

Arachnid envenomation

John R. Saucier, MD, FACEP^{a,b,*}

^a89 Beaumont Avenue, University of Vermont School of Medicine,
Burlington, VT 05405, USA

^bDivision of Disaster and Wilderness Medicine, Department of Emergency Medicine,
Maine Medical Center, 22 Bramhall Street, Portland, ME 04102, USA

The class Arachnida of the phylum Arthropoda comprises an estimated 70,000 species. The class is divided into nine orders: Acari (mites and ticks), Araneae (true spiders), Scorpiones (true scorpions), Opiliones (harvesters, daddy-long-legs), Pseudoscorpionidae (false scorpions), Pedipalpi/Uropygi (whip scorpions), Palpigradi (microwhipscorpions), Ricinulei (hooded tickspiders), and Solpugida (sun spiders; Fig. 1). Of these the Acari, Araneae, and Scorpiones have had a direct impact on human health as vectors of disease (Acari) or through direct envenomation (Araneae and Scorpiones). This latter process, and true spiders and scorpions, is the subject matter of this article.

The interaction between man and spiders or scorpions, although ancient, is not one of predator and prey but rather one of accidental encounter. That human envenomation occurs at all is the result of two factors: (1) humans share various neuronal and cellular components with insects and small mammals which are the normal prey for these arachnids (ie, the venom works on humans too) and (2) the wide-ranging habitats for both species places humans and spiders in proximity to one another, resulting in the inadvertent triggering of the animal's defensive mechanism (ie, being in the wrong place at the wrong time). Annually thousands of human deaths result from these chance meetings with perhaps a hundred times that number causing significant morbidity. In the United States, perhaps because of a combination of our mostly nontropical climate and relatively rapid access to health care, there have been no recorded deaths from envenomation since 1968 [1–3].

This article focuses on the medically relevant arachnid species found in North America and selected other arachnids from around the world. While

* Division of Disaster and Wilderness Medicine, Department of Emergency Medicine, Maine Medical Center, 22 Bramhall Street, Portland, ME 04102.

E-mail address: saucij@mmc.org

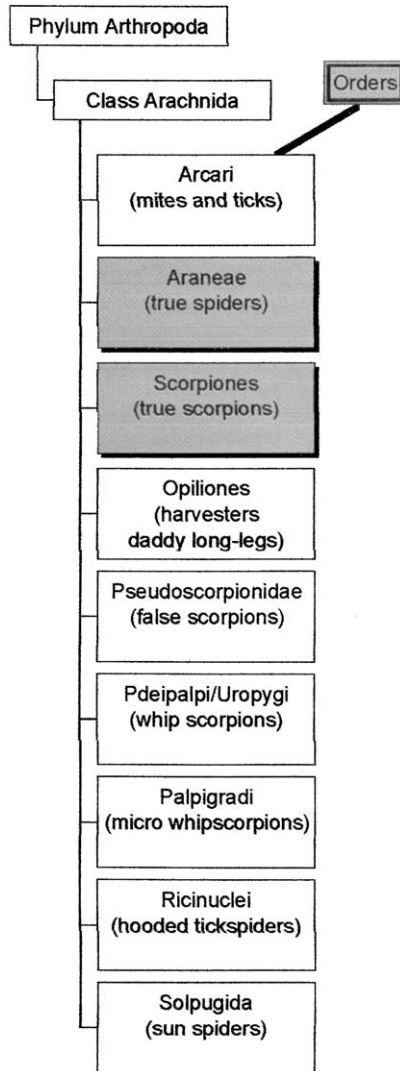


Fig. 1. Taxonomy of arachnids.

it is largely still true that the geographic location of the envenomation assists in determining the species responsible, the booming trade in arachnids as exotic pets should prompt the clinician to inquire into this possibility [4]. Expert advice should be sought in either case; species identification is critical in determining the need for and proper type of antivenom therapy.

Beneath every stone there lurks a scorpion.
Sophocles, c. 450 BC

Scorpions

The order Scorpiones consists of about 1200 species grouped into seven families. Of these Buthidae contain the majority of clinically significant scorpions. In its last available annual report (2001) the American Association of Poison Control Centers (AAPCCC) lists 14,569 calls relating to scorpion stings, of which 851 (6%) required medical attention. No deaths were recorded in this report [5]. There are, however, an estimated 5000 deaths worldwide caused by scorpion stings per annum.

Scorpions vary in size from a few millimeters to 15 cm. They are typically night stalkers and remain hidden during the day under rocks, plant matter, or in shallow burrows that shield them from temperature extremes and predators. They are extremely tolerant of heat but can also live in colder settings and at altitudes of more than 14,000 feet in the Andes. Their flattened and segmented body plan enables them to conform to small enclosures. Clothing, particularly shoes, ground cloths, cooking pots, and so forth make convenient hideaways when in the proximity of people. Resembling miniature lobsters (with which they are only distantly related), scorpions have small heads (prosoma), variably sized claws (pedipalps), eight paired legs, and a segmented “tail” (actually part of the abdomen) ending in a venom-containing telson (Fig. 2). The telson is composed of a vesicle that stores the venom and a stinger (aculacea). It is a single, slender spike that pierces shell or skin, allowing muscular action to squeeze the venom into its prey through twin openings at its base. The size of the scorpion does not correlate with its aggressiveness when confronted or the potency of its venom. In the United States and Mexico it is the relatively small *Centruroides* genus that accounts for the majority of severe envenomations. *Tityus*, *Leirus*, *Mesobuthis*, and

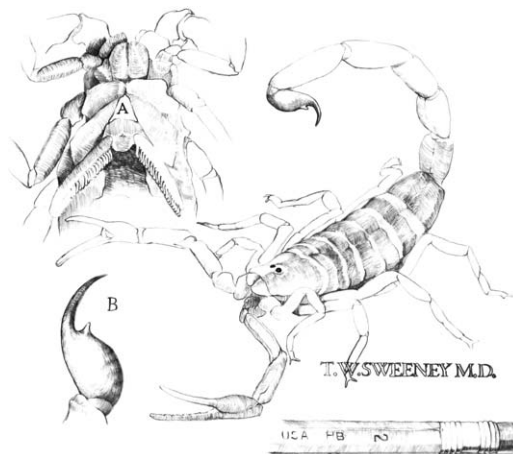


Fig. 2. The size and common features of the *Centruroides* scorpion. Note the typical triangularly-shaped sternum and (B) the tubercule near the base of its stinger. Illustration by Timothy Sweeney, MD.

