Venomous Snakebites in the United States

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Venomous snakebite treatment is controversial. Venomous snakebites are known to occur in all but a few states. Approximately 10 to 15 individuals die from snakebites each year, with bites from diamondback rattlesnakes accounting for 95 percent of fatalities. The identification of the two endogenous classes of venomous snakes are discussed in detail to aid in determining the proper treatment for each class. Approximately 25 percent of all pit viper bites are "dry" and result in no envenomation.

The best first aid is a set of car keys to get the victim to a facility where antivenin is obtainable. Incision and suction should be limited to very special situations; cryotherapy and use of tourniquets applied by laymen should be avoided. Proper medical management at a health care facility requires establishing whether envenomation has occurred and to what extent, followed by appropriate dosing of antivenin. The use of corticosteroids and antibiotics is controversial. Tetanus immunization should be updated, if necessary. Although research in developing a more purified antivenin is under way, the best treatment for snakebite is prevention.

A venomous snakebite—rarely does any subject draw more attention and controversy in an emergency department. Frequently two or more proponents of different treatments may feel the need to defend zealously their specific school of thought, and anyone who attempts even the simplest care of a venomous snakebite may be called on to defend his or her actions in a sometimes emotional public arena. The family physician should be aware of the current thoughts on all aspects of snakebite treatment.

Venomous snakebites occur in every state in the continental United States, except Maine, Michigan, and Delaware, and rarely in the New England states,[1] with venomous snakes being most common in the South and in rural areas. An estimated 45,000 snakebites are reported each year in the United States, approximately 8,000 of which are venomous.[2–4] At least 1,000 additional venomous snakebites go unreported.[5] Mortality from snakebites ranges between 9 to 15 persons per year (less than 1 percent) in the United States. Most snakebite deaths occur in children, the untreated, the mistreated, or those with other underlying medical problems.[3] Members of certain religious sects also routinely handle venomous snakes and refuse treatment in the hope that their religious beliefs will effect a cure for the snakebite.

Approximately 75 percent of all snakebites occur in people aged between 19 and 30 years, 1 percent to 2 percent occur in women, and less than 1 percent occur in blacks. Approximately 40 percent of all snakebites occur in people who are handling or playing with snakes, and 40 percent of all people bitten had a blood alcohol level of greater than 0.1 percent. Sixty-five percent of snakebites occur on the hand or fingers, 24 percent on the foot or ankle, and 11 percent elsewhere.[6] One case was reported of a rattlesnake bite on the glans penis.[7]

Copperheads inflict about 3,000 bites per year because of their propensity to invade human habitat. Fortunately their venom is less toxic and rarely fatal.[8] Diamondback rattlesnakes account for 95 percent of fatalities, although they account for only 10 percent of total bites.

IDENTIFICATION

There are two endogenous classes of venomous snakes in the United States: the Crotalidae, which includes all rattlesnakes, water moccasins, and copperheads, is the most significant; the Elapidae, which includes the coral snakes, plays a less significant role in the morbidity and mortality of venomous snakebites.

