The diagnosis and management of snakebite in dogs – a southern African perspective

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ABSTRACT

Cases of snakebite envenomation are frequently presented to veterinary practitioners in southern Africa. Despite this, no published guidelines exist on how this medical emergency should be managed. Southern African snake venoms can be classified into 3 main types based on the main mechanism of venom action and clinical presentation. A polyvalent antivenom is manufactured in South Africa and contains antibodies against the most important southern African snake venoms. The cytotoxic venoms are represented mainly by the puff-adder (*Bitis arietans*), Mozambique spitting cobra (*Naja mossabica*), black-necked spitting cobra (*Naja nigricollis*) (in the Western Cape and Namibia) and the stiletto snake (*Atractaspis bibronii*). These venoms may cause dramatic local swelling, high morbidity and low mortality and infrequently require the use of antivenom for survival (the only cytotoxic venoms used to prepare the antivenom are the puff-adder and Mozambique spitting cobra). The neurotoxic venoms (represented chiefly by the non-spitting cobras and mambas) cause high mortality due to rapid onset of paresis and require antivenom and mechanical ventilatory support which is life-saving. The boomslang (*Dispholidus typus*) and the vine snake (coagulopathic venom) rarely bite humans but dogs may be bitten more frequently. These venoms cause a consumption coagulopathy and successful treatment of boomslang bites requires the use of snake species-specific monovalent antivenom. There is no antivenom available for treating vine snake (*Thelotornis capensis*), berg adder (*Bitis atropos*), night adder (*Causus* spp.), stiletto snake and other lesser adder bites. There are some important differences between the way snakebites are managed in humans and dogs.

Key words: dog, snakebite, southern Africa.

Leisewitz A L, Blaylock R S, Kettner F, Goodhead A, Goddard A, Schoeman J P **The management of snakebite in dogs – a southern African perspective**. *Journal of the South African Veterinary Association* (2004) 75(1): 7–13 (En.). Department of Companion Animal Clinical Studies, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

INTRODUCTION

Snakebite is a common medical emergency[16] with significant morbidity and mortality in small animal practice in southern Africa. In Australia, a country with similar climatic conditions to southern Africa, snakebite is also reported as a common presentation[20]. There is almost no veterinary literature addressing this problem in dogs in southern Africa. Because the interaction between the pet and the snake is often not witnessed, the diagnosis of snakebite is most often presumptive, based on clinical presentation. The snake itself, or a good description of the snake, is usually not presented with the patient, necessitating syndromic management. Veterinarians should have knowledge of the poisonous snake species most commonly causing bites in their area of practice and be able to identify them. Of 175 species of snakes in the subregion, only a handful are regarded as poisonous enough to cause death[7]. There are some important differences between humans and dogs in the way snakebite is managed.

EPIDEMIOLOGY

Being ectotherms (cold blooded), the level of activity of a snake is determined by the temperature of its environment. For this reason the highest incidence of snakebite in humans and dogs is during the warmer summer months[5,9,18,21]. There is a consistent annual peak in late summer and early autumn in the area served by the Onderstepoort Veterinary Academic Hospital (OVAH)[16]. This is thought to be due to increased pre-hibernation activity.

The most common bite site in dogs is cranial to the shoulders, especially the face and the loose skin around the neck as dogs attack snakes with their mouths. Standing humans are most commonly bitten below the knee[5,9,18]. The different anatomical location of this bite distribution has important consequences for the outcome of bites, specifically cytotoxic ones.

Snakebite is confined almost completely to rural environments and is rarely a problem in built-up urban areas[11]. Hunting dogs and dogs that are inquisitive by nature are especially prone to bites. There is no study to evaluate snakebite predilection for breed or sex of dog.

In humans, cytotoxic bites outnumber neurotoxic bites by about 10:1[5,9,18,21]. This is likely to be similar for dogs. Morbidity is high in the case of bites from cytotoxic snake species but mortality is low. The opposite is true of bites due to the neurotoxic snake species where mortality is high and morbidity is low. It is probable that many snake-envenomated dogs die prior to veterinary care.

SNAKE IDENTIFICATION

This can be a daunting task for the novice but some simple, general rules may be helpful in deciding if a southern African snake is venomous or not. The following usually indicate a dangerous snake:

- The snake raises its head from the ground and makes a hood when threatened (cobras, *Naja* spp.; rinkhals, *Haemachatus haemachatus*; black mamba, *Dendroaspis polylepis*). The boomslang (*Dispholidus typus*) and vine snake (*Thelotornis capensis*) will inflate the front portion of the body.
- The snake is banded as opposed to striped along the length of the body. A chevron-like pattern dorsally (adders) is an indication of danger.
- Heavy bodied snakes with a distinct neck and triangular head (adders).

