# V316
# Snakes, Spiders and Scorpions, Oh My! Or, Managing Envenomations

Ahna Brutlag, DVM
Pet Poison Helpline, Bloomington, MN, USA

## KEY POINTS

- Rattlesnake venom can cause significant local tissue destruction, coagulopathy, and hypotension
- 25% of rattlesnake snake bites are "dry" or nonenvenomating
- If facial or laryngeal swelling is evident, be prepared to quickly intubate
- Black widow spider envenomation causes muscle cramping, extreme pain, and paralysis
- Brown recluse spider envenomation causes extensive local tissue destruction
- While all scorpions can sting, only the bark scorpion in AZ causes systemic toxicity in the US

## INTRODUCTION

While certain geographical regions of the US have are more burdened with venomous animals, only Alaska can claim to have no venomous snakes, spiders, or scorpions. Noting this, with the exception of Maine and Hawaii, every state in the U.S. harbors at least one venomous snake and all have black widow spiders. Given this distribution, the possibility of companion animal encounters with venomous creatures is widespread.

This lecture will cover envenomations from rattlesnakes, coral snakes, black widow spiders, brown recluse spiders, and bark scorpions. Of all of these, rattlesnake envenomations are the most deadly and common. The next most clinically serious/frequent is likely black widow envenomations in cats.

## NORTH AMERICAN CROTALIDS (RATTLESNAKE, COPPERHEAD, COTTONMOUTH)

Crotalids, also called pit vipers (named for their characteristic facial features), comprise the largest family of venomous snakes in the US (Crotalidae). This family includes rattlesnakes (*Crotalus* and *Sistrurus*), copperheads (*Agkistrodon picivorus)*, and cottonmouths, also called water moccasins (*A. contortrix)*. Their geographic territory is immense with only Maine, Alaska and Hawaii being free of native venomous snakes.

In the US, 99% of all venomous snake bites are from crotalids. While copperheads are responsible for the majority of venomous snake bites in the U.S., rattlesnakes cause the highest number of annual snake-bite deaths due to the potency of their venom. It is estimated that 150,000–300,000 companion animals are bitten by crotalids every year in the US, with 90% of the bites occurring between April and October (Gwaltney-Brant, 2007; Peterson, 2006b). While all rattlesnake species in the continental U.S. have "rattles," not all will rattle before striking. The correct identification of the snake is helpful for predicting potential clinical signs.

Crotalid venom is not intended to kill prey, but to immobilize it and predigest its tissues. Thus, venom is comprised of a "stew" of enzymes, myotoxins, cytotoxins, hemorrhagic toxins, cardiotoxins, and neurotoxins. These toxins may work as a single toxic agent or collectively to achieve a toxic effect on a given (or multiple) physiological systems.

Crotalids envenomations in the US are typically manifested by 3 different clinical pictures:

1. Significant local tissue damage, coagulopathy, hypotension
2. Severe neurotoxicosis (with little local tissue damage and no coagulopathy)
3. All of the above

The toxicosis seen depends on the species and age of the snake, the amount of venom injected, and the geographic location of the snake. Clinicians should be prepared for any of the above scenarios following crotalid envenomation.

The clinical signs associated with crotalid envenomation may include tissue swelling, pain, necrosis, ecchymosis, and petechiation near the site of envenomation (see Figure 1). The severity of a local reaction

does not reflect the potential severity of systemic toxicity. Additionally, since ¼ of bites are "dry," fang marks do not necessarily predict envenomation. When envenomation is present cardiovascular signs may include severe hypotension which is poorly responsive to fluid administration, tachycardia, dysrhythmias and shock. Hypertension may also be noted. Neurological signs include muscle fasciculations, an absence of pupillary reflexes, weakness, ataxia, seizures, and coma. Finally, beware of airway obstruction secondary to facial edema. This is a common occurrence in companion animals as they are frequently bit on the nose or face.

Though it is typical for local or systemic signs of envenomation to appear within 30 min to 2 hours, cases with a delayed onset of signs (up to 8 hrs) have been reported. Therefore, it is important to have all patients with a suspected crotalid bite present to the hospital immediately and be observed for at least 8–12 hours. Baseline laboratory evaluation including a CBC with a platelet count should be performed. Additionally, careful notation, measurements, and/or pictures of the wound should be documented to note progressing swelling and lesion size. An IV catheter should be placed to allow for the administration of medication and antivenin.

The only proven therapy against crotalid envenomations is antivenin. Antivenin is highly effective at reversing the neurological, coagulopathic, cardiovascular and necrotizing effects of venom if given early. In the U.S., Fort Dodge Laboratories made and marketed a veterinary specific North American crotalid antivenin. However, at the time these notes were written in 2009, Fort Dodge (Wyeth) had been recently purchased by Pfizer Animal Health and the status of its antivenin was not known. In the absence of this product, CroFab™ or Antivipmyn™ (by Bioclon; a Mexican product) will be effective substitutes. CroFab™ may be available from area hospitals and Antivipmyn™ is stocked by some zoos. Begin antivenin administration if significant swelling/necrosis or coagulation defects, fluid loss, change in neurologic status, or cardiac conduction abnormalities are noted. Follow the label guidelines for dilution and administration. Be prepared for a possible anaphylactic reaction, especially when using the veterinary-specific product.

While hospitalized, laboratory values including a CBC with platelet count, baseline serum chemistry, coagulation profile with fibrinogen, and urinalysis should be monitored frequently (especially the coagulation profile, q 12 hrs). Other in-hospital monitoring may include an ECG and frequent checking of blood pressure. Therapies may include the administration of colloids, analgesics (use opioids, not NSAIDS), aggressive IV fluid therapy and blood products. Steroids and drugs interfering with coagulation are best avoided.

Care of the bite wound itself should include clipping and cleaning of the site. Broad spectrum antibiotics may be needed, especially for gram-negative coverage. Surgical excision or suction of the bite wound, the application of hot/cold compresses, or arterial tourniquets, or the administration of aspirin or tranquilizers are not recommended.

The recurrence of symptoms may occur in severe envenomations or when inadequate doses of antivenin have been given. Coagulopathy recurrences have occurred days after antivenin administration. Thus, it may be important to restrain dogs and cats from being too active for the first couple of weeks following a bite. The prognosis in cats and dogs is relatively good provided there is early medical intervention.

**Figure 1. Bite to the face and subsequent envenomation from a prairie rattlesnake. Note the extensive local tissue damage and edema. The dog was not treated with antivenin and the wound took many months to heal. (Photo courtesy of Barney Oldfield, DVM, Totah Animal Hospital, Farmington, New Mexico)**

### RATTLESNAKE VACCINE

In 2004, Red Rock Biologics released a rattlesnake vaccine aimed at reducing the morbidity and mortality of crotalid envenomations in dogs. The vaccine is designed to elicit an immune reaction to the major protein fractions found in the venom of the Western diamondback rattlesnake. It claims no protection against the Mojave rattlesnake. However, to date there is little published evidence regarding the efficacy of this vaccine and many in the toxicological community remain skeptical. No scientific literature about the product could be found in a Pub Med literature search and repeated requests by the author to speak with a technical services representative from Red Rocks Biologics went unanswered.

### CORAL SNAKES (*MICRUROIDES EURYXANTHUS* AND *MICRURUS* SPP.)

In the US, the following coral snakes may be of clinical significance: The Eastern coral snake, the Texas coral snake, the South Florida coral snake (*Micrurus* spp.); and the Sonoran coral snake (*M. euryxanthus*). These snakes are brightly colored. They have a black head followed by fully circumferential rings of black, red, and yellow (or white). While other, non-venomous snakes share this coloration, the coral snake can be differentiated as the yellow and red bands are directly adjacent ("Red touches yellow, dangerous fellow").

Coral snakes are diurnal and passive but, if provoked, they will strike. However, coral snake envenomations are not common. Even among human beings in N. America, coral snake envenomations make up less than 1% of all venomous snake bites.

The method of envenomation by coral snakes is much different than that of the crotalids. Because they have a rudimentary system for envenomation, chewing action is required to inject venom. In 85% of human coral snake bites, the snake must be aggressively shaken or pulled of the skin (Peterson, 2006a). Companion animals may present to the veterinarian with the snake still attached. It is estimated that 60% of coral snake bites are "dry" or nonenvenomating (Peterson, 2006a). However, the amount of venom in one envenomation may prove fatal for dogs, cats or humans.

Coral snake venom is primarily neurotoxic with possible hematologic complications. Cats may be more prone to life-threatening envenomations than dogs. In cats, the signs of envenomation are primarily neurological and include CNS depression, ascending flaccid quadriplegia, anisocoria and hypothermia (Peterson, 2006a). Hypotensions, respiratory depression followed by failure, and myoglobin release have also been reported. Dogs may exhibit more hematologic signs such as intravascular hemolysis, anemia, and hemoglobinuria in addition to the other listed signs. Also, it is more common to see vomiting, intense salivation, and tachycardia in dogs than in cats (Peterson, 2006a).

Treatment is largely symptomatic and supportive as coral snake antivenin is no longer available in the U.S. Coralmyn™, a Mexican antivenin, is effective and may be available from zoos. Follow manufacturer's guidelines for administration. Surgical excision or suction of the bite wound, the

application of hot/cold compresses, or arterial tourniquets, or the administration of aspirin or tranquilizers are not recommended.

The bite wound site should be clipped and cleaned well. Broad spectrum antibiotics may be needed, especially for gram-negative coverage, though it is rare to have significant local tissue damage from coral snake bites. Baseline labs such as a CBC, chemistry panel, and urinalysis should be performed. The onset of clinical signs may be delayed for 10–18 hours; thus, patients should be hospitalized and observed for a minimum of 24–48 hours.

Attentive monitoring of respiratory, cardiac, hematologic, and CNS systems is imperative. Death is most often due to respiratory failure; intubation and assisted ventilation may be needed. Intravenous fluids should be used to treat dehydration, maintain tissue perfusion and manage myoglobinuria. If an abnormal heart rate or rhythm is noted, ECG monitoring is necessary. The use of corticosteroids in the absence of anaphylaxis is controversial and not often recommended.

### Black Widow Spiders (*Latrodectus* spp.)

Black widow spiders are found in every U.S. state except Alaska. They are commonly found west of the Rocky Mountains, from Canada to Mexico. East of the Rocky Mountains, they are also common the southern half of the country and from Maryland southward on the east coast. They are not easily found in New England and the upper Midwest (Vetter, 2009).

Females, the larger sex, are 2–2.5 cm long. They are shiny and black with a characteristic red or orange hourglass shape on their ventral abdomen (becomes more predominant with age). Immature females are brown and do not have the hourglass marking. However, they are still capable of causing severe envenomation. Black widow spiders are not typically aggressive but will bite defensively when their web is disturbed or they are provoked.

Only the female black widow spider is large enough to cause envenomation in mammals and one bite may prove lethal to cats or dogs. Animals may be bitten externally or, perhaps, while trying to ingest the spider. Cats suffering envenomation have been known to vomit up the spider. Cats are especially sensitive to black widow spider venom. One study reported death in 20 out of 22 cats following black widow spider envenomation. The mean survival time was 115 hours (Peterson, 2006c).

Overall, envenomation leads to systemic toxicity, not local tissue damage (as compared to the brown recluse spider). Sings of toxicity begin within 8 hours of envenomation and may persist for days. Signs in dogs include regional numbness, cramping of the abdominal, lumbar, and thoracic muscles, possible respiratory distress, seizures, and extreme pain (Peterson, 2006c). Abdominal rigidity in the absence of pain or tenderness, has been dubbed a hallmark sign of widow spider envenomation (Peterson, 2006c). Cats may be more prone to display salivation, restlessness, vomiting, diarrhea, and severe pain with tremors, muscle cramping, and ataxia preceding compete paralysis. Hypertension and tachycardia are common to both cats and dogs.

Treatment is primarily symptomatic and supportive though antivenin exists and has shown great success in human beings. Antivenin was used in at least one cat with suspected widow spider envenomation that had been clinical for 26 hours. Neurological function was restored within 30 minutes of administration (Gwaltney-Brant, 2007). Supportive care includes diazepam and methocarbamol for muscle relaxation and opioids for analgesia.

### Brown Recluse Spider (*Loxosceles reclusa*)

Brown recluse spiders (BRS), so named for their reclusive nature, are shy, nocturnal hunters. They spin small webs used which they use as a retreat, not to capture prey. The entire spider is 2–3 cm in diameter, light brown/yellow, with a characteristic violin shape on its dorsal cephalothorax (see Figure 2). Their geographical range is well delineated (see Figure 3). However, in spite of this well established range, BRS envenomations continue to be diagnosed well outside of this area (Pace, 2009). Given the strict range of the BRS, these are likely erroneous diagnoses as the skin lesion can mimic the bull's eye lesion of Lyme disease, chemical burns, pressure sores, diabetic ulcers, cutaneous anthrax, and bacterial infections.

The brown recluse spider may easily cohabitate with humans. When in the home, it is often in boxes, under piles of clothing, or in bedding. A most remarkable case report discusses the finding of 2,055

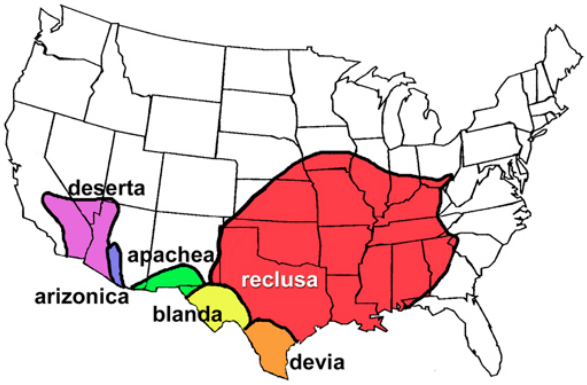
brown recluse spiders inside one Kansas home over a 6 month period of time (Vetter, 2002). Of these, 300–400 spiders were large enough to cause envenomation. During this time, no inhabitants reported being bitten. Similar in-home infestations of 30–40 BRS also yielded no reported envenomations.

Predominant clinical signs of BRS envenomation in humans involve local dermal necrosis. Dogs may be slightly more resistant to the effects of BRS venom than humans (Pace, 2009). Initially, the bite may appear as an edematous "bull's eye" lesion, progressing to a hemorrhagic bulla in 1–3 days. The subsequent eschar may be present for weeks, giving way to an indolent ulcer. Systemic signs following envenomation are rarer but may include hemolytic anemia, hemoglobinuria, fever, vomiting, weakness, dehydration (Peterson, 2006d).

Treatment for BRS envenomation is largely supportive. Antivenin is being investigated but is not yet commercially available. Dapsone, a leukocyte inhibitor, has shown some success when used experimentally for the treatment of dermal lesions (Peterson, 2006d). However, in spite of this, its use remains highly controversial. Surgical excision of dermal lesions is no longer recommended. Additional treatments may include analgesics, antibiotics, wound debridement, and antiemetics, antipyretics, and intravenous fluids for systemic signs. Drugs that disrupt coagulation should be avoided in cases of systemic toxicity.

**Figure 2. Brown recluse spider (*Loxosceles recluse)*. Note the characteristic violin shape on the cephalothorax. This shape is not well demarcated in immature spiders. (Courtesy of Richard Vetter, Dept. of Entomology, University of California, Riverside)**

**Figure3. Geographic distribution of *Loxosceles* spiders, including the brown recluse (*L. reclusa)*, in the United States. (Courtesy of Richard Vetter, Dept. of Entomology, University of California, Riverside)**




### BARK SCORPION (*CENTRUROIDES EXILICAUDA*)

Scorpions are arachnids. They have large claws or pinchers with which to grasp prey but, more concerning, is the telson. The telson is an appendage attached at the caudal abdomen which contains two venom glands and a stinger. Hence, scorpions do not "bite," they "sting." All scorpions are capable of

stinging and causing local pain. However, not all cause systemic envenomation in humans or companion animals.

In the U.S, it is widely believed that there is only one scorpion capable of systemic envenomation—the bark scorpion (*Centruroides exilicauda*), so named because it prefers to hide under tree bark. This scorpion is commonly encountered both in the home and outside and is the only species in the U.S. capable of climbing. The geographic range of the bark scorpion is located almost exclusively within Arizona's state boundaries.

Scorpion venom blocks voltage-gated potassium and sodium channels in nervous tissue. The degree of envenomation is highly variable. Clinical signs associated with envenomation in humans are correlated to body size, with children being the most adversely affected. This seems to hold true with companion animals as well as cats and small dogs develop more severe systemic signs than do large dogs (Holzman, 2009).

Clinically, humans and animals may exhibit neurological signs along with local pain following bark scorpion envenomation. However, bark scorpion stings do not cause significant local inflammation. If this is seen, stings from other species of scorpion or other causes should be suspected. Common CNS signs include nystagmus, paresthesia, referred pain, and asymmetric jerking of the limbs (not seizures) (Holzman, 2009 and Ruha, 2009). Other sings include excessive salivation, tachycardia, fever, hypertension, and increased respiratory secretions.

Treatment is primarily symptomatic and supportive. However, in human beings, antivenom (Anascorp) is gaining popularity and showing great success (Ruha, 2009). Other treatments include benzodiazepines for sedation and opioids for analgesia.

## Poison Control Resources

For assistance managing a potentially poisoned patient, a number of resources are available. In the US and Canada, veterinarians or pet owners may call Pet Poison Helpline ($35/case) at (800) 213-6680 or the ASPCA's Animal Poison Control Center ($60/case) at (888) 426-4435. Both of these services are available 24/7 and are staffed with experts in the field of veterinary toxicology. Pet Poison Helpline is additionally staffed with veterinary specialists in emergency and critical care and internal medicine, as well as PharmDs. The author recommends using either of these services in order to obtain the most accurate and current veterinary-specific clinical advice.

## References


1. Gwaltney-Brant SM, Dunayer EK, Youssef HY. Terrestrial zootoxins. In: Gupta RC, ed. *Veterinary toxicology: Basic and clinical principles*. New York: Elsevier Inc, 2007; 785–807.
2. Holzman D. Personal phone conversation regarding her experience managing both human and companion animal *Centruroides* envenomations over 20 yrs with the Arizona Poison and Drug Information Center. Nov, 2, 2009.
3. Pace LB, Vetter RS. Brown recluse spider (*Loxosceles reclusa*) envenomation in small animals. *J Vet Emerg Crit Care* 2009;19(4):329–336.
4. Peterson ME. Snake bite: Coral snakes. In: Peterson ME, Talcott PA, eds. *Small Animal Toxicology* 2nd ed. St. Louis, MO: Elsevier Inc, 2006a; 1039–1048.
5. Peterson ME. Snake bite: North American pit vipers. In: Peterson ME, Talcott PA, eds. *Small Animal Toxicology* 2nd ed. St. Louis, MO: Elsevier Inc, 2006b; 1017–1038.
6. Peterson ME. Spider envenomation: Black widow. In: Peterson ME, Talcott PA, eds. *Small Animal Toxicology* 2nd ed. St. Louis, MO: Elsevier Inc, 2006c; 1063–1069.
7. Peterson ME. Spider envenomation: Brown recluse. In: Peterson ME, Talcott PA, eds. *Small Animal Toxicology* 2nd ed. St. Louis, MO: Elsevier Inc, 2006d; 1070–1075.
8. Ruha A. F(ab)2 antivenom for reversal of neurotoxicity following bark scorpion sting. *NACCT Conference 2009*. San Antonio, TX. Sept. 24, 2009.
9. Vetter RS, Barger DK. An infestation of 2,055 brown recluse spiders (Aranea: Sicariidae) and no envenomations in a Kansas home: Implications for bite diagnoses in nonendemic areas. *J Med Entomol* 2002; 39(6):948–951.
10. Vetter RS. Personal email exchange regarding the geographic distribution of black widow spiders in the US. Oct. 29, 2009.