhaphy (repair of splenic laceration or rupture with sutures), nonoperative management of solid organ injury, and angiography with embolization, either singularly or in combination.[5-8,10] Reports of salvage results, adjusted for the grade of injury severity, demonstrate significant success rates in both adults and children, with the overall rate approaching 80%.[6,10]

Despite the predominantly favorable statistics that have arisen in support of methods of splenic salvage, debates concerning these techniques have occurred. Criteria for decision-making between nonoperative and surgical treatment, imaging options, and follow-up care all have been subject to numerous studies and discussions. The current approach to the management of splenic injury will be reviewed in this issue, with the aid of these previous studies.

Initial Evaluation and Resuscitation

For a patient who is the victim of a traumatic injury, the importance of initial evaluation and resuscitation efforts cannot be overstated. Expeditious transportation to a medical facility with appropriate trauma verification as well as assessment and resuscitation by first responders are key initial steps to achieving a positive outcome for the patient.

The primary survey and initial stabilization should be followed during the evaluation of a traumatically ill patient. Cervical and spinal stability should be maintained throughout the survey. The airway must be patent to ensure a pathway for adequate oxygenation. If a clear airway cannot be established, endotracheal intubation or a surgical airway may need to be performed. Breathing and ventilation efforts should be examined and supported if necessary, with supplemental oxygen being provided for every injured patient.[1] Circulatory status should be confirmed next through pulse checks, skin color, and level of consciousness. Two large-bore peripheral intravenous lines should be established to allow for administration of fluids and retrieval of blood for labs. Disability, or the neurologic state, must be appraised using the Glasgow Coma Scale (GCS). Finally, the patient must be fully exposed to gain a complete examination. However, care with blankets and a warm environment must be taken to protect against hypothermia.

When a patient presents with an abdominal injury, the diagnosis may not be readily evident. Thus, the crucial step in the first phase of patient management is to consider such an injury. Pertinent information regarding the mode of injury should be collected. Following the ABCs, the trauma responder should commence with a thorough physical examination. A complete abdominal assessment should consist of examination for ecchymosis, lacerations, distention; auscultation for normoactive bowel sounds; percussion for dullness, which would suggest intraperitoneal fluid or hemoperitoneum, and hypertympanic regions, which would denote dilatation; and palpation to assess for voluntary and involuntary guarding, rebound tenderness, and abnormal masses.[1] Though a patient may pass this portion of the complete examination without raising suspicion for abdominal trauma, it is imperative that repeat examinations be performed; a significant number of patients with hemoperitoneum are noted to have had "normal" initial abdominal findings.[12] If the initial physical exam is normal, yet the suspicion is high for an abdominal injury, further workup is warranted.

Splenic injury, like abdominal injury in general, is not always apparent. However, there are "classic" findings that should alert trauma responders to the possibility of damage to the spleen. Left upper quadrant tenderness and ecchymosis or left lower chest tenderness may be manifestations of splenic injury. Left lower rib fractures also should raise the consideration of splenic trauma. Injuries to other organs, such as the liver or left kidney, increase the possibility of splenic injury, with thoracic injuries being the most concomitant.[13] Kehr's sign (pain referred to the left shoulder following splenic injury) or Balance's sign (dullness to percussion in the left upper quadrant) may be found. It is important to remember that splenic trauma also may present with nonspecific signs, such as hypotension. Again, the consideration of the possibility of splenic injury often is paramount to the initial management and eventual diagnosis.

The hemodynamic status of a patient ultimately determines the approach that is taken upon presentation. Hemodynamically unsta-

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ble patients first require prompt establishment of airway, breathing, and circulation. Evaluation and resuscitation occur simultaneously. A trauma patient with hypotension is considered to be hypovolemic until proven otherwise, and a search for hemorrhage must take place. If the abdomen is the obvious area of blood loss, emergent operative exploration must occur. If the clinician is unable to confirm abdominal injury, yet the patient is unstable, a diagnostic peritoneal lavage or abdominal ultrasound may aid in the decision regarding whether to perform a laparotomy.[13] A patient who is hemodynamically stable should undergo evaluation of the ABCs, as well as resuscitation as needed. Imaging studies, such as CT scans, may then be completed to determine their specific injuries. Thus, the hemodynamic stability of a patient presenting with abdominal trauma will play the most significant role in guiding the trauma physician to choose an appropriate plan of action.

Organ Injury Scoring

Following the initial resuscitation and evaluation of patients with abdominal trauma, the decision-making process regarding management strategies must be employed. As has already been noted, certain physical presentations should spur the healthcare provider to choose the route of emergent laparotomy over other evaluative modalities. However, for the majority of patients with abdominal trauma, the presenting picture may be less clear. To facilitate a more uniform approach to these patients, the American Association for the Surgery of Trauma (AAST) formed the Organ Injury Scaling (OIS) Committee in 1987 to create a scoring system tailored to individual organs. A revised organ injury scale for the spleen was adopted in 1995;[9] it is based on the size, degree, and depth of lacerations and/or hematomas in the spleen observed at surgery.

In their revision, the OIS Committee stated that the purpose of scaling an organ injury is to allow for a better comparison between different treatment strategies for a comparable injury.[9] While prognosis cannot be determined based on the splenic injury scale, it does aid physicians in choosing treatment options. For example, patients with Grade 5 splenic injuries often are managed by splenectomy.[13] A subsequent splenic injury scale was devised in 2007 that utilizes multidetector CT images;[14] it will be discussed shortly.

The splenic injury scale provides medical personnel with a systematic approach to determine the extent of damage, and is a component in deciding the next appropriate course of action. Imaging studies are utilized to assign a splenic injury score, as well as to diagnose and treat injuries in select cases. These options are an invaluable extension of the initial physical examination in judging the extent of traumatic abdominal injury.

Imaging Options

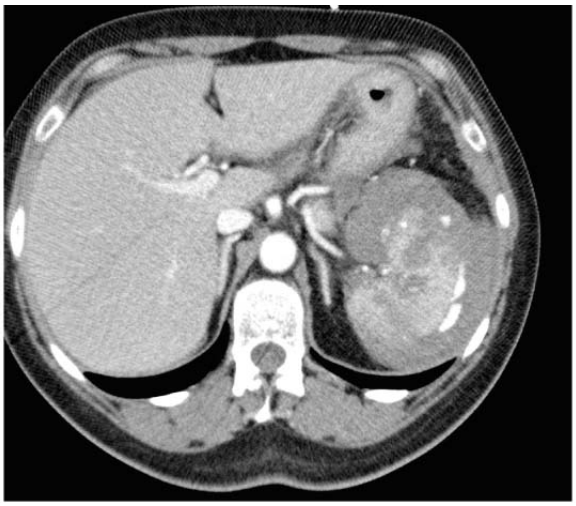
Along with physical examination, there are three major tools employed in the assessment of blunt abdominal trauma: diagnostic peritoneal lavage (DPL), computed tomography (CT), and focused abdominal sonography for trauma (FAST). DPL has been used for more than 40 years as a rapid and accurate method to confirm the presence of traumatic intraperitoneal hemorrhage, with an accuracy of 92-98%.[15] The greatest impact of DPL is in expediting unstable trauma patients to emergent laparotomy. Limitations of DPL include significant false-positive cases, the poor sensitivity of visual inspection of fluid, and its limited usefulness in hemodynamically stable patients. FAST scans are noninvasive, portable, can be done concurrently with resuscitation efforts, and have high accuracy for detecting free intraperitoneal fluid (96-98%).[15] Emergent exploratory laparotomy also is indicated in unstable patients with positive FAST scans.

