attempt to obviate endotracheal intubation. Acetylcholinesterase inhibitors are not a substitute for administration of an appropriate antivenom when available.

Care of the bite wound includes simple cleansing with soap and water; application of a dry, sterile dressing; and splinting of the affected extremity with padding between the digits. Once antivenom therapy has been initiated, the extremity should be elevated above heart level to reduce swelling. Tetanus immunization should be updated as appropriate. Prophylactic antibiotics are generally unnecessary after bites by North American snakes, as the incidence of secondary infection is low. In some regions, secondary bacterial infection is more common and the consequences are dire; in these regions, prophylactic antibiotics (e.g., cephalosporins) are used commonly. Antibiotics may also be considered if misguided first aid efforts have included incision or mouth suction of the bite site. Pain control should be achieved with acetaminophen or narcotic analgesics. Salicylates and nonsteroidal anti-inflammatory agents should be avoided because of their effects on blood clotting.

Most snake envenomations involve SC deposition of venom. On occasion, however, venom can be injected more deeply into muscle compartments, particularly if the offending snake was large and the bite occurred on the lower leg, forearm, or hand. Intramuscular swelling of the affected extremity may be accompanied by severe pain, decreased strength, altered sensation, cyanosis, and apparent pulselessness—signs suggesting a muscle compartment syndrome. If there is clinical concern that subfascial muscle edema may be impeding tissue perfusion, intracompartmental pressures should be measured by a minimally invasive technique (e.g., wick catheter or digital readout device). If the intracompartmental pressure is high (>30–40 mmHg), the extremity should be kept elevated while antivenom is administered. A dose of IV mannitol (1 g/kg) can be given in an effort to reduce muscle edema if the patient is hemodynamically stable. If the intracompartmental pressure remains elevated after 1 h of such therapy, a surgical consultation should be obtained for possible fasciotomy. Although evidence from animal studies suggests that fasciotomy may actually worsen myonecrosis, compartmental decompression is still necessary to preserve nerve function. Fortunately, the incidence of compartment syndrome is very low after a snakebite, with fasciotomies required in <1% of cases. Nevertheless, vigilance is essential. If a fasciotomy is deemed necessary, it should be undertaken with the patient's informed consent whenever possible.

Wound care in the days after the bite should include careful aseptic debridement of clearly necrotic tissue once coagulation has been restored. Intact serum-filled vesicles or hemorrhagic blebs should be left undisturbed. If ruptured, they should be debrided with sterile technique. Any debridement of damaged muscle should be conservative because there is evidence that such muscle may recover to a significant degree after antivenom therapy.

Physical therapy should be started as soon as possible so that the victim can return to a functional state. The incidence of long-term loss of function (e.g., reduced range of motion, impaired sensory function) is unclear but is probably quite high (>30%), particularly after viperid bites.

Any patient with signs of envenomation should be observed in the hospital for at least 24 h. In North America, a patient with an apparently "dry" viperid bite should be watched for at least 8 h before discharge, as significant toxicity occasionally develops after a delay of several hours. The onset of systemic symptoms commonly is delayed for a number of hours after bites by several of the elapids (including coral snakes, *Micrurus* species), some non–North American viperids (e.g., the hump-nosed pit viper [*Hypnale hypnale*]), and sea snakes. Patients bitten by these snakes should be observed in the hospital for at least 24 h. Patients whose condition is not stable should be admitted to an intensive care setting.

At hospital discharge, victims of venomous snakebites should be warned about symptoms and signs of wound infection, antivenom-related serum sickness, and potential long-term sequelae, such as pituitary insufficiency from Russell's viper (*D. russelii*) bites. If coagulopathy developed in the acute stages of envenomation, it can recur during the first 2–3 weeks after the bite. In such cases, victims should be warned to avoid elective surgery or activities posing a high risk of trauma during this period. Outpatient analgesic treatment, wound management, and physical therapy should be provided.

MORBIDITY AND MORTALITY

The overall mortality rates for victims of venomous snakebites are low in regions with rapid access to medical care and appropriate antivenoms. In the United States, for example, the mortality rate is <1% for victims who receive antivenom. Eastern and western diamondback rattlesnakes (*Crotalus adamanteus* and *Crotalus atrox*, respectively) are responsible for the majority of snakebite deaths in the United States. Snakes responsible for large numbers of deaths in other countries include cobras (*Naja* spp.), carpet and saw-scaled vipers (*Echis* spp.), Russell's vipers (*D. russelii*), large African vipers (*Bitis* spp.), lancehead pit vipers (*Bothrops* spp.), and tropical rattlesnakes (*C. durissus*).

The incidence of morbidity—defined as permanent functional loss in a bitten extremity—is difficult to estimate but is substantial. Morbidity may be due to muscle, nerve, or vascular injury or to scar contracture. Such morbidity can have devastating consequences for victims in the developing world when they lose the ability to work and provide for their families. In the United States, functional loss tends to be more common and severe after rattlesnake bites than after bites by copperheads (*Agkistrodon contortrix*) or water moccasins (*Agkistrodon piscivorus*).

GLOBAL CONSIDERATIONS

In many developing countries where snakebites are common, scarce access to medical care and antivenom resources contributes to high rates of morbidity and mortality. In many countries, the available antivenoms are inappropriate and ineffective against the venoms of medically important indigenous snakes. In those regions, further research is necessary to determine the actual impact of venomous snakebites and the specific antivenom needs in terms of both quantity and spectrum of coverage. Without accurate statistics, it is difficult to persuade antivenom manufacturers to begin and sustain production of appropriate antisera in developing nations. There is evidence that antivenoms can be produced in much more cost-effective ways than those currently being used. Just as important as getting the correct antivenoms into underserved regions is the need to educate populations about snakebite prevention and to train medical care providers in proper management approaches. Local protocols written with significant input from experienced providers in the region of concern should be developed and distributed. Appropriate antivenoms must be available at the likely first point of medical contact for patients (e.g., primary health centers) in order to minimize the common practice of referring victims to more distant, higher levels of care for the initiation of antivenom therapy. Those who care for snakebite victims in these often-remote clinics must have the skills and confidence required to begin antivenom treatment (and to treat possible reactions) as soon as possible when needed.

MARINE ENVENOMATIONS

Much of the management of envenomation by marine creatures is supportive in nature. A specific marine antivenom can be used when appropriate.

INVERTEBRATES

Cnidarians The Golgi apparatus of the cnidoblast cells within cnidarians, such as hydroids, fire coral, jellyfish, Portuguese men-of-war, and sea anemones, secretes specialized living stinging organelles called *cnidae* (also referred to as *cnidocysts*, a term that encompasses nematocysts, ptychocysts, and spirocysts). Within each organelle resides a stinging mechanism ("thread tube") and venom. In the stinging process, cnidocysts are released and discharged upon mechanosensory stimulation. The venoms from these organisms contain bioactive substances such as tetramine, 5-hydroxytryptamine, histamine,