Observations on the bite of the Mozambique spitting cobra 
(Naja mossambica mossambica)

C. R. TILBURY

Summary

Experience in the treatment of bites by the Mozambique spitting cobra, Naja mossambica mossambica Peters, is described, and it is shown that the predominant effect of the venom is cytotoxic. The gross pathology of the bite is presented.

A large percentage of the bites (94%) occurred inside human dwellings, and of these, 81% occurred while the victims were asleep.

The efficacy of the SAIMR's polyvalent antivenom was tested, and the evidence suggests that its effectiveness is indirectly proportional to the time which has elapsed between the bite and the administration of the antivenom.

A comparison of the data with those presented by Warrell et al. in respect of bites by Naja nigricollis in Nigeria, shows that the clinical effects of bites by N. mossambica and N. nigricollis are closely related.

The effects of snakebite venom in southern Africa can be divided into haemotoxic, cytotoxic and neurotoxic groups, these effects in humans being caused by the back-fanged snakes, adders and elapid snakes respectively. During the past few years this straightforward 'traditional' approach has become more complex, since the bite of the berg adder (Bitis atropos) has been shown to cause neurotoxic symptoms, and bites of some of the elapids, notably the spitting cobras of the genus Naja, cause cytotoxic effects.

Published reports of the effects of spitting cobra bite are rare, and the literature generally infers that the venom is neurotoxic, in keeping with that of other cobras. However, a few recent reports have demonstrated clinical signs of swelling and necrosis following spitting cobra bites, without obvious neurological involvement.

In this article a review of 17 cases of envenomation by the Mozambique spitting cobra, Naja mossambica mossambica, is presented.

The snake

The spitting cobras and the ringhals (Hemachatus haemachatus) have the ability to 'spit' their venom. This is accomplished with the aid of specially adapted fangs, in which the venom canals open at right-angles. The stream of venom ejected forms a fine spray which may be accurate at up to 3 - 4 m. The fangs are relatively short, averaging about 3 mm, but may reach 6 mm in large specimens. The southern African spitting cobras of the genus Naja are currently referable to four taxa:

1. Naja nigricollis nigricollis: only recorded in the eastern Caprivi region of South West Africa.
3. Naja nigricollis woodii: a black form inhabiting semidesert regions of southern South West Africa and Great Namakaland, down to Citrusdal in the Cape Province.

The Mozambique spitting cobra (N. m. mossambica) is a very common snake in Zululand and is well known to the rural inhabitants by the name M'fesi. It is generally a small cobra, rarely exceeding 1,5 m. The colour varies from pale grey to dark olive on the dorsum, with each scale being edged in black. The ventral surface may be from pale yellow to salmon pink, with irregular black cross-bars on the throat. The snake is essentially nocturnal in habit, although it may be encountered abroad, often basking, during daylight hours. It has a varied diet, consisting of small mammals, other snakes, lizards, birds and sometimes even insects, but its main diet is toads.

Patients

During the period 1 October 1979 - 11 December 1980, 16 cases of confirmed or presumed M'fesi bite were treated at the Ngwelezana State Hospital, Empangeni, and 1 case at Mandini, Zululand (Table I). Positive identification of the snakes involved has posed problems, owing to the tendency of the rural Black to confuse the species and throughout the Transvaal, Botswana and north-eastern South West Africa into southern Angola.

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The other cobras found in the area are the Egyptian cobra, N. haje annulifera, and the rare forest cobra, N. melanoleuca. The venom of the former is known to be potent and predominantly

