

## Review Article

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### Poisonous Snakes and Snakebite in the U.S.: A Brief Review

#### Abstract

This article briefly reviews some current ideas regarding snakebite in the United States. Twenty species of native venomous snakes occur and include 15 species of rattlesnakes, the copperhead and cottonmouth, two species of coral snakes, and one seasnake. Snake venoms contain a variety of enzymes and non-enzymatic toxins. Composition may vary geographically, ontogenetically, and individually. As determined by mouse toxicity, most lethal venoms are those of the Mohave rattlesnake (*Crotalus scutulatus*), tiger rattlesnake (*C. tigris*), and pelagic seasnake (*Pelamis platurus*). Venomous snakes may bite without injecting venom and rarely inject more than half their available venom. Some colubrid snakes generally presumed nonvenomous, such as garter snakes, can in rare instances inflict venomous bites.

Most snakebites in the United States are sustained close to the victim's home and usually reach medical aid within an hour. Fishermen, hunters, and backpackers do not seem to be a high risk group. Almost half the bites result from deliberate contact with a venomous snake. Symptoms of pit viper envenomation are reviewed. A persistent drop in blood pressure is the single most reliable indication of dangerous envenomation. About half the coral snake bites do not result in envenomation, but it is serious when it occurs.

Snakebite first aid measures and principles of treatment are reviewed. Two relatively new procedures, elastic bandaging of a bitten limb and use of a powerful small suction device are discussed. Procedures under development include ELISA tests to improve diagnosis and evaluate therapy, antivenoms of higher potency and lower allergenicity, and immunization for high risk individuals. Because of the complexity of evaluating snakebite and the chance that a layman might attempt a naive treatment more harmful than helpful, the emphasis in first aid for snakebite is to get the stricken individual promptly to a hospital.

#### Introduction

Although venomous snakebites are uncommon in the United States (6000-7000 cases annually), they generate a disproportionate amount of interest, anxiety, and controversy. There are several reasons for this. First, snakebite is a situation where a complex organism (a snake) injects a complex secretion (venom) into another complex organism (a human). This generates a staggering number of variables and allows for outcomes that can range from death in less than an hour to a totally trivial injury. Second, the composition and amount of venom a snake injects varies with geographic locality, season, and the age, size, and health of the reptile. Third, there are no good animal models (aside from monkeys) for evaluating the action of snake venoms on man and the effectiveness of treatments. The mouse is widely used, but it is not a very small human being. Finally, the unpredictable course of snakebite and the limited knowledge that most physicians have of venomous snakes and their bites may cause uncritical acceptance of treatment plans.

#### The Snakes and Their Venoms

There are 20 species of unquestionably venomous snakes known from the United States—15 species of rattlesnakes, the copperhead and cottonmouth, two other pit vipers that are closely related to each other, and two species of coral snakes. The pelagic seasnake (*Pelamis platurus*) is occasionally reported in Hawaiian waters and has been recorded once on the coast of extreme southern California. Additionally there are several snake species that are essentially harmless but can, on rare occasions, inflict a mildly venomous bite. Rattlesnakes occur in at least 45 of the contiguous states and four Canadian provinces, although they are usually quite local in distribution, and their numbers have been greatly reduced in the last half century. Species that present the greatest danger to man are the eastern and western diamondbacks (*Crotalus adamanteus* and *C. atrox*), timber rattlesnake (*C. horridus*), prairie rattlesnake (*C.v. viridis*), northern and southern Pacific rattlesnakes (*C.v. oreganus* and *C.v. helleri*), and Mohave rattlesnake (*C. scutulatus*). The pigmy rattlesnake (*Sistrurus miliarius*) causes many bites in the southern states, but

fatalities are unknown. The other species (see Table 1) either are uncommon or occur in regions where they have little contact with man. The copperhead (*Agkistrodon contortrix*), with a wide range in the eastern United States and ability to survive in well populated areas, probably causes more bites than any one species of rattlesnake, but fatalities are virtually unknown. The cottonmouth (*A. piscivorus*) has a more limited range

and prefers swamps and sluggish waterways. Nevertheless it accounts for a significant number of snakebites in the southeastern states and fatalities are on record. Coral snakes are highly secretive and seldom seen. The eastern species (*Micrurus fulvius*), which may reach 1.2 m in length, is potentially lethal; the smaller Arizona species (*Micruroides euryxanthus*) has never caused a human fatality.