It is important to remember that some snakes will sham death when threatened (particularly the rinkhals) and no snake should be assumed dead and handled. If the snake is presented along with the dog, advice on identification should be sought as this would be helpful in predicting the

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Received: July 2003. Accepted: December 2003.

clinical course and outcome of the patient. In this regard owners are poor sources of reliable information. In most cases, snakebite is strongly suspected but never confirmed, therefore the clinical presentation and progression of clinical signs often confirms the diagnosis.

VENOM TYPES

There are essentially 3 important groups of venoms in terrestrial snakes of southern Africa. Each leads to a particular clinical envenomation syndrome.

1. Cytotoxic venoms result in progressive swelling. The 3 important snakes in this group are the puff-adder (*Bitis arietans*) and spitting cobras (*Naja mossambica, N. nigricollis*), with the stiletto snake (*Atractaspis bibronii*) and night adder (*Causus* spp.) causing lesser swelling.
2. Neurotoxic venoms of the family Elapidae produce progressive paresis. This group is represented by the cobras (snouted, Cape and forest cobras, *Naja annulifera, Naja nivea* and *Naja melanoleuca,* respectively) and the mambas (*Dendroaspis* spp.).
3. Coagulopathic venoms of the family Colubridae, which includes the boomslang and vine snake, produce bleeding. Puff-adder[15] and Gaboon adder (*Bitis gabonica*) bites[17] may cause a coagulopathy.

The berg adder (*Bitis atropos*)[12] and rinkhals[25] have both neurotoxic and cytotoxic venom. The snouted cobra venom may also have mixed effects.

PATHOGENESIS AND DIAGNOSIS

Progressive swelling (cytotoxic envenomation)

The time course of disease following a bite in these cases is relatively long. Most cases will only present several hours after the bite with the venom effect being almost exclusively local. Systemic signs seen are most likely the result of extensive fluid extravasation and not due to systemic venom absorption.

The classical presenting complaints are:

- ***Local swelling that is typically non-painful.*** The degree of swelling varies and may be mild and completely inconsequential or massive and life-threatening (Figs 1, 2, 3). Swelling is usually present within 2 hours of the bite, peaks between 12 and 24 hours and is significantly reduced by 72 hours (without the use of antivenom). Swelling is typically located around the head and neck. Bites in humans are typically very painful and may be associated with serious tissue loss, both of which are rarely seen in dogs. We postulate that the layer of fur and looseness of a dog's skin around the neck may make deep muscle deposition of the venom more difficult in dogs as opposed to humans. Human furless skin requires more pain receptors for protection which are especially prevalent on the hands. Some venom components are hyperalgesic. In the few bites involving muscle bellies in dogs (shoulder and gluteal region), pain has been severe although tissue loss was not remarkable. A further exception is bites by spitting cobras[27] which are extremely painful and cause significant skin loss (Fig. 4).
- ***Infection***. It is somewhat surprising that secondary bacterial infections are uncommon in dogs. This is counter-intuitive as free blood, anaerobic tissue and bacteria from a snake's mouth would provide grounds for sepsis. Snake venom is, however, antibacterial in action[2] and there are few bacteria in snake mouths[4]. In 1 study of infected snakebite wounds only aerobic bacteria which were either Gram positive cocci or Gram negative bacilli were isolated[3].
- ***Systemic signs associated with massive blood loss and hypotension.*** Progressive swelling from a puff-adder bite is rapid and associated with haemorrhage and oedema (Figs 2, 3). Swelling from a Mozambique spitting cobra (*Naja mossabica*), stiletto snake and night adder is slower in progression and not associated with haemorrhage. Swelling in a 20 kg dog's neck following a puff-adder bite may contain upwards of half a litre of blood. This is a significant proportion of the circulating volume and this rapid loss may lead to hypotension (oligaemic shock). Signs associated with hypotensive shock include weakness, pallor, tachycardia, hypothermia, reduced urine production and eventual complete collapse. Poor organ perfusion and tissue hypoxia may result in multiple organ failure. An awareness of these complications has important implications for monitoring and treatment. Similar complications have been described in humans[15].
- ***Upper airway obstruction***. Dogs that have been bitten around the face or neck region, may develop swelling, which causes upper airway compromise and eventually death by asphyxiation (Fig. 3). Cervical swelling may dissect down the trachea into the mediastinal space which may have serious consequences for cardiac venous return as the large veins contained in the cranial mediastinum collapse easily.
- ***Haematological consequences***. Swelling is due to the loss of whole blood and this does not initially affect haematocrit. The physiological response to blood loss and a fall in blood pressure result in fluid retention, haemodilution and a later fall in haematocrit. In serious cases, haematocrit should be monitored twice daily.

