

Surgery in Africa - Monthly Review

Snakebites in children in Africa: a practical approach to management

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Introduction

The management of snakebite in children is controversial, with advocates of differing protocols vigorously defending their views (1). The controversies are however, at least in part, due to the fact that the fauna in the innumerable micro and macro environments on this continent are unique and not directly comparable with those of other areas and that it is not possible to develop an algorithm that caters for all possible variations. In a space as large and as geographically diverse as Africa there are habitats that suit widely differing populations of reptiles. Similarly resources for the management of patients differ considerably across the continent and management protocols must be regionally developed and regionally specific.

As it is impossible to derive an algorithm suitable for all parts of the continent some guiding principles are described.

Risk factors

Children are at risk of snakebite for several reasons. Firstly their natural curiosity, and in some a testosterone effect, means that children may innocently invade the reptile's space and be perceived as a threat. Secondly, particularly in rural areas where small boys are used as herdsmen, there is an increased risk due to the increased potential for interaction, and very frequently the lack of protective footwear. Thirdly, the small mass of the child means that any given dose of venom will have a greater effect as it will be distributed throughout a smaller volume (2).

It is important to remember that not all snakes are venomous, and not all bites in children are caused by snakes. Inspection of the wound is important. It should be clinically possible to distinguish a snake's bite from a scorpion sting or spider bite.

When a snake envenoms a victim it is an offensive strategy designed to paralyse, kill and make digestible a potential meal. However no venomous snake seriously believes that it can eat prey as large as even a very small child. Therefore, biting a child is a defensive action. When biting defensively a snake may not inject venom into the victim, a so called "dry bite". This

probably happens more often than realized as many well documented bites by venomous species have resulted in no clinical morbidity (3). In Guinea a dry bite rate of 12% has been reported amongst 76 patients (4) and in Cameroon in 25% of more than 300 patients (5). Whatever the regional situation it is important to recognize that dry bites do occur and to avoid over-treating patients who are not at risk.

Many other factors such as the size of the reptile, the length of time since it last emptied its venom sacs and the state of its dentition will affect the volume of venom injectable. Thus not all venomous snakebites, even from the same species, are equal.

Two principles can be derived. Firstly snakebite is in many instances preventable and energy spent on educating children, particularly small boys, about snakes and how to avoid conflict with them, is energy well spent. Secondly the panic and pandemonium that surrounds snakebite is frequently unjustified.

The habit of killing snakes on sight, the “only good snake is a dead one” philosophy, is unfortunate and unnecessary. Unfortunate: because it perpetuates the notion that these animals are uniquely dangerous, and unnecessary: because reduction of conflict between our species can be achieved with no deterioration in the quality of our lives at all.

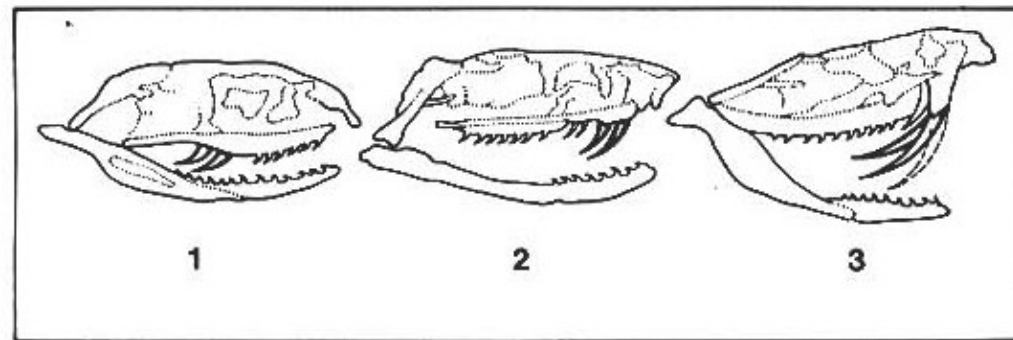
Rational management is not served by emphasis on the rare and spectacular. Most snakebite victims can be safely managed in hospitals without tertiary care facilities (6).

