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27. THE CLINICO-PATHOLOGY AND TREATMENT OF SNAKEBITE IN SOUTHERN AND CENTRAL AFRICA

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Introduction

If there are no fairies at the bottom of one's garden, in South Africa there may be a snake or two. Africa shares with other world areas a high attainment of its herpetologists and a paucity of detailed medical observations. Indeed, there is seldom opportunity to study the early and often urgent symptoms of snake bite as those exposed are mainly primitives and rural dwellers.

A local difficulty is apparent. Elsewhere, a certain family or even species is outstanding as the cause. In Southern Africa, by the many varieties available, these with a wide variation in colour and markings within the species (e.g. the cobras), by the frequent mimicking of venomous by harmless snakes, and by the fact that the snake usually flits after an attack, it is not surprising that snakes are rarely identified.

In our hospital experience also, it has not been possible to differentiate the elapine snakes on clinical grounds but from the scanty evidence previously available, the very rapidity and severity of a venom action has tended to point to a particular species, the black mamba. However, the puff adder has often been identified by its sluggish grossness and we have assumed that our most serious cases of local tissue death were caused by it when the snake was not identified. Similarly the night adder has been identified often enough to apportion blame. Elapine bite victims have tended to report early enough (Fig. 1) for the clinician to give a fair description of the clinical features but where adder bite victims have reported late (and only because of persistence of swelling or because of gangrene), there have been few competent early observations.

Though it is often claimed that amateurs can easily recognise our cobras and adders, most victims do not qualify, are bitten in their ignorance, often in the dark, and are often children to whom all snakes are large.

The common venomous snakes

Southern and Central Africa have the dubious distinction of harbouring some 137 venomous snakes comprised in three families, COLUBRIDAE, ELAPIDAE and VIPERIDAE but not more than 25 species however, are capable of causing death (Table 1). Of all areas Natal, my home province, has the greatest variety of snakes and highest incidence of bite.

TABLE I — COMMON POISONOUS SNAKES OF SOUTHERN AFRICA

COLUBRIDAE	ELAPIDAE	VIPERIDAE
Boomslang Bird snake Skaapstekers	True Cobras (4) Rinkals "Cobra" Mambas (3)	Puff Adder Night Adder Gaboon Viper Berg Adder * Mole Viper ** Carpet Viper *
Haemotoxic	Neurotoxic	Local cytotoxic

* and ** Also has special elapine effects.
* Has dominant colubrid effects.

The three COLUBRIDAE shown in Table 1 are all peculiar to Southern Africa. They are back-biters with a limited capacity for a successful strike but the boomslang and the bird snake have at least extremely potent venoms which have a marked effect on blood coagulation. Our peculiar experience is fortunately tempered by the docility of these snakes.

The ELAPIDAE have small permanently erect fangs and are all dangerously venomous with a serious neuro-toxic effect. Many are small inoffensive burrowers but included are 4 mambas and 4 true cobras. The Rinkals "cobra" and the black-necked cobra also spit accurately to cause conjunctivitis; but this can be adequately dealt with by simple lavage.

The vipers show the highest development of fangs which are mobile, large and hollow. Undoubtedly the Puff Adder is rightly feared for ubiquity, the potency of its cytotoxic venom and is productive of the majority of our serious bites. The Gaboon viper is our largest viper and very venomous. A fearsome creature but reputedly good natured, it is reluctant to strike except when trodden on. Found in rain forest areas, it is fortunately rare. There are two species of night adder which frequently cause the less serious bites. The berg adder has also a special elapine effect. Mole vipers abound and cause many of our lesser bites. The carpet viper, found in the more Northern areas, appears to have a special colubrid effect.

The highest incidence of snakes is in the more populous areas where land has been cleared for cultivation and where habitation has invited rodents.

Epidemiology

I present here evidence of a 7-year study of over 1,000 cases of snake bite, 892 of which were admitted to our hospital. Table II presents the cases according to each family, together with an analysis of those with serious effects, and the fatal cases. It is seen that overall there was a high incidence of morbidity and mortality though relative to the size of the population at risk the incidence of bites was low. Noted too is that in 484 instances the snake was not identified.