In-saline-agglutination-positive, secondary immune mediated haemolytic anaemia (IMHA) has occasionally been observed following bites. It is necessary to monitor cases with a falling haematocrit with regular in-saline agglutination tests (ISA) and look for signs of free haemoglobin in serum or urine. It may be necessary to treat this complication with glucocorticoids.

A non-DIC thrombocytopenia is very common in puff-adder bites. In the baboon the venom has been shown to contain a potent irreversible platelet aggregation-inducing component[8] that may be associated with active haemorrhage seen in humans[30] which has also been observed in a dog at the OVAH. Immune-mediated platelet destruction may occur as a few cases develop intravascular haemolysis as a result of secondary IMHA.

Typically the leukogram observed in the OVAH following puff-adder bites is inflammatory (neutrophilia often with a left shift) that is most likely the consequence of tissue damage and the systemic inflammatory response syndrome (SIRS) and not of infection.

Progressive weakness (neurotoxic envenomation)

Many cases of neurotoxic envenomation probably die before presentation. The time between when a dog is bitten by a neurotoxic snake and the onset of life threatening collapse is often so short that unless the owner actually observes the interaction between dog and snake, we suspect many dogs die without veterinary care. The time from bite to the onset of signs is invariably less than an hour and in severe cases less than 30 minutes. Systemic absorption of venom results in pre- or post-synaptic toxin blockade at the neuro-muscular junction (NMJ)[19,26]. This group of snakes have short, fixed front fangs and venom is deposited superficially in tissues.

- ***Generalised weakness.*** Usually the 1st neurological signs to become obvious typify bulbar paralysis (loss of the swallowing reflex, paralysis of the tongue and jaw) with saliva pouring from the mouth as it cannot be swallowed (Fig. 5). This is followed by limb weakness and finally flaccid paralysis. Breathing is shallow and rapid and the dog's mucous membranes become cyanotic and death follows. Similar signs have been reported in humans[5,9,18].

Fig. 1: **The typical presentation following an adder bite is a non-painful facial and ventral neck swelling. Most cases recover without complication and require no treatment.**

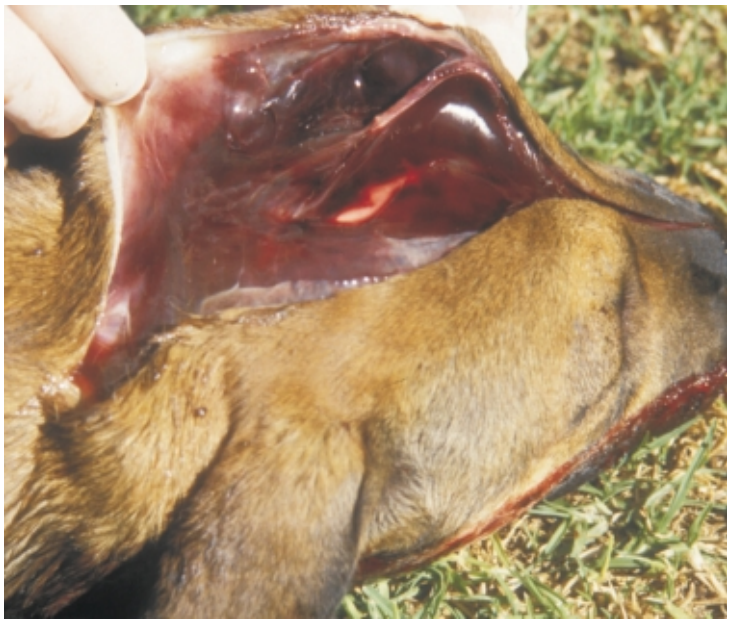

Fig. 2: **A young Staffordshire terrier dog died due to asphyxiation following a puff-adder bite. The figure illustrates the massive haemorrhage that is responsible for the large swellings seen following adder envenomation.**

The differential diagnosis for flaccid quadriplegia would include:

1. Polyradiculoneuritis (rarely so acute and seldom severe enough to cause respiratory embarrassment).
2. Myasthenic crisis (rare).
3. Tick bite toxicosis.
4. Organophosphate toxicity (the flaccid form of the disease or chronic intoxication).
5. Botulism (rare in dogs).

- ***Local signs***. These are usually limited to mild swelling and the bite marks may be unidentifiable. Exceptions to this are spitting and snouted cobra bites where swelling may be significant and necrosis may occur. On occasions a 'dry bite' may be delivered by the snake, *i.e.* no venom injected in spite of a clear bite, making it important to withhold treatment until clear signs of envenomation are apparent.
- ***Other complications***. On occasion, focal intracranial CNS signs are seen on recovery. The complications of mechanical ventilation can be serious (especially if improperly applied) and life-threatening. The most common outcome, however, is complete and quick recovery if treatment is early and aggressive.