TABLE 1. Venomous snakes of the United States and Canada and their geographic distribution.

Scientific Name	Common Name(s)	Distribution
<i>Crotalus adamanteus</i>	Eastern Diamondback Rattlesnake	Coastal plain, from se. North Carolina to Louisiana
<i>Crotalus atrox</i>	Western Diamondback Rattlesnake	Western Arkansas & s. Oklahoma thru most of Texas to se. California*
<i>Crotalus cerastes</i>	Sidewinder	Southern Arizona & s. California deserts*
<i>Crotalus horridus</i>	Timber Rattlesnake Canebrake Rattlesnake (southern populations)	New England to n. Florida, west to central Texas, north to se. Nebraska, s. Wisconsin & se. Minnesota
<i>Crotalus lepidus</i>	Rock Rattlesnake	Mountains and canyons from s. central Texas to se. Arizona*
<i>Crotalus mitchelli</i>	Speckled Rattlesnake Panamint Rattlesnake	Western Arizona, se. Utah, s. Nevada, s. California*
<i>Crotalus molossus</i>	Blacktail Rattlesnake	South central Texas to w. Arizona*
<i>Crotalus pricei</i>	Twin-spotted Rattlesnake	Mountains of se. Arizona*
<i>Crotalus ruber</i>	Red Diamond Rattlesnake	Southwestern California*
<i>Crotalus scutulatus</i>	Mohave Rattlesnake	Trans-Pecos Texas to s. Nevada & adjacent California*
<i>Crotalus tigris</i>	Tiger Rattlesnake	South central Arizona*
<i>Crotalus v. viridis</i>	Prairie Rattlesnake	West Texas and New Mexico north to w. North Dakota, Montana, se. Saskatchewan & adjacent Alberta*
<i>Crotalus v. helleri</i> <i>C.v. oregonus</i>	Pacific Rattlesnake	Most of California north to s. central British Columbia*
<i>Crotalus v. lutosus</i>	Great Basin Rattlesnake	Nevada, w. Utah & Oregon, s. Idaho**
<i>Crotalus willardi</i>	Ridge-nosed Rattlesnake	Mountains of se. Arizona & adjacent New Mexico*
<i>Sistrurus catenatus</i>	Massasauga Rattlesnake	Southern Ontario, w. New York & nw. Pennsylvania southwest to se. Arizona & s. Texas*
<i>Sistrurus miliarius</i>	Pygmy Rattlesnake	Eastern North Carolina to e. Texas & Oklahoma, s. Missouri, sw. Kentucky
<i>Agkistrodon contortrix</i>	Copperhead	Southern New England to Florida panhandle, west to Trans-Pecos Texas, north to se. Nebraska, central Indiana & Ohio
<i>Agkistrodon piscivorus</i>	Cottonmouth Water Moccasin	Southeastern Virginia to central Texas mostly at low elevations, north to central Missouri & sw. Indiana
<i>Micrurus fulvius</i>	North American Coral Snake	Coastal plain from se. North Carolina to sw. Texas, north to sw. Arkansas*
<i>Micruroides euryxanthus</i>	Arizona or Sonora Coral Snake	Southern Arizona & sw. New Mexico*
<i>Pelamis platurus</i>	Pelagic Sea Snake Yellow-bellied sea snake	Hawaii, extreme sw. California coast. Few records, probably based on strays.*

\*Species also occurs in Mexico.