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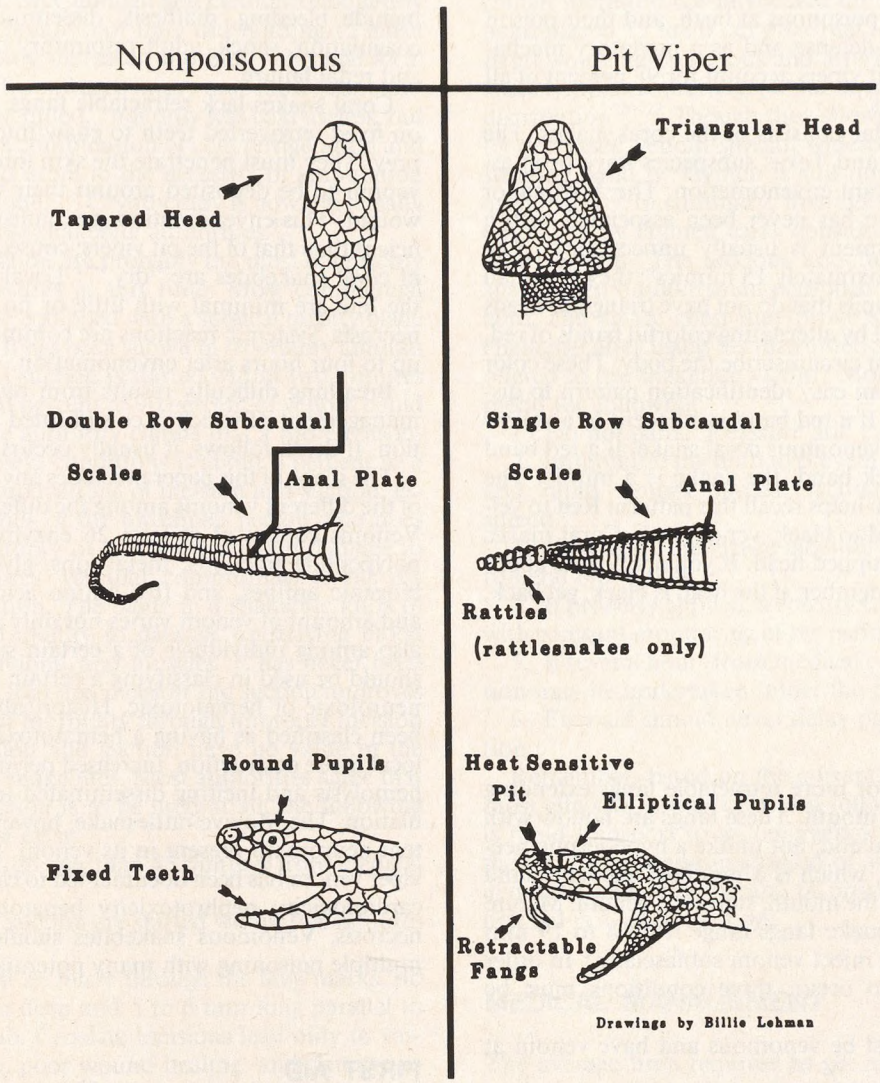

Figure 1. Identifying characteristics of nonpoisonous snakes and pit vipers

The family Crotalidae, or the pit vipers, is so named because of a heat-sensitive pit located midway between the eye and the nostril on each side of the head. This heat-sensitive structure is able to detect temperature changes as small as 1 °F at 14 inches.[6] The pit vipers are easily identified by retractable fangs, elliptical pupils (all native North American snakes with elliptical pupils are poisonous), a single row of subcaudal scales distal to the anal plate, and a triangular-shaped head. Frequently, a snake will be brought in decapitated and a distinction between single and double row of scales distal to the anal plates becomes important (Figure 1). Caution must be used whenever examining a pit viper's head, as the striking reflex may be present for 60 minutes after death.[9]

There are about 30 species of rattlesnakes, the most dangerous being the Eastern and Western diamondbacks.[10] Copperheads and water moccasins make up the rest of the Crotalidae.[1] These pit vipers can be very small, as in the case of a pygmy rattler, which measures 10 inches as an adult, or very large, as in the case of the Eastern

diamondback rattlesnake, which grows in excess of 6 to 7 feet. Pit vipers are poisonous at birth, and their poison is used both for self-defense and as a predatory mechanism.[6] As a group, pit vipers account for 98 percent of all venomous snakebites.[1]

The family Elapidae consists of the coral snakes. The Eastern coral snake and Texas subspecies have been associated with significant envenomation. The Arizona, or Sonoran, coral snake has never been associated with a mortality, and treatment is usually unnecessary.[4] The coral snake has approximately 15 mimics[6]; they are small snakes with round pupils that do not have triangular heads and are distinguished by alternating colorful bands of red, black, and yellow that circumscribe the body. These color variations allow for an easy identification pattern to distinguish the species. If a red band is adjacent to a yellow band, the snake is a venomous coral snake. If a red band is adjacent to a black band, the snake is a mimic. The following pneumonic helps recall this pattern: Red to yellow, kill a fellow. Red to black, venom lack. Coral snakes always have a black-tipped head. If you are unsure about the band pattern, remember, if the head is black, get back, Jack!