Fang anatomy/species

Venomous snakes have fangs and leave fang-marks. Non-venomous snakes do not have fangs. They have small teeth and leave a bite similar to a small rodent bite. Clinical examination of the unadulterated site of the injury can thus determine whether the offending reptile was venomous or not. When there is local swelling or the area has been secondarily assaulted by a traditional healer or misguided first-aider this important clinical sign may be masked.

Venomous snakes either have their fangs at the front of their mouths, or at the back. (Fig 1) If the fangs are fixed they must, of necessity, be short or the snake would be unable to close its mouth. Many adders have hinged fangs that can be folded back against the palate when not needed. In some species such as the Gaboon adder (*Bitis gabonica*) and Puff Adder (*Bitis arietans*) such fangs may be very long and penetrate deeply into the tissues during a bite. Fixed fanged snakes very often deposit venom into subcutaneous tissues rather than deep to the fascia of a limb although in the small child even short fangs may penetrate the fascia.

Back-fanged snakes, such as the boomslang (*Dispholidus typus*), typically are unable to release their victim which is ensnared in the caudally placed fangs. This technique of restraining prey is ideal for a snake that lives in trees and eats birds but when a human is bitten it appears as if the snake is clinging on and it must be forcibly removed. As such snakes must draw their victim into their mouth to use their posteriorly placed weaponry they cannot bite on flat body surfaces or large limbs and rarely, if ever, bite more proximally than the wrist or ankle. In spitting snakes the venom duct ends on the anterior surface of the fang rather than at its tip.



Snake fangs: 1. Posterior 2. Anterior-fixed 3. Anterior-hinged

Figure 1. The anatomy of snake fangs represented by 1. Boomslang (*Dispholidus typus*) 2. Black mamba (*Dendroaspis polylepis*) 3. Puff adder (*Bitis arietans*)

Tourniquets

Many patients in Africa seek primary advice from a traditional healer and various potions and incisions are prescribed (7). These serve only to delay presentation to hospital and delay is a recognized poor prognostic factor (8). Regrettably the use of a tourniquet has become the standard first-aid measure applied to snakebite victims. Concerns revolve around the tightness of the tourniquet and the duration for which it is applied. Clearly a tourniquet applied at a pressure greater than systolic pressure will rapidly result in irreversible ischaemia in the affected limb. Due to the long interval between injury and presentation to hospital, incipient gangrene of the leg below a tourniquet is not an uncommon presenting feature, and it is hard to blame the snake for this morbidity. Eventual release of a tourniquet results in the ischaemia-reperfusion injury associated with the washout of the products of anaerobic metabolism. Reperfusion injury itself may be responsible for considerable toxicity, circulatory instability and metabolic derangement (9) and myoglobinuria following reperfusion of ischaemic muscle may lead to pigment nephropathy and renal failure. It has been argued that application of a tourniquet whilst delaying entry of venom into the general circulation, by confining the venom in the bitten area, may increase the local concentration of venom and therefore the local toxic effect. In bites by snakes with a predominantly cytotoxic venom profile this would result in greater tissue damage (10).

Current practice is to advise primary care givers to regard the affected limb as if it was fractured and to treat it accordingly with splintage, a firm crepe dressing from digits to the root of the limb and to prevent usage. This effectively limits lymphatic flow without compromising vascular supply to the limb (6).

Venom/Antivenom

Venom is a complex mixture of biologically active peptides representing modified saliva. In many instances it is difficult to distinguish between a primary venom effect and the response to the injection of these peptides in terms of cytokine and other endogenous peptide release.

Traditionally snakes are classified according to the primary action of their venom into cytotoxic, neurotoxic and haemotoxic varieties. In fact this classification recognizes only the dominant feature of the venom and all snakes inject a wide range of toxic peptides or evoke a range of “toxic” responses from the tissues. Thus it is not unusual to see bleeding or nystagmus following a puff adder bite, which is supposedly purely cytotoxic (11); or necrosis surrounding a viper bite, that is supposedly haemotoxic (12) or a cobra bite, that is supposedly neurotoxic (13).