\*\*Other subspecies of *Crotalus viridis* occur in parts of Arizona, Utah, Colorado and Wyoming. These are *C.v. concolor* (Midget Faded Rattlesnake), *C.v. nuntius* (Hopi Rattlesnake), *C.v. cerberus* (Arizona Black Rattlesnake), and *C.v. abyssus* (Grand Canyon Rattlesnake).

Snake venoms are the most complex of animal toxins, containing up to twenty or so biologically active proteins and polypeptides. Many are enzymes. Those that are most important and widespread include phospholipase A and proteases (endopeptidases) that account for some of the hemorrhagic and necrotizing activity, arginine ester hydrolases that contribute to hypotensive and anticoagulant activity, and hyaluronidase that facilitates the spread of venom in tissues. There are also non-enzymatic myotoxins and neurotoxins. It must be emphasized, however, that the effects of snake envenomation result from many venom components acting in concert. The classification of venoms as neurotoxic, hemorrhagic, or myotoxic is usually an oversimplification.

As judged by mouse toxicity, the most lethal rattlesnake venom is that of the tiger rattlesnake (*Crotalus tigris*) followed by that of the Brazilian rattlesnake (*C. durissus terrificus*), Mohave rattlesnake, and midget faded rattlesnake (*C. viridis concolor*). All these venoms contain a powerful neurotoxin. In South American rattlesnakes, it is known as crotoxin; in North American rattlesnakes as Mohave toxin. Recent work shows the two are immunologically very similar (Weinstein *et al.* 1985). Mouse lethal doses for some rattlesnake and other snake venoms are shown in Table 2. Venoms of many snake species show individual, ontogenic, and geographic variation in lethality and other properties. Venoms of two gravid timber rattlesnakes collected the same day on the same hilltop showed a five-fold difference in lethal toxicity (Minton 1953). In at least three rattlesnake species, venoms of young snakes differ markedly from those of adults in protein composition (Minton 1967, Fiero *et al.* 1972, Minton & Weinstein 1986). In all, lethality seems to peak in snakes 6-9 months of age, then decline to adult levels. Reports of unexpectedly severe reactions to bites of young rattlesnakes indicate this variation may be of clinical significance (Reid & Theakston 1978). Over a large part of its Arizona range, venom of the Mohave rattlesnake does not contain Mohave toxin and is much like venom of the western diamondback rattlesnake in lethality and proteolytic and hemorrhagic activity (Glenn *et al.* 1983). Venom from western diamondback rattlesnakes from west Texas and Arizona has greater lethality but lower proteolytic activity than that of snakes from north Texas and Oklahoma (Minton & Weinstein 1986).

TABLE 2. Venom yields from adult snakes of average size and mouse lethal doses. Based chiefly on data from the author's laboratory and from Glenn & Straight (1982).

Species	Average Venom Yield (mg)	Mouse LD/50 mg/kg	
		intra-venous	sub-cutaneous
<i>Crotalus adamanteus</i>	250-500	1.68	14.55
<i>Crotalus atrox</i> (NE)	200-400	3.15	19.52
<i>Crotalus atrox</i> (SW)	100-250	2.07	14.16
<i>Crotalus cerastes</i>	30-60	2.25	12.35
<i>Crotalus horridus</i>	100-200	2.63	9.15
<i>Crotalus lepidus</i>	5-30		11.55 <sup>1</sup>
<i>Crotalus mitchelli</i>	75-150		10.90 <sup>2</sup>
<i>Crotalus molossus</i>	150-300		16.42
<i>Crotalus pricei</i>	2-12	0.95	11.39
<i>Crotalus ruber</i>	200-400	3.72	21.25
<i>Crotalus scutulatus</i>	40-100	0.16	0.31 <sup>3</sup>
<i>Crotalus tigris</i>	5-15	0.06	0.21
<i>Crotalus v. viridis</i>	50-100	1.61	16.15
<i>Crotalus v. helleri</i>	75-150	1.29	3.56
<i>Crotalus v. concolor</i>	10-25	0.28	
<i>Crotalus willardi</i>	2-15	1.61	
<i>Sistrurus catenatus</i>	15-40	0.25	5.25 <sup>4</sup>
<i>Sistrurus miliarius</i>	7-25	3.65	24.25
<i>Agkistrodon contortrix</i>	40-70	10.92	25.60
<i>Agkistrodon piscivorus</i>	100-150	5.11	25.80
<i>Micrurus fulvius</i>	4-16	0.38	1.30
<i>Micruroides euryxanthus</i>	0.5-3	0.90	
<i>Pelamis platurus</i>	0.25-1		0.70 <sup>5</sup>