Bleeding (haemotoxic/coagulopathic envenomation)

The onset of clinical disease following injection of these venoms takes longer than that due to neurotoxicity (may be minutes) or cytotoxicity (hours). Most patients would be presented to a veterinary practitioner the next day.

Severe bleeding is usually the result of venom haemorrhagins as well as procoagulant enzymes with boomslang and vine snake venom causing activation of clotting factors II and X[23,28] and Gaboon venom containing a thrombin-like substance[24]. Disseminated intravascular coagulation ensues and once coagulation factors are depleted, active bleeding occurs. Disseminated microvascular thrombi may produce multiple organ failure. Thrombocytopenia may lead to bleeding from a puff-adder bite. With all

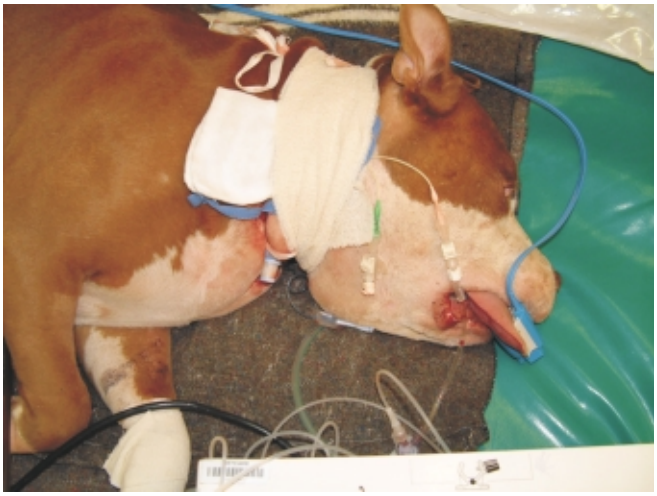

Fig. 3: **Puff-adder** (*Bitis arietans)* **bites are sometimes severe enough to cause upper airway obstruction. The pitt bull terrier developed upper airway obstruction and had a tracheostomy tube surgically placed** (arrow)**. Also note the massive swelling cranial to the shoulders.**

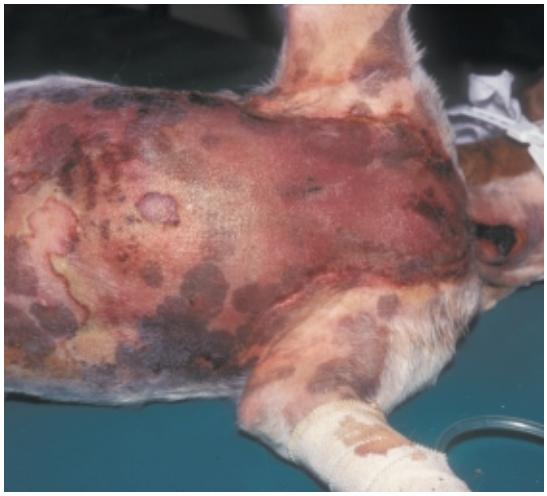

Fig. 4: **Mozambique spitting cobra** (*Naja mossambica*) **bites are remarkably cytotoxic and associated with severe pain. This small dog sloughed all the skin on the ventral cervical region, ventral chest and abdomen and ultimately succumbed.**

these bites, bleeding can only occur if the action of haemorrhagins cause capillaries to leak.

There is 1 reported case of a boomslang bite in a dog[28]. Bite site bleeding was present from the moment of the bite, with systemic bleeding commencing 12 hours later. The dog collapsed approximately 24 hours after the bite and was bleeding freely from the gingiva, was pale and severely anaemic. A case of vine snake envenomation has been reported[23]. The dog showed a mild haemorrhagic tendency, an abnormal coagulation profile and made an uneventful recovery on the 3rd day without treatment. Active bleeding due to a puff-adder bite has been seen at the OVAH.

TREATMENT

General comments on snakebite antivenom

The following snake venoms are used in the polyvalent antivenom manufacturing process by the South African Vaccine Producers (Pty) Ltd.: puff-adder, Gaboon adder, rinkhals, snouted cobra, Cape cobra, forest cobra, Mozambique spitting cobra, green mamba (*Dendroaspis angusticeps*), Jameson's mamba (*D. jamesoni*) and black mamba (*D. polylepis*).* A monovalent antivenom is produced for boomslang envenomation. Antivenom comprises pepsin-refined immunoglobulins prepared from the serum of horses that have been hyper-immunised with the various snake venoms.

Appropriate antivenom can prevent further spread of swelling (cytotoxicity), prevent the onset of respiratory failure and reduce the period of ventilation (neurotoxicity) and smother coagulopathic mechanisms (haemotoxicity). Antivenom does not prevent necrosis.