Hemodynamically stable patients with equivocal physical examination, FAST, or DPL results are best managed by CT scan. CT also is the diagnostic modality of choice for nonoperative management of solid organ trauma and for serial surveillance of injuries in stable patients. CT scans require a reasonably cooperative patient who is hemodynamically stable and can be transported to the radiology suite. The accuracy of CT scan in stable abdominal trauma patients is quite high. CT has the ability to detect clinically unsuspected injuries, such as retroperitoneal injuries, that may modify treatment plans. The major limitations to CT scan are in detection of mesenteric and hollow-organ injuries and the potential for adverse events related to contrast media administration.

Abdominal CT scan is considered to be a sensitive, specific, and accurate modality for delineating the extent and severity of traumatic injury to the liver and spleen.[16] CT scoring systems were proposed for grading blunt splenic injury in the late 1980s,[16-18] correlating the CT features with the common surgical observations of traumatized spleens. CT grading systems were recognized as being fairly accurate in depicting the extent of splenic injury and degree of hemoperitoneum compared with surgical observations in these studies. However, the CT grade was deemed unreliable as a prognostic factor for predicting the success of nonoperative management, with a sizable percentage of high-grade splenic injuries in hemodynamically stable patients successfully managed nonoperatively and some low-grade splenic injuries still requiring laparotomy and splenectomy.

By the late 1990s, several investigators tried to understand why, in hemodynamically stable patients selected for nonoperative management, the grade of splenic injury or degree of hemoperitoneum on CT scan did not predict the outcome of successful nonoperative management. Gavant et al[19] retrospectively analyzed 263 patients with blunt splenic injuries, 82 of whom underwent immediate emergent surgery and 181 of whom were stable enough to be evaluated with emergent CT. Of the 181 CT scan patients, 72 were treated nonsurgically. Eleven of these patients failed nonsurgical therapy, 9 (82%) of whom had active extravasation or the presence of a traumatic pseudoaneurysm. The authors of the study concluded that the failure rate for nonoperatively managed blunt splenic trauma may be markedly reduced if patients with active extravasation or traumatic pseudoaneurysms are treated with emergent surgical or endovascular repair. Federle et al[20] performed a retrospective study of 270 splenic injury patients, 150 of whom underwent rapid, dynamic, bolus contrast-enhanced CT scans, with 100 selected for nonoperative management. Of the 96 patients who had no evidence of active extravasation, 83 recovered without surgery or other intervention, and the splenic salvage rate among the 100 patients selected for nonoperative management was 92%. The authors of this study con-

Figure 1. Blunt Splenic Injury, Grade 5, with Active Extracapsular Extravasation



cluded that the absence of active extravasation was an important predictor of successful nonoperative management. Further studies[21-23] defined the appearance of active extravasation *(see Figure 1)*, and defined a role for delayed phase CT scans to differentiate active splenic hemorrhage from contained vascular injuries, and reinforced the detection of active hemorrhage as indicating the need for emergent operative or embolization therapy.

How good is modern-day multidetector CT (MDCT) with bolus intravenous contrast for detecting active arterial bleeding? This question was addressed in a 2007 article by Roy-Choudhury et al[24] that compared the detection of active extravasation in a physiologic flow phantom using a 16-slice MDCT and digital subtraction angiography (DSA). The accepted rate of bleeding required for detection on angiography is 1 mL/min, demonstrated in experimental celiac and mesenteric angiographic studies in dogs in the 1960s.[25] Roy-Choudhury et al found in their model that a simulated first order aortic branch selective DSA study detects hemorrhage at 0.96 mL/min, verifying the earlier animal studies. However, a simulated intravenous contrast-enhanced MDCT study was more sensitive than DSA, detecting hemorrhage at 0.35 mL/min. MDCT with a simulated first order arterial branch contrast administration and simulated superselective DSA with the catheter tip right at the bleeding site both can detect active hemorrhage at rates < 0.05 mL/min. The authors concluded that intravenous bolus contrast-enhanced MDCT can be used as the initial imaging technique to diagnose active arterial bleeding in hemodynamically stable patients with suspected splenic injury. Marmery et al[14] proposed a modified MDCT grading system in 2007 targeted at optimizing the selection of patients for nonoperative management. This grading system incorporates key features of the AAST system with respect to the splenic parenchymal injuries, but elevates the presence of active arterial bleeding and direct splenic vascular injuries to the highest grade (Grade 4), with recommendation for splenic surgery or angiography and embolization *(see Table 1)*. ROC analysis of the modified MDCT grading system and the AAST grading system showed an improvement in the discrimination of patients requiring embolization or surgery with the modified MDCT grading system.

CT scans have greatly contributed to the changing strategies of splenic management. Their ability to recognize minor injuries and aid in the scoring of splenic injuries has allowed the continuum of care to shift from routine splenectomy to operative splenic salvage and nonoperative management. The risk of overwhelming post-splenectomy sepsis[2-4] has further contributed to the increased usage of salvage techniques, of which nonoperative management has been perhaps the most controversial.

Nonoperative Management

Nonoperative management in the care of the injured spleen has undergone intense scrutiny as researchers have attempted to determine which criteria should be utilized to prescribe a patient to more conservative treatment. Various parameters, such as advanced age and neurologic status, which were first thought to be indicative of the potential outcome of nonoperative management, have since been contested. The value of routine repeat abdominal CT scanning also has been debated, with the current practice supporting the side of decision-making based on clinical progression.

During the 1980s, studies that approached nonoperative management using strict guidelines for patient selection resulted in varying success rates.[7] Cogbill et al[6] suggested that hemodynamic stability, the absence of other considerable abdominal organ injuries, and a full abdominal examination in the absence of other confounding factors would allow nonoperative treatment of Grades 1 through 3 splenic injuries with initial success rates of greater than 80% in adults. Smith et al[7] added to this criteria in 1992 with their study, which achieved a 93% nonoperative management success rate by excluding patients older than age 55 years. Hunt et al[26] found that the majority of adult patients with an Injury Severity Score (ISS) of less than or equal to 15 could be treated nonoperatively for their splenic injuries during a five-year study. However, it would not be long before some of these guidelines were challenged.

The criteria that suggested age older than 55 is adverse contributor to the success of splenic nonoperative management has been tested. In their study, which resulted in an overall 98% nonoperative success rate, Pachter et al[8] reported a 100% nonoperative success rate among the 17 patients included who were older than age 55. Similarly, Gaunt et al[11] found no statistical difference with respect to age between a group of patients who were successfully managed nonoperatively versus a group of patients who required immediate surgical exploration. They also reported that among the patient group that failed nonoperative management, all were younger than age 55 years. Subsequent studies[27] have provided further evidence in support of allowing patients over the age of 55 to undergo nonoperative management on a case-by-case basis.