Androctonus genera are responsible for venom-laden stings in other parts of the world (Table 1) [6].

The venom of scorpions is designed to immobilize their prey so that the scorpion can begin the procedure of mincing it and introducing it into a preoral cavity, where the pieces are partially digested. The pureed portions are then sucked into the gut for further processing. Scorpions do not have teeth or fangs that could bite a human, although in some species the pincers could result in a nasty nip. The scorpion can control the amount of venom released per sting depending on the victim's size [7]. In addition, several rapid stings can deplete the venom to such an extent that subsequent stings introduce minimal additional amounts. Dry stings with no apparent envenomation occurring are common [3].

Scorpion venom is quite variable from species to species, but it is, in general, a mixture of single-chain polypeptides containing neurotoxins that block ion channels, particularly sodium and potassium. Secondary effects caused by a pronounced acetylcholine and catecholamine release also occur. A small amount of hyaluronidase, which allows the spread of the toxin, is present. A variety of other substances including serotonin, lipids, nucleotides, and amino acids have been identified [7–10].

The most notable aspect of a scorpion sting is significant pain at the puncture site that increases markedly with tapping lightly but with minimal noticeable local redness or edema. The typical adult experiences local pain and some paresthesias extending along the affected limb that can last for several hours but with minimal systemic effects. Systemic envenomation, when it does occur, usually causes more morbidity and the majority of deaths in children and the elderly. Initially there is a transient excess cholinergic tone at the neuromuscular junction resulting in salivation, lachrimation, urinary incontinence, defecation, gastroenteritis, and emesis (SLUDGE syndrome). It is, however, the subsequent norepinephrine release causing tachycardia, hypertension, hyperpyrexia, myocardial depression, and pulmonary edema

Table 1
Common venomous scorpions

Scorpion (genera)	Country	Prominent symptoms
Centruroides (bark scorpion)	US, Mexico, West Indies, Northern South America	Local pain, paresthesias, hypertension, tachycardia, confusion, saccadic eye movements
Leiurus (death stalker)	North Africa, Middle East	Hypertension, tachycardia, pulmonary edema, hyperglycemia
Tityus (devil scorpion)	Central and South America, West Indies	Local pain, tachycardia, hypertension, pulmonary edema, pancreatitis
Androctonus (fat-tailed scorpion)	North Africa, Middle East, Pakistan and western India	Local pain and burning, diaphoresis, convulsions, confusion
Mesobuthis (red scorpion)	India	Local pain, paresthesias, hypertension, tachycardia, hyperkalemia, hyperglycemia

that can be fatal. Central nervous system (CNS) effects commonly include confusion, agitation, ataxia, and myoclonic and dystonic movements (“the restless child with roving eyes”) [11]. Other complications including myocardial infarction without coronary thrombosis [12–14], hyperglycemia, pancreatitis [15], and ischemic strokes [16,17] have also been described. The onset of systemic symptoms is usually within 6 hours of the sting, with a crisis in about 12 hours then gradual recovery, although pain, paresthesias, and tachycardia can persist for 2 weeks.

In the United States, *Centruroides exilicauda* (also known as *Sculpurata*), one of 41 species of bark scorpions, is responsible for the bulk of severe envenomations. This slim yellow–brown arachnid is 4 to 7.5 cm in length at maturity and has slender pincers, a triangular-shaped sternum, and a tubercle near the base of its stinger (Fig. 2). Its range is limited to southern Arizona, the bottom of the Grand Canyon, and the area surrounding Las Vegas and western New Mexico [18]. Five thousand incidents of scorpion stings occur in Arizona each year with only 5% resulting in systemic symptoms.

Field management of suspected scorpion envenomation should start with calming the victim and determining the potential for systemic symptoms by proper identification of the scorpion involved, which might be difficult if the sting is from an imported exotic scorpion, if was found in the clothing or luggage of a returning traveler, or even if the sting occurs in an endemic area when the offending scorpion has been reduced to a battered pulp. Recognizing that 95% of incidents will yield only local or mild systemic symptoms should reassure the provider and victim, even if the scorpion does resemble *C. exilicauda*. If the sting is immediately recognized, the use of a Sawyer extraction device (Sawyer Products, Safety Harbor, Florida) can be used, although its effectiveness has not been tested for scorpion envenomation. Local cold application, immobilization, light compression wrapping, and acetaminophen for discomfort should suffice during a 6-hour observation period [10,19–22]. Anaphylaxis to scorpion stings is rare but should be anticipated in victims who have a prior history of stings.

There is considerable controversy surrounding the use of antivenom for scorpion stings, even for patients who have severe systemic symptoms. Antivenom appears to be species specific and should be administered within 1 hour of envenomation [23], which limits its use to regions that have a known single species and access to a local health station where the appropriate antivenom can be stored, which seems to be an effective policy in some parts of Mexico and Morocco [24]. In the United States, the University of Arizona can provide antivenom for *Centruroides* species for severe systemic envenomation. The antivenom is derived from goat serum and has a 3% incidence of immediate sensitivity and a 60% chance of serum sickness following its use [25]. In addition, this antivenom is not U.S. Food and Drug Administration approved, so it cannot be transported across state lines, limiting its use to Arizona only. Antivenom, while helping with local pain and paresthesias, does not seem to be effective in combating more severe

complications such as stroke and pulmonary edema, most likely because the venom does not have a major direct toxic effect on the brain and heart (those complications are secondary to the pronounced catecholamine release), which is why the antivenom should ideally be administered on site within 1 hour of envenomation, to curtail the discharge of norepinephrine. The Catch 22 is that one might not know if envenomation has occurred at that point, whereas if one waits for severe symptoms it might be too late. Local medical practice parameters might be helpful in aiding clinical judgment [26–29].