<sup>1</sup>Based on specimens from the Big Bend region of Texas. Evidence indicates much variation in toxicity among populations of this species.

<sup>2</sup>Based on specimens from San Diego Co., California. A subspecies with much more toxic venom occurs in Baja California.

<sup>3</sup>Based on specimens from populations whose venom contains Mohave toxin.

<sup>4</sup>Based on Indiana specimens. Venom of western populations appears to be less toxic.

<sup>5</sup>Based on specimens from the Coral Sea.

## Who Gets Bitten and Why

Those whose work or recreation takes them outdoors and into remote areas often consider themselves at special risk from snakebite. Unless their activities include hunting snakes, this is not the case. Snakebite throughout the world is largely a matter of contact hours. The snake that bites you is most likely one that lives in your garden or under your house. Most snakebites in the United States, unless they involve deliberate

contact with venomous snakes, occur within a half-mile of the victim's home. Writing of his extensive experience in California, Russell (1980 p. 269) says: "Contrary to popular opinion, most snakebites in the United States occur within a short distance from medical care. In California, over 90 percent of the rattlesnake bites occur within city limits or within 2 miles of city limits in foothill area... Few bites occur in backpackers, serious hunters, or fishermen... In the past 20 years, there has been only one backpacker in the Sierras of California, who I know of, who has been bitten by a rattlesnake, and this happened when he was changing a tire at the end of his hike."

Until about 1950, most snakebites in the United States were associated with what could broadly be called agricultural activities. When I was growing up in southern Indiana, the first snakebite of the season might occur with mushroom hunting in late April but was more likely to be associated with strawberry or blackberry picking in late May or June. Other bites were sustained while lifting rocks, clearing weeds or rubbish, or doing other chores about the farm. Today there are many fewer small farms where most of the work is done by hand, and there has been a great increase in large scale mechanized agriculture. On the other hand, there has also been a decided increase in permanent or seasonal rural living. Rural children ages 5-12 have always been a comparatively high risk group if they live where venomous snakes are relatively plentiful. Children of this age are more likely to go barefoot and be less than careful where they step or where they put their hands. Many small children are insatiably curious about snakes and today are more likely to try to catch reptiles their grandparents at the same age would have killed forthright. Today a much higher percentage of bites involve deliberate contact with venomous snakes. Snake catching, snake handling, and the keeping of venomous snakes in captivity attract people whose motives vary from serious scientific interest to exhibitionism and religious fervor. With a few, abuse of alcohol and drugs may contribute to the risk.

### **The Bite and Its Effects**

An important fact not always appreciated by physicians or laymen is that venomous snakes,

including the most dangerous species, may inject little or no venom when they bite. According to one study, only about half the bites by the widely-feared Asian cobra result in poisoning (Reid 1964). In 16 of 31 coral snake bites in the southeastern U.S., no poisoning developed (Neill 1957, Parrish & Khan 1967). Rattlesnakes, copperheads, and cottonmouths appear to be more efficient biters with about 75 percent of bites resulting in envenomation. Experiments in laboratories in several parts of the world agree that snakes rarely inject more than half their available venom in a single bite. There is some suggestion that a defensive bite directed against a predator or an animal too large to serve as food results in less venom injection than a bite intended to kill prey. However, rattlesnakes attacking prey may also deliver relatively ineffective strikes (Kardong 1986). The degree to which snakes control the amount of venom injected in various situations remains essentially unknown.