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TABLE II — SNAKE BITE — 7 years 1957-1963 — Natal

Species		Died	Severe effects
Total Cases	1068	21	80
COLUBRIDAE	5	0	0
Skaapsteker	3	0	0
Natal Black Snake	1	0	0
Herald Snake	1	0	0
ELAPIDAE	41	10	16
True cobra	18	3	9
Rinkals cobra	14	0	5 (4)
Black Mamba	7	7	—
Green Mamba	2	0	2
VIPERIDAE	538	11	64
Berg Adder	1	0	0
Puff Adder	210	11	56
Night Adder	8	0	1
Puff or Night Adder	319	0	7
Unknown			
venomous	314	0	0
harmless	170	0	0

Some human attitudes to snakes

Let us consider some important human attitudes which influence the incidence and severity of snake bite.

Whether as some would claim we are not born to fear snakes but are conditioned to it, fear is there and fascination too. Both no doubt lead to trouble. In our first aid we advise calm and immobilisation, yet the emotional distress is real and if only to get to a snake-bite outfit, the impulse is to run. In my series, some adults and many children were frightened long hours after the attack.

Only a few of us surely think snakes are beautiful but many are attracted and mainly perhaps by the possible danger.

So boys become amateur herpetologists and as they grow they become indifferent to the possible dangers. Ignorance reaches its zenith in the case of snake-handlers even to their ignoring bites when working in snake parks because they think they "know" their snakes and falsely believe in immunity from previous bites. Nor can snakes be tamed and an usually docile one can take offense at any time. Thus has one experienced herpetologist and at least 4 snake catchers lost their lives in Southern Africa. Snake catching and handling are obviously hazardous and it does not seem that enough care is taken.

Except when dangerously close, identification appears quite difficult. Our local snake park received its prize black mamba, sent in error as a rather handsome specimen of a mole snake.

The white population is snake conscious at least with regard to the facilities of protection and those such as nature lovers and fishermen carry snake-bite outfits, no doubt though, many with out-of-date antivenoms and dirty instruments. Unfortunately the main population at risk are the rural Bantu and Asiatics who in fact, do seem ready enough to seek help from mission hospitals, agricultural hospitals and farms, all of which are equipped.

Age incidence of bite

Children run the greatest risk in their play (usually barefooted) and through their curiosity of moving things (Fig. 2). Though the smaller children have no fear, they are bitten in large numbers. The older children also molest snakes. One, a Bantu herd-boy tried to strike a more agile mamba with his hand and died by his audacity.

Some snakes such as the rinkals "cobra" can sham death and some bite when they are dead. A Bantu male adult claimed the severed head of a black mamba which had just been shot for the purpose of fortifying a native remedy. He placed it in his pocket and taking it out a few minutes later, he was promptly bitten and was immediately very ill with neurotoxic symptoms. He survived.

A snake census is meritous but not entirely satisfactory. Numbers can be readily increased by the reward — conscious deliberately rearing less harmful snakes — even to enterprising schoolboys selling back to our snake park, snakes stolen from it the day before.

No doubt to allay our fears, herpetologists stress that only some 300 of the world's 3,000 species are dangerous to man and few will attack unless molested. This is cold comfort to the 1,000 victims of this series.

Seasonal incidence

Snakes may be more shy than those they bite but there is ample evidence of aggression in the breeding season when it is warmer and more humid (Fig. 3) and when they are cut off from their lairs. Whatever the basis for attack, snake bites do occur in large numbers wherever their paths cross with other animals. Yet the overall risk is small (Table III). In South Africa one has nine times the chance of injury travelling by car to a picnic than from the snakes there; and 40 times the chance of dying.

TABLE III

	Injury	Death
Snake bite	24	0.35
Road accidents	211 . 3	14 . 4
	×9	×40

Situation of the bite

The very great majority of bites (Fig. 4) occur on the feet and lower legs, 84%. The principal remainder are on the hand (7.7%). The risk is with terrestial snakes, often through treading on them, well camouflaged as they are, and most commonly with the more lazy adders which strike lower down than do the cobras (and especially the mambas) which rear to strike. We share with other areas, alarm over head and trunk bites and even those of the upper limb, situations where a tourniquet cannot be applied, and where better absorption is likely by more abundant vascular connections.