Other parameters also have been challenged, favoring the application of more nonoperative management plans. In the same study that assessed the significance of age in nonoperative outcomes, Gaunt et al[11] found no statistical difference with respect to the GCS score or systolic blood pressures between patients

Table 1. Proposed New Grading System Incorporating Splenic Vascular Injury*

Grade	Criteria
1	Subcapsular hematoma < 1 cm thick Laceration < 1 cm parenchymal depth Parenchymal hematoma < 1 cm diameter
2	Subcapsular hematoma 1- to 3-cm thick Laceration 1-3 cm in parenchymal depth Parenchymal hematoma 1-3 cm in diameter
3	Splenic capsular disruption Subcapsular hematoma > 3 cm thick Laceration > 3 cm in parenchymal depth Parenchymal hematoma > 3 cm in diameter
4a	Active intraparenchymal and subcapsular splenic bleeding Splenic vascular injury (pseudoaneurysm or arteriovenous fistula) Shattered spleen
4b	Active intraperitoneal bleeding

* Reproduced with permission from the *American Journal of Roentgenology.* Marmery H, Shanmuganathan K, Alexander MT, Mirvis SE. Optimization of selection for nonoperative management of blunt splenic injury: comparison of MDCT grading systems. *AJR Am J Roentgenol* 2007;189:1421-1427.

managed surgically versus nonoperatively. In their research on splenic trauma management, Pachter et al[8] found that 90% of patients admitted to the study with an ISS of greater than 15 successfully underwent nonoperative care. Furthermore, they suggested that CT scoring of splenic injury may not definitively predict the outcome of nonoperative treatment.

In view of the many studies that have been performed to aid the healthcare provider in correctly choosing patients who may successfully undergo nonoperative management, we suggest certain guidelines. First, it is prudent to consider the hemodynamic status of the patient, their response to fluids, and their risk of further bleeding. Next, the patient chosen for nonoperative care of the injured spleen should not have concomitant significant injury of another abdominal organ, due to the common association.[13] Finally, while injury scoring does not predict the patient's prognosis,[9] CT scanning may be used to assess the severity of injury, and in turn help the trauma team in deciding which course of treatment to follow.

A patient who is selected for nonoperative management is best cared for in the hospital setting. This will ensure that the patient attains bed rest and may be closely monitored through hemoglobin results and serial abdominal exams to determine their progression.[13] The necessity of routinely repeating abdominal CT scans has been contested,[28] with the current approach being to repeat the CT scan if the patient's condition remains unchanged or deteriorates. The exception in the routine usage of follow-up CT scanning is to clear patients with splenic trauma who wish to return to contact sports following their recovery.[8]

The recent success rates of nonoperative management parallel its popularity as a component of the treatment repertoire for patients with splenic injury. However, literature also has addressed the failures of nonoperative management. The presence of a "vascular blush" on an abdominal CT scan or a splenic artery pseudoaneurysm are two of the documented causes of nonoperative failure.[10] As a result, selective angiographic embolization has arisen as a treatment modality that may prevent a patient from failing nonoperative management.

Splenic Angiography and Embolization

Recognizing that direct arterial injuries and extravasation were associated with failure of nonoperative management, Sclafani et al,[29] in 1991, proposed that angiography could help differentiate those patients who could be reliably treated with bed rest alone from those who required some form of directed hemostasis. He further proposed that transcatheter splenic artery embolization could successfully gain hemostasis, and could function as an alternative to laparotomy. In his study of 44 hemodynamically stable splenic injury patients (including patients that rapidly stabilized with resuscitation), 17 patients were found to have active arterial extravasation on angiography, and they were managed with proximal (main) splenic artery embolization with coils, with successful control of hemorrhage in all patients. The overall splenic salvage rate of those in whom nonoperative management was attempted was 35/36 patients (97%).[29] This study established a role for transcatheter splenic artery embolization in the nonoperative management of blunt splenic injury.

Splenic artery embolization (SAE) is performed as an emergent interventional procedure in the early hospitalization phase, often within the first hour or two following presentation. These usually are hemodynamically stable or transiently stable patients. Immediately after the CT is reviewed, the decision about emergent splenic artery angiography and embolization is made, commonly because of CT evidence of contrast extravasation, vessel injury, pseudoaneurysm, arteriovenous fistula, or significant hemoperitoneum *(see Figure 2).* Additionally, a small percentage of nonoperative management patients who had more minor splenic injuries, without extravasation on initial CT, continue to show evidence of bleeding, with persistent transfusion requirement. These patients also are candidates for embolization, even several days after presentation, and SAE still can be performed in this time frame with good clinical results. Initially, selective splenic artery angiograms are performed. Abnormal angiographic findings include active extravasation; pseudoaneurysms; arteriovenous fistulas; abrupt vessel cut off; and an abnormal "pointillism" type parenchymal appearance *(see Figure 3),* which represents tiny dots and puddles of parenchymal contrast extravasation in regions of splenic contusion. If any of these features are present, there generally is indication for SAE. Proximal main SAE typically is performed in the mid splenic artery, usually distal to the dorsal pancreatic artery. Numerous conventional and microcatheter techniques are effective, and multiple embolic materials have been used; however, coil embolization and Gelfoam embolization predominate. If a pseudoaneurysm or AV (arteriovenous) fistula is found, superselective embolization may be considered *(see Figure 4)*. Following embolization, final angiograms are performed to confirm hemostasis. A follow-up CT scan is performed 24-48 hours post embolization.

Figures 2a-2d. Grade 3 Splenic Injury with Pseudoaneurysm

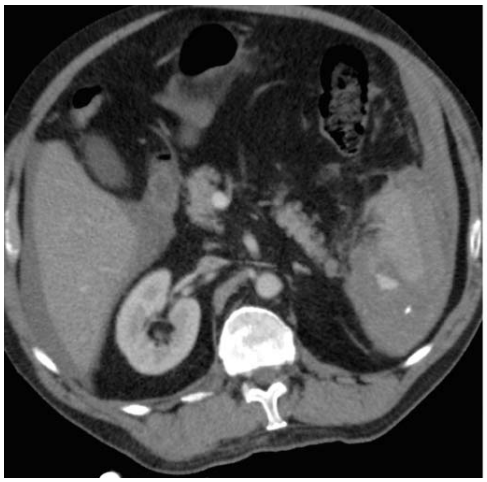


Figure 2a. MVA patient with grade 3 splenic injury, with abnormal intrasplenic contrast extravasation suggesting contained vessel rupture or pseudoaneurysm.

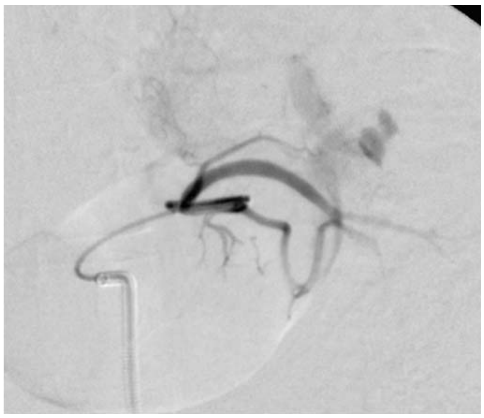


Figure 2b. Selective splenic artery angiogram, arterial phase, demonstrates a 2 centimeter, bilobular pseudoaneurysm with extravasation in the periphery of the spleen.

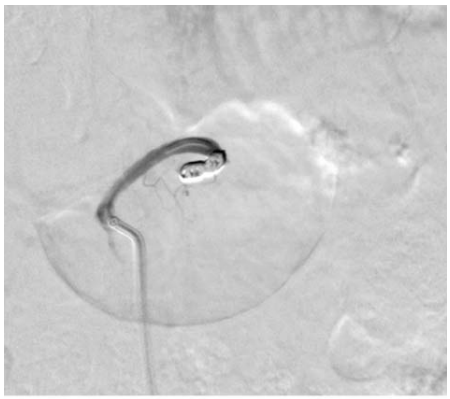


Figure 2c. Splenic artery angiogram following coil embolization of the distal main splenic artery shows cessation of primary arterial flow to the spleen.