When a rattlesnake or other pit viper delivers an effective bite, pain is usually immediate and intense, although occasionally, and usually with a very severe bite, there may be numbness around the area of the bite lasting as long as 30 minutes. Puncture wounds are obvious and bleed longer and more freely than a nonvenomous wound. Swelling usually begins within a few minutes and spreads both peripherally and centrally. Discoloration around the bite usually is evident within a few minutes. If pain, swelling, and discoloration are absent 15-30 minutes after a pit viper bite, the odds are very good no venom was injected. Important exceptions are individuals bitten by rattlesnakes whose venoms are low in those toxins that are responsible for local swelling and discoloration. This includes some populations of the Mohave rattlesnake, speckled rattlesnake, rock rattlesnake, and tiger rattlesnake. Swelling and discoloration may also be minimal in the rare instance when most of the venom is injected intravenously. However, the individual who sustains such a bite quickly shows signs of generalized poisoning, and the diagnosis of envenomation is rarely in doubt. Individuals under the influence of alcohol or drugs may not have normal pain perception and may display bizarre symptoms. A reliable sign of envenomation, usually seen within the first hour after a bite, is painful swelling of lymph nodes in the groin if the bite is on the foot or leg and in the axilla if on the hand or arm.

Tingling of the face and metallic taste in the mouth are common symptoms of rattlesnake bite. Nausea, vomiting, chills, sweating, and thirst are common symptoms of pit viper bites within the first hour. Hemorrhagic blebs may appear within two hours after a bite or be delayed much longer. These symptoms definitely indicate poisoning but not necessarily a serious or life-threatening bite.

A persistent fall in blood pressure is probably the most reliable indication of a serious pit viper bite. This may manifest itself as faintness or loss of consciousness with pallor and a weak, rapid pulse. Other ominous signs are generalized muscular twitching, widespread appearance of large hives (angioneurotic edema), diarrhea, contracted pupils, and sensation of yellow vision. All may develop within an hour after the individual is bitten.

Coral snake bites are not very painful; there is little or no swelling and no discoloration. Fang punctures bleed little and may be almost undetectable a few hours after the bite. Characteristically there is an asymptomatic period that can last up to seven hours. In fact, in about half the cases, no definite signs of poisoning ever develop. Not many cases of coral snake bites have been reported in detail, but symptoms seem to be quite variable. Sometimes, there are pains radiating from the location of the bite or in the abdomen. Serious manifestations include drooping of the eyelids, difficulty in speaking and swallowing, generalized muscular weakness and incoordination, drowsiness, and difficulty in breathing. This can progress to a complete and fatal paralysis.

A curious and not well understood phenomenon is that of venomous bites by presumably nonvenomous snakes of the family Colubridae. Some colubrids have enlarged, grooved fangs in the rear of the upper jaw, and at least two African species are unequivocally dangerous. A few species with this type venom apparatus just enter the United States along the Mexican border, and a few cases of mild venomous bites by them have been reported. The most serious cases reported in the United States have resulted from bites of Asian snakes such as the yamakagashi (*Rhabdophis tigrinus*) and red-necked keelback (*R. subminiatus*) purchased as innocuous pets (Mittleman & Goris 1974, Cable *et al.* 1984). These snakes have enlarged but ungrooved teeth in the rear of the upper jaw. Poisoning by these snakes results in a dramatic decrease in cer-