Supportive care is probably of more benefit than antivenom [30,31]. When the patient has been transported to the hospital, the key is to watch for signs of severe envenomation and to blunt their effects before more damage is done. Concerning signs are ECG changes (ST elevation, tented T waves, and ventricular arrhythmias) [11,32,33], elevated CK-MBs or troponin, hyperglycemia, altered mental status, or seizures. Extreme hypertension, hyperpyrexia, and hypotension should be managed aggressively. Nitroprusside is the agent of choice for emergent hypertension. Bawaskar strongly recommends the early use of prazosin 0.25–0.5 mg p.o. in suspected severe red scorpion envenomation and claims to have seen significantly reduced mortality in his hospital in India with its use [34–38]. Pulmonary edema is felt to be a combination of myocardial depression and the direct action of norepinephrine on the pulmonary vascular bed. Treatment is with oxygen, mechanical ventilation as needed, digitalis, diuretics, and the use of β -blockers if the ejection fraction is not severely depressed ($>50\%$). The hypotensive patient who has pulmonary edema might require dobutamine [39] or dopamine. Insulin, particularly in the setting of hyperglycemia, might have some advantage [40]. Steroids have not proved to be of benefit, although they are still used. Angiotension Converting Enzyme (ACE) inhibitors seem to be detrimental by increasing the levels of bradykinin. Morphine can provoke arrhythmias and nifedipine can initiate heart block and significant hypotension [41]. None of these interventions or the avoidance of others has been subjected to double-blind, prospective study.

Prevention of stings in areas where scorpions live is based on knowledge of their habitats and lifestyles. Shake out clothing well each morning before wearing, particularly footwear. Turn over ground cloths carefully because scorpions can attach themselves to the undersides. Wear appropriate closed-toe footwear when walking through potential habitats. Use gloves and long-sleeved shirts when gathering firewood or clearing brush, and monitor the exploratory wanderings of children [42].

...and along came a spider...

Spiders

True spiders (order Araneae) are a diverse group of arachnids comprising 34,000 named species divided into 105 families [2,7]. All true spiders have

characteristic unsegmented bodies and the ability to make silk, although not all use the silk to produce webs. Spiders produce venom that they use to subdue their prey and to start the digestion process. Spiders must liquefy their victims before they can ingest them because they lack teeth. For the venom to be a significant risk for humans, the spider must have strong enough fangs to break the skin and have sufficient venom injected to exert an effect. Several families of spiders will cause at least some local reaction after envenomation: Hexathelidae (funnel web spiders), Salticidae (jumping spiders), Lycosidae (wolf spiders), Clubionidae (running or sac spiders), Sparassidae (huntsmen), and Araneidae (orb-weaving spiders). Theridiidae (widow spiders) will result in systemic symptoms but with minimal tissue damage. Agelenidae (hobo and grass spiders) present a mixed bag, with hobos resulting in some severe tissue and systemic symptoms in a minority of cases, whereas grass spiders cause mild systemic symptoms only. Sicariidae (Loxosceles; recluse and fiddlehead spiders) cause significant local necrosis and systemic symptoms. Tarantulas (family Theraphosidae), although having a painful bite, do not cause local necrosis but will initiate local dermatitis from their urticating hairs (Table 2).

True spiders have a worldwide distribution and many thrive in heavily populated areas, resulting in many biting episodes per year. The AAPCC data for 2001 lists 20,204 calls for spider bites [5]. More than 50% of these were not linked to a specific species. One thousand eight hundred ninety patients (19.7%) sought medical attention for these bites, but there were no deaths. The worldwide incidence of spider bites is unknown. The importance of identifying the spider species responsible for the individual bite cannot be overemphasized. Even squashed spiders can be identified, so it is worth preserving the arachnid in a container with alcohol for expert reviews. This practice will spare the patient considerable anxiety and might obviate the need for antivenom injections. The following sections of this article divide spider envenomations into two major categories: necrotic arachnidism and arachnidism without tissue necrosis. Individual species are discussed further under the general categories. A final section focuses on tarantulas and the clinical effects of their urticating hairs.

Table 2
Venomous spiders

Family	Common name	Tissue injury	Systemic symptoms
Hexathelidae	Funnel web spiders	+/-	++
Salticidae	Jumping spiders	+/-	-
Clubionidae	Running/sac spiders	+/-	+/-
Araneidae	Orb-weaving spiders	+	-
Agelenidae	Hobo spiders	++	+/-
Agelenidae	Grass spiders	+	+/-
Sicariidae	Fiddlebacks	++++	++
Theridiidae	Widow spiders	-	+++
Theraphosidae	Tarantulas	+	-
Sparassidae	Huntsmen	+/-	-

Necrotic arachnidism

Necrotic arachnidism is defined as local necrosis caused by a spider bite. Venom in these spiders contains varying amounts of hyaluronidase and a suspected levarterenol-like substance (although this might be a secondary effect) that causes significant local ischemia, enhancing the liquefaction process. A mixture of bacteria from the spider fangs might also further tissue necrosis as it starts to process its prey [43]. Subsequent infection only adds to the extent of tissue injury. In some spider species (eg, jumping spiders), local tissue damage is primarily the result of external digestion or histamine release and is self-limited. *Loxocoles* species represent the other end of the spectrum, with progressive ulceration and deepening necrosis secondary to significant envenomation. Differential diagnoses include necrotizing fasciitis, erythema chronicum migrans from *Borrelia*-infected tick bites, and anthrax. Significant management differences result from the actual diagnosis made. History might be helpful, even if the spider is not available for identification.