Polyvalent antivenom is available in southern Africa from the SAIMR and includes the venom of a wide range of snakes prevalent in the region. In many other parts of Africa appropriate antivenoms are available, but not in all. No antivenom can include the venom of all toxic snakes. In southern Africa notable exclusions are the small adders and the boomslang. In the case of the latter specific antivenom is available. As bites from the small adders in both southern and western Africa are unlikely to result in morbidity their inclusion is unnecessary (14).

So it is still important to take a history in an attempt to define the species responsible for an attack to avoid placing the patient at risk of unnecessary administration of antivenom. The singular feature of a bite by a back-fanged snake has already been mentioned. Giving polyvalent antivenom to a patient with a history suggestive of a back-fanged snakebite does no good and may do considerable harm. Similarly antivenom is not indicated, and is in fact contraindicated, if history or clinical findings suggest assault by one of the many small adders. The puff adder, bitis arietans, which is responsible for many bites in children in southern Africa (15), is notoriously sluggish and reluctant to move away from paths etc. until trodden on. It then reacts with lightning speed and has long hinged fangs that allow it to deposit venom deep into the tissues. In West Africa the principal offender is Echis Carinatus, the saw-scaled viper which can have local cytotoxic as well as an invariable haemotoxic effect (16).

Antivenom is raised by the incremental injection of venom into horses. Like many other products of horse serum it has the potential to stimulate severe anaphylactic reactions. In our own experience 50% of patients to whom antivenom was administered developed an anaphylactic response. In a similarly large experience from northern KwaZulu-Natal, Blaylock found that only 3% of 333 patients required antivenom and 41% of those given antivenom suffered from adverse effects (17). Similarly, high incidences of complications have been reported from other centres using the South African antivenom (18); whilst in Cameroon, using an entirely different antivenom, a 6% incidence of adverse effects is reported (19). In a pilot study in Northern Nigeria four of seven patients given a monovalent antivenom against Echis Ocellatus experienced adverse reactions requiring adrenaline (20) and in a larger series using an older monovalent echis carinatus antivenom 21 % of patients developed immediate adverse reactions (12). In an intensive care setting these are manageable complications. On the side of a mountain they are not.

The ultimate tragedy would be to lose a patient to an anaphylactic reaction in the field following horse serum injected for a bite by a snake whose venom is not represented in the antivenom cocktail. It is preferable to avoid antivenom in situations that are not immediately life-threatening, to limit its use to cases in which the responsible snake has been identified either by direct observation or by recognition of the clinical diathesis, and then use it only with the knowledge that the venom of the responsible snake is included in the cocktail of antivenom that is available. Ideally facilities to treat anaphylaxis should be at hand (18). When antivenom is to be administered it is important to be aware that the dose required is determined by the amount of venom injected, not the size of the victim. Therefore the dose required for a child should be the same as for an adult (2). Antivenom should only be injected intravenously.

Most children with snakebites do not need antivenom in order to make a complete recovery. Exposing them to the risk of anaphylaxis therefore seems difficult to justify. In many parts of the world envenomation can be diagnosed by “detection kits” that identify venom degradation products in urine (22).

No such tests are available for Southern African snakes. Envenomation must then be inferred from the clinical findings or indirectly from investigations such as the thromboelastogram (TEG).

As all snake venom has at least some coagulopathic effect the TEG, which measures functional defects in the process of coagulation (22, 23) will identify those patients who have been envenomed or are suffering from the ischaemia-reperfusion syndrome that will also impair coagulation. A normal TEG means that there has been no interference with blood coagulation from any cause (24). Dry bites can thereby be effectively recognized and overtreatment avoided. A caveat is the boomslang from whom the venom effect on the haemostatic cascade can take up to four days to become clinically apparent (25). Salooja provides a comprehensive review of the philosophy and practicalities of thromboelastography. (23)

Compartment Syndrome

Although when strictly defined a compartment syndrome implies loss of function of structures within an affected osseo-fascial compartment it is a term loosely used to denote an increase in compartmental pressure (26).

