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Elapid Snake Envenomation: North American Coral Snakes and Australian Elapids (Tiger Snakes, Brown Snakes, Taipans, Death Adders, and Black Snakes)

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Introduction

Elapids are venomous front-fanged snakes found in Australia, North America, South and Central America, Asia, Africa, India, and the Middle East. Fangs are often small, resulting in minimally traumatic bites. Therefore, failure to identify a bite site does not rule out envenomation. Elapid snakes may also give a 'dry bite' without injection of venom into the patient. The authors recommend that antivenom is used when there is clinical evidence of Australian elapid envenomation in dogs and cats. Because of the potential for anaphylactic reactions to equine immunoglobulins, antivenom should not be used in the absence of definitive signs of envenomation, and epinephrine should always be available when antivenom is going to be used. In America, prompt treatment with antivenom is recommended for dogs found in close contact with a recently found dead or mutilated coral snake.

This chapter primarily discusses elapid envenomations in Australia and North America. The basic principles of treatment of envenomation may be helpful to guide treatment decisions for elapid envenomations in other countries, with the proviso that the correct antivenom needs to be administered (Table 142.1). Worldwide veterinary-specific information about elapids is often limited; a useful source of information about the human envenomation syndromes for individual species of snakes worldwide and recommended antivenoms can be found at the following website: www.toxinology.com

Table 142.1 Australian and North American elapid snakes, toxins, and recommended antivenoms.

Genus	Species	Venom toxins	Potential clinical effects	Recommended antivenom type and starting dose
North America elapids				
Coral snakes *(Micruroides; Micrurus)*	Western coral snake or Arizona coral snake (*Micruroides euryxanthus*) North American coral snake or Eastern coral snake or harlequin coral snake *(Micrurus fulvius)* Texas coral snake (*Micrurus tener*)	Neurotoxin (dogs, cats) Hemolysin (dogs)	Paralysis (severe) Hemolysis (mild, uncommon)	Suero Antiofidico Anticoral – Liquido- Solucion injectable- Instituto Clodomiro Picado, Universidad de Cosa Rica Coralmyn, Polivalent Anticoral Fabotherapic, Instituto Bioclon SA de CV, Mexico Both require special permission to import 1–2 vials. Increase dose to 5 vials if severe signs such as paralysis and hypoventilation, if the owners can afford

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Genus	Species	Venom toxins	Potential clinical effects	Recommended antivenom type and starting dose
Australian elapids				
	There are over 100 different species of elapids within Australia; only those commonly associated with envenomation in animals are discussed below			Australian polyvalent snake antivenom 40 000 units (CSL) can be used to treat envenomation by all Australian elapid species. It is extremely expensive compared to monovalent antivenoms listed below
Brown snakes (*Pseudonaja*)	Dugite (*Pseudonaja affinis*) Eastern brown snake (*Pseudonaja textilis*) Western brown snake or Gwardar *(Pseudonaja nuchalis*) Speckled brown snake (*Pseudonaja guttata*)	Procoagulant Neurotoxin	Coagulopathy (severe) Paralysis (severe)	Brown snake antivenom 1000 units. If requires intubation and ventilation, 3000 units
Black snakes (*Pseudechis*)	Red-bellied black snake (*Pseudechis porphyriacus*)	Procoagulant (mild?) Hemolysin Myotoxin Neurotoxin	Hemolysis (severe) Anemia (severe) Rhabdomyolysis (severe) Paralysis (minimal to severe)	Tiger snake antivenom 3000 units (black snake antivenom 18 000 units can be used but is more expensive)
	Mulga snake or king brown snake (*Pseudechis australis*)	Hemolysin Myotoxin Neurotoxins Anticoagulant	Hemolysis (mild to severe) Anemia Rhabdomyolysis (severe) Paralysis (mild to severe)	Black snake antivenom 18 000 units
Copperheads (*Austrelaps*)	Common copperhead or lowlands copperhead (*Austrelaps superbus*)	Neurotoxins Myotoxins Hemolysins Anticoagulant	Paralysis (mild to severe) Rhabdomyolysis (severe) Hemolysis (severe) Anemia (severe)	Tiger snake antivenom 3000 units
Death adders (*Acanthophis*)	Common death adder (*Acanthophis antarcticus)*	Neurotoxins Anticoagulant	Paralysis (severe)	Death adder antivenom 6000 units
Taipan (*Oxyuranus*)	Coastal taipan or common taipan (*Oxyuranus scutellatus*)	Neurotoxins Myotoxin Procoagulants Hemolysins	Paralysis (severe) Rhabdomyolysis (severe) Coagulopathy (severe) Hemolysis (mild)	Taipan antivenom 12 000 units
Tiger snake (*Notechis*)	Mainland tiger snake, Western tiger snake, island tiger snake (*Notechis scutatus*)	Procoagulant toxins Neurotoxins Myotoxin	Coagulopathy (severe) Paralysis (severe) Rhabdomyolysis (severe) Hemolysis (rare, mild)	Tiger snake antivenom 3000 units. If requires intubation and ventilation, 9000 units