ENVENOMATION

Pit vipers have two or more retractable fangs extending from the roof of the mouth. These fangs are hollow with exit ports at the distal end, not unlike a hypodermic needle. A venom gland, which is a modified salivary gland located in the roof of the mouth, stores the venom. Mature diamondback rattlesnake fangs range from 8 to 19 mm long. Rarely do they inject venom subfascially.[6] In order for envenomation to occur, three conditions must be present:

1. The snake must be venomous and have venom at the time of the bite.
2. The fangs must penetrate the skin.
3. Venom must be injected during fang penetration.

Despite this teleologically advanced mechanism for envenomation, between 20 percent and 25 percent of all pit viper bites are "dry" and result in no envenomation.[11] A recent meal, failure of fang penetration, and improper venom expulsion are all factors that account for this discrepancy. Pit viper envenomation is characterized by both local effects and toxic systemic reactions. Once injected into the subcutaneous tissue, venom exerts a necrotizing effect on lymphatic vessels and lymphoid tissue, the cells, and the small blood vessels. Progressive swelling and pain are universally encountered within one hour. Hemorrhagic blebs, petechiae, and ecchymoses from the destruction of red blood cells occur in the skin within a few hours at the site of the bite.[9] Toxic systemic reactions include bleeding, diathesis, disseminated intravascular coagulation, shock, adult respiratory distress syndrome, and renal failure.

Coral snakes lack retractable fangs. Instead, they rely on fixed retroverted teeth to gnaw into the flesh of their prey. They must penetrate the skin long enough for their venom to be deposited around their teeth and into the wound. This envenomation mechanism is much less efficient than that of the pit vipers; consequently, 50 percent of coral snakebites are "dry."[11] Local manifestations of the bite are minimal with little or no pain, swelling, or necrosis. Systemic reactions are common and may occur up to four hours after envenomation.

Breathing difficulty results from bulbar paralysis, the management of which is complicated by marked salivation. If death follows, it usually occurs within 24 hours.[9]

The scope of this paper precludes any lengthy discussion of the different venoms among the different snake species. Venoms consist of at least 26 enzymes and numerous polypeptide fractions, metal ions, glycoproteins, lipids, biogenic amines, and free amino acids.[11] The strength and amount of venom varies not only among species but also among individuals of a certain species.[5,12] Caution should be used in classifying a certain species' venom as neurotoxic or hematotoxic. Historically, pit vipers have been classified as having a hematotoxic venom, causing local tissue destruction, increased permeability of vessels, hemolysis and inciting disseminated intravascular coagulation. The Mojave rattlesnake, however, has a very potent neurotoxin present in its venom. Likewise other pit viper venom has been documented to cause neurotoxicity, cardiotoxicity, nephrotoxicity, hepatotoxicity, and myonecrosis. Venomous snakebites should be viewed as a multiple poisoning with many potential consequences.

FIRST AID

Following a snakebite, the urge to do something must be quite compelling, as rarely does a victim enter an emergency department without the dubious benefit of a tourniquet, cold packs, an incision, or some other exotic therapy administered by well-intended individuals.[11] First aid for snakebite remains somewhat controversial, although most authorities agree that minimizing field procedures and quickly transporting the victim to a medical facility need to be emphasized. Most snakebite cures in the last 25 years are rooted more closely in folklore and anecdotal experience than in scientific verification.