Dose

A unit of antivenom neutralises a fixed quantity of venom and has nothing to do with the size of the patient. In view of venom / mass ratio, small dogs often are worse off than large dogs. The dose of antivenom cannot be set in mg/kg or m*l*/kg as for traditional drugs. Guidelines used in the OVAH for antivenom administration are as follows:

- Do not use in dogs showing no clinical signs.
- Use in cytotoxic snakebites if the patient is deteriorating and/or there are evolving systemic consequences of the bite.
- All neurotoxic snakebite victims that are symptomatic should be treated.
- Give as much antivenom as the owner can afford up to a maximum of 8 vials. One vial is better than none.
- Always give the antivenom intravenously.
- The response to a small test dose does not predict the outcome to the main dose and is therefore unnecessary.
- Injection should always be slow.
- Do not inject locally in or around the bite site.

Boomslang bite: 1 or 2 ampoules of monospecific boomslang antivenom.

Puff-adder bite: 5 ampoules of polyvalent antivenom is usually adequate.

Gaboon adder bite: up to 20 ampoules of polyvalent antivenom may be necessary.

Vine snake bite: there is no effective antivenom.

Adverse effects to South African-manufactured antivenom and antivenom manufactured elsewhere are common in humans. Adverse effects include a rash, urticaria, angio-oedema, bronchospasm and hypotension[1]. It is recommended in humans that atopic individuals be premedicated with adrenaline[1]. At the OVAH, adverse effects are rare, but when they do occur they vary from full-blown anaphylaxis (rare) to more common mild allergic responses characterised by urticaria or angioedema (Fig. 6) and occasionally vomiting and diarrhoea. Anaphylaxis requires prompt treatment with adrenaline, which is life-saving. Milder reactions are effectively managed with a single parenteral dose of a short-acting glucocorticoid. In humans, serum sickness is described and usually occurs about 10 days after treatment. It is characterised by pruritic skin rash, pyrexia and arthralgia[10,22,31]. Although there is no reason why this should not occur in dogs, it has never been specifically diagnosed or suspected at the OVAH.

Progressive swelling (cytotoxic envenomations)

Most cases of progressive swelling require very little medical attention and recover without treatment. It is important that cases that are showing signs are carefully observed for progressive swelling. Antivenom is usually not necessary. Despite previous veterinary recommendations that antivenom use in puff-adder bites in dogs was not beneficial, subsequent experience has proven otherwise. Similar observations have been made for humans[30]. Standard treatment applied at the OVAH consists of the following:

1. All cases showing swelling (unless resolving) are admitted for observation.
2. Place a cephalic catheter for crystalloid fluid administration at maintenance rates.
3. Antibiotics are unnecessary unless there is obvious necrosis (spitting cobra bites).
4. Analgesics are not widely used in the management of progressive swelling in dogs as pain seems to be minimal. Potent analgesia (buprenorphine; morphine) may be needed in dogs bitten by a spitting cobra.
5. If a case deteriorates, aggressive treatment is necessary. Signs of deterioration include:
   - Worsening weakness or depression (a deterioration in habitus).
   - A rising pulse rate, respiratory rate, a drop in rectal temperature or pale mucous membranes with sudden changes in capillary refill time.
   - Swelling that begins to impinge on the upper airway.
   - A haematocrit that continues to fall or the appearance of haemoglobinuria, haemoglobinaemia or a positive ISA test.
   - Evidence of spontaneous haemorrhage.

Recommendations for the management of the above scenarios are:

1. Volume replacement by whole blood transfusions to replace lost blood. If blood is unavailable, preferably use a synthetic colloid (hetastarch) or lastly crystalloids at shock doses.
2. Intravenous administration of as much antivenom as the owner can afford (1 vial may be life-saving but as many as 8 (or more) vials may be given). Administration even in the late phase of disease may well be helpful and should not be withheld.
3. Cases with upper airway obstruction will require tracheostomy tube placement. In many cases the ventral cervical swelling is so severe that a traditional tracheostomy tube is too short. In such cases an endotracheal tube (ET) inserted through the ventral cervical surgical tracheostomy site is useful. If an ET tube is placed by mouth, full anaesthesia will normally be necessary, which is best avoided as many of these cases will need an artificial airway for longer than a day. A tracheostomy tube needs *very* good nursing care (nebulisation to keep secretions moist, regular suction and daily replacement) to prevent complications such as blockage with dried mucous plugs.
4. Critically ill dogs should have intravenous broad-spectrum antibiotic cover. Critical illness is associated with immunosuppression and an increased risk of infection.

*Antivenom may be acquired directly from the Antivenom Unit; the contact details are: Tel +27 (0)11 386 6000; Fax +27 (0)11 386 6016; 1 Modderfontein Road, Edenvale, Gauteng; PO Box 28999, Sandringham, 2131 South Africa.