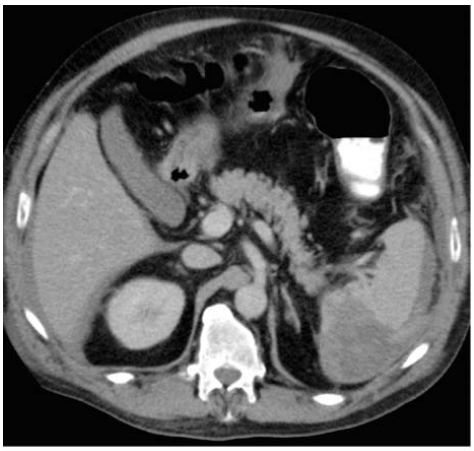


Figure 2d. CT scan 4 days following splenic embolization demonstrates resolution of the psuedoaneurysm, and no extravasation, with the resolving contusion and a component of minor splenic infarction in the embolized segment.

The rich collateral circulation of the spleen helps limit splenic infarction, and post embolization, the spleen is still functional reticuloendothelial system tissue. Hagiwara et al[30] studied 31 patients with blunt splenic injury: 3 who required emergent surgery, 13 who required bed rest alone, and 15 who were undergoing transcatheter arterial embolization. Nonoperative management of patients with blunt splenic injury employing transcatheter arterial embolization was successful in 93% of patients, and splenic function, as determined by technetium sulfur-colloid examinations, was preserved in all patients after embolization. The authors encouraged more extensive use of transcatheter embolization and the management of splenic injury without surgery.[30] In a subsequent article in 2005, Hagiwara et al[31] reported positive results of transcatheter SAE in hemodynamically unstable patients in whom there had been an initial transient response to fluid resuscitation. All 15 patients had extravasation at angiography, and all were embolized (several patients had additional non-splenic injuries requiring further embolization; i.e., pelvic fractures, renal injury, liver injury). All patients survived without operative intervention and all had functional splenic tissue on follow-up scintigraphy. These positive reports on SAE, even extending to the initially unstable patient, spurred considerable controversy among trauma physicians; some of whom believed the pendulum on splenic preservation had swung too far with nonoperative and angiographic embolization-assisted management.

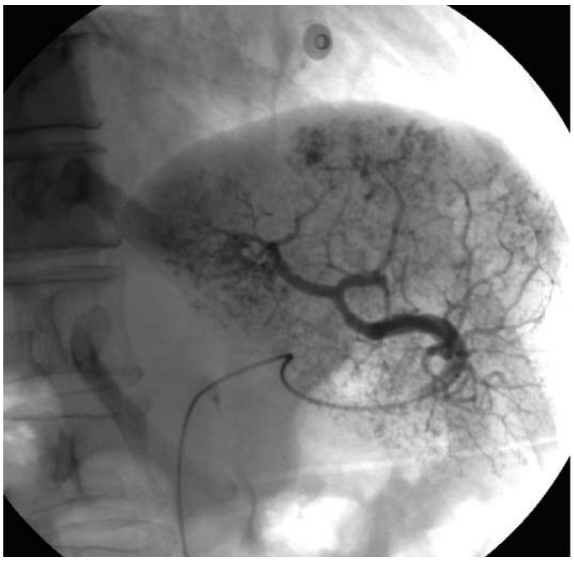
Critical reviews of nonoperative management of blunt splenic trauma began to focus on failures of nonoperative management, and challenged the apparent major positive impact of SAE. Cooney et al[32] reported on the selective use of SAE over a four-year period in which 194 adults with splenic injuries were treated, 48 of whom went to surgery for splenectomy or splenorrhaphy. Nonoperative management with bed rest was used in 137 patients, 11 of whom (8%) failed. SAE was employed in only 9 patients, and 3 of the 9 embolization patients failed, requiring subsequent splenectomy (one for bleeding, and two for infarction), for a failure rate of 33%. They concluded that SAE was useful in only 5% of their patients, "saving" 6 spleens with high-grade injuries, but that it had a 33% failure rate. Failures of nonoperative management were most commonly caused by errors in judgment and selection, frequently from failure to recognize "high-risk" injury patterns on CT and from attempting nonoperative management in anticoagulated or coagulopathic patients. The causes and consequences of failure of nonoperative management of splenic injuries were further discussed in a retrospective descriptive study by McIntyre et al in 2005[33] involving 2243 patients identified in a statewide trauma registry with blunt

splenic trauma over a 6-year period. Of the 2243 patients, 610 (27%) required immediate splenectomy, splenorrhaphy, or splenic embolization. Of the remaining 1633 patients admitted for nonoperative management, 252 (15%) failed. Among the injury and patient characteristics reviewed, age > 55, ISS > 25, and admission to Level III or IV trauma centers (compared to Level I centers) were associated with significant risk of failure of nonoperative management. Smith et al,[34] in a 2006 paper entitled "Splenic artery embolization: Have we gone too far?" reported results of 221 patients over a four-year period at a Level I trauma center. At this center, 165 (75%) of the patients were selected for nonoperative management, 41 of whom (25%) underwent SAE. The failure rate for splenic embolization was 27%, and among high AAST grade splenic injuries (grades 3, 4, and 5) and patients with moderate or large hemoperitoneum, the failure rate with embolization was 43%. The failure rate among splenic embolization patients with observed angiographic extravasation was 59%, and the failure rate in embolization patients with transient hypotension was 57%. They concluded that any hypotension probably warrants laparotomy, and that the combination of high grade injury, significant hemoperitoneum, and extravasation on angiogram predict a high risk of failure. They also noted that there should be a low threshold for splenectomy if bleeding persists.[34] Two other critical studies found no difference in mortality, no difference in adjusted cost, and no difference in the nonoperative management success rate with or without embolization.[35,36]

Numerous studies supportive of SAE in the nonoperative management of blunt splenic trauma continued to show high splenic salvage rates in the 80-90% range, even among high AAST grade splenic injury patients.[37-39] The secondary splenectomy rate is lower in nonoperatively managed patients after successful angiographic embolization,[39] though it has been recognized that patients with an arteriovenous fistula have a higher failure rate, even after apparent successful embolization.[38] A recent Norwegian study[40] reported the results of a prospective study with two groups: group 1 with routine operative and nonoperative management methods; and group 2 employing a splenic injury algorithm with mandatory angiographic embolization of all grades 3, 4, and 5 injuries, and all patients with signs of ongoing bleeding regardless of injury grade. In group 1, 43% of patients underwent emergent laparotomy, and the splenic salvage rate of the nonoperative management group was 79%. In group 2, with SAE, there was a 27% emergent laparotomy rate, a 4% failure rate for embolization, and a nonoperative management splenic salvage rate of 93%. They concluded that SAE increases the percentage of patients in whom nonoperative management was attempted, increased the nonoperative management success rate, and increased the splenic salvage rate.[40]

CT scans performed in follow-up of nonoperative management splenic trauma patients reveal a combination of organization and resolution of parenchymal injuries, as well as parenchymal, subcapsular, and intraperitoneal hemorrhages. Killeen et al[41] described the CT scan findings following SAE in 80 patients, and found splenic infarctions occurred in 63% of patients following proximal embolization and in 100% of patients following distal embolization. Distal embolizations were associated with larger infarcts in the embolized segment, whereas infarcts after proximal embolization tended to be smaller and more peripheral. Another important observation was the presence of gas bubbles within an infarct or subcapsular fluid collection, which clearly can be seen related to the use of Gelfoam as an embolization agent *(see Figure 5)*. However, it is virtually impossible on an imaging basis to differentiate this from an abscess complicating a region of splenic injury or infarction. In Killeen's study,[41] seven patients developed gas collections, four of whom resolved uneventfully, two of which were drained percutaneously and found to be sterile, and of which one was found to be an abscess at laparotomy. The presence of an air-fluid level in a subcapsular collection, or associated with free intraperitoneal air, is more suggestive of abscess formation. Complications arising from SAE after blunt splenic trauma are common, and as some institutions have expanded the use of SAE, it becomes more important to understand the full spectrum of potential complications from the embolization procedure. Ekeh et al[42] described major complications of splenic bleeding, splenic infarction, splenic abscess, and contrast-induced renal failure in 4 of 15 (27%) patients undergoing embolization. Minor complications of fever, pleural effusion, and uneventful, non-target coil migration occurred in 8 of 15 (53%) of the embolization patients. They still concluded that although major and minor complications of SAE occur frequently, it is still a safe and effective adjunct in the nonoperative management of splenic trauma.