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### TABLE I. DETAILS OF VICTIMS OF N. M. MOSSAMBICA ENVENOMATION

<table>
<thead>
<tr>
<th>No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Bite site</th>
<th>Activity when bitten</th>
<th>Time from bite to admission (h)</th>
<th>Swelling</th>
<th>Other symptoms noted</th>
<th>SAIMR serum (ml)</th>
<th>End-result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12</td>
<td>M</td>
<td>Upper arm</td>
<td>Asleep</td>
<td>27</td>
<td>Severe</td>
<td>Drowsiness (hypoavolaemia), diarrhoea</td>
<td>—</td>
<td>Superficial necrosis (20 cm²)</td>
</tr>
<tr>
<td>2</td>
<td>40</td>
<td>M</td>
<td>Dorsum of foot</td>
<td>Bitten at entrance to house</td>
<td>9</td>
<td>Mild</td>
<td>—</td>
<td>—</td>
<td>Superficial necrosis (8 cm²)</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
<td>F</td>
<td>Outer thigh</td>
<td>Asleep</td>
<td>3</td>
<td>Mild</td>
<td>—</td>
<td>100</td>
<td>Marble-sized abscess</td>
</tr>
<tr>
<td>4</td>
<td>65</td>
<td>M</td>
<td>Above ankle</td>
<td>Asleep</td>
<td>46</td>
<td>Severe</td>
<td>—</td>
<td>—</td>
<td>Superficial necrosis (± 260 cm²)</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
<td>F</td>
<td>Dorsum of foot</td>
<td>Asleep</td>
<td>9</td>
<td>Severe</td>
<td>Drowsiness</td>
<td>100</td>
<td>Superficial necrosis (± 120 cm²)</td>
</tr>
<tr>
<td>6</td>
<td>13</td>
<td>M</td>
<td>Mid-lumbar over L3</td>
<td>Asleep</td>
<td>2.5</td>
<td>Mild</td>
<td>Vomiting</td>
<td>—</td>
<td>Superficial necrosis (10 cm²)</td>
</tr>
<tr>
<td>7</td>
<td>5</td>
<td>F</td>
<td>DIP joint of middle finger</td>
<td>Asleep</td>
<td>1.5</td>
<td>Mild</td>
<td>—</td>
<td>—</td>
<td>Hypermobile DIP joint</td>
</tr>
<tr>
<td>8</td>
<td>80</td>
<td>F</td>
<td>Calf posterior</td>
<td>Asleep</td>
<td>7</td>
<td>Severe</td>
<td>Vomiting</td>
<td>100</td>
<td>Superficial necrosis (± 200 cm²)</td>
</tr>
<tr>
<td>9</td>
<td>6</td>
<td>M</td>
<td>Dorsum of foot</td>
<td>Walking inside house</td>
<td>96</td>
<td>Moderate</td>
<td>—</td>
<td>—</td>
<td>Superficial necrosis (50 cm²)</td>
</tr>
<tr>
<td>10</td>
<td>11</td>
<td>M</td>
<td>Lateral side of foot</td>
<td>Stood on snake inside house</td>
<td>3.5</td>
<td>Mild</td>
<td>—</td>
<td>—</td>
<td>Superficial necrosis (4 cm²)</td>
</tr>
<tr>
<td>11</td>
<td>28</td>
<td>M</td>
<td>Dorsum of foot</td>
<td>Asleep</td>
<td>2</td>
<td>Mild</td>
<td>Vomiting</td>
<td>100</td>
<td>No sequelae</td>
</tr>
<tr>
<td>12</td>
<td>12</td>
<td>M</td>
<td>Above inner canthus, left eye</td>
<td>Asleep</td>
<td>3</td>
<td>Severe</td>
<td>—</td>
<td>—</td>
<td>Soft-tissue necrosis (2,5 cm²)</td>
</tr>
<tr>
<td>13</td>
<td>30</td>
<td>M</td>
<td>Base of first toe</td>
<td>Asleep</td>
<td>2.75</td>
<td>Mild</td>
<td>—</td>
<td>—</td>
<td>Superficial necrosis (± 2 cm²)</td>
</tr>
<tr>
<td>14</td>
<td>9/12</td>
<td>F</td>
<td>Over ramus of mandible</td>
<td>Asleep</td>
<td>7</td>
<td>Severe</td>
<td>Drowsiness</td>
<td>—</td>
<td>Soft-tissue necrosis (± 70 cm²)</td>
</tr>
<tr>
<td>15</td>
<td>40</td>
<td>M</td>
<td>Forefinger</td>
<td>Handling snake</td>
<td>25 min.</td>
<td>None</td>
<td>—</td>
<td>80</td>
<td>No sequelae</td>
</tr>
<tr>
<td>16</td>
<td>16</td>
<td>F</td>
<td>Dorsum of foot</td>
<td>Asleep</td>
<td>3.5</td>
<td>Moderate</td>
<td>Drowsiness</td>
<td>100</td>
<td>Superficial necrosis (± 50 cm²)</td>
</tr>
<tr>
<td>17</td>
<td>9/12</td>
<td>M</td>
<td>Base of thumb</td>
<td>Asleep</td>
<td>5.5</td>
<td>Severe</td>
<td>Drowsiness, vomiting</td>
<td>55</td>
<td>Superficial and deep necrosis (± 12 cm²)</td>
</tr>
</tbody>
</table>