Cryotherapy, a treatment promoted primarily in the 1950s, was based on the observation that if cooled enough in vitro, snake venom lost its potency. Victims were

treated by immersing the bitten extremity into an ice bath and refrigerating it. McCullough and Gennaro eloquently showed that cryotherapy not only did nothing to effect mortality, but greatly increased morbidity through local tissue destruction from freeze injury and subsequent amputation. They concluded, "not only was cold useless, but in some instances a combination of cold refrigeration and venom contributed more to the necessity of amputation than did venom alone."[13] Cryotherapy is now universally condemned by most snakebite authorities.[14–17]

Incision and suction are probably the most commonly used first-aid measures at this time. Most commercial snakebite kits come equipped with a surgical quality blade and suction bulbs. It has been shown in laboratory animals that if incision and suction is started within two minutes of the snakebite, 50 percent to 90 percent of venom could be removed.[13] One authority claims that a more reasonable figure under field conditions is about 5 percent to 6 percent.[11] The complications of incision and suction, especially in the hands of the untrained person who does not know the anatomy of the body, include damage to underlying structures, vascular compromise to the extremity, and infection. The blade in a snakebite kit is of sufficient size and quality to damage underlying blood vessels, nerves, tendons, and muscles. It has never been shown in a clinical trial that incision and suction improves mortality, although morbidity through improper incision is increased. Incision and suction have no value in the treatment of coral snakebites. Most authorities agree that if incision and suction is to be used at all, the following qualifications must exist:

1. The victim is greater than 45 minutes from medical attention.
2. Incision and suction are started within five minutes of the snakebite.
3. Incision must be made through the fang marks, no greater than 3 mm deep and 5 to 6 mm long parallel to the axis of the limb. Cruciate incisions lead only to vascular compromise, poor wound healing, and damage to underlying structures.[5,11,18]
4. Suction should be continued for 30 to 60 minutes.
5. First aid should not delay transport to a medical facility.

Use of tourniquets enjoyed popularity in the 1960s. The theory behind the value of using a tourniquet was that by confining venom to the bitten extremity and preventing systemic spread, mortality would be prevented. This was found to be true in some studies with dogs; however, a correlation to these studies showed that tourniquets increased the morbidity in the affected limb.[13] Even if the tourniquet were applied properly by a layman, swelling resulting from a snakebite may cause vascular compromise to the extremity, thus allowing the venom to degrade a now ischemic extremity.[19] The use of tourniquets applied by laymen should be condemned. Recently the Australian literature has advocated the use of a constricting band placed 15 to 20 cm proximal to the snakebite. This band would allow venous and arterial blood flow but restrict lymphatic drainage, a major mechanism in venom distribution.[20–22] Though they showed significant benefit by decreased venom spread, widespread use of the constricting band by laymen is still not advocated by most authorities.[23] In summary, first-aid measures commonly employed by laymen carry considerable risk with a dubious benefit. The anxious and frequently intoxicated snakebitten outdoorsman equipped with a sharp knife, a cord, and an ice chest may prove to be quite a medical challenge after using his instruments.[5] What then can be offered to the snakebite victim? Most authorities recommend the following:

1. Do not panic. Reassure and calm the victim.
2. Immobilize the limb to prevent increased blood flow and rapid venom spread. Activity increases venom spread.[9]
3. Never give the victim alcohol, as it will increase peripheral blood flow.
4. If properly applied, a constricting band may be used with constant monitoring of the peripheral vascular status.
5. If several hours from medical care, incision and suction may be undertaken under the previous guidelines.
6. First aid should never delay prompt medical attention.

Remember, based on the current literature, the single most effective course of action following a pit viper bite is rapid transport to an emergency department because the intravenous administration of antivenin remains the definitive and only therapy of proven value.[24] The best first aid is a set of car keys.

MEDICAL MANAGEMENT

The average time required to get medical attention following a snakebite is between 15 to 60 minutes in the United States.[5] The most important initial treatment would be to stabilize the patient and determine whether envenomation has occurred. Snakebite victims are usually apprehensive and can be hysterical. Fang marks must be present for envenomation to occur. At times it is difficult to determine whether fang penetration has actually occurred, especially if the victim has sustained another injury to the area in fleeing or received first aid by some enthusiastic bystander.

Pit viper bites exhibit swelling as an initial response, usually within 10 minutes, but always within 30 minutes.[5] Pain is a frequent symptom, but may be absent. The physician should establish that envenomation has occurred on the basis of clinical findings rather than on the presence

of fang marks and identification of the snake. Russell[5] has proposed the following rating scale for pit viper bites:

No envenomation—No local or systemic reactions, although fang marks may be present.