Figure 3. Selective Splenic Angiogram in Patient with Extensive Contusion/Laceration



Selective splenic angiogram in patient with extensive splenic contusion / laceration demonstrates the abnormal "pointillism" type parenchymal appearance.

Operative Splenic Salvage and Splenectomy

As we have previously mentioned, the management of splenic injury has evolved from routine splenectomy to surgical salvage techniques to nonoperative treatment with the aid of SAE in select cases. The catalyst for this change was the recognition of the risk of overwhelming post-splenectomy sepsis, first studied in chil-

Figures 4a-4d. Splenic Contusion/Laceration with Pseudoaneurysm and AV Fistula

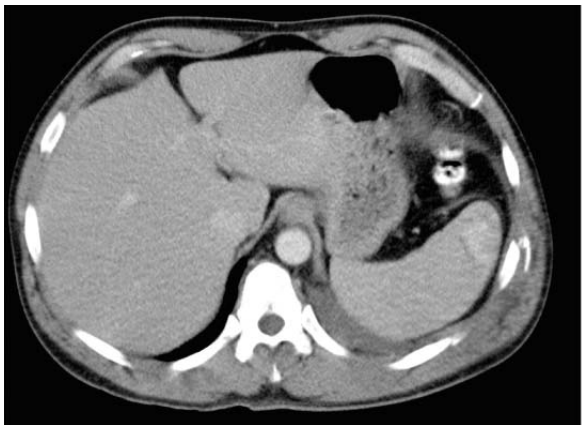


Figure 4a. CT scan of 38-year-old patient with grade 2 splenic injury; a 2 cm deep splenic contusion/laceration in the anterior spleen, with mildly abnormal contrast enhancement, no extravasation, no significant intraperitoneal hemorrhage, and a left sided hemothorax in the posterior costophrenic sulcus.

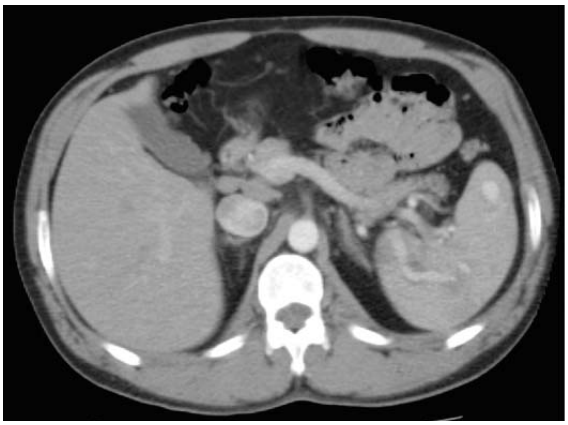


Figure 4b. Patient developed a chronic nonunion of one of the rib fractures, which was still painful, and presented for evaluation of persistent left sided pain approximately 2 months following the initial accident. An abdominal CT scan demonstrates the development of a contained splenic pseudoaneurysm with arteriovenous fistula.

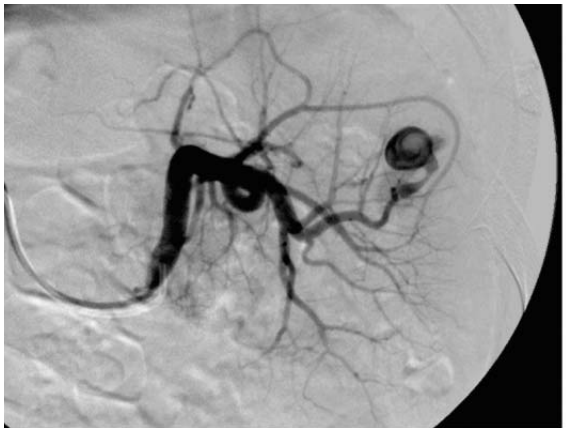


Figure 4c. Splenic angiogram, arterial phase, demonstrating the pseudoaneurysm. There is a single dilated arterial pedicle supplying the pseudoaneurysm.

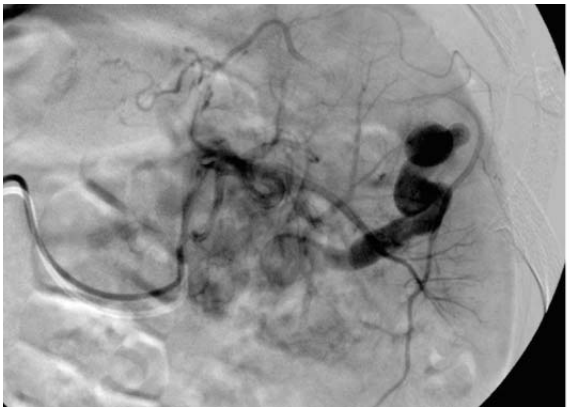


Figure 4d. Splenic angiogram, late arterial phase, demonstrating the arteriovenous fistula component, with the dilated, serpeginous draining vein. The angioarchitecture is fairly simple, with a single arterial pedicle and single draining vein.

dren[2] and then in adults.[3] While the overall incidence of fulminant sepsis in otherwise healthy splenectomized adults is low,[43-45] it is generally preferable to avoid this potentially fatal complication through measures taken to salvage splenic function. However, it must be remembered that certain circumstances, such as uncontrollable hemorrhage, continue to necessitate splenectomy. Thus, whether through splenorrhaphy, partial splenic resection, or splenectomy, operative management of splenic injury remains an important component of care of the injured spleen.

Methods of operative splenic salvage, namely splenorrhaphy and partial splenic resection, are invaluable aids in the preservation of splenic function when possible. Usage of these procedures has declined over the years due to the popularity of nonoperative management.[8,46] Yet, in the patient who does not qualify for nonoperative management based on the aforementioned guidelines, operative splenic salvage may still be an option. In 1985, Feliciano et al[47] concluded that splenorrhaphy could safely be performed in patients with few concomitant intraabdominal injuries and low-grade splenic injury (grades 1, 2, and 3 with success rates of 61.5% to 88.5%). They further discovered that the risk of rebleeding in patients who underwent splenorrhaphy was extremely low. Subsequent literature has suggested that partial splenic resection is most useful for grade 4 splenic injuries or for severe injury located at either splenic pole, and should only be performed if at least one-third of the spleen can be saved.[13] As a result, the decision to enact operative splenic salvage should be made based on classification of the severity of splenic injury, the overall hemodynamic stability of the patient, and the presence of associated abdominal injuries.