Snake positively identified as Naja mossambica mossambica Peters. DIP = distal interphalangeal (joint).
Fig. 1. Area of the study indicating patient distribution.

0 - 3 hours: moderate-to-severe pain at the site of the bite, associated with a progressively enlarging area of swelling and warmth of the affected area.

3 - 5 hours: first signs of impending tissue necrosis may appear at the site of the bite, either as a dark discoloration or as a raised blister. Serosanguineous fluid may begin to ooze from the bite or from self-inflicted cuts.

5 - 9 hours: the discoloration begins to spread from the bite site, often caudad and occasionally in a 'skipping' fashion, leaving areas of normal skin in between (Fig. 2).

9 - 48 hours: plum-coloured discoloration reaches its maximum extent between 36 and 48 hours, and becomes sharply demarcated.

48 - 72 hours: swelling reaches its maximum. The plum-coloured discoloration darkens to black. Blisters may now appear within the discolored area, but usually first appear at the periphery of the necrotic area (Fig. 3).

Day 3 onwards: the swelling begins to gradually subside. As time goes by, the blisters coalesce to form a blistered rim around the edge of the lesion. From day 6 - 8 onwards, debridement of the necrotic area may be carried out. The subcutaneous tissues under viable skin may be destroyed, forming subcutaneous burrows which may extend quite far from the demarcated area of necrosis. After debridement the resultant ulcer invariably has peripheral undermining (Figs 4 - 6).

Fig. 3. Patient 16 — photograph taken at approximately 72 hours showing early blistering at periphery which is beginning to coalesce, and two small areas of necrosis separated from the main area below the lateral malleolus.

Fig. 4. Patient 16 — at 8 days after the bite, necrotic slough prior to debridement can be seen.

Fig. 2. Patient 17 — photograph taken approximately 14 hours after the bite showing serosanguineous blister just above the bite site, discoloration of the thenar eminence and two islands of discoloration along the distal palmar crease.

Fig. 5. Patient 14 photographed on day 10 during debridement, showing relatively deep soft-tissue necrosis and undermined periphery of the lesion.
Fig. 6. Patient 12 on day 14, 3 days after debridement, showing deep undermining of the lesion.

The necrosis usually involves only the skin and subcutaneous tissues, sparing the muscles and deeper structures. This is presumably because, considering the *M. feaei*’s relatively short fangs, the venom is deposited mainly in the subcutaneous space and spreads through the superficial lymph drainage system.

The other effects of envenomation as described by Warrell et al., viz. failure of clot retraction, prolongation of clot lysis time and haemorrhage, were not looked for in our patients. However, in patient 4 determination of the partial thromboplastin time on day 9 showed elevation (75 seconds; control 39 seconds). The level of fibrin degradation products was increased to > 40 g/ml (normal < 10 g/ml), and the plasma fibrinogen level was 10.1 g/l. At this stage there was advanced necrosis of the dorsum of the foot and lower leg.

Five of the patients vomited, 1 after drinking a home-made potion and 1 after receiving antivenom. Two patients reported diarrhoea. Drowsiness was noted in 4 patients; 3 were children and part of the dorsal surface of the hand. On admission 7 hours after being bitten, patient 8 already had signs of impending necrosis at the bite site. This heraldic patch of about 6 cm² blossomed to involve most of the palm and part of the dorsal surface of the hand. On admission 9 hours after being bitten did not prevent a necrotic ulceration.