5. In very ill patients a high level of nursing care is required for assessment of urine production (urinary catheter), regular turning, toilet care of artificial airways and attention to nutrition (by naso-gastric or oesophagostomy tube) if the patient does not eat for longer than a day. Monitoring and pharmacological management of blood pressure is helpful.
6. The treatment of Mozambique spitting and other spitting cobra bites is somewhat different from the typical puff-adder bite. Haematoma formation does not occur and hypovolaemic shock is unusual. There is extreme pain (much like *Hyalomma* tick bites) and often large areas of skin necrosis. These cases should be given antivenom and a large area around the bite site should be shaved in anticipation of slough. The problems that require management relate to pain, fluid, electrolyte and protein loss that occur through the large wounds. Secondary infection is a potential risk.

Treatments that are generally not recommended include:

1. Corticosteroids. There is no rationale for the use of these drugs. They may worsen muscle weakness, enhance catabolism and cause immunosuppression. An indication for 1 dose of a short-acting steroid would be a mild adverse reaction to antivenom.
2. Non steroidal anti-inflammatory drugs are contra-indicated in volume-challenged patients. Use morphine-like drugs if analgesia is required.
3. Antihistamines.
4. Under no circumstances should large incisions be made in an attempt to drain venom or the haematoma. When humans are bitten in muscle, fasciotomy may be indicated to relieve pressure and prevent necrosis (compartment syndrome). Although this is theoretically possible in the dog (bitten in a muscle belly surrounded by fascia), the authors have never felt the need to perform this procedure. The management of such large open wounds would require expensive specialist ICU facilities.

Progressive weakness (neurotoxic envenomations)

All dogs suspected of having been bitten should be *very* carefully observed and *no* treatment should be initiated until it is obvious that signs are present. The approach at OVAH to managing symptomatic cases is as follows:

1. Place an intravenous catheter for venous access.
2. As soon as the dog becomes weak in the limbs or begins to show shallow breathing or reduced respiratory effort, an intravenous general anaesthetic is administered and an ET tube placed. An AMBU bag or a closed circuit anaesthetic machine allows manual ventilation followed by as much antivenom as the owner can afford given slowly intravenously (over about half an hour). While this is being done a mechanical ventilator can be set up (Figs 7A, 8). Ventilation (bag or mechanical) may be required for 6–12 hours while antivenom is reversing paralysis. In the OVAH general anaesthesia with pentobarbitone (or the more expensive propofol by continuous rate infusion) is maintained for 6–12 hours before attempting to wean the dog off the ventilator. Good nursing care is necessary and includes keeping the body temperature normal, regular turning, maintenance intravenous fluid and bladder care. The use of prophylactic antibiotics is controversial and most likely unnecessary.

Is it worth trying to treat these cases without a mechanical or manual ventilator?

No, as ventilatory support is crucial to success in spite of using antivenom.

Is it worth trying to treat these cases without antivenom?

Yes, as ventilatory support is life-saving but this strategy is not encouraged.

In 2 cases of human Cape cobra envenomation, up to 19 vials of antivenom had no effect in reversing NMJ blockade and long-term ventilation was required. Once complete flaccid paralysis has become established it seems the antivenom has minimal effect[6].

It is clear from our experience at the OVAH that some dogs develop obvious local swelling and even skin necrosis in addition to neurotoxic effects, requiring ventilator support. This dual neuro- and cytotoxic effect may be ascribed to the snouted cobra in our hospital (Figs 7B, 9).

The complications associated with these bites usually relate to mechanical ventilation or acute adverse antivenom reactions. The haematological consequences seen in cytotoxic envenomations are not a feature of neurotoxic bites.

Bleeding (haemotoxic envenomations)

Antivenom is more important in the bleeding syndrome than in the syndromes of progressive swelling or progressive weakness where good supportive measures alone may be life-saving. Antivenom is indicated if there is active bleeding (internal or external), blood fails to clot in a test tube or there is laboratory evidence of significant coagulopathy (prothrombin index less than 50%, partial thromboplastin time more than double the control).

Be aware of haematoma formation at injection sites and gain peripheral venous access so that possible bleeding is more easily controlled. Fresh whole blood or fresh plasma transfusion may be necessary. Monitor urine output in case of renal failure.

Heparin is contraindicated as venom-induced thrombin is resistant to its action[29]. Fibrin-stabilising drugs may convert a DIC with a good prognosis to one with a bad prognosis. Thrombolytics would aggravate the situation.

In humans, antivenom is effective even in late-stage disease when active haemorrhage is well established[15] and the same observation has been made in the OVAH.