Despite the success rates of operative splenic salvage in select cases[47] and the ensuing avoidance of overwhelming post-splenectomy sepsis, there remain clear-cut indications for splenectomy.[8] Massive injury to the spleen, complex multi-system injury, hemodynamic instability, and failure of strategies for splenic salvage prompting laparotomy continue to occur, mandating removal of the spleen as good surgical practice. Consideration of

Figures 4e-4f. Selective Coil Embolization

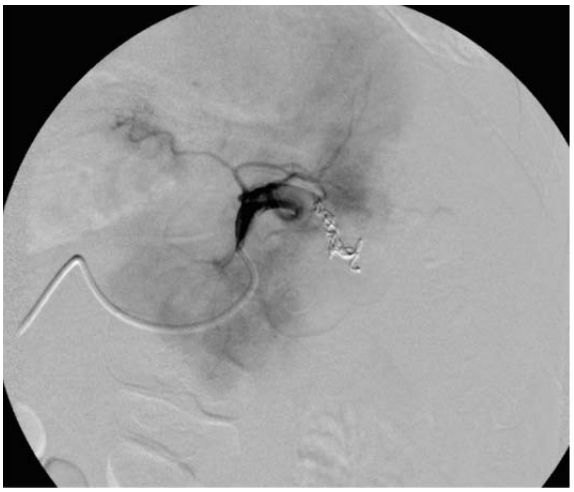


Figure 4e. Selective coil embolization of the arterial pedicle supplying the pseudoaneurysm and arteriovenous fistula.

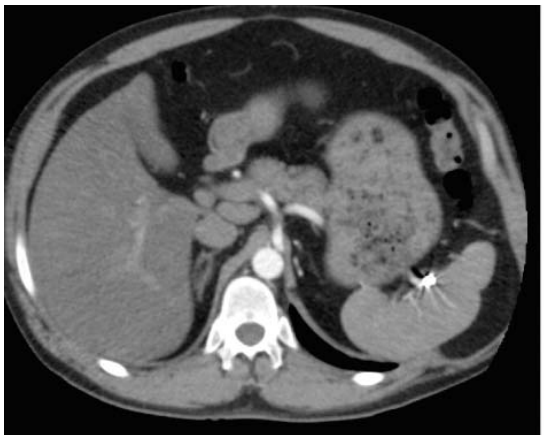


Figure 4f. CT scan of the abdomen 2 years following the ATV accident demonstrates no evidence of residual pseudoaneurysm or arteriovenous fistula, patent flow in the main splenic artery, and homogeneous enhancement of the splenic parenchyma. There is a focal cortical scar in the area of splenic injury and embolization.

splenectomy should occur in the presence of more than 1000 mL of hemoperitoneum, continuously decreasing hemoglobin levels, hemodynamic instability, or the need for more than 2 units of transfused blood.[48] Attempts to salvage the spleen during laparotomy should be abandoned if these methods fail to attain hemodynamic stability or severe splenic injury or associated abdominal injuries are identified.

As part of a comprehensive approach to the management of splenic injury, a strategy exists for the management of patients who must undergo splenectomy. This involves documentation (alert identification) of the asplenic state, vaccination for encapsulated organisms (*Neisseria meningitidis, Haemophilus influenzae,* and *Streptococcus pneumoniae*); antibiotic prophylaxis availability; and documentation to family, friends, and primary care providers of the risk for post-splenectomy sepsis. Furthermore, white blood cell counts in the postoperative period may be useful in early recognition of an infection. This was determined by Horowitz et al,[51] who found that white blood cell levels were consistently greater than 16 and began to rise on postoperative day 4 in infected patients, 2 days later than the peak of physiologic leukocytosis. It also has been suggested that thrombotic complications may be prevented with low-dose aspirin regimens.[49] Thus, through a comprehensive and scrupulous post-splenectomy protocol, serious complications may be avoided.

Finally, despite the advances in nonoperative and operative splenic management, variability in the success rates of splenic salvage and the routine practice of splenectomy have been reported.[26] Despite the widespread popularity of guidelines to follow, there are no recommendations for improving rates of splenic salvage in situations in which the rate is below published studies. At our institution, we hypothesized that by using statistical process control (SPC), we could achieve a splenic salvage rate of greater than 80%. First, data collected on the incidence of splenectomy in one year served as our benchmark (45%). Following intensive education regarding the concept of splenic salvage, current splenic salvage rates were displayed at monthly performance improvement meetings attended by trauma surgeons and other specialists, such as radiologists. The meetings also served to allow for evaluation of every instance of splenic injury and management. At the conclusion of our three-year period, rates of successful splenic salvage had risen from 45% to 92%. Thus, we submit that SPC using control chart data display and intensive education, coupled with critical incident review of each instance of splenectomy, may result in an improvement in the overall splenic salvage rate and lead to improved care for trauma patients.

The advances in nonoperative management of the traumatically injured spleen should not negate the importance of operative methods of splenic salvage and splenectomy in select cases. Post-splenectomy complications of overwhelming sepsis and thrombotic pathology underscore the need to attempt splenic salvage unless faced with uncontrollable hemodynamic instability, severe splenic injury, or severe associated intraabdominal pathology. The principles of post-splenectomy care demand a cooperative effort between the trauma surgeon, primary care provider, and patient to ensure that proper practices are followed to minimize the risk of complications. Additionally, we recommend that each trauma center should undertake a process of performance improvement utilizing statistics, education, and peer review meetings to achieve levels of splenic salvage that will benefit their patients.

Summary

Our understanding of the physiologic role of the spleen has grown since the first documented splenectomy due to traumatic injury in 1590.[52] Its function as a member of the reticuloendothelial system and the immune deficiency and thrombotic complications that have arisen in asplenic patients have caused the med-

Figure 5. CT Scan of Gelfoam Embolization

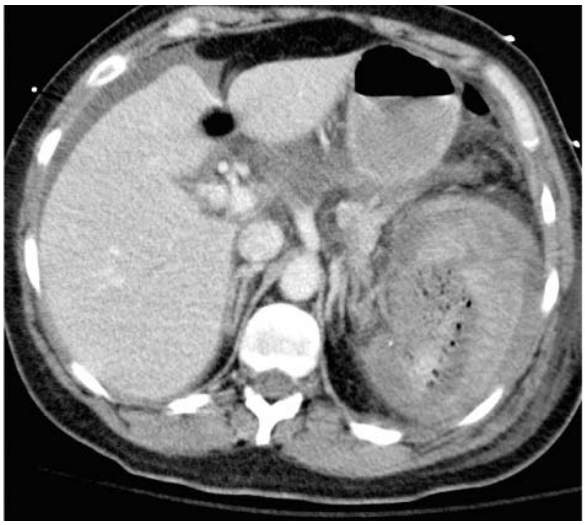


Figure 5. CT scan one day following Gelfoam embolization of the same patient as in Figure 1, demonstrates resolution of the active extravasation and multiple small air bubbles in the spleen due to the Gelfoam material. This appearance is not indicative of an abscess, and this patient had an uneventful recovery with successful splenic salvage.

ical community to reassess the performance of routine splenectomy following injury. Splenic injury scoring recommendations, along with imaging techniques, such as abdominal CT scanning and selective angiographic embolization, have enabled healthcare providers to not only recognize various nuances of splenic injury better, but also to successfully apply operative and nonoperative methods aimed toward preserving splenic function.

The recommendations made in this review reflect the ongoing efforts of medical research to develop techniques to ensure a more favorable outcome in patients who are victims of splenic trauma. Progress in the management of these patients has been achieved through the work of practitioners from various fields, and reflects the maxim that trauma patients require the efforts of an array of healthcare providers, beginning in the field and continuing through their care following discharge, to attain the best possible outcome. It is anticipated that present and future studies will continue to enlighten healthcare providers regarding appropriate management of patients with splenic injury.