Spiders that cause minimal local injury

There are 2800 species of Salticidae (jumping spiders), most of which are tropical, but 300 species live in temperate climates. These spiders are small (<1.5 cm long; length given will represent body length only; leg length in addition is usually at least that of body length) and are active in daylight hours. They have characteristic jumping movements as they hop on their fourth pair of legs. Looking for prey in plants, they might be frequent biters of gardeners, resulting in a local small vesicle or wheal that will resolve in 1 to 2 days without treatment. *Phidippus audax* is the most common species encountered in North America [2,44].

There are 2000 species of Lycosidae (wolf spiders) with about 100 living north of Mexico. Medium in size, these hairy spiders are, on average, 2.5 cm long. They are primarily daytime hunters in the United States, running over rocks and ground cover seeking prey. Their bite is painful and results in some local necrosis that might be related to digestive juices rather than venom. Local wound care is usually sufficient. There is no available antivenom. Bites occur most commonly on hands and feet while clearing shrubs or walking unprotected through wolf spider habitats. *Lycosida tarantula* of Italy is the spider of legend that causes envenomated persons to become lethargic then to dance wildly in the streets as part of their cure [45]. It is, however, not venomous and might have been confused with a local spider of the widow family.

There are 2500 species of Araneidae, also known as Argiopidae (orb-weaving spiders) with several hundred inhabiting North America. These are the families of spiders (as well as two others) that build traditional orb-shaped webs. They are web-based hunters of small to medium size (usually <3 cm long). The golden orb weaver *A aurantia* is a typical garden spider whose

painful bite can result in a local, self-limited vesicle that disappears in 24 hours [46].

Sparassidae (huntsmen) are a family of house-dwelling, small (1 cm body length), long-legged spiders. They are responsible for the majority of bites in Northern Australia. They cause a self-limited local erythematous skin lesion with rare mild systemic symptoms.

Spiders that cause local necrosis and systemic symptoms

There are 1500 species of Clubionidae (running or sac spiders) with less than 100 in North America. These small nocturnal spiders often mimick large ants in their movements, and they can dwell in buildings or vegetation, where they form tube-shaped webs in which to hide. The genus *Chicaricanthium* has been responsible for a number of urban bites in the Boston area. The bite is painful and results in a pruritic wheal that is short lived. Mild systemic symptoms of nausea or abdominal pain have been rarely reported [47,48].

There are 500 species of Agelenidae (hobo spiders, grass spiders [see below]), about 300 of which are found in the United States. These small (1 cm long) spiders are dwellers in cities and suburban woods, building funnel-shaped webs from which they pounce out upon prey that disturb their net. Their bite causes local blistering and their venom is associated with systemic findings of headache, lethargy, and hallucinations. The hobo spider (*Tegenaria agrestis*), an introduced European house spider, lives predominately in Washington, Oregon, Utah, Idaho, and Montana, and most severe bites occur during the winter months. Local wound care and supportive treatment is recommended [49,50].

Spiders that cause extensive tissue necrosis and systemic symptoms

There are 129 species of Sicariidae (crab, fiddleback, brown, and recluse spiders) in two genera, of which *Loxosceles* is the most medically significant in North America. Two species *L. reclusa* and *L. arizonica* in the United States have been identified for at least 50 years as causing significant tissue necrosis in humans. The AAPCC's 2001 data notes that 13% of the calls concerning spider bites were related specifically to the brown recluse with fully 39% of those victims seeking medical attention. Their venom contains a mixture of enzymes including hyaluronidase, which causes tissue spread and sphingomyelinase D, which is responsible for cellular destruction. Hemolysin contributes to the systemic effect that can be seen, although sphingomyelinase D can also stimulate hemolysis indirectly through complement activation [51,52]. *L. reclusa* inhabits much of the south central United States, sparing the Atlantic and Pacific coasts and states along the Canadian border. Transients can occur, hiding in travelers' clothing and luggage. The recluse is a small spider (1.5 cm long) with the characteristic violin (fiddle) design on its back and six eyes in three pairs (dyads) instead of the usual eight (Fig. 3) [53]. It is a night stalker, so bites usually occur as the spider is tangled in clothing or

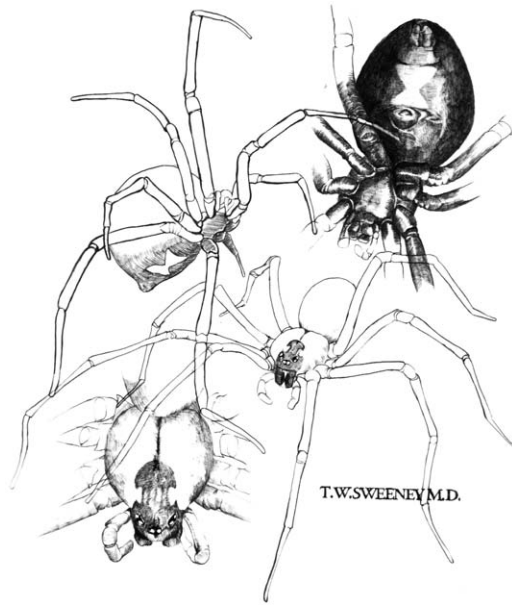


Fig. 3. Illustration of recluse (*Bottom*, dorsal violin shape) and widow (*Top*, ventral hour-glass shape) spiders. Illustration by Timothy Sweeney, MD.

bedding and strikes out defensively. There might be no knowledge of the spider's bite, making the diagnosis particularly difficult. If recalled, the bite is initially painful but then the pain subsides in several hours.