Those patients who claimed to have been bitten by a snake in the neurotoxic group, but who had no neurological signs, were admitted for 24 hours’ observation. It was in this group that the unusual nature of the response to the bite by the *M. feaei* was discovered.

Depending on which doctor was on duty when the patient arrived, some were given antivenom, some were admitted for observation, while others were diagnosed as having adder bite and admitted on a conservative regimen. Thus, of the 16 snakebite patients seen at Ngwelezana Hospital during the period of study, only 7 received antivenom. The Mandini patient (case 15) was given antivenom 25 minutes after the bite in anticipation of neurotoxic effects.

When serum was given, it was preceded by 5 - 10 minutes by an intravenous injection of promethazine HCl (Phenergan). The serum was mixed with 200 ml of normal saline and administered intravenously over 15 - 30 minutes. The following dosage regimen was used: weight > 20 kg - 100 ml; weight 10 - 20 kg - 5 ml/kg; weight < 10 kg - 30 ml.

Two of the patients (cases 5 and 16) developed a generalized urticarial rash about 30 minutes after the administration of antivenom; the rash responded to promethazine HCl and hydrocortisone. Patient 11 had an episode of vomiting shortly after the serum was infused. Vomiting was also a feature in 4 other patients and its relationship to the antivenom may therefore be coincidental.

Where necessary, additional supportive measures were used. Patient 14 was intubated with a nasal intratracheal tube because of severe constrictive oedema causing mechanical obstruction to respiration. Patients 1, 5, 14 and 17 required resuscitation with plasma volume expanders. Central venous pressure was monitored via subclavian intravenous lines.

In patients with necrosis, antibiotic cover with soluble penicillin and gentamicin was given. Sloughectomy was performed once the area was demarcated, and when large areas were involved a Eusol drip onto the bandages was employed with good effect. Skin grafts were performed in patients 1, 4, 5, 8 and 9. Patient 14 was sent to a larger centre for plastic surgery. Patients 2, 6, 10, 12 and 13 had lesions small enough to allow spontaneous granulation and closure. Patients 16 and 17, under treatment at the present time, have areas of necrosis which will require skin grafting. Patients 3, 7, 11 and 15 experienced minimal morbidity and will be discussed under ‘Effects of antivenom’.

There were no deaths.

### Effects of SAIMR polyvalent antivenom

Of those patients given antivenom (patients 3, 5, 7, 8, 11, 15, 16 and 17), those who presented ‘late’ (patients 5, 8, 16 and 17) went on to develop necrosis in spite of having received large volumes of polyvalent antivenom. Patient 16 had a faintly discernible area of discoloration of approximately 3 cm² on admission 3½ hours after being bitten and 100 ml of SAIMR polyvalent serum was administered intravenously. Necrosis eventually involved approximately 50 cm² of the side and dorsum of the foot. Patient 17 presented 5½ hours after being bitten, with a serosanguineous blister over the site of the bite. Fifty-five millilitres of polyvalent serum was administered intravenously on admission, yet necrosis eventually involved most of the palm and part of the dorsal surface of the hand. On admission 7 hours after being bitten, patient 8 already had signs of impending necrosis at the bite site. This heraldic patch of about 6 cm² blossomed to involve most of the calf, an area of ± 200 cm². A similar occurrence was seen in patient 5, who was bitten twice on the foot. The intravenous administration of 100 ml of serum on admission 9 hours after being bitten did not prevent a necrotic

### Treatment of bites

In a guide to the treatment of snakebites seen in our outpatient department, I advocated a conservative approach to bites showing signs of cytotoxic envenomation. This entails clear intravenous fluids, analgesics, anti-tetanus toxoid, elevation of the affected limb and observation of blood pressure and pulse rate at regular intervals. When necessary, plasma volume expanders (fresh-dried plasma, blood, Plasmalyte B), are given to counteract hypovolaemia. Antibiotic cover is provided if the patient has self-inflicted cuts or if necrosis is present.

SAIMR polyvalent anti-snakebite serum is to be given only to patients showing signs of neurotoxic envenomation, i.e. ptosis, hypersalivation, slurred speech, drowsiness or depression of respiration.

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area of about 120 cm² from developing. In these cases, whether the antivenom mopped up free toxins and prevented even further spread of necrosis is debatable.