Elapid Species Identification

North America

The venomous Eastern coral snake can be easily identified as having a black snout, and yellow, black, and red bands encircling the body. The red and black rings are wider than the interposed yellow rings (Figure 142.1). Care should be taken not to become confused with the harmless scarlet king snake in which the yellow and red rings are separated by black rings ("Red on yellow kill a fellow, red on black, venom lack"). The other species of coral snakes in the Americas and Mexico will have color pattern variations specific to the species in a particular geographic location.

Australia

Snake coloration or relying on the public's description is notoriously inaccurate for identifying snake species within Australia, with the majority of snakes being brown in colour even if they are a member of the Black (*Pseudechis*), Taipan (*Oxyuranus*), Copperhead (*Austrelaps*) or Brown (*Pseudonaja*) snake family or one of the many mildly venomous species of snakes. The following methods are recommended to help determine which type of antivenom should be administered.

- Identification of the snake by an experienced herpetologist. Veterinarians can attempt to identify dead snakes using a snake identification key for their local area which identifies snakes according to scale counts, body shape, and color. Catching, attempting to kill or examination of live elapid snakes by inexperienced snake handlers should be strongly discouraged due to the significant risk of life-threatening human envenomation.
- Use of the Commonwealth Serum Laboratory (CSL) snake venom detection kit (SVDK) on the bite site, urine or blood. This kit determines the most appropriate antivenom to administer. The SDVK is not suitable for use outside Australia and Papua New Guinea.
- Knowledge of elapid snakes within the specific locality together with clinical signs can help determine the most appropriate antivenom to use [1]. See Table 142.1 for clinical signs of the major venomous elapid species. However, failure to respond appropriately to antivenom should always raise concerns that the wrong antivenom has been administered.

Figure 142.1 The Eastern coral snake, *Micrurus fulvius fulvius*, is a venomous elapid snake characterized by the red and yellow colors lying adjacent to one another. It is geographically distributed in the south-eastern part of the United States.

Pathophysiology of Elapid Venoms

Elapid snake venoms are a complex combination of toxins, with the venom of many species containing multiple neurotoxins, peptides, and enzymatically active and non-active compounds [2]. Despite the prodigious amount of research into elapid venom toxins, their mechanisms of action and treatment efficacy *in vitro* and recent studies of antivenom efficacy in human envenomations, there is limited research into the clinical syndrome of envenomation in dogs and cats. Additionally, there are many mild to moderately venomous elapids whose venom has not been researched.

There appear to be significant clinical differences in the envenomation syndromes of humans and other species, with humans being less susceptible to the neurotoxin effects of envenomation in comparison to dogs and cats [3]. It is also possible that the high incidence of severe paralysis in dog and cat envenomation is due to higher amounts of venom being injected by the snake, potentially due to the unrelenting attacking behavior of dogs and cats when hunting snakes.

Neurotoxins

Neurotoxins are classed as either presynaptic or postsynaptic and exert their effects at the neuromuscular junction, causing a rapid onset of lower motor neuron (LMN) paralysis. Many venoms contain both presynaptic and postsynaptic neurotoxins. Presynaptic neurotoxins belong to the phospholipase A_2 group of toxins and cause structural changes to the nerve terminal, preventing release of acetylcholine. *In vitro* studies have shown that presynaptic neurotoxins can become irreversibly bound and unresponsive to antivenom. Postsynaptic neurotoxins (as found in the western hemisphere elapids and some Australian elapids) act as antagonists at

acetylcholine receptors, and *in vitro* studies do not support irreversible binding, so theoretically there should be a rapid response to administration of appropriate antivenom.

Clinical experience in dogs and cats with Australian brown (*Pseudonaja*) and tiger (*Notechis*) snake envenomations which contain presynaptic neurotoxins indicates that severe acute paralysis can rapidly reverse with high doses of antivenom, and irreversible binding is likely to take longer than 24 hours and potentially several days [4]. Conversely, reports of envenomations when postsynaptic neurotoxins are present in both dogs and humans (in Australasia) have revealed cases in which improvement of paralysis has been delayed post antivenom [5,6].