The initial lesion is small and erythematous and might be surrounded by a lighter, reddened ring resembling that of Lyme disease. (The geographic distribution of the two processes in the United States shows little overlap, which might be helpful diagnostically) [54]. There is a central depressed area of bluish necrosis with surrounding blanching then a circling erythema. A central nodular or raised wheal-like lesion is unlikely to be *Loxosceles*. The lesion can progress to a prominent serous or frankly bloody bulla in 24 to 72 hours. In 3 to 5 days there can be significant spreading eschar that sloughs to reveal a deep ulcerative base. Lipolysis by the venom might continue to extend the ulcer. Unless bacterial infection occurs, the ulcer might be more burning or itching rather than painful. Despite a significant ulcer, the patient does not appear to be toxic [55]. Healing can take more than 2 weeks to occur and excision and grafting has been considered in the treatment of chronic ulcers.

The primary concern should be in identifying whether or not the lesion is caused by the recluse spider. Several authors feel that in endemic (and even nonendemic) areas that any ulceration is labeled as a recluse bite if no other cause is readily found. This practice has led to ineffective and dangerous treatment, not counting the financial expense and marked anxiety for the patient [55–57]. As Sean Bush, MD recently said, “Show me the spider” is the

proper position to take in evaluating recluse bites and the literature surrounding them (Wilderness Medicine Newsletter, Spring 2003). Lacking a “smoking gun,” ELISA assays of the venom in the wound hold some diagnostic promise but are not yet widely available [58]. If the lesion is a recluse bite, then treatment is initially ice, elevation, immobilization, and loose compression wrapping. Systemic symptoms seem to be related to hemolysis and platelet aggregation with resulting renal damage, but these are rare, occurring less than 1% of the time. Adequate hydration following renal function parameters and thrombosis profiles can be helpful in preventing more severe clotting events or renal failure.

Much controversy surrounds the treatment of significant ulcers. Early excision does not seem to be useful and can result in more severe tissue damage than conservative treatment. No benefit could be found in an animal model comparing dapsone, hyperbaric oxygen, or the immune modulator cyproheptadine with placebo [56]. Prednisone seems to be of little use, but antihistamines might be helpful for pruritis. Local skin care is paramount with close follow-up for signs of infection. Prophylactic antibiotic use is probably unnecessary in immune-competent patients, and admission to the hospital should rest on clinical judgment. Antivenom is not available in the United States, but early (<4 hours) administration of F(ab) and monoclonal antivenom seems to limit necrosis in animal models [59,60].

Spiders that cause predominantly systemic symptoms and minimal tissue injury

Agelenopsis (grass spiders; subfamily of Agelenidae) have similar hunting techniques and size (1 cm length) to hobos, described previously. They have a bite that does not result in tissue damage but does produce a syndrome consisting of headache, disorientation, and nausea. These symptoms can easily be confused with encephalitis, CNS tumor, or toxin ingestion without appropriate patient history. No specific treatment is recommended other than supportive care and a search for other etiologies [61].

The 74 species of Hexathelidae (funnel web spiders of Australia) living in Eastern Australia are notably aggressive large spiders (4 cm long). Bites of one species, *Atrax robustus*, has resulted in all the deaths from this family. Its 4- to 5-mm fangs are sturdy enough to penetrate fingernails and tenacious to the point of making the spider difficult to remove from its victim. There is severe pain at the site of envenomation but no local necrosis [62–64]. Its venom is complex and includes a substance that, while not an anticholinesterase, acts to increase acetylcholine at synaptic receptors. There is often an initial phase of hyperstimulation with tachycardia, hypertension, vomiting, and salivation that might progress within 2 hours of the bite to hypotension, respiratory depression, and pulmonary edema.

Males wander from the web in search of females and contain the more potent venom of the two genders. Deaths have occurred within 4 hours of

envenomation and are usually children under the age of 12 years. Thirteen deaths were recorded in Australia from 1927 to 1984. Initial treatment consists of wrapping the involved extremity, immobilization, and transporting to a health care facility. Depending on the length of the transport, ventilatory support might be needed and should be anticipated. The use of a Sawyer extractor on the scene might have some theoretical benefit. Antivenom, readily available in Australia, is derived from rabbit serum and reverses systemic symptoms rapidly.

There are 2000 species of Theridiidae (comb foot spiders, widows), of which 200 species live in North America. These small- to medium-sized (1 cm long) spiders are often found suspended from their irregularly shaped webs. They are typically found around dwellings and there are frequent interactions with humans. The archetypical scenario is the perineal bite sustained during a visit to the camp outhouse; however, in reality most bites actually occur on the extremities. The bite is painful but leaves only a small, flat wheal on the skin surface (Fig. 4). A minority of individuals who experience envenomation will not recall a bite at all. The AAPCC 2001 data noted, as for the brown recluse, that 13% of their calls for spider bites involved a suspected black widow (*Lactrodectus mactans*). Thirty percent of these victims sought medical attention.

The black widow large enough to envenomate a human is indeed the female of the species and is identified by its glossy, black body and red hourglass marking on its ventral surface (see Fig. 3). The male of the species is much smaller and nonvenomous to humans. It treads lightly around its mate so that it will not live up to the legend. There are also brown or gray widows with slightly different coloration and markings. The venom of *L. mactans* contains α Lactrotoxin, a neurotoxin that results in presynaptic neurotransmitter release [65,66]. The resulting clinical effects are rapid in onset, and within 1 hour there are notable muscle spasms and severe abdominal pain with rigidity enough to simulate peritonitis on examination. Hypertension and diaphoresis are prominent. A facial swelling called *Lactrodectus facies* is described. Recovery is excellent, but 3 to 7 days of pain and prostration can occur. Antivenom derived from horse serum is available through Merck in the United States and is reserved for victims who have severe systemic symptoms. Allergic reactions, particularly an arthrus reaction, occurring 7 to 14 days after administration of the antivenom is seen in 75% of patients who receive it. One death has been reported as an immediate anaphylaxis response to the antivenom. A preliminary dose to test for allergy is required [67–69]. Because black widow bites rarely result in death, supportive care should be the rule until a safer antivenom is available. Initial treatment is supportive with local ice, loose compressive wrapping, immobilization, and the considered use of a Sawyer extractor. One must be careful not to remove the wrapping until the patient is in a monitored situation because sudden deterioration can occur. Severe pain can be treated with analgesics as needed. Intravenous calcium gluconate is ineffective for pain in most case reports.



