VIRGINIA SNAKE ENVENOMATIONS

INTRODUCTION
In the United States (US), approximately 45,000 snake bites occur each year, with about 7000-9000 bites resulting from venomous snakes. Death from a venomous snake bite in the US is very rare, with only 5-10 deaths per year. Four species of venomous snakes are indigenous to Virginia: the Copperhead (*Agkistrodon contortrix mokasen*), Timber rattlesnake (*Crotalus horridus horridus*), Canebrake rattlesnake (*Crotalus horridus atricaudatus*), and Eastern Cottonmouth (*Agkistrodon piscivorus piscivorus*). These snakes all fall within the category of pit vipers (subfamily *Crotalinae*), which share venom characteristics and certain anatomic features including a heat sensing pit between the eye and nostril, elliptical shaped pupils, and a single row of caudal scales on the ventral surface of the snake.

CLINICAL PRESENTATION
The spectrum of clinical presentations from *Crotalinae* envenomations ranges from asymptomatic to cardiovascular collapse and death. The presentation is dependent upon the amount and properties of the venom injected, the location of the bite, and the size and general health of the victim. The components of snake venom vary not only with the different species of snakes, but also from one snake to another, depending on the season, nutritional status, and age. It is therefore impossible to accurately predict the extent of local tissue damage a patient will develop following a snake bite. In Virginia, the Timber rattlesnake, Canebrake rattlesnake and Cottonmouth are regarded as having more toxic venom than the Copperhead and are more likely to result in systemic and local morbidity and, while rare, mortality. Bites from *Crotalinae* species which do not introduce venom (“dry bites”) have been estimated to occur in up to 20% of exposures.

TISSUE INJURY
Tissue damage at the site of the bite is the most common complication following envenomation by Virginia’s venomous snakes. Numerous enzymes have been isolated from the venom of snakes. The mechanism...
of each of these enzymes is directed at the breakdown of specific components of the tissue in which the venom has been injected, allowing the venom to penetrate further. As a result, patients will initially experience edema at the bite site that will progressively spread to adjoining tissues. Ecchymosis may also develop and give the area around the bite a bluish hue. In addition, hemorrhagic blebs may develop and become quite large. Lymphangitis and lymphadenopathy may progress secondary to lymphatic spread of venom components, causing the clinician to incorrectly assume the occurrence of secondary bacterial infection.

**COAGULOPATHY**

Following envenomation, coagulopathy has been reported, especially in association with the Timber Rattlesnake and the Canebrake Rattlesnake. Venom-induced thrombocytopenia, elevation in PR and PTT, fibrinolysis, and disseminated intravascular coagulation (DIC) have all been reported.

**TREATMENT**

Initial snakebite treatment is associated with numerous myths and dangerous practices that can contribute to the morbidity of the patient. The most important step to assure a good outcome is immobilization and rapid transport of the victim for evaluation by trained medical personnel. Many first-aid measures taught in the past are no longer recommended. Capture of the snake for identification may lead to another victim. Thorough examination of the patient with proper observation will dictate treatment regardless of the species involved. Performing incisions through the wound, application of suction devices, electric shock therapy, tourniquets, cryotherapy and heat application should all be avoided in the management of North American *Crotalinae* envenomations.

The bite area should be gently cleansed. Circumferential measurement at several points along the affected limb should be started shortly after the patient’s arrival and repeated at intervals until progression has ceased. A useful technique to assure consistent measurement is to place a small mark on either side of the paper tape. The bitten extremity should be immobilized and elevated to a level above the heart with a pillow or other means. When elevated, the edema commonly will move proximally as components of the venom follow the lymphatics.

**ANALGESIA**

Pain control usually requires parenteral opioid agents such as hydromorphone during the first 24-48 hours of therapy. Non-steroidal anti-inflammatory medications are typically avoided due to the hematologic effects of snake venom.

**INFECTION AND ANTIBIOTICS**

Case series have shown the incidence of infection to be low (approximately 3%) following snakebites. Venom itself has been demonstrated to have antibacterial properties. Currently, prophylactic antibiotics are not recommended in cases of snakebites.

**FASCIOTOMY**

Appropriate use of fasciotomies in the snakebite victim is a topic wrought with misconceptions. The literature is clear that fasciotomy should not be performed unless elevated muscle compartment pressures are documented.

**FLUID REPLACEMENT**

Hypotension may be caused by fluid loss due to third spacing, vomiting, hemorrhage secondary to coagulopathy, or vasovagal effects. Crystalloid administration should begin immediately in these patients.
ANTIVENOM
The most critical decision facing the clinician treating snakebite victims is when to administer antivenom. In general, antivenom should be used to treat moderate and severe envenomation. This includes those patients with rapid swelling or ecchymosis of the bitten area, progression of swelling or ecchymosis away from the site of the bite (especially that crossing a joint), systemic signs and symptoms (excluding those related to stress response), and coagulopathy/thrombocytopenia.

There is only one commercially available pit viper antivenom currently available in the US: Crotalidae Polyvalent Immune Fab (FabAV), brand name CroFab. FabAV is made by inoculating sheep with 4 different US snake venoms and is indicated for the treatment of all pit vipers indigenous to the US. Generally for significant Crotalinae envenomation an initial 4-6 vials of AV are mixed and diluted in 250cc of normal saline. Dosing depends on the severity of envenomation and not on patient weight. Infusion should begin slowly (20-30 cc/hr) over the first 20 minutes; if no reaction occurs the remainder can be administered over 60-90 minutes.

While allergic reactions do occur, the immunogenicity of this antivenom is overall low. Prior to infusion patients should be asked about a history of sheep, papaya or papain allergy, which may result in a high risk of reaction to the FabAV. Acute and delayed (serum sickness) hypersensitivity reactions have been rarely described. The most common side effect of FabAV is a rate related anaphylactoid reaction. These reactions may include urticaria, flushing, itching, diaphoresis, and in severe cases wheezing and hypotension. FabAV related anaphylactoid reactions are typically well controlled by stopping infusion temporarily and treating with antihistamines.

Local tissue symptoms and coagulopathy should be rechecked 1 hour following completion of the FabAV infusion. Repeat dosing of FabAV may be required in select cases with clinically relevant progression of local tissue injury and/or continued coagulopathy.

FOLLOW UP
Once the patient’s local symptoms have stabilized, coagulopathy (if initially present) has improved, and the patient is able to take oral pain medications they can be safely discharge from the hospital. If available, physical or occupational therapy consult prior to discharge may help with functional activities during patient recovery. All patients in whom coagulopathy developed should have follow up labs checked 2-3 days after discharge to monitor for recurrence of coagulopathy. Patients with only local symptoms should typically follow up with their primary care physician within the next week to monitor for progression of symptoms. Patients who received antivenom should be instructed to monitor for signs of serum sickness and all patients should be counselled on signs of potential infection.

For any questions please call the Blue Ridge Poison Center at 1-800-222-1222. Cell users may call 1-800-451-1428.

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