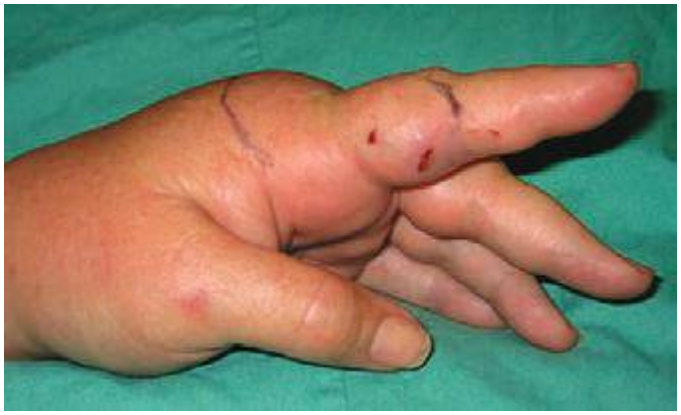
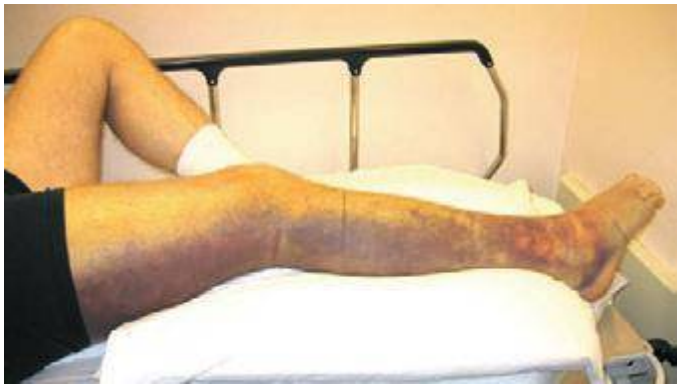
Proteolytic enzymes cause local tissue necrosis, affect the coagulation pathway at various steps, and impair organ function. Hyaluronidases promote the spread of venom through connective tissue. Myocardial depressant factors reduce cardiac output, and bradykinins cause vasodilation and hypotension. Neurotoxins act either pre- or postsynaptically to block transmission at the neuromuscular junction, causing muscle paralysis. Most snake venoms have multisystem effects on their victims.

After a venomous snakebite, the time to symptom onset and clinical presentation can be quite variable and depend on the species involved, the anatomic location of the bite, and the amount of venom injected. Envenomations by most viperids and some elapids with necrotizing venoms cause progressive local pain, swelling, ecchymosis (Fig. 474-1), and (over a period of hours to days) hemorrhagic or serum-filled vesicles and bullae. In serious bites, tissue loss can be significant (Figs. 474-2 and 474-3). Systemic findings are extremely variable and can include tachycardia or bradycardia, hypotension, generalized weakness, changes in taste, mouth numbness, muscle fasciculations, pulmonary edema, renal dysfunction, and spontaneous hemorrhage (from essentially any anatomic site). Envenomations by neurotoxic elapids such as kraits (*Bungarus* species), many Australian elapids (e.g., death adders [*Acanthophis* species] and tiger snakes [*Notechis* species]), some cobras (*Naja* species), and some viperids (e.g., the South American rattlesnake [*Crotalus durissus*] and some Indian Russell's vipers [*Daboia russelii*]) cause neurologic dysfunction. Early findings may consist of nausea and vomiting, headache, paresthesias or numbness, and altered mental status. Victims may develop cranial nerve abnormalities (e.g., ptosis, difficulty swallowing) followed by peripheral motor weakness. Severe envenomation may result in muscle paralysis, including the muscles of respiration, and lead to death from respiratory failure and aspiration. Sea snake envenomation results in local pain (variable), generalized myalgias, trismus, rhabdomyolysis, and progressive flaccid paralysis; these manifestations can be delayed for several hours.



A



B

FIGURE 474-1 Northern Pacific rattlesnake (*Crotalus oreganus oreganus*) envenomations. *A.* Moderately severe envenomation. Note edema and early ecchymosis 2 h after a bite to the finger. ***B.*** Severe envenomation. Note extensive ecchymosis 5 days after a bite to the ankle.

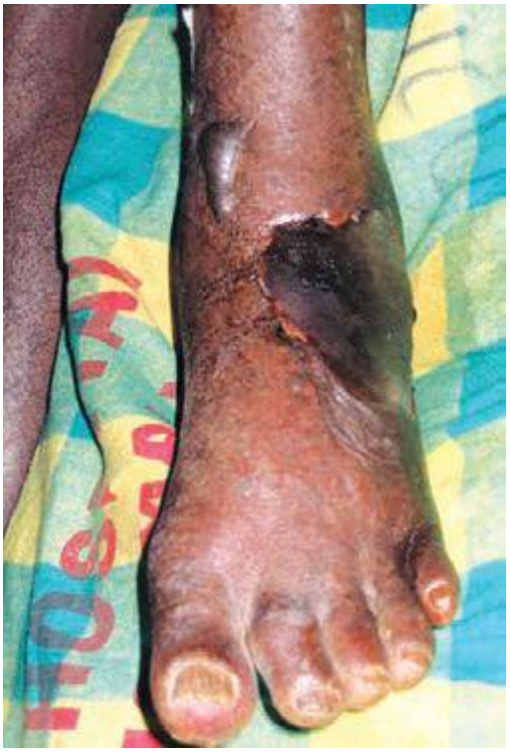


FIGURE 474-2 Early stages of severe, full-thickness necrosis 5 days after a Russell's viper (*Daboia russelii*) bite in southwestern India.

TREATMENT VENOMOUS SNAKEBITE

FIELD MANAGEMENT

The most important aspect of prehospital care of a person bitten by a venomous snake is rapid transport to a medical facility equipped to provide supportive care (airway, breathing, and circulation) and antivenom therapy. Most of the first-aid measures recommended in the past are of little benefit, and some actually worsen outcome. It is reasonable to apply a splint to the bitten extremity to lessen bleeding and discomfort and, if possible, to keep the extremity at approximately heart level. In developing countries, indigenous people should be encouraged to seek immediate care at a health care

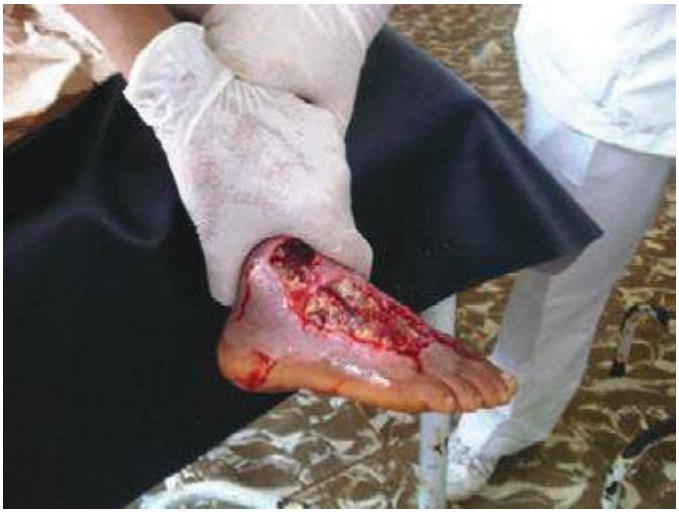


FIGURE 474-3 Severe necrosis 10 days after a pit viper bite in a young child in Colombia. *(Courtesy of Jay R. Stanka; with permission.)*