Venom ophthalmia

Ocular envenomation is caused by squirted venom from the spitting cobras and the rinkhals. All cases seen at the OVAH had large corneal erosions, oedema and severe chemosis. Venom is a caustic (basic) agent that forms soaps with the lipids of the corneal cell membranes and disrupts glycosaminoglycan ground substance, causing softening of the tissue and devitalisation of corneocytes. These actions may continue after liberal ocular irrigation[13,14]. Treatment involves the use of liberal amounts (around 1 ℓ per eye) of a sterile isotonic irrigation agent (such as Ringers lactate). Pain is intense and the dog may well require sedation to allow proper irrigation. Before using any other topical agent, the cornea should be stained with fluorescein. Topical corticosteroids should not be used in the presence of corneal ulceration but oral corticosteroids are beneficial in managing chemosis. A sterile uveitis and/or hypopyon frequently develops within a day. Topical antibiotics are indicated and atropine assists in pain management. Pain is as a result of the loss of corneal epithelium which stimulates the ophthalmic branch of the trigeminal nerve, causing a reflex miosis. The spasm of the iris muscles drives the pain response felt by the patient. Effective relief can be achieved by paralysing the iris with atropine that will cause cycloplegia and mydriasis. Epinephrine has similar but far weaker effects. The longer the time interval between envenomation and treatment the longer the period for which treatment will be required (possibly weeks). The cornea is a resilient tissue and has a remarkable ability to regenerate.

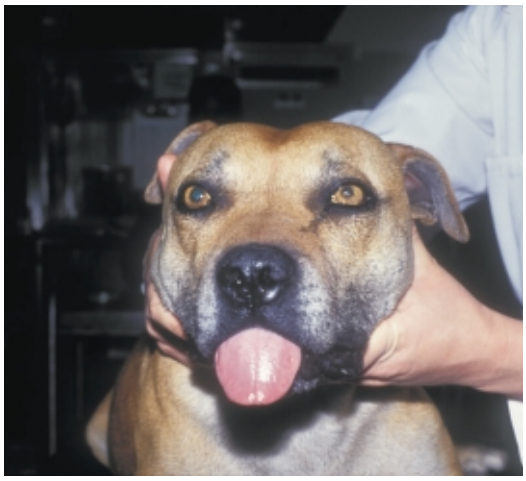

Fig. 5: **Bulbar paralysis following neurotoxic snake bite. Note the tongue paralysis and anisocoria. This dog was unable to swallow and was minutes away from requiring a general anaesthetic, tracheal entubation and assisted ventilation.**

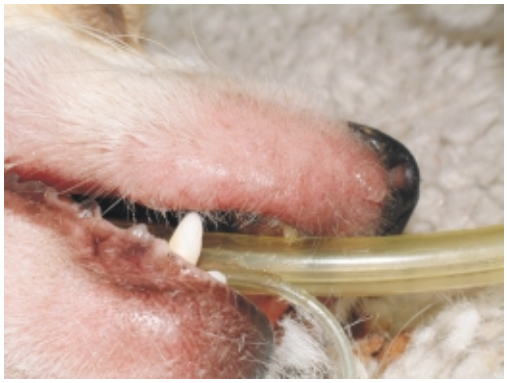

Fig. 6: **Adverse reactions to the use of polyvalent anti-serum are fairly common but mild. Following a large dose given intravenously to this small dog bitten by a cobra, moderate facial angio-oedema and hyperaemia developed. Such reactions are independent of antivenom dose.**