The moral is plain — when in the country to wear boots or at least shoes and never open sandals; also thick long trousers wherever the vegetation is abundant. To tread warily is not enough. Such precautions would reduce the risk by at least one half. One wonders whether snake catchers and herpetologists in their field work so protect themselves, and do they always wear gauntlets?

The clinical effects of bite

Table IV considers the general highlights of clinical action for both elapine and viperine snakes in our experience, and using the evidence of the fatal cases.

TABLE IV — SNAKE BITE DEATHS

1 — Long delay in treatment 2 — Profound effect in children
3 — Antivenom — too little, too late, unsuitable site, wrong type
4 — Negative *distant* necropsy findings

	ELAPINE (8)	VIPERINE (9)
Hospital admission	quite early	late
Clinical, onset	rapid	late
march	rapid	slow
Dramatic signs	consistent	lacking
Local, pain	little	often severe
swelling	little	rapid, massive extravasation

A. *Bites by Colubrid snakes*

Attack by these snakes is rare in any Southern African experience. All 5 cases in my series of such bite were almost symptom free and did not show the special haemotoxic effect.

B. *Bites by Elapine snakes* (Table V)

The essential effect is distant from the bite, neurotoxic and rapidly produced by venous spread. We relate our first aid to this — the tourniquet (the vital first step) and the antivenom given intravenously.

TABLE V — THE CLINICAL EFFECTS OF SOUTHERN AFRICAN ELAPINE SNAKES *

Clinical	No.	Fatal	Clinical	No.	Fatal
Severe pain	2	—	Respiratory distress	15	8
Swelling, none	14	4	Breathless	7	3
little	18	4	Shallow respiration	8	5
moderate	1	—	Low blood pressure	16	7
severe	2**	—	Cardiac arrest	1	1
Vertigo	7	—	Increased sweating	12	
Drowsy	1	—	Hyperpyrexia	3	
Restless	16	3	Nausea	1	
Unconscious	12	5	Severe vomiting	4	1
Convulsions	2	1	Pallor	3	1
Headache	1	—	Nose bleed	1	
Hallucinations	1	—			
Difficult swallowing ***	11	2			
Difficult speech	7	1			
Throat pain	2	—			
Increased salivation	14	8			
Ptosis	8	4			

* Hospital group of 35, of which 25 identified and 10 likely.

** Both associated with long applied tourniquet.

*** Excludes those unconscious.

Experimentally all South African cobras have more potent venoms than have the mambas but the mamba gains its rightful dreaded reputation on account of the greater volume of venom it can more rapidly inject.

Death from a cobra or a mamba bite is in anything from minutes to an average of 8 hours and the majority of those alive at 24 hours will survive and without ill effects.

There is a paralysis of motor action by a curare-like effect peripherally on motor nerve-endings or by a central brain-stem action, probably both. And there is plenty of evidence of widespread effects on the cerebrum — vertigo, convulsions and unconsciousness often occurring before respiratory failure is developed well enough to give cerebral anoxia. The noticeable eyelid ptosis, strabismus and speech inco-ordination are useful diagnostic features but not harmful.

The outstanding effect is a paralysis of respiration and of deglutition so that a victim embarrassed by the former can drown in his own saliva. Salivation is not increased but only seems to be; just as in other instances of interference of deglutition (e.g. oesophageal cancer, head injuries).

Once commenced, respiratory palsy is rapid in progress but there is probably no march of palsy from one muscle group to another. That diaphragmatic action appears to last the longest is probably because it is seen best and especially when

it is aided by vigorous struggles of abdominal muscles. In our cases it was not easy to test the tone of limb muscles but it was seen that these also share in the increasing flaccid palsy from lassitude to inertia. Muscles which need to overcome the effect of gravity are most easily seen to fail — in many cases the neck muscles tire and the head falls. Ptosis of the eyelids may well represent merely such a failure. Finally the basic sphincters fail, to give incontinence of urine and faeces.

Respiratory failure seems to take different forms depending on the venom dose. Most usually, the failure is quiet, breathing becomes increasingly shallow, the subject also lying quietly unconscious, as seen in many of our cases. But there can be more of a fight and an increased labour of respiration.