In the limbs compartmental pressure can be raised by a variety of insults but relevant here are the swelling associated with venom injection deep to the deep fascia on one hand and the swelling associated with the reperfusion injury on the other. Subcutaneous injection of venom causes considerable swelling that is reflected in an increase in compartmental pressure but this is rarely sufficient to threaten the limb (27).

It is the early recognition of impaired nutrient delivery to muscle cells that is the aim of clinical monitoring. Increasing pain particularly on passive stretching of the muscles within the compartment, paraesthesia, paralysis, and a feeling of hardness or pressure over the compartment are strongly suggestive of increased compartmental pressure. Because capillary occlusion occurs at a lower pressure than axial artery occlusion, the pulses will be present. This is important as it is not uncommon for clinicians to have a false sense of security because they can feel peripheral pulses. Capillary blood flow can be completely obstructed and the muscle cells profoundly hypoxic or already dead, in the presence of bounding peripheral pulses.

In the preverbal or fractious child clinical assessment of compartment pressure may be impossible. It is also apparent that the critical pressure at which the microcirculation ceases, leading to tissue death, must be related to, although considerably lower than, the systemic blood pressure. In children, and particularly in the hypotensive child following snakebite, microcirculatory arrest may occur at compartmental pressures that would not cause any concern in an adult (28).

It is ideal therefore to measure both the intracompartmental pressure and the systemic blood pressure and to make a therapeutic decision based on their relationship. We advise fasciotomy when the compartmental pressure has risen to within 30mm Hg of the mean arterial pressure. This balance can be altered in the child's favour by increasing the systemic blood pressure. Fluid resuscitation is therefore a cornerstone of therapy (29).

Measuring compartmental pressure need not be a complicated or difficult procedure. A hand held needle manometer is an elegant tool for the procedure but one can use the same apparatus that anaesthetists use to measure direct arterial pressures, attached to a fluid filled needle inserted into the suspect compartment (30), remembering to accurately zero the apparatus at the level of the needle tip, or, if pushed, a water filled piece of plastic tubing attached to a water filled needle that has been inserted into the compartment, and a ruler. It is wise to take three measurements and calculate the mean (31). As snakebite injury is an evolving rather than a static pathology it is possible to repeat the measurements at will.

Many children in our experience arrive after a prolonged application of a tourniquet with established muscle death in the affected limb. No amount of resuscitation will restore dead tissue to vitality but it may protect marginal tissue allowing a useful limb to be salvaged. Thus even in late presenters aggressive resuscitation is essential.

It should be remembered that compartment syndrome following snakebite or tourniquet use is the commonest reason for amputation in children in many areas of Africa (28) and vigilance is the best defence against pressure induced cell death.

Fasciotomy and fasciotomy closure

Increasing compartmental pressure results in failure of the microcirculation within the compartment, and it is upon a patent capillary circulation running at capillary pressures that cells depend for their survival. Oxygen provision to cells is therefore impaired at much lower pressures than the systemic blood pressure. Although capillary pressures are clearly related to systemic blood pressure they must always be lower and it is again emphasized that cell death may occur whilst peripheral pulses remain easily palpable.

If critical compartment pressures are confirmed by direct measurement or strongly suspected on clinical grounds, fasciotomy should not be delayed. Whilst theatre is being arranged it is important to continue aggressive fluid resuscitation, provide oxygen and to keep the limb at heart height – not elevated - in order to maximize oxygen delivery to muscle cells (32).

On incision of the fascia in a normal individual the muscle will bulge out and this alone does not predict successful decompression. Incisions should encompass the whole length of the compartment and in the leg all four compartments should be released (33) particularly if there is a history of a tourniquet having been applied. In patients with a venom-related bleeding diathesis this should be addressed prior to surgery.

Following fasciotomy wounds can be left to heal by second intention with an acceptable cosmetic and functional result (34).