Minimal envenomation—Local swelling but no systemic reactions.

Moderate envenomation—Swelling that progresses beyond the site of the bite together with as systemic reaction or laboratory changes (such as a fall in hematocrit, fibrinogen level, or platelets or hematuria).

Severe envenomation—Marked local and systemic reactions such as bleeding, diathesis, or shock, with marked laboratory changes.

If no envenomation is suspected, the patient should be observed for a minimum of four hours before discharge from the emergency department, and sent home with specific instructions to return if systemic signs or symptoms develop.[5]

Once envenomation has been determined, the patient should be monitored closely for systemic signs. A complete blood count, fibrinogen level, platelet count, urinalysis, prothrombin time-partial thromboplastin time, serum electrolytes, blood urea nitrogen, and bilirubin should be determined. Of these tests, the fibrinogen level and platelet count are the most acutely sensitive for severe envenomation.[25] The bitten extremity should be monitored for swelling by means of circumferential measurements every 30 minutes. Prompt and adequate antivenin polyvalent (Wyeth) should be administered intravenously.

Antivenin polyvalent is a hyperimmunized horse serum active against all pit viper species envenomations. Skin testing as described in the packet insert should precede intravenous administration. Caution must always be used, as there is a 10 percent false-negative rate for skin testing, and life-support equipment should be at the bedside.

The intravenous route of antivenin has been well demonstrated in man and animals to work on both systemic and local toxicity.[26,27] Antivenin is most effective if administered within four hours, less effective after eight hours, and of questionable efficacy if administered after 24 hours.[28] Initial dosage should be given according to Table 1. The initial dose must be diluted in 500 mL of intravenous fluids and given over approximately one hour, starting slowly and observing for any adverse reactions.

Unlike most medications, antivenin dosage is not weight or age dependent, but parallels the total amount of venom injected. Children tolerate snake bites poorly and frequently require larger doses of antivenin.[29] Total dosage needs to be individualized to effect a control of the progression of envenomation.[30] The two most common mistakes in snakebite management are giving antivenin unnecessarily or not giving enough when it is indicated.[31] Life-support measures, intravenous epinephrine, and intravenous diphenhydramine should be at the bedside. Two intravenous access catheters should be in place, one for antivenin and the other for emergency drugs and plasma expanders. All support measures should be instituted as soon as they are indicated. It is better to err on the side of giving too much antivenin for a case of moderate to severe envenomation than not giving enough.[5] Once the decision to give antivenin has been made, the patient should be hospitalized and closely observed in an intensive care unit setting until all symptoms have resolved.[32] In moderate to severe envenomations, the patient's blood should be typed and crossed for four

TABLE 1. EXTENT OF ENVENOMATION AND DOSAGE OF ANTIVENIN

Grade	Signs and Symptoms	Initial Treatment
No envenomation	Fang marks present; no local or systemic reactions	No antivenin; local care. Tetanus prophylaxis when indicated. Observation in emergency department for at least four hours
Mild envenomation	Fang marks present; local swelling but no systemic reaction. Pain may be present or absent	3 to 5 vials antivenin
Moderate envenomation	Swelling that progresses beyond the site of the bite with systemic reaction or laboratory changes (eg, fall in hematocrit and fibrinogen levels or platelets or hematuria)	6 to 10 vials antivenin
Severe envenomation	Marked local and systemic reaction. Bleeding diathesis, disseminated intravascular coagulation, shock, or adult respiratory distress syndrome with marked laboratory changes	15 + vials antivenin

units, and urine output should be closely monitored.[33] Heparin has no use in the treatment of disseminated intravascular coagulation associated with snakebite.[5] Pain should be controlled with conventional analgesics.[33] Plasma expanders are frequently necessary to treat shock and may be used in conjunction with vasopressors. Colloids tend to work better than crystalloid plasma expanders because of increased membrane permeability.[5,12,19,34]

Copperhead envenomations are much less severe than rattlesnake envenomations, and they frequently do not require antivenin and rarely cause death.[8]