Higher doses of neurotoxic venom have been associated with a more rapid onset of paralysis in dogs envenomated with tiger snake venom and the same is thought to occur in other elapid envenomations [7]. Dogs and cats which are active post envenomation tend to have a faster onset of clinical signs, probably due to muscular activity increasing lymphatic flow and absorption of the venom from the bite site. Paralysis is non-painful and humans have been reported to progress to severe paralysis while sleeping in a hospital bed.

Procoagulant Toxins

Prothrombin activators which are present in some Australian elapid venoms are either classed as group C prothrombin activators (*Oxyuranus* and *Pseudonaja* venoms), similar to factor XaVa prothombinase complex, or group D prothrombin activators (*Notechis* venom) which is similar to factor Xa and requires the presence of the patient's factor Va for the procoagulant effects to be manifested. Procoagulant toxins cause diffuse intravascular thrombosis, consuming clotting factors and then resulting in venom-induced consumptive coagulopathy (VICC) with depletion of fibrinogen, factor V and factor VIII. Coagulation testing reveals prolongation of PT and aPTT commonly beyond the limits of test detection [4,8]. VICC and cerebral haemorrhage are the most common causes of human death in Australian snake bite. Acute renal failure occasionally occurs in patients with VICC and is potentially due to renal ischemia secondary to intrarenal microthrombosis.

Normalization of the patient's clotting times post antivenom administration takes a significant period of time due to the requirement for hepatic synthesis of replacement clotting factors, commonly at least 12 hours in dogs and cats after antivenom and 24–36 hours in humans [4,8]. Current human guidelines and clinical experience with animals indicate that one vial of antivenom should be sufficient to neutralize procoagulant toxins. Clinical signs of hemorrhage are infrequent, and fresh frozen plasma is rarely indicated and should never be given before antivenom as it may worsen thrombosis formation [9]. Because of the severe coagulopathy and the potential for life-threatening bleeding, strict confined rest is recommended until clotting times normalize [10].

Anticoagulant Toxins

Several phospholipase A_2 toxins in Australian elapids have an anticoagulant effect on blood, causing prolongation of aPTT and potentially PT. Spontaneous hemorrhage does not occur and the prolonged bleeding times improve rapidly after antivenom administration [11,12].

Myotoxins

Various phospholipase A_2 toxins in Australian elapids can cause severe rhabdomyolysis. Increases in creatinine kinase (CK) are generally delayed for at least 3–6 hours post bite, and minimizing patient muscular activity and early administration of antivenom prevent the development of rhabdomyolysis; delayed administration of antivenom is still worthwhile as it potentially prevents the worsening of rhabdomyolysis. CK can be severely elevated >5000 U/L, and in some patients massive elevations >500 000 U/L can occur. Elevations in CK do not immediately resolve post administration of antivenom; instead, CK plateaus and then gradually decreases [11,13]. Myoglobinuria is common and severe myoglobinuria may cause acute kidney injury (AKI) to develop [7,13,14]. Megaoesophagus secondary to myotoxins has been reported as a complication of Australian tiger snake envenomation in dogs, with reports of resolution taking up to 5 weeks [15].

Hemolytic Toxins

Some phospholipase toxins in North American and Australian elapids cause lysis of red cells, resulting in severe anemia, hemoglobinemia and hematuria, and potentially AKI [16]. Echinocytes or burr cells and evidence of red cell damage may be visualized on blood smear examination (see Chapter 66). The risk of development of anemia from exposure to hemolytic toxins appears to be species specific, with dogs having an increased susceptibility compared to humans and cats [11,13,16]. Rare cases of immune-mediated delayed hemolytic anemia in dogs have also been reported [17]. In envenomations due to Australian tiger snake bites and US coral snake bites, hemolysis may occur without significant anemia developing. Severe hemolytic anemia can develop in bites from Australian black snakes, copperheads, and taipans.

Local-Acting Cytotoxins

Localized tissue reactions or injury around the bite site are not a significant component of North American or Australian elapid envenomations and in most cases the bite is difficult to identify in dogs and cats. Mild local tissue swelling may occur associated with envenomating bites from Australian black snakes, taipans, and copperheads. Severe local tissue injury can occur to secondary to African and Asian cobra bites, which are also members of the elapid family [18].