Fig. 4. Black widow bites. Wheals on skin surface are designated by arrows. (Courtesy of Kevin Wallace, MD).

Spiders that cause local skin irritation but no envenomation

There are 800 species of Theraphosidae (tarantulas, baboon spiders) worldwide, with 30 species in the Southwest United States. These large (>5 cm long), hairy spiders, though native in the United States to only its Southwest region, make popular pets and so are found throughout the country. The 2001 AAPCC data lists only 1% of the calls for spider bites related to tarantulas with 16% of victims seeking medical attention. Although the bite of the tarantula is quite painful, there is little evidence of envenomation in humans, perhaps because these spiders are large enough to subdue their prey with only minimal use of venom. Unless infection occurs,

there is little inflammation at the puncture site. The primary health concern for these spiders is the urticating hairs that cover their body. As a defensive mechanism they can rub the hairs off the dorsum of their bodies and fling them at their harasser. For humans this results in a local urticarial response. If the hairs penetrate the cornea, however, local keratitis or even uveitis can occur as well as the more subacute ophthalmia nodosa with granulomatous lesions surrounding the hairs in the cornea. Suspected eye involvement should prompt an ophthalmologic referral. Field treatment makes one more use for duct tape in assisting the removal of the offending hairs from the skin (not the cornea). Diphenhydramine, or systemic steroids in more severe reactions, might attenuate the histamine-related irritation [70–74]. The puncture wounds from the spider's fangs require local wound care, follow-up for signs of infection, short-term analgesia, and a tetanus booster.

Arachnophobia

In reviewing the Index Medicus for current references for this article, 15% of the articles retrieved contained research done on arachnophobia. It is estimated that 6% of the population has significant fear of spiders. This fear in children might appear as early as age 5 and is related to some conditioning event or modeling similar reactions of their parents. The prevalence of this phobia might contribute to the anxiety, tachycardia, and hypertension seen in the early stages of suspected envenomation. It certainly hinders identification of the offending species as the victim swiftly retreats or pulverizes the arachnid beyond ready recognition. It might also result in unanticipated trauma because avoidance behaviors can be extreme. In any organized outdoor experience, particularly with children or adolescents, it is worth spending some time discussing these fears and how to not be victims to them [75–77].

Summary

Arachnid envenomation represents a significant health problem in North America and worldwide. Good access to care, supportive treatment, and (rarely) antivenom provide the necessary ingredients for a full recovery. Understanding the value of arachnids in the ecosystem might allay fears that can exacerbate the potential seriousness of an envenomation and lead to a more pleasant experience with nature in general. Leaders of outdoor expeditions should consider acquainting their students with the arachnid hazards in their region. Everyone should be aware of preventative measures to avoid bites and stings and simple first aid principles of wrapping and immobilization should they occur. The clear need for identifying the spider or scorpion involved is pivotal in beginning any treatment protocol (Box 1).

Box 1. Care of arachnid envenomation

1. Sawyer extractor
2. Local ice
3. Loose compressive wrapping
4. Immobilization
5. Hydration
6. Six-hour observation for systemic symptoms
7. Supportive care
 - a. Consider prazosin, 0.25 mg, for hypertension
 - b. Benzodiazepines for convulsions or agitation
 - c. Temperature control
 - d. Oxygen and ventilatory support as needed
 - e. Dobutamine or dopamine for refractory hypotension
8. Antivenom
 - a. Reserve for severe systemic symptoms refractory to supportive measures
 - b. Monitor carefully for anaphylaxis

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References

- [1] Suchard JR, Connor DA. Scorpion envenomation. In: Auerbach PS, editor. Wilderness medicine. 4th edition. St. Louis (MO): Mosby; 2001. p. 839–63.
- [2] Levi HW, Levi LR. Spiders and their kin. New York: Golden Press; 1968.
- [3] Otten EJ, Blomkalns AL. Venomous animal injuries. In: Adams J, Barkin RM, Barsan WG, et al, editors. Rosen's emergency medicine: concepts and clinical practice. 5th edition. St. Louis (MO): Mosby; 2002. p. 785–800.
- [4] Rubio M. Scorpions. Happaage (NY): Barron's Educational Series, Inc; 2000.
- [5] Litovitz TL, Klein-Schwartz W, Rodgers GC, et al. 2001 Annual report of the American Association of Poison Control Centers. W.B. Saunders, Philadelphia (PA) *Am J Emerg Med* 2002;20(5): p. 429.
- [6] Farghly W, Ali F. A clinical and neurophysiological study of scorpion envenomation in Assiut, Upper Egypt. *Acta Paediatr* 1999;88(3):290–4.
- [7] Boyer LV, McNally JT, Binford GJ. Spider bites. In: Auerbach PS, editor. Wilderness medicine. 4th edition. St. Louis (MO): Mosby; 2001. p. 807–38.
- [8] Becerril B, Marangoni S, Possani L. Toxins and genes isolated from scorpions of the genus *Tityus*. *Toxicon* 1997;35(6):821–35.