tain blood clotting factors with resultant internal and external hemorrhages and related complications. *Rhabdophis* is closely related to the North American garter snakes (*Thamnophis*) and water snakes (*Nerodia*), so it is not surprising that a few cases of envenomations characterized by pain, swelling, and ecchymosis have been reported following bites of garter snakes. Two plentiful and widely distributed species, the wandering garter snake (*T. elegans vagrans*) and common garter snake (*T. sirtalis*) have been implicated. Similar cases have been reported following bites by hognose snakes (*Heterodon*). However, perhaps only one in several thousand bites by these presumably nonvenomous colubrids results in poisoning. The answer may be in the nature of the colubrid venom apparatus. Duvernoy's gland, roughly the equivalent of the pit viper venom gland, has a highly toxic secretion in at least some colubrid snakes. But it has a very small lumen for venom storage. And a solid tooth is not so effective for introducing venom into a wound as one that is grooved or hollow. When a colubrid snake feeds, it often holds and chews its prey. This gives time for Duvernoy's gland to secrete venom which is worked into wounds made by the teeth. It probably is significant that a majority of human envenomations by colubrids involve snakes that hung on for several seconds. And in some cases, a captive snake bit when being offered food.

### First Aid and Definitive Treatment

Stated simply, the objectives of snakebite treatment are: (1) Remove venom before it can combine with target tissues. (2) Neutralize venom that cannot be removed. (3) Counteract effects of venom that cannot be neutralized. (4) When all the above fail, repair the damage.

Snakebite treatment in the United States has seen a number of colorful and downright dangerous procedures advocated, sometimes by scientists and physicians. In southern Indiana in the 1920's and 30's, whiskey was a popular remedy; nearly every farmer kept a bottle or two "just in case." Other popular treatments were application of the split body of a freshly-killed chicken to the bite or soaking the bitten part in kerosene or turpentine. First aid techniques involving ligature, incision, and suction have been widely advocated and are sound in principle but

of dubious value in practice. Ligatures increase pain and have little effect in retarding spread of venom. Incision followed by suction does facilitate removal of some venom. However, incisions may damage large blood vessels, nerves, or tendons, provide entry for infection, and be followed by serious blood loss particularly since many snake venoms markedly impair blood coagulation. Excision—cutting out the entire area around a bite—is almost certain to do more harm than good and cannot be recommended. Chilling the area around a bite by use of ice or chemical spray with or without a ligature is ineffective and can do serious damage if continued for a prolonged period. Recently, electric shock using high voltage, low amperage current from an outboard motor, lawn mower, or other source has been suggested as first aid for snakebite. The method is under investigation but cannot currently be advocated.

As already mentioned, most persons bitten by snakes can reach definitive medical care within 30-50 minutes. Realizing this, I have maintained for the last decade or so that the most useful snakebite first aid kit consists of car keys and some coins for a call to a hospital. If at all possible, the actual driving to the hospital should be entrusted to someone else. The stress of driving in traffic does not help a snakebite and vice versa. Calling ahead is important. It gives the emergency room time to prepare for the patient's arrival, and the physician on duty can consult with colleagues or call a Poison Information Center if he wishes. The snake should be brought to the hospital with the victim if possible so its identity can be verified. It is better if the reptile is dead. A live snake in a hospital emergency room is a needless distraction and sometimes a hazard. Identification is especially important if a coral snake is suspected, for harmless mimics of coral snakes occur in many parts of the United States. In cases of bites by exotic snakes kept as pets, identification of the reptile is very important also.

Two relatively new procedures seem to be effective and safe enough that their use can be cautiously advocated for the small number of snakebites that occur under circumstances such that a delay of 30 minutes or more in reaching a medical facility can be anticipated. One is the use of elastic bandages such as are widely used to treat sprains wrapped snugly over the area of

the bite and extended up the bitten limb to the trunk if there is enough bandage. I am told that panty hose make an acceptable substitute. The limb is then immobilized with a splint. The bandages are left in place until the victim reaches the hospital and preparations for beginning intravenous administration of antivenom have been made. Both experimental and clinical evidence indicate the technique is quite effective in preventing absorption of venom from a bitten limb (Sutherland 1983, pp. 22-32). The method was developed in Australia where the important snakes have venoms that are highly lethal but not very destructive locally. If used for treating bites by rattlesnakes and other pit vipers whose venoms cause significant local damage, it must be with the realization that generalized poisoning may be prevented at the cost of increasing local damage. It is the recommended procedure for bites by coral snakes whose venoms are more like those of Australian snakes.