It has often been reported that for the less rapidly effective cobra venoms, the heart can continue to beat strongly long after respiration has ceased, which of course, is common to other forms of respiratory failure. Such, at least would mean that if respiration could be maintained artificially, the outlook need not be so gloomy especially if the venom could be neutralised, which for cobras we have proof that it can. We had one such case who in fact "died" 4 hours after a mamba bite (one hour after admission), was revived by exposed cardiac massage to what we thought was normal heart action and was maintained for two days on a respirator before he succumbed. Two others survived after 5 days of respiratory support and I can now report three other successes from the past two years.

More than half our cases had some disturbance of deglutition or speech and nearly all of these had profuse salivation. General cerebral effects were also common. One-third were unconscious on or after admission, 4 for very long periods and yet survived.

Restlessness was seen most often with conscious subjects and the usual demeanour to death was of quiet unconsciousness and shallow respiration.

Many had profuse sweating which, with a low blood pressure, constituted for some observers the picture of "shock", but most of these cases also had fever.

Eyelid ptosis was seen only in 8 cases. Pupillary changes were very variable or absent and were not considered of diagnostic importance.

Half the cases had respiratory distress, all severe, and half of these survived.

In this elapine series, the local effects of bite were minimal or not existent. Slight swelling was occasional and pain was rare.

Though all South African venoms are essentially anticoagulant *in vitro*, except in the case of the COLUBRIDAE, the haemorrhagic action is not important. No elapine cases ever had haemorrhages nor jaundice and in fact the haemorrhagic action in viperine bites was always local and not, I think, related to a coagulation defect.

The value of antivenom

I consider from my series that we now have abundant proof of the great value of the antivenom available (Table VI). Fourteen of 25 serious cases of elapid bite responded to antivenom, some very dramatically. One noted too the pleasant freedom from sensitivity reactions (1%) as contrasted with figures as high as 30% in some world areas.

TABLE VI — DETAILS OF ANTIVENOM THERAPY IN 892 CASES (Polyvalent *B. lachesis* — *H. haemachatus* — *N. nivea* antivenom. S.A.I.M.R.)

A.	Given	Not given *	No record
Antivenom	712	142	38
Not required **	186		
Required		6	
> 6 hours after bite	45		
30 mls or more	29		
Intravenous	14		

* Includes 26 reporting 24+ hours after bite and 1 Rinkal Cobra spitting.

** These cases had few or no signs. No observation period.

	Early	Late	Fatal
Sensitivity 7	5	2	0

B. ELAPINE SERIES 35

Severe 25

Died 9

Good response 14

Also other treatment 3

It is also seen that the doctors of Southern Africa seem shy of giving antivenoms intravenously, yet it is by this route that success will come in elapine bite cases. They also tend to prescribe doses which are low. We can see in Table VI how few were given 30 mls of the antivenom or more, yet this is the advised minimal dose for serious cases. Children also must be given at least an adult dose, a dose dependent on their size relative to the size of the snake.

I would never withhold this polyvalent serum from cases of puff adder bite but it is difficult to evaluate the benefit. It was problematic whether it was ever of any help in this series. Antivenom never appeared to resolve or indeed limit any moderate sized or severe swellings. Many injections were obviously of course, given too late.

In Southern Africa we now have a polyvalent anti-mamba serum, but it will be harder still to prove its value in the face of the very rapid fatal action of the mamba venoms. It was only used in one case in this series and did not alter the march of events.

Fig. 5, records in pictorial form, the march to death or recovery in severe cases in both the elapine and viperine series. Of 25 identified elapine bites, 8 died rapidly while 14 were resolved dramatically by the use of antivenom, 2 more succeeding by the aid of assisted respiration; and one, it was thought, by the exhibition of steroids.

C. *Bites by Viperine Snakes* (Table VII)

The dominant clinical effect of a viper venom is local, cytotoxic and self-limiting. There is no general bleeding diasthesis. General effects are uncommon, usually late and not neurotoxic (except in the case of 2 species). Spread of toxin is by lymphatics, which was shown well in 43 cases where lymphangitis was the main effect. This is a very important fact in management and immobilisation must be encouraged both of the part bitten and of the subject as a whole.