Coral snake envenomations must be managed quite differently from the pit viper's. Coral snake venom is primarily a neurotoxin and acts by inhibiting acetylcholine at the neuronal synapse.[35] Initial swelling is either absent or very minor. Toxicity may be delayed several hours, and once it becomes apparent, it is frequently too late to reverse the effect.[4] Consequently, in someone who has definitely been bitten by a coral snake, five vials of coral snake antivenin should be administered. This quantity is sufficient to neutralize all the venom a coral snake is capable of injecting during a bite.[13,31] Because 50 percent of coral snakebites are "dry," 50 percent of the victims will be overtreated. The alternative of watching a progressive coral snake envenomation and not being able to control it, however, is much less desirable.[5] Signs and symptoms of coral snake envenomation include parasthesias, increased salivation, fasciculations of the tongue, difficulty with swallowing and phonation, visual disturbances, and a bulbar-type paralysis with ptosis, respiratory distress and failure, convulsions, and shock.[5] As with pit vipers, the earlier the antivenin is administered, the better the response.[31] A minimum 24-hour hospitalization for close observation is indicated in all patients with coral snake envenomations.

A small following of surgeons in the United States still use wound excision and fasciotomy as a primary treatment of snake envenomations.[36,37] This method of excision and fasciotomy has no proponents abroad. By excising the wound one inch equidistant from fang marks to the muscle fascia, it has been shown experimentally in dogs to recover a substantial amount of venom if performed within two hours of envenomation.[38,39] No data, however, show that this approach is superior to appropriate antivenin therapy.[1] Furthermore, to route a person from the snakebite area into the operating room with appropriate workup within two hours is difficult. It is quite likely that in the case of a minimal envenomation, this treatment would cause more morbidity than the envenomation itself. Russell and others,[5,11] who have treated over 700 venomous snakebites successfully and have the largest series of snakebites documented worldwide, condemn the use of excision and fasciotomy as unnecessary and disfiguring. Fasciotomy may be necessary in the event of a true compartment syndrome, although some studies show this condition to be quite rare, as snake envenomation rarely penetrates muscular fascia.[11] Surgical debridement of necrotic tissue should be instituted on the third to sixth day.[5,11] In a computerized search of snakebite literature over the past ten years, only four articles have recommended wound excision with fasciotomy,[36–38,40] whereas 26 regard it as unnecessary in the face of good medical management.[33,41–45]

Intravenous corticosteroids have proven to be both beneficial and useless.[1] In the only controlled trial on their use, they were found to be of no benefit.[46] Most authorities do not recommend their use.[16,47] Eighty percent of patients treated with antivenin will develop serum sickness reaction consisting of fever, malaise, arthralgia, joint effusion, urticaria, abdominal pain, and lymphedema. Three percent of these patients will require hospitalization for severe reactions and require intravenous steroids.[48] Since this reaction is a true serum sickness reaction, corticosteroids are the treatment of choice and diphenhydramine should be used only for symptomatic pruritis.[10,35]

The use of antibiotics is also controversial. There are no controlled studies of their prophylactic efficacy. In the face of tissue necrosis, the judicious use of broad-spectrum antibiotics is indicated. Tetanus immunization is also routinely recommended.[41]

New developments in purified antivenin are currently under way and show hope for more efficacious and safe management of snakebites. When an offending snake is an imported species, the physician should consult the Oklahoma City Zoo (Telephone 405-424-3344) for guidance on availability and choice of antivenin.[9]

The best treatment for snakebite is prevention. Most snakebites could be prevented by observing the following guidelines:

1. Never handle snakes unless you are capable of identifying all the local species and recognize both venomous and nonvenomous snakes.
2. Wear shoes and clothing to protect the lower legs in snake-inhabited areas.
3. Walk slowly through wooded areas, and never put your feet or hands in a place you cannot visualize first. Most snakes would rather avoid you as much you would avoid them.
4. Avoid hiking alone in a snake-infested area.
5. Avoid walking at night without a flashlight in snake-infested areas. Snakes are nocturnal feeders and are more active at night.

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