With regard to the other 4 patients given antivenom, patient 3 presented 3 hours after a bite on the lower thigh with mild swelling and warmth at the bite site. The swelling did not progress any further. Seven hours after the administration of antivenom the patient stated that the pain was subjectively much less. She was discharged 2 days after admission, with mild swelling. She was readmitted 7 days later for drainage of a marble-sized abscess under the site of the bite. Patient 7 presented 1 ½ hours after being bitten over the distal interphalangeal joint of the middle finger. Mild swelling was noted on admission and 100 ml of polyvalent serum was given intravenously. On the 4th day after admission a small area of fluctuation was noted under the site of the bite. On day 5 the terminal phalanx was noted to be hypermobile and radiography showed the joint space to be obliterated. On day 6 the epidermal layer over the site of the bite peeled off to reveal normal skin. She was discharged. Patient 11 presented 2 hours after being bitten on the foot and 100 ml of polyvalent serum was given intravenously. Apart from mild swelling and warmth, nothing further developed and he was discharged on day 6. Patient 15, an amateur snake collector from Mandini, sought help within 25 minutes of being bitten on the index finger. He was given 80 ml of polyvalent serum intravenously. Apart from local pain, nothing further developed.

Discussion

In all patients with M'fesí bites seen at Ngwelezana Hospital, the only neurological sign perhaps directly related to the venom has been drowsiness. This was very marked in patient 16, 3 hours after the bite. Four other patients who became drowsy (cases 1, 5, 14 and 17) were all children. Patient 14, a 9-month-old baby bitten on the face 6 hours earlier, was drowsy on admission. When drowsy, patients 1 and 5 were both hypovolaemic. Russell noted drowsiness as the only systemic sign in 2 patients bitten by N. nigricollis.

The dominant feature of the M'fesí bites seen has been a characteristic cytotoxic lesion, the venom having a cellular toxicity far more destructive than that experienced in puff adder (Bitis arietans) bites. In fact, it is suspected that a lot of M'fesí bites have been wrongly attributed to the puff adder in the past, merely on the assumption that the puff adder caused tissue necrosis and the M'fesí neurotoxic effects — a sentiment similarly voiced by Warrell et al. in respect of N. nigricollis and the puff adder.

In a series of 1 067 bites treated at King Edward VIII Hospital, Durban, between 1957 and 1963, it was assumed that the most serious local tissue damage was caused by the puff adder if the offending snake was unidentified. Two hundred and ten cases were thus attributed to the puff adder, and not one to the M'fesí.

In the light of earlier experience with puff adder bites, any associated necrosis usually involves a relatively small local area — usually occurring in bites on or near the extremities. Necrosis may follow prolonged ischaemia due to the 'compartment syndrome', resulting in either gangrene of an affected limb or a Volkmann's contracture, both of which are rare occurrences. Warrell et al. described the local necrosis in cases of spitting cobra bite as being detectable between 36 hours and 10 days (mean 5 days) after being bitten. In our experience, the necrosis may be heralded by a dark spot within 3 hours, and by 9 hours a port-wine-type discolouration may be detected, enlarging to its maximum extent between 36 and 48 hours.

The exact mechanism whereby necrosis is caused is uncertain. Snake venoms contain a number of toxic protein fractions which have enzyme action. Among them have been identified various proteases which cause cellular damage, and hyaluronidase which encourages the spread of the toxins through the tissues. Birdsey, Lindorder and Gewurz showed that complement activation by an alternative pathway occurred in vivo with 7 different elapid venoms, including N. nigricollis. Warrell et al. found this to be the case in vivo. The activated complement enzymes cause cell damage, while C3 and C5 are thought to give rise to the formation of anaphylatoxins which cause release of histamine from mast cells and increase vascular permeability. Other complement reaction products induce polymorphonuclear infiltration. The release of lysozymes from dead polymorphs also causes vascular damage and tissue injury.