- [9] Batista C, Gomez-Lagunas F, Lucas S, et al. TcI, from *Tityus* Cambridge, is the first member of a new subfamily of scorpion toxin that blocks K(+) -channels. *FEBS Lett* 2000; 486(2):117–20.
- [10] Bentur Y, Taitelman U, Aloufy A. Evaluation of scorpion stings: the poison center perspective. *Toxicon* 2003;41(3):261–7.
- [11] Das S, Nalini P, Ananthakrishnan S, et al. Scorpion envenomation in children in southern India. *J Trop Med Hyg* 1995;98(5):306–8.
- [12] Cupo P, Hering S. Cardiac troponin I release after severe scorpion envenoming by *Tityus serrulatus*. *Toxicon* 2002;40(6):823–30.
- [13] Blum A, Jawabreh S, Gumanovsky M, et al. Scorpion envenomation and myocardial damage. *Isr Med Assoc J* 2000;2(4):318–9.
- [14] Benvenuti L, Douetts K, Cardisi J. Myocardial necrosis after envenomation by the scorpion *Tityum serrulatus*. *Trans R Soc Trop Med Hyg* 2002;96(3):275–6.
- [15] Joshi S, Shah S, Pichumoni C. Scorpion sting pancreatitis/pancreatopathy. *J Assoc Physicians India* 1999;47(3):352–4.
- [16] Thacker A, Lal R, Misra M. Scorpion bites and multiple cerebral infarcts. *Neuro India* 2002;50:100–1.
- [17] Fernandez-Bouzas A, Morales-Resendiz M, Llamas-Ibarra F, et al. Brain infarcts due to scorpions stings in children: MRI. *Neurorad* 2000;42:118–20.
- [18] Dodge NN. Poisonous dwellers of the desert. Globe (AZ): Southwest Parks and Monuments Association; 1974.
- [19] Tilton B, Hubbell F. Bites and stings. In: *Medicine for the backcountry*. 3rd edition. Guilford (CT): The Globe Pequot Press; 1999. p. 158–62.
- [20] Schimelpfenig T, Lindsey L. Poisons, bites, and stings. In: *Wilderness first aid*. Lander (WY): NOLS Publications; 1991.
- [21] Forgey WW. Arthropod envenomation. In: *Practical guidelines for wilderness emergency care*. 2nd edition. Guilford (CT): The Globe Pequot Press; 2001. p. 87–91.
- [22] Holliman CJ, Dey CC. Environmental illnesses. In: VanRooyen MJ, Kirsch TD, Clem K, et al, editors. *Emergency field medicine*. New York: McGraw-Hill; 2002. p. 693–8.
- [23] Mazzei de Davila C, Davila D, Donis J, et al. Sympathetic nervous system activation, antivenom administration and cardiovascular manifestations of scorpion envenomation. *Toxicon* 2002;40(9):1339–46.
- [24] Osnaya-Romero N, de Jesus Medina-Hernandez T, Flores-Hernandez S, et al. Clinical symptoms observed in children envenomated by scorpion stings, at the children's hospital from the State of Morelos, Mexico. *Toxicon* 2001;39(6):781–5.
- [25] Lovecchio F, Welch S, Kimmons J, et al. Incidence of immediate and delayed hypersensitivity to *Centruroides* antivenom. *Ann Med* 1999;34(5):615–9.
- [26] Gueron M, Ilia R. Is antivenom the most successful therapy in scorpion victims? *Toxicon* 1999;37:1655–7.
- [27] Banner W Jr. A scorpion by any other name is still a scorpion. *Ann Emerg Med* 1999; 34(5):669–70.
- [28] Ghalim N, El-Hafny B, Sebti F, et al. Scorpion envenomation and serotherapy in Morocco. *Am J Trop Med Hyg* 2000;62(2):2777–83.
- [29] Borges A, Tsushima R, Backx P. Antibodies against *Tityus discrepans* venom do not abolish the effect of *Tityus serrulatus* venom on the rat sodium and potassium channels. *Toxicon* 1999;37(6):867–81.
- [30] Banner W. A scorpion by any other name is still a scorpion. *Ann Emerg Med* 1999;34: 669–70.
- [31] Gibly R, Williams M, Walter F, et al. Continuous intravenous midazolam infusion for *Centruroides exilicauda* scorpion envenomation. *Ann Emerg Med* 1999;34(5):620–5.
- [32] Mahadevan S. Scorpion sting. *Indian Pediatr* 2000;37(5):504–14.
- [33] Mathur A, Verma G, Cehlot R, et al. Non-cardiac pulmonary oedema in scorpion bite. *J Assoc Physicians India* 1993;41(6):398.