Supportive measures

It seems reasonable to ensure prophylaxis against tetanus for all victims of snakebite (35). Care must of course be taken in the use of anti-tetanus serum which is again a horse serum product. Antibiotic cover against anaerobes also seems sensible despite the lack of anaerobes in the oral flora of a small sample of snakes in South Africa (36) Anaerobes and gram negative rods were the dominant oral flora in snakes in Thailand (37). Broad spectrum antibiotics are necessary in patients with ulceration present or imminent (38).

Analgesia is essential, and this should be intravenously administered, as a regular dosage, not on a prn basis. It will not mask the signs of developing compartment syndrome.

Intravenous fluid volume must be restored to replace fluid sequestered in the affected limb. Oedema may be progressive and replacement must keep up with these losses. As they are not directly measurable losses the aim is to keep the circulatory space full. Direct clinical signs that this has been achieved include a normal mental status, warm pink peripheries and a urine output of 1 ml/kg/hr on an ongoing basis.

Thus in patients with moderate or severe envenomation it is ideal to have a secure intravenous line and a urinary catheter inserted so that this balance can be regularly reassessed. ECG and oxygen saturation monitoring should be used if available.

Monitoring

There is no doubt that snakebite as a clinical syndrome is not static but evolves and the patient is entitled to regular repeated clinical observation. If deterioration is noted and this deterioration can be ascribed to progressive envenomation, the use and particularly the dose of antivenom must be reviewed. It is again emphasized that antivenom dosage is dependent on the dose of venom injected at the time of injury, not the size of the victim, and that failure to respond may indicate that further antivenom is necessary. However deterioration can also be due to fluid depletion, sepsis or pain.

Management protocol

Figure 2 outlines our management algorithm that has at its core clinical evaluation. Initially it is essential to make the diagnosis of a bite by a venomous snake. Thereafter, clinical diagnosis of envenomation determines future management. Those of us fortunate enough to have access to TEG monitoring use it at this level to aid in the exclusion of children who have not been envenomed (23). Clinical assessment is also key to deciding whether antivenom is justified in terms of the patient's clinical course, the likely or confirmed species responsible for the attack and a knowledge of the snakes represented in the locally available polyvalent antivenom. There is no time limit after which antivenom is no longer used and

we have effectively used antivenom up to five days post injury. When antivenom use is deemed necessary a test dose is often given. The dose of antivenom to be injected intravenously is determined by the snake not the patient. We routinely start with four ampoules of SAIMR antivenom, irrespective of the size of the patient, and are prepared to repeat the dose as required.

All patients should have the compartments of the affected limb clinically monitored for increasing pressure. Remember that the presence of peripheral pulses is not helpful. If there is clinical difficulty in establishing the presence or absence of increasing compartment pressures, and in our experience there usually is, it is helpful to measure the pressures by whatever means are available. This must be related to the mean arterial pressure of the victim. Fasciotomy is not a benign procedure and we prefer to perform it only when we have documented the need for it, but it is always preferable to an amputation.