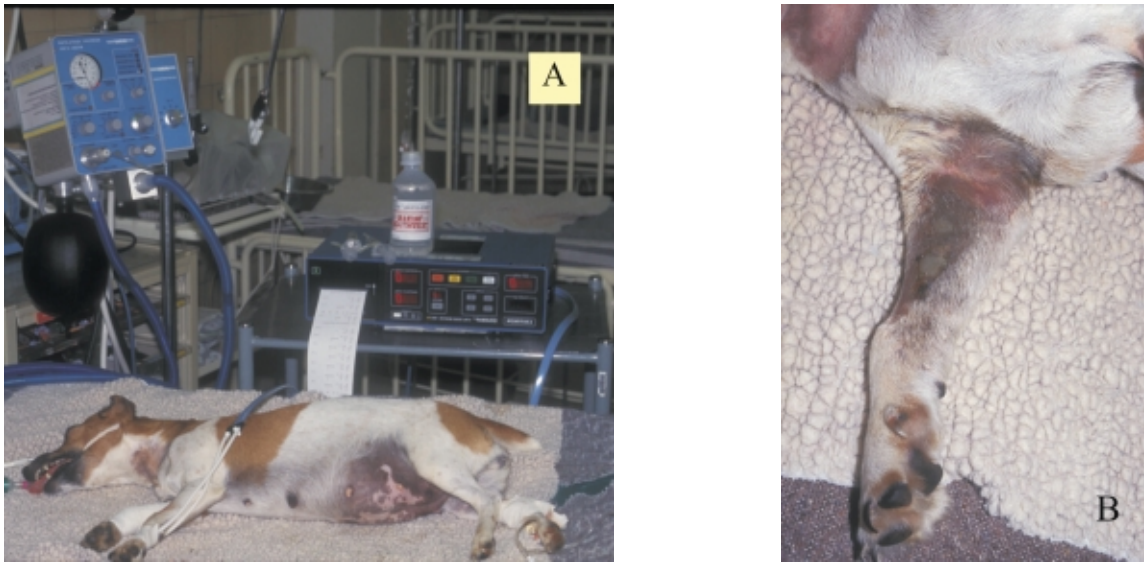

Fig. 7: **Bites from the snouted cobra** (*Naja annulifera*) **are classically neurotoxic. Treatment of these cases requires intravenous anti-serum and mechanical ventilation** (A)**. Occasionally signs of cytotoxic injury are seen as illustrated in** (B) **where a well circumscribed area of tissue necrosis can be seen medially on the front leg.**

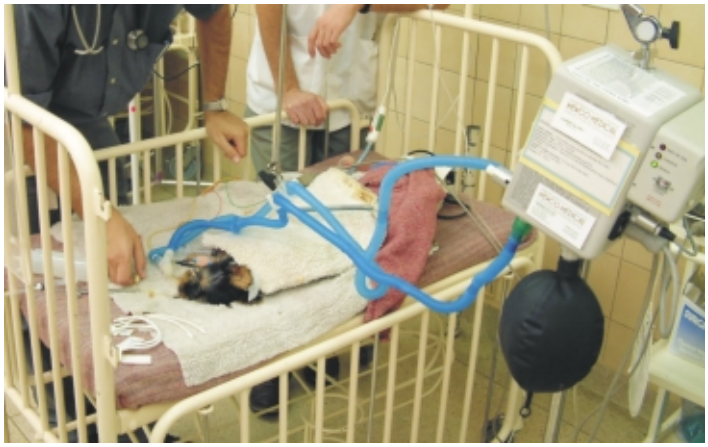

Fig. 8: **Neurotoxic bite victims require mechanical ventilation for up to 12 hours following the use of antivenom. Smaller patients have a worse prognosis.**

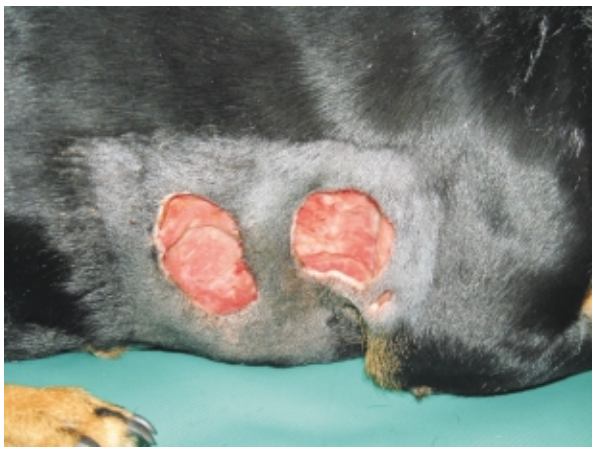

Fig. 9: **Following a bite from a snouted cobra** (*Naja annulifera*), **this dog developed classic neuromusucular junction blockade and required a large dose of antivenom and assisted ventilation. A few days after this 2 large areas of skin necrosed on the lateral chest wall – probably the bite site. Snouted cobra** (*Naja annulifera*) **bites may be associated with obvious cytotoxic effects.**

CONCLUSIONS

There are many similarities in terms of clinical presentation, treatment and outcome between dogs and humans following a venomous snakebite. Key differences include: (1) the way a diagnosis is made (humans can usually provide an accurate history of snakebite and in dogs the diagnosis is usually made based on presenting clinical signs); (2) whereas humans are usually bitten below the knee, dogs are usually bitten around the head and neck, which in the case of cytotoxic bites may result in asphyxiation; (3) bites due to the boomslang or vine snake (coagulopathic venoms) may well be more common in dogs than in man. Polyvalent antivenom is definitely indicated in all neurotoxic envenomations where signs of NMJ blockade are clear; in all complicated cases of cytotoxic bites and in intractable bleeding following coagulopathic envenomations. Neurotoxic envenomations require some form of mechanical ventilatory support in addition to antivenom treatment and complicated cytotoxic bites may well require surgical intervention to maintain a patent airway in addition to vigorous intensive care. It is interesting to note that the snouted cobra may well be responsible for local cytotoxic effects (including tissue necrosis) and the typical NMJ blockade associated with neurotoxic venoms (the Cape cobra and rinkhals have also been similarly implicated in human bites). Generally morbidity (in terms of cost to the owner and suffering to the dog) due to snakebite in dogs is high but mortality is low.

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