The effect is a destruction of all tissues especially blood vessels and their contents but the cytolysis with its tissue necrosis, the coagulation and thrombosis of the blood produces its own barrier to spread. Bleeding adds to the internal pressure to increase the ischaemia. So produced are the swelling, induration, haemorrhages as ecchymoses and blisters, and necrosis.

TABLE VII — LOCAL EFFECTS OF SNAKE BITE
(7 years: 1957-1963 — Natal)

Cases	892
Swelling	726 (slight, 290; moderate, 300; severe, 136)
Extravasation	67
Abcess	20 (initial, 12; later, 8)
Necrosis	23 (local, 11; extensive, 12)
Oligaemic shock	25 (died, 9; dramatic response, 9)
Lymphangitis	43
"Cellulitis"	104 (by snake, 100; by bacteria, 4)
Thrombophlebitis	4 (superficial, 1; deep, 3)

Tissue necrosis and oligaemic shock

Though an overwhelming envenomation is possible, for instance by an intravenous injection, with consequent convulsions and unconsciousness, each death in this series had a rapidly expanding extravasation of a limb. None had frank gangrene but one had marked ischaemia distal to the main blood collection.

Nearly all took some time to die as contrasted with the elapine series and in my opinion death was due to *decompensated oligaemic shock* caused by the rapid massive blood loss, either not treated at all or in those who came too late for help.

There is a strong inclination however, in cases who have apparently been doing well and who many hours after a bite have a sudden and fatal collapse,

for clinicians to invoke a new special factor of venom action now with a distant as against a previous local effect. This is unlikely and no evidence is in fact available. Three of the 9 fatal cases collapsed in hospital and others were rushed in when it had occurred at home.

If enough cases could be shown to develop collapse following removal of a long applied tourniquet, we might imply a release of breakdown products of muscle metabolism but in the many instances we released tourniquets, collapse did not follow.

It has seemed that the role of such breakdown products is no greater in the production of general effects in viperine bite than with other examples of infarction when, during the body's struggle to reject particularly frank gangrene, the patient can become very ill — but he does so gradually and does not die rapidly.

As I have indicated, if it occurs, any coagulation defect must be local, perhaps localised by the barriers of necrosis etc. and no cases had jaundice. At necropsy no distant effects were found and there was never evidence of pyogenic invasion. Yet many had severe anaemia when first reporting.

Consider a limb rapidly filling with blood from the general circulation while its own contents are immobilised by what amounts to an enclosed catastrophe of tissue death. If a surgeon acquainted with crush injuries was to witness this, he would not hesitate to replace the blood lost and he would do it quickly. It does not surprise that the volume increase of a part can represent fully half the original blood volume of the patient. In my series some extravasations reached well into the trunk after involving a whole limb.

The majority of victims are fortunately young adults who can adequately compensate for the sudden oligaemia and at first, will appear quite normal. But compensation cannot be kept up indefinitely and when it breaks, "shock" is then often irreversible. The picture of collapse in viperine bites is identical with such decompensation after crush injuries and extensive burns. The clinician should be aware that the *signs* usually described for oligaemic shock are either those of intense compensation or of decompensation — but shock is actually present from the onset of swelling, building as it goes. We should treat the situation and not wait for the signs. Those who survive on their own (many must hover on the brink), when they are admitted late, often appear toxic and their condition is not unlike the "illness of trauma" following crush injuries.

Except where the swelling is due to the inflammatory oedema consequent on the tissue insult by venom, when improvement will come from infusions of other fluids such as plasma, only blood will suffice. I have indicated that the infusions may have to be very large. Care of course is required with late arrivals — for these, packed cells should be used.

Vasopressor substances or steroids cannot restore blood volume depleted of blood and in fact the former are dangerous. They do not increase cardiac output but do increase the work of the heart and most important, produce profound renal ischaemia. Antivenom neither, can replace lost blood.

The swelling

This is two main types:

(1) Little or no surface extravasation with the limb soft or hard.

(2) Haemorrhages evident as ecchymoses when the swelling is usually more massive and solid.

Necrosis is induced in either type by the very concentration of venom but found more often in the second type. It is then often extensive, superficial or deep.