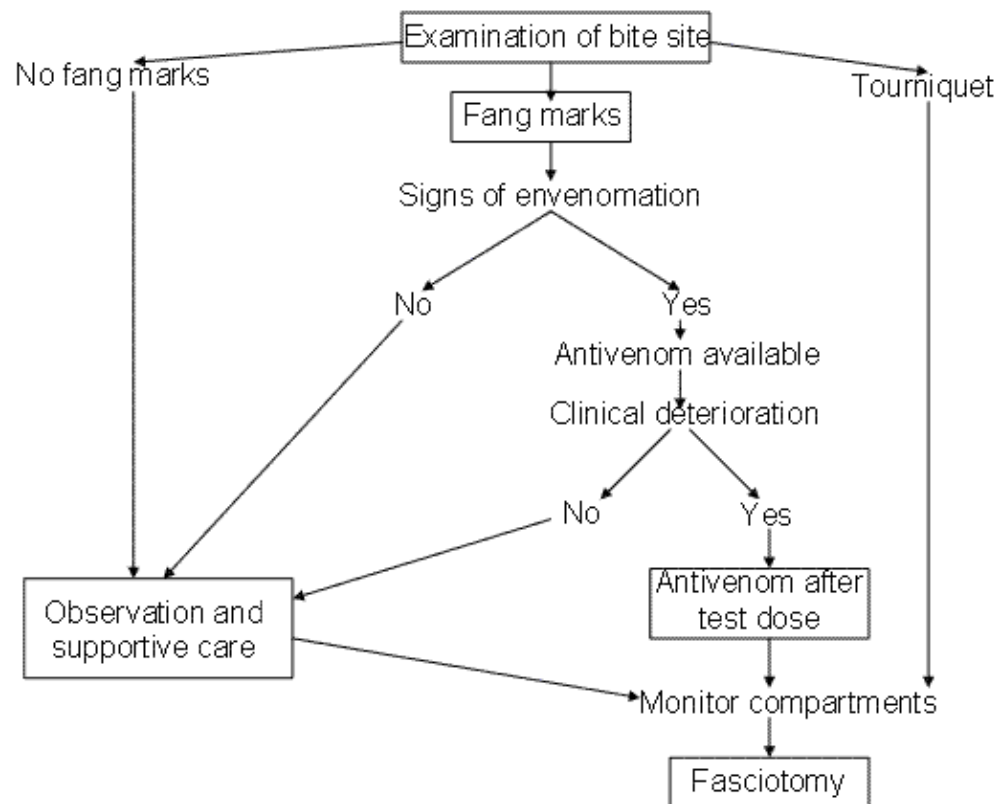


Figure 2; Algorithm of care used in Durban for the management of children with snakebite. “Signs of envenomation” in our practice include assessment of the TEG. “Monitoring of Compartments” includes serial invasive pressure measurements whenever clinical signs are unreliable. Fasciotomy is performed if the compartment pressure rises to MAP – 30 mmHg (where Mean Arterial Pressure is equivalent to the Diastolic Pressure plus one third of the pulse pressure.)

Conclusion

There is a wide variety of snakes in Africa occupying a wide variety of habitats. Health care facilities in different countries vary as do the availability of appropriate antivenom and other

elements of emergency care. Thus there can be no universal protocol of management.

Fortunately most snakebite victims require supportive care only and can be safely managed in hospitals without tertiary facilities. The principal risk, after envenomation has been identified and managed, is the risk of a compartment syndrome either due to the snake or an ill-advised tourniquet. In children clinical signs of increasing compartmental pressures are difficult to elicit and direct compartmental pressure measurement is able to give objective evidence to reassure the attending physician or to mandate fasciotomy.

Our traditional healers should be targeted for education on primary care as they appear to be consulted early by many victims.

Recommendations

1. Snake populations, and therefore the patterns of injuries they cause, are regionally specific and require regionally specific protocols of management.
2. Throughout Africa primary care following snakebite is often sought from traditional healers who should be encouraged to incorporate modern concepts of care into their regimens, or at least to avoid harmful interventions. They currently contribute to the frequent delay between injury and presentation to hospital.
3. Dry bites are common and patients who have not been envenomed must be recognized early to avoid over-treatment that may be dangerous. We have found the TEG to be helpful in this decision but acknowledge that it is not widely available.
4. The use of tourniquets should be strongly discouraged as they make the diagnosis of envenomation difficult, threaten the affected limb and have no demonstrable beneficial effects.
5. Antivenom, if available, should be used judiciously. There is a risk of adverse or anaphylactic response that seems to vary with the antivenom being studied but is always considerable. Antivenom should only be considered when the patient is confirmed to have been envenomed, is at risk of major morbidity or mortality and facilities for resuscitation are at hand. In essence this means that antivenom should only be administered in hospital, not in the field.
6. Victims of snakebite should be offered tetanus prophylaxis and broad spectrum antibiotics. Analgesia should not be forgotten.
7. Patients must be monitored for the signs of increasing intra-compartmental pressure and the care-giver must be prepared to perform a fasciotomy should the ICP rise to within 30mmHg of mean arterial pressure.
8. Patients should however be aware that generally mortality from snakebite is rare and with appropriate care morbidity should be minimal.

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