- [34] Bawaskar H, Bawaskar P. Scorpion sting: a review of 121 cases. *J Wilderness Med* 1991;2: 164–74.
- [35] Bawaskar H, Bawaskar P. Scorpion sting: a review of 121 cases. *J Wild Med* 1991;2: 164–74.
- [36] Bawaskar H, Bawaskar P. Scorpion sting. *J Assoc Physicians India* 1998;46(4):338–92.
- [37] Kulkarni A. Prazosin therapy and scorpion envenomation. *J Assoc Physicians India* 2001; 49:1213.
- [38] Bawaskar H, Bawaskar P. Indian red scorpion envenoming. *Indian J Pediatr* 1998;65(3): 383–91.
- [39] Souheil E, Nouirs S, Besbes-Ouanes L, et al. Dobutamine in severe scorpion envenomation: effects on standard hemodynamics, right ventricular performance and tissue oxygenation. *Chest* 1999;116(3):748–53.
- [40] Murthy K, Hase N. Scorpion stings and role of insulin. *J Assoc Physicians India* 1994; 42(2):172–3.
- [41] Deshpande S, Alex A. On the management of scorpion stings. *Heart* 2000;83(5):582.
- [42] Groshong T. Scorpion envenomation in eastern Saudi Arabia. *Ann Emerg Med* 1993; 22(9):89–95.
- [43] Monteiro C, Rubel R, Cogo L, et al. Isolation and identification of *Clostridium perfringens* in the venom and fangs of *Loxosceles intermedia* (brown spider): enhancement of the dermonecrotic lesion in loxoscelism. *Toxicon* 2002;40(4):409–18.
- [44] Huntley A. Jumping to unfortunate conclusions: *Phidippus audax*, the most common cause of spider bites. *Dermatol Online J* 1997;3(2):5.
- [45] Anonymous. The tarantula—poison spider or a case of mistaken identity? *Adverse Drug React Toxicol Rev* 1996;15(4):199–202.
- [46] Haddad C. Symptoms of the bite of an orb-web spider *Araneus apricus* (Araneae: Araneidae). *S Afr Med J* 2002;92(7):528–9.
- [47] Reifsnnyder D. Spider bites. *Hosp Pract* 1994;29(9):15.
- [48] Foradori M, Keil L, Wels R, et al. An examination of the potential role of spider digestive proteases as a causative factor in spider bite necrosis. *Comparative Biochemistry & Physiology* 2001;130(2):209–18.
- [49] Sadler M, Force R, Solbrig R, et al. Suspected *Tegenaria agrestis* envenomation. *Ann Pharmacother* 2001;35(11):1490–1.
- [50] Vetter R, Roe A, Bennett R, et al. Distribution of the medically-implicated hobo spider (Araneae: Agelenidae) and a benign congener, *Tegenaria duellica*, in the United States and Canada. *J Med Entomol* 2003;40(2):159–64.
- [51] Gomez H, Krywko D, Stoecker W. A new assay for the detection of *Loxosceles* species (brown recluse) spider venom. *Ann Emerg Med* 2002;39(5):469–74.
- [52] Barbaro K, Ferreira M, Cardoso D, et al. Identification and neutralization of biological activities in the venoms of *Loxosceles* spiders. *Brazilian Journal of Med Bio Research* 1996; 29(11):1491–7.
- [53] Vetter R. Identifying and misidentifying the brown recluse spider. *Dermatol Online J* 1999; 5(2):7.
- [54] Vetter R, Barger D. An infestation of 2,055 brown recluse spiders (Araneae: Sicariidae) and no envenomations in a Kansas home: implications for bite diagnoses in nonendemic areas. *J Med Entomol* 2002;39(6):948–51.
- [55] Anderson P. Missouri brown recluse spider: a review and update. *Mo Med* 1998;95(7): 318–22.
- [56] Phillips S, Kohn M, Baker D, et al. Therapy of brown spider envenomation: a controlled trial of hyperbaric oxygen, dapsone, and cyproheptadine. *Ann Med* 1995;25(3):363–8.
- [57] Vetter R, Bush S. The diagnosis of brown recluse spider bite is overused for dermonecrotic wounds of uncertain etiology. *Ann Emerg Med* 2002;39(5):54–6.
- [58] Gomez H, Krywko D, Stoecker W. A new assay for the detection of *Loxosceles* species (brown recluse) spider venom. *Ann Emerg Med* 2002;39(5):469–74.

- [59] Guilherme P, Fernandes I, Barbaro K. Neutralization of dermonecrotic and lethal activities and differences among 32–35 kDa toxins of medically important *Loxosceles* spider venoms in Brazil revealed by monoclonal antibodies. *Toxicon* 2001;39(9):1333–42.
- [60] Elston D. What's eating you? *Loxosceles reclusa* (brown recluse spider). *Cutis* 2002;69(2): 91–2,94–5.
- [61] Vetter R. Envenomation by a spider, *Agelenopsis aperta* previously considered harmless. *Ann Emerg Med* 1998;32(6):739–41.
- [62] Browne G. Near fatal envenomation from the funnel-web spider in an infant. *Pediatr Emerg Care* 1997;13(4):271–3.
- [63] Miller M, Whyte I, White J, et al. Clinical features and management of *Hadronyche* envenomation in man. *Toxicon* 2000;38(3):409–27.
- [64] Nicholoso G, Graudins A. Spiders of medical importance in the Asia-Pacific: atracotoxin, latrotoxin and related spider neurotoxins. *Clin Exp Pharmacol Physiol* 2002;29(9):785–94.
- [65] Bonnet M. The toxicology of the *Latrodectus hasselti* spider—the Australian red back spider. *Br Homeopath J* 1999;88(1):2–6.
- [66] Gueron M, Ilia R, Margulis G. Arthropod poison and the cardiovascular system. *Am J Med* 2000;18(6):708–14.
- [67] Allen R, Norris R. Delayed use of antivenin in black widow spider envenomation: a case report. *J Wilderness Med* 1991;2:187–92.
- [68] Clark R, Wethern-Kestner S, Vance M, et al. Clinical presentation and treatment of black widow spider envenomation: a review of 163 cases. *Ann Emerg Med* 1992;21(7):782–91.
- [69] Isbister G, Churchill T, Hirst D, et al. Clinical effects of bites from formally identified spiders in tropical Northern Territory. *Med J Aust* 2001;174(2):79–82.
- [70] Kelley T, Wasserman G. The dangers of pet tarantulas: experience of the Marseilles Poison Centre. *J Toxicol Clin Toxicol* 1998;36(1–2):55–6.
- [71] Waggoner T, Nishimoto J, Eng J. Eye injury from tarantula. *J Am Optom Assoc* 1997; 68(3):188–90.
- [72] Belyea D, Truman D, Ward T, et al. The red eye revisited: ophthalmia nodosa due to tarantula hairs. *South Med J* 1998;91(6):565–7.
- [73] Castro F, Antila M, Croce J. Occupational allergy caused by urticating hair of Brazilian spider. *Journal of Allergy & Clinical Immunology* 1995;95(6):1282–5.
- [74] Shrum K, Robertson D, Baratz K, et al. Keratitis and retinitis secondary to tarantula hair. *Arch Ophth* 1999;117(8):1096.
- [75] Ost L, Stridh B, Wolf M. A clinical study of spider phobia: prediction of outcome after self-help and therapist-directed treatments. *Behav Res Ther* 1998;36(1):17–35.
- [76] Merckelbach H, Muris P. The etiology of childhood spider phobia. *Behav Res Ther* 1997; 35(11):1031–4.
- [77] Mineka S, Mystkowski J, Hladek D, et al. The effects of changing contexts on return of fear following exposure therapy for spider fear. *J Consult Clin Psychol* 1999;67(4):599–604.