Clinical Signs of Envenomation

Any of the following clinical signs are an indication for antivenom administration.

Preparalytic Signs

Preparalytic signs most commonly occur within 30 minutes or less of the bite, and can include one or more of vomiting, salivation, inappropriate urination or defecation, trembling, tachypnea or collapse and then a period of apparent recovery. In dogs, preparalytic signs have been associated with an LD_{50} dose or higher of *Notechis* venom or progression to clinical signs of envenomation [4,7].

Paralysis

Elapid venoms are primarily neurotoxic, resulting in paralysis, and in severe cases death from ventilatory failure. Any signs of LMN paralysis, even if mild, are an indication for antivenom if there is known contact with an elapid. Elapid snake envenomation should be considered as a differential diagnosis for any animal which develops rapidly progressive LMN paralysis in endemic areas.

Australia

Classically, clinical signs start with a decreased gag, weak or absent palpebral reflex, then weakness of the limbs and a mildly ataxic gait, followed by recumbency, inability to hold the head up, difficulties swallowing and ptyalism due to pharyngeal paralysis, loss of all withdrawal reflexes and complete LMN paralysis with signs of respiratory distress, hypoventilation, and then finally respiratory arrest. Mydriasis is a relatively late clinical sign. Brachycephalic breeds can develop respiratory distress as an early sign when envenomated. Because of the potential for irreversible binding of neurotoxins and requirement for prolonged periods of hospitalization or ventilation, sufficient antivenom should be administered early in the course of envenomation. The onset and speed of progression of paralysis is highly variable. Dogs occasionally require ventilation within 30 minutes of being bitten or can take 6–12 hours to develop signs with rare reports of longer time periods. Therefore, the patient must never be left unobserved when monitoring for potential snake bite.

Cats occasionally have a more delayed presentation and may be presented with signs of severe paralysis after their owners notice that the cat hasn't moved off the same piece of furniture for 12–24 hours.

Complications of paralysis include aspiration pneumonia, corneal ulcers, and detrusor atony. If ventilation is required then multiple vials of antivenom are associated with a more rapid improvement in tiger and brown snake envenomations [4].

North America

Coral snake envenomations have a similar progression of paralysis as above and also have a variable time course for onset of clinical signs of paralysis, varying from immediately post bite to 36 hours later, with the majority showing clinical signs within 2–4 hours of being bitten. Any sign of weakness or illness after close contact with a coral snake is an indication for antivenom, especially if the venous PCO_2 is increasing toward 65 mmHg.

Coagulopathy

Coagulopathy can occur with Australian elapid envenomations and is an indication for administration of antivenom (see Chapter 70). VICC is associated with severe prolongations of PT, aPTT, and ACT test beyond the limits of test detection. Clinical bleeding occurs infrequently in dogs and cats and can include bleeding from oral wounds, hematuria, hyphema, gastrointestinal hemorrhage and, very rarely in small animals, internal bleeding or central nervous system hemorrhage [4,17]. Large hematomas can occur associated with venepuncture, cystocentesis, and oral wounds; therefore jugular venepuncture should be avoided if possible and pressure bandages placed over sites of venepuncture. Occasionally, dogs and cats will not develop coagulopathy despite other clinical signs of envenomation from a species of snake known to have procoagulant toxins. Anticoagulant toxins may just cause a mild prolongation of APTT which does not progress to a severe coagulopathy.

Rhabdomyolysis

Rhabdomyolysis commonly occurs with Australian elapid envenomations at least 3–6 hours post bite and is primarily detected by recognition of myoglobinuria or elevated CK on testing. Myoglobinuria can be severe, with urine

dark brown to black in color. Humans often report significant muscle pain secondary to venom-induced rhabdomyolysis although this is more difficult to appreciate in dogs and cats. Regurgitation secondary to megaesophagus may occur in dogs and take weeks to resolve.

Hemolysis and Anemia

Severe hemolysis and anemia can develop in bites from Australian black snakes, copperheads and taipans; antivenom is indicated and blood transfusions may be required.

In envenomations due to Australian tiger snake and US coral snake bites, hemolysis may occur without significant anemia developing. Hemolysis only occurs in approximately 50% of the dogs that are bitten by North American coral snakes. The hemolysis is usually not life-threatening but pigmenturia should be treated with intravenous crystalloid solutions and is an indication for antivenom (see Chapter 167).

First Aid Advice for Owners

Because of the potential for rapid onset of clinical signs of paralysis, any owner who phones to say that their pet has been bitten or may have been in close contact with a snake should be advised to minimize activity and present their pet for immediate veterinary examination. If owners describe an already collapsed patient, provide instructions on how to perform mouth-to-nose ventilation if required en route, as the most common cause of acute death in dogs and cats is hypoxemia secondary to respiratory paralysis. A firm crepe pressure bandage can be applied to a bitten limb and has been shown to slow venom absorption; however, in dogs and cats, most bites are found on the head, neck, and thorax and in these locations pressure bandages are contraindicated.

Treatment of Envenomation in Dogs and Cats

Patients should be under constant observation because of the significant risk of rapid onset of paralysis. Dogs occasionally progress from walking to respiratory arrest within 15–30 minutes.

Asymptomatic Patients

An IV catheter should be placed (avoid the jugular vein) and blood collected to evaluate PT, aPTT or ACT, PCV, TP, hemolysis, and CK (where geographically indicated). Urine should be collected to assess for pigmenturia. In Australia, a SVDK is recommended if signs of envenomation are present and there is an uncertainty regarding which antivenom to administer. Cystocentesis should be avoided, if coagulopathic. Neurological assessment is performed to assess for evidence of paralysis. The presence of paralysis, coagulopathy, hemolysis or anemia and known contact with an elapid snake are indications for antivenom.

When a severely envenomated patient presents, the first steps in treatment follow the ABC approach (see Chapter 2). Assess if the patient has a patent airway (suction/swab any saliva present), intubate if the patient is apneic or severely paralyzed and unable to breathe and start manual or machine ventilation with 100% oxygen if apneic or hypoventilating ($ETCO_2$>60 mmHg) (see Chapter 188). Most severely envenomated dogs and cats will respond well to oxygen support and assisted ventilation, allowing time to perform diagnostics and start appropriate treatment. Bradycardia is common during severe envenomations, is not associated with hypotension and normally resolves with improvement in oxygenation. Hypotension can occur but normally resolves with appropriate correction of hypoxemia or a conservative fluid bolus.

Antivenom is administered diluted at least 1:1 in Hartmann's/lactated Ringer's or 0.9% NaCl and administered over 20–30 minutes with close monitoring for evidence of anaphylaxis occurring, unless cardiac arrest has occurred in which case it is administered as a bolus. Epinephrine should always be readily available. If anaphylaxis is suspected 0.01 mg/kg epinephrine is administered IM, and it may be necessary to repeat it in 15–20 minutes if the vital signs do not stabilize. Intravenous adrenaline should be avoided except in the situation of cardiac arrest because of the potential for cardiac arrhythmias and intracranial hemorrhage if VICC is present.

There is significant variation between the purity and potential risk of anaphylaxis between antivenoms from different countries. If dexamethasone and antihistamines are recommended by the manufacturer prior to administration, this recommendation should be followed. In Australia, antivenom is highly purified and the incidence of anaphylactic reactions in dogs and cats is 10%; when they do occur, reactions are rarely severe [4].

The patient's neurological status should be reassessed immediately after administration of antivenom. If there is deterioration in neurological status, further antivenom should be administered.

In Florida, cost restrictions usually limit the administered dose to 1–2 vials of coral snake antivenom [19]. The Costa Rican product used is purified and requires no preventative hypersensitivity treatment. In Australia, if VICC or anticoagulant coagulopathy is present, a single dose of antivenom should be sufficient to neutralize

the effects of the procoagulant or anticoagulant toxins. With VICC, the coagulopathy normally improves within 12 hours in dogs and cats although normalization of clotting times may take up to 24 hours post antivenom. Fresh frozen plasma is only recommended if it has been at least 6 hours post bite and only if life-threatening hemorrhage is suspected and antivenom has already been administered [9]. Strict confined rest is recommended until the patient's clotting times have normalized.

If myolysins are present and CK continues to rise significantly 6 hours after antivenom, and there is severe myoglobinuria, further antivenom should be administered and intravenous crystalloid should be infused at an approximate twice maintenance dose of 120 ml/kg/day until the pigmenturia clears. The urine output should be closely monitored because of the risk of AKI developing. Pigmenturia secondary to myoglobinuria often improves rapidly once sufficient antivenom has been administered. Humans report significant muscle pain associated with myotoxins and opiate analgesia should be administered to dogs and cats (see Chapter 191).

Dogs and cats should remain under close observation until all signs of envenomation have resolved. Strict confined rest for 1–2 weeks is recommended after discharge.

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