References

1. American College of Surgeons Committee on Trauma. Advanced Trauma Life Support For Doctors: Student Course Manual, 7th Edition. Chicago, IL: American College of Surgeons, 2005.
2. King H, Shumaker HB Jr. Splenic studies I. Susceptibility to infection after splenectomy performed in infancy. *Ann Surg* 1952;136:239-242.
3. O'Neal BJ, McDonald JC. The risk of sepsis in the asplenic adult. *Ann Surg* 1981;194:775-778.
4. Chaikof EL, McCabe CJ. Fatal overwhelming post splenectomy infection. *Am J Surg* 1985;149:534-539.
5. Lange DA, Zaret P, Merlotti GJ, et al. The use of absorbable mesh in splenic trauma. *J Trauma* 1988;28:269-275.
6. Cogbill TH, Moore EE, Jurkovich GJ, et al. Nonoperative management of blunt splenic trauma: a multicenter experience. *J Trauma* 1989;29:1312-1317.
7. Smith JS Jr, Wengrovitz MA, DeLong BS. Prospective validation of criteria, including age, for safe, nonsurgical management of the ruptured spleen. *J Trauma* 1992;33:363-368.
8. Pachter HL, Guth AA, Hofstetter SR, Spencer FC. Changing patterns in the management of splenic trauma: the impact of nonoperative management. *Ann Surg* 1998;227:708-717.
9. Moore EE, Cogbill TH, Jurkovich GJ, et al. Organ injury scaling: spleen and liver (1994 revision). *J Trauma* 1995;38:323-324.
10. Davis KA, Fabian TC, Croce MA, et al. Improved success in nonoperative management of blunt splenic injuries: embolization of splenic artery pseudoaneurysms. *J Trauma* 1998;44:1008-1013.
11. Gaunt WT, McCarthy MC, Lambert CS, et al. Traditional criteria for observation of splenic trauma should be challenged. *Am Surg* 1999;65:689-691.
12. World Health Organization. Essential Surgical Care: Primary Trauma Care Manual, Abdominal Trauma. http://www.steinergraphics.com/surgical/manual09.html (Accessed on 9/30/08.)
13. Malangoni MA, Fallon WF. Management of splenic trauma in adults. In: *Shackelford's Surgery of the Alimentary Tract,* Vol. III, 5th Ed. Saunders, 2001.
14. Marmery H, Shanmuganathan K, Alexander MT, et al. Optimization of selection for nonoperative management of blunt splenic injury: comparison of MDCT grading systems. *AJR Am J Roentgenol* 2007;189:1421-1427.
15. Hoff WS, Holevar M, Nagy KK, et al. Practice Management Guidelines for the Evaluation of Blunt Abdominal Trauma. http://www.east.org/tpg/bluntabd.pdf (Accessed on 9/30/08.)
16. Mirvis SE, Whitley NO, Gens DR. Blunt splenic trauma in adults: CT-based classification and correlation with prognosis and treatment. *Radiology* 1989;171:33-39.
17. Buntain WL, Gould HR, Maull KI. Predictability of splenic salvage by computed tomography. *J Trauma* 1986;28:24-34.
18. Resciniti A, Fink MP, Raptopoulos V, et al. Nonoperative treatment of adult splenic trauma: development of a computed tomographic scoring system that detects appropriate candidates for expectant management. *J Trauma* 1988;128:828-831.
19. Gavant ML, Schurr M, Flick PA, et al. Predicting clinical outcome of nonsurgical management of blunt splenic injury: using CT to reveal abnormalities of the splenic vasculature. *AJR Am J Roentgenol* 1997;168:207-212.
20. Federle MP, Courcoulas AP, Powell M, et al. Blunt splenic injury in adults: clinical and CT criteria for management, with emphasis on active extravasation. *Radiology* 1998;206:137-142.
21. Willmann JK, Roos JE, Platz A, et al. Multidetector CT: detection of active hemorrhage in patients with blunt abdominal trauma. *AJR Am J Roentgenol* 2002;179:437-444.
22. Yao DC, Jeffrey Jr. RB, Mirvis SE, et al. Using contrast-enhanced helical CT to visualize arterial extravasation after blunt abdominal trauma: incidence and organ distribution. *AJR Am J Roentgenol* 2002; 178:17-20.
23. Anderson SW, Varghese JC, Lucey BC, et al. Blunt splenic trauma: delayed-phase CT for differentiation of active hemorrhage from contained vascular injury in patients. *Radiology* 2007;243:88-95.
24. Roy-Choudhury SH, Gallacher DJ, Pilmer J, et al. Relative threshold of detection of active arterial bleeding: in vitro comparison of MDCT and digital subtraction angiography. *AJR Am J Roentgenol* 2007;189:W238-W246.
25. Nusbaum M, Baum S, Blakemore WS, et al. Demonstration of intra-abdominal bleeding by selective arteriography: visualization of celiac and superior mesenteric arteries. *JAMA* 1965;191:389-390.
26. Hunt JP, Lentz CW, Cairns BA, et al. Management and outcome of splenic injury: the results of a five-year statewide population-based study. *Am Surg* 1996 Nov;62:911-917.

27. Bee TK, Croce MA, Miller PR, et al. Failures of splenic nonoperative management: is the glass half empty or half full? *J Trauma* 2001;50:230-236.
28. Allins A, Ho T, Nguyen TH, et al. Limited value of routine followup CT scans in nonoperative management of blunt liver and splenic injuries. *Am Surg* 1996;62:883-886.
29. Sclafani SA, Weisberg A, Scalea TM, et al. Blunt splenic injuries: nonsurgical treatment with CT, arteriography, and transcatheter arterial embolization of the splenic artery. *Radiology* 1991;181:189-196.
30. Hagiwara A, Yukioka T, Ohta S, et al. Nonsurgical management of patients with blunt splenic injury: efficacy of transcatheter arterial embolization. *AJR Am J Roentgenol* 1996;167:159-166.
31. Hagiwara A, Fukushima H, Murata A, et al. Blunt splenic injury: usefulness of transcatheter arterial embolization in patients with a transient response to fluid resuscitation. *Radiology* 2005; 235:57-64.
32. Cooney R, Ku J, Cherry R, et al. Limitations of splenic angioembolization in treating blunt splenic injury. *J Trauma* 2005;59:926-932.
33. McIntyre LK, Schiff M, Jurkovich G. Failure of nonoperative management of splenic injuries: causes and consequences. *Arch Surg* 2005;140:563-568.
34. Smith HE, Biffl WL, Majercik SD, et al. Splenic artery embolization: have we gone too far? *J Trauma* 2006;61:541-544.
35. Wahl WL, Ahrns KS, Chen S, et al. Blunt splenic injury: operation versus angiographic embolization. *Surgery* 2004;136:891-899.
36. Harbrecht BG, Ko SH, Watson GA, et al. Angiography for blunt splenic trauma does not improve the success rate for nonoperative management. *J Trauma* 2007;63:44-49.
37. Haan JM, Biffl W, Knudson MM, et al. Splenic embolization revisited: a multicenter review. *J Trauma* 2004;56:542-547.
38. Haan JM, Bochicchio GV, Kramer N, et al. Nonoperative management of blunt splenic injury: a 5 year experience. *J Trauma* 2005;58:492-498.
39. Bessoud B, Denys A, Calmes JM, et al. Nonoperative management of traumatic splenic injuries: is there a role for proximal splenic artery embolization. *AJR Am J Roentgenol* 2006;186:779-785.
40. Gaarder C, Dormagen JB, Eken T, et al. Non operative management of splenic injuries: improved results with angioembolization. *J Trauma* 2006;61:192-198.
41. Killeen KL, Shanmuganathan K, Boyd-Kranis R, et al. CT findings after embolization for blunt splenic trauma. *J Vasc Interv Radiol* 2001;12:209-214.
42. Ekeh AP, McCarthy MC, Woods RJ, et al. Complications arising from splenic artery embolization after blunt splenic trauma. *Am J Surg* 2005;189:335-339.
43. Schwartz PE, Sterioff S, Mucha P, et al. Postsplenectomy sepsis and mortality in adults. *JAMA* 1982;248:2279-2283.
44. Holdsworth RJ, Irving AD, Cuschieri A. Postsplenectomy sepsis and its mortality rate: actual versus perceived risks. *Br J Surg* 1991;78:1031-1038.
45. Bisharat N, Omari H, Lavi I, et al. Risk of infection and death among post-splenectomy patients. *J Infect* 2001;43:182-186.
46. Brasel KJ, DeLisle CM, Olson CJ, et al. Splenic injury: trends in evaluation and management. *J Trauma* 1998;44:283-286.
47. Feliciano DV, Bitondo CG, Mattox KL, et al. A four-year experience with splenectomy versus splenorrhaphy. *Ann Surg* 1985;201:568-575.
48. National Guideline Clearinghouse. Indications for splenectomy. http://www.guidelines.gov/summary/summary.aspx?ss=15&doc_id=5505&string=spherocytosis#s23 (Accessed on 9/30/08.)
49. Linet MS, Nyren O, Gridley G, et al. Causes of death among patients surviving at least one year following splenectomy. *Am J Surg* 1996;172:320-323.
50. Pisters PW, Pachter HL. Autologous splenic transplantation for splenic trauma. *Ann Surg* 1994;219:225-235.
51. Horowitz J, Leonard D, Smith J, et al. Postsplenectomy leukocytosis: physiologic or an indicator of infection? *Am Surg* 1992;58:387-390.
52. Wilkins B. The spleen. *Br J Haematol* 2002;117:265-274.