N. nigricollis venom has been shown to contain a curariform neurotoxin which is probably responsible for the rapid death by respiratory paralysis in its prey. It is suggested that a similar toxin in the M'fesí venom exerts a local effect in humans — as evidenced in our patient who, after being in the eyes, subsequently developed a unilateral temporary paresis of the pupil constrictors. Russell noted some local neurological signs in 2 patients bitten by N. nigricollis, including depressed deep reflexes and muscle weakness in the affected limbs.

Since 1971 the venoms of all the major South African elapids, including the M'fesí, have been used in the manufacture of SAIMR polyvalent anti-snakebite serum. Christensen claims satisfactory neutralizing ability of the serum against N. nigricollis venom (then encompassing M'fesí studies). Our in vivo experience would appear to confirm this claim.

Warrell et al., however, were unimpressed by Christensen's claim. Two of their patients received 40 ml of SAIMR polyvalent serum 10 and 6 hours post-bite, respectively, and both developed fairly extensive subcutaneous necrosis. One of their patients was given 40 ml of FitzSimons polyvalent serum (Bitis, Hemachatus, Naja) 24 hours after the bite and still developed necrosis. A further patient was given 40 ml of FitzSimons polyvalent serum 18 hours after the bite and yet developed no necrosis. The former 3 bites were associated with moderate-to-severe swelling. One patient received no serum and did not develop necrosis, although swelling was present.

In 2 cases of envenomation from N. nigricollis in snake-handlers in America, administration of antivenom (Institut Pasteur; Anti Bitis-Echis-Naja) within 1 hour prevented necrosis from developing.

Because of the various factors affecting the amount of venom injected as well as variable inherent resistance to toxins in individuals, it is difficult to predict the final extent of necrosis in a particular bite if it remains untreated. When and if necrosis occurs, it may extend deeply over a wide area or be a small superficial skin lesion. Because of this variability, the effect in vivo of the SAIMR polyvalent serum has been difficult to assess. The severity of the clinical symptoms will obviously be related to the amount of venom injected. It may be argued that the last 40 f FitzSimons polyvalent serum (Bitis, Hemachatus, Naja) 24 hours after the bite and still developed necrosis. A further patient was given 40 ml of FitzSimons polyvalent serum 18 hours after the bite and yet developed no necrosis. The former 3 bites were associated with moderate-to-severe swelling. One patient received no serum and did not develop necrosis, although swelling was present.

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Of bites occurring inside homes during the same period, the M'fesí accounted for 16, the side-stabbing snake (Atractaspis bibrioni) for 9, the black mamba (Dendroaspis polyphyletis) for 1, the puff adder (Bitis arietans) for 4 and unidentified snakes for 7, including 2 bites that strongly suggested that the M'fesí was the
culprit. All of the 'legitimate' bites from the *M'fezi* occurred inside the homes of the victims, as did the case of ophthalmia due to snake venom. In the series of Warrell et al.¹ of the 6 victims bitten while awake, 3 were bitten on the hand and 3 on the foot. This suggests a tendency for these snakes to strike at the nearest part of a supposed attacker, which usually happens to be a foot or a hand. This would tend to contradict the widely held view that the larger elapids, noted for the habit of rearing the anterior part of their bodies when threatened, would usually strike at sites above the foot.

**Conclusion**

It has been the custom to treat snakebite victims presenting with swelling and pain conservatively, as is correct with adder bites. However, because of the morbiditity that may follow the untreated bite of the *M'fezi*, it is urged that more time be spent in trying to establish the identity of the offending snake. Of several occasions the patient's correct identification of the snake was dismissed because it was thought that the symptoms were wrong. These patients could possibly have been saved long periods in hospital, skin grafts and disfigurement. If the bites occur inside the home, there is a good chance that the snake is an *M'fezi*; in all doubtful cases it may be wise to give antivenom.

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Particular attention must be given to the prevention and early detection of hypovolaemic shock, which is especially liable to occur in children within 24 hours of being bitten.

In my experience in Ovamboland, SWA/Namibia, bites from the barred spitting cobra, *N. nigricollis nigracinca*, although not documented, also show a severe cytotoxic effect. Perhaps further studies will show this snake to have a similar venom to that of *N. mossambica* and *N. nigricollis*. I have not encountered or heard of victims of a bite from *N. nigricollis woodii*.

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**REFERENCES**