The lower limb has often been seen markedly flexed at the knee and by the hardness of the swelling, we feared serious muscle involvement by haemorrhage or coagulation but in most of these cases, residual induration was rare.

The resolution of swelling

Many of those admitted with severe and extensive swellings had persistence of these for over 10 days. The possible importance of venous occlusion as the basis of slow resolution is suggested by the outcome in 4 illustrative cases. One child had a thrombosis of the long saphenous vein of a lower limb as the solitary clinical feature. Three women who were bitten on the feet had swelling of the limbs which had not been considered severe at the onset, still present at the end of 3 months (Table VII). Solid swelling involved the greater part of a lower limb with a tendency to subside after rest and elevation. In all 3, venography showed irregularity of and evidence of recanalisation in the deep venous system (as though after thrombosis). Two of these women in the earlier phase of their venous oedema had severe pain and marked tenderness on palpation of the popliteal vein behind the knee. All 3 adults were back to normal after a further 4 months.

Fourteen cases had an extremely rapidly developed soft swelling of much of a limb, with almost as rapid a subsidence over some 12 hours. There were judged to have had lymphatic oedema caused by bites of the lesser adders.

Necrosis

Occlusion of a main vessel was only rarely the cause of the infarction seen, which even when large, tended to be patchy. The action is mainly on smaller vessels, sparing at least some tissue. Some of the deleterious effect is perhaps due to the explosive effects of disruption. Twelve cases of necrosis were extensive, 4 involving the whole lower limb below the knee.

The role of bacteria

Bacteria come no doubt on snake fangs and by dirty incisions but are they important? Though abscesses were found in 20 cases, only 8 had had an incision and some of the others almost certainly represented lysis of necrotic tissue.

Local temperature rise and redness were seen frequently. Diagnosed by some observers as "cellulitis" they occurred far too early after the snake attack to incriminate bacteria. Why not an inflammatory response to snake toxin?

Including those cases coming to necropsy, septicaemia or pyaemia were never encountered. Gas gangrene and tetanus never occurred either.

Only 9 cases of viperine snake bite had general effects separate from any distant influence of the local lesion. These were considered sensitivity reactions to the venom before any antivenom had been given. Six had vomiting, 2 had abdominal colic and there was one instance each of dizziness, headache, sweating, urticaria, hyperpyrexia, drowsiness.

Fig. 5 shows graphically the outcome of 18 severe viperine bites when the snake was identified. Though 9 died, some were admitted only after 24 hours when they had collapsed at home, presumably from the decompensation of blood loss. Nine others responded well to blood infusion.

Treatment

A summary of "A System of Management of Snake Bite in Southern and Central Africa", which includes a "Simple Regime of Early Treatment", is appended.

My criticisms of past and present modes of treatment is given in the light of my experience of snake habits, the probable routes of snake venom action, and the facilities available in our areas. From all the work, the simple regime was devised.

Discussion

A. Barrio: "In envenomation by ELAPIDAE neostigmine is useful, as demonstrated in neuro-muscular preparations, in 1949, in collaboration with O. Vital Brazil?"

D. Chapman: "I have no experience with *Micrurus*-envenomation. Prostigmine has been used in some of our cases of elapid snakes, but not with any measurable effect."

F. Kornalik: "Corroborate your opinion about the cause of death in viper-bites being oligaemic shock. We had the same results in experimental animals in which, even when a sublethal dose of venoms has been applied, anemia occurs with a rise of the haematocrit from 45% to even 80%."

D. Chapman: "The oligaemic shock is of great importance and is the primary cause of the illness and death which follows our African viper bites."

P. J. Deoras: "Have you any data about the areas the tourniquet has been applied by various workers?"

P. J. Deoras: "Have you any data about the areas the tourniquet has been aping our African viper bites."

Table legends

Table I — The common poisonous snakes of Southern and Central Africa.

Table II — Snake bite in Natal in a 7-year period showing the morbidity and mortality incidence, relative to each snake species.

Table III — Contrast the risk. A contrast of the chance of snake bite and its mortality with road accidents in South Africa.

Table IV — The outstanding events seen in those dying from elapine and viperine snake bites.

Table V — The clinical effects of Southern African elapine snake bite.

Table VI — How the available antivenom was used or abused. Its success in identified severe elapine bites is given. Note the low sensitivity incidence.

Table VII — The local effects of snake bite. Though this analysis includes all cases of bite, swelling was not a feature of elapid bite and the figures given here virtually represent the viper bite experience.

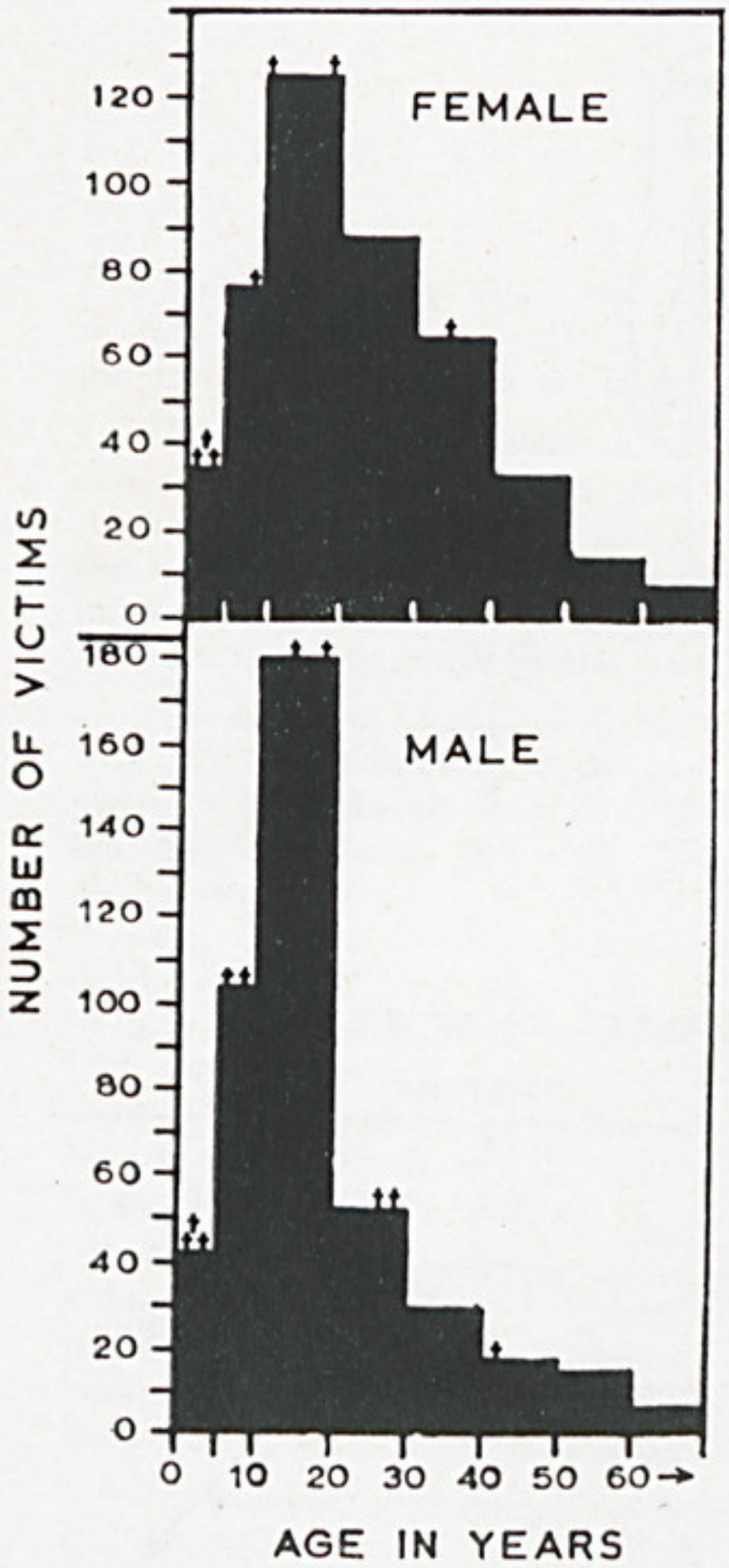

Fig. 2 — Age and Sex Incidence. Note that children of both sexes were the commonest victims and that most deaths occurred in young subjects.