Another new technique is use of a two-chambered suction device that produces one atmosphere negative pressure. This is applied to the bite as quickly as possible *without* making any incisions and left in place about three minutes. Suction can be repeated as necessary. Preliminary animal experiments indicate up to half an injected dose of rattlesnake venom can be removed. The device was also tested on three persons bitten during a rattlesnake roundup in the Southwest with apparently good results (Bronstein *et al.* 1985). Sawyer Products of Long Beach, California, markets the device in a small, light, durable kit suitable for field use.

Scientific evaluation of these and other first aid measures has been possible through development of very sensitive and specific enzyme-linked immunoassay (ELISA) for snake venoms. Although these tests are not generally available in the United States, enough experimental work has been done to demonstrate their value both in diagnosis of snakebite and evaluation of treatment. Since the test can be done in about two hours, determining presence of venom in blood and urine can be helpful in distinguishing a relatively minor bite from a potentially serious one. These assays have also contributed to better understanding the pathophysiology of snake envenomation. One interesting observation is that venoms of some snakes can be detected at the site of a bite after several days.

Although it is not the purpose of this review to discuss hospital management of snakebite, something should be said about antivenom. This is the only clinically effective antidote for snake venom poisoning and is produced commercially by hyperimmunization of horses with snake venoms. The product commonly used in the United States is made against venoms of three species of rattlesnakes and a large neotropical pit viper of the genus *Bothrops*. It is at least somewhat effective in neutralizing venoms of all North American pit vipers and the most important neotropical species as well as those of a few Old World vipers. A coral snake antivenom is available in limited quantity in those states where coral snakes occur. Antivenoms are by no means ideal therapeutic agents. Their neutralizing capacity is relatively low, hence large doses—400 ml or more—sometimes must be given in a very severe envenomation. Not all venoms and venom fractions are equally well neutralized. Antivenoms have little effect against the factors in pit viper venoms that cause local swelling and necrosis. Being foreign proteins, antivenoms commonly cause serum sickness, less frequently anaphylaxis and other forms of immunologic injury. Significant improvement in antivenoms can be anticipated. Experimental antivenom prepared by affinity chromatography has much better neutralizing capacity and is less likely to cause anaphylaxis (Russell *et al.* 1985). A commercial product could be available in a relatively short time. Monoclonal antibodies have been produced against several snake venom toxins, but their therapeutic use has not been explored.

Except in most unusual situations, antivenom should be given in a hospital. This is but one of numerous reasons why hospitalization is recommended for all but obviously trivial snakebites. Blood pressure and other vital signs are more easily monitored, and hypovolemic shock, the commonest cause of death in pit viper bites, can be detected early and corrected. Other dangerous complications of snakebite such as coagulopathy, renal failure, respiratory failure, and infection can also be detected early and dealt with more effectively. In a series of 9 fatal rattlesnake bites in Arizona, two individuals refused to go to the hospital and three others reached the hospital after delays of 1.5 to 29 hours (Hardy 1986).

A single nonfatal snakebite confers no effective immunity, and evidence of immunity in professional and religious snake handlers who have survived many bites is equivocal. Some have died of snakebite after surviving ten or more previous bites. Immunization with toxoid, a strategy highly successful in infections such as diphtheria and tetanus, has been attempted with detoxified snake venoms. The only large scale trial, in the Ryukyu Islands, was not particularly successful (Sawai *et al.* 1969), but the procedure is being considered in some other regions of very high snakebite incidence. In the United States it would be appropriate only for a very small number of individuals. The best single preventative measure is to avoid deliberate contact with venomous snakes. "Illegitimate" snakebites, those sustained by individuals who knowingly place themselves at risk, make up roughly half the snakebites reported in the United States.

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