CNE/CME Objectives

Upon completing this program, the participants will be able to:

a.) discuss conditions that should increase suspicion for traumatic injuries;
b.) describe the various modalities used to identify different traumatic conditions;
c.) cite methods of quickly stabilizing and managing patients; and
d.) identify possible complications that may occur with traumatic injuries.

CNE/CME Questions

1. Which of the following statements regarding the management of a patient with a possible abdominal injury is correct?
   A. They should be taken for immediate exploratory laparotomy even if they are stable due to the potentially disastrous outcomes following a missed diagnosis of a splenic rupture.
   B. Patients with stable vital signs and a nonspecific abdominal examination on presentation should be released with specific instructions to follow-up with a primary care physician.
   C. A chest x-ray is adequate in patients with suspected rib fractures, as thoracic injuries do not increase the likelihood of associated abdominal injuries.
   D. Stable patients with a high possibility of abdominal injury should undergo repeated abdominal exams and appropriate imaging studies due to the high incidence of patients with hemoperitoneum who had benign initial examinations.

2. Which of the following statements is correct regarding the importance of hemodynamic stability when assessing a patient with abdominal trauma?
   A. Airway patency, breathing, and circulation do not need to be considered first because an unstable patient taken for exploratory laparotomy will be intubated and placed on IV fluids in the operating room.
   B. A hemodynamically unstable patient with obvious abdominal blood loss should be taken for immediate exploratory laparotomy.
   C. A hemodynamically unstable patient with obvious abdominal blood loss should be taken for an emergent abdominal CT scan.
   D. The hemodynamic stability of a patient with abdominal trauma does not factor into the decision-making process regarding the diagnosis and management of injury.

CNE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a letter of credit.** When your evaluation is received, a letter of credit will be mailed to you.

3. Which statement regarding splenic injury is correct?
   A. Consideration of possible splenic injury should only occur when a patient presents with obvious left upper quadrant abdominal trauma.
   B. Patients with splenic injury often experience long-term sequelae of the inability to become angry and lack of imagination, due to the loss of splenic function and its role in achieving various emotional states.
   C. Nonoperative management, surgical salvage techniques, and splenectomy are utilized to balance the importance of splenic preservation with the need to prevent splenic hemorrhage following traumatic injury.
   D. The spleen is rarely injured following blunt abdominal trauma.

4. Which of the following statements regarding imaging techniques in the assessment of abdominal trauma is correct?
   A. FAST is performed most often due to the need to move patients through the emergency room quickly.
   B. CT scans have the ability to detect certain abdominal injuries which were not detected during the initial examination, and should only be performed in patients who are hemodynamically stable.
   C. Active extravasation viewed during an abdominal CT scan should have little bearing on the choice of management of splenic injury.
   D. A hemodynamically unstable patient with unconfirmed abdominal injury may undergo either an abdominal CT scan or a FAST evaluation to aid in the decision regarding possible emergent laparotomy.

5. Which of the following statements regarding guidelines for nonoperative management of splenic injury is correct?
   A. Patients who undergo nonoperative management should not have associated significant injury of another abdominal organ.
   B. Patients with hemodynamic instability may be initially managed nonoperatively with avoidance of all operative procedures due to the risk of postoperative infection.
   C. Splenic injury scoring should not be used when deciding which course of treatment to follow.
   D. Patients greater than 50 years of age should never be managed nonoperatively.

6. Nonoperative management of splenic injury is:
   A. most successfully accomplished on an outpatient basis in young compliant patients.
   B. rarely used in adults due to the limited role of the spleen as a person matures.
   C. best carried out with the aid of weekly abdominal CT scans in order to assure that the patient achieves complete resolution of injury.
   D. aided by SAE if a vascular blush is identified during the abdominal CT scan.

7. Operative management in patients with splenic injury:
   A. has evolved and essentially obliterated the performance of splenectomy, even in the case of uncontrollable hemorrhage.
   B. includes splenorrhaphy, which should be performed in patients with persistently declining hemoglobin levels or those requiring more than 2 units of transfused blood.
   C. should be determined based on hemodynamic stability, severity of splenic injury and the presence of concomitant abdominal injuries when choosing between operative salvage techniques and splenectomy.
   D. should not be performed on patients greater than 55 years of age with a Grade 5 splenic injury, due to the decreased wound healing potential in patients with advanced age.

8. Which statement regarding postsplenectomy care is correct?
   A. Patients should be vaccinated for encapsulated organisms, and family and primary care providers should be educated regarding the signs and risk of postsplenectomy sepsis.
   B. Vaccination for encapsulated organisms is not necessary prior to the age of 50 due to the loss of immune function which accompanies aging.
   C. Overwhelming sepsis is the only complication that has observed in patients who have undergone splenectomy.
   D. Due to the overall low incidence of overwhelming postsplenectomy sepsis, there are no routine precautions which need to be followed.

9. Which of the following statements regarding the initial evaluation of patients with abdominal trauma is correct?
   A. Palpation of the abdomen is rarely helpful and should be avoided due to the risk of causing more trauma in a potentially precarious presentation.
   B. Left upper quadrant pain and dullness to percussion, left shoulder pain, and hypotension are possible presentations seen following splenic injury.
   C. Disability does not need to be assessed in these patients and supplementary oxygen is usually not required.
   D. The physical exam may be postponed until after an abdominal CT scan is performed.

Answers: 1. D; 2. B; 3. C; 4. B; 5. A; 6. D; 7. C; 8. A; 9. B

In Future Issues:

Approaching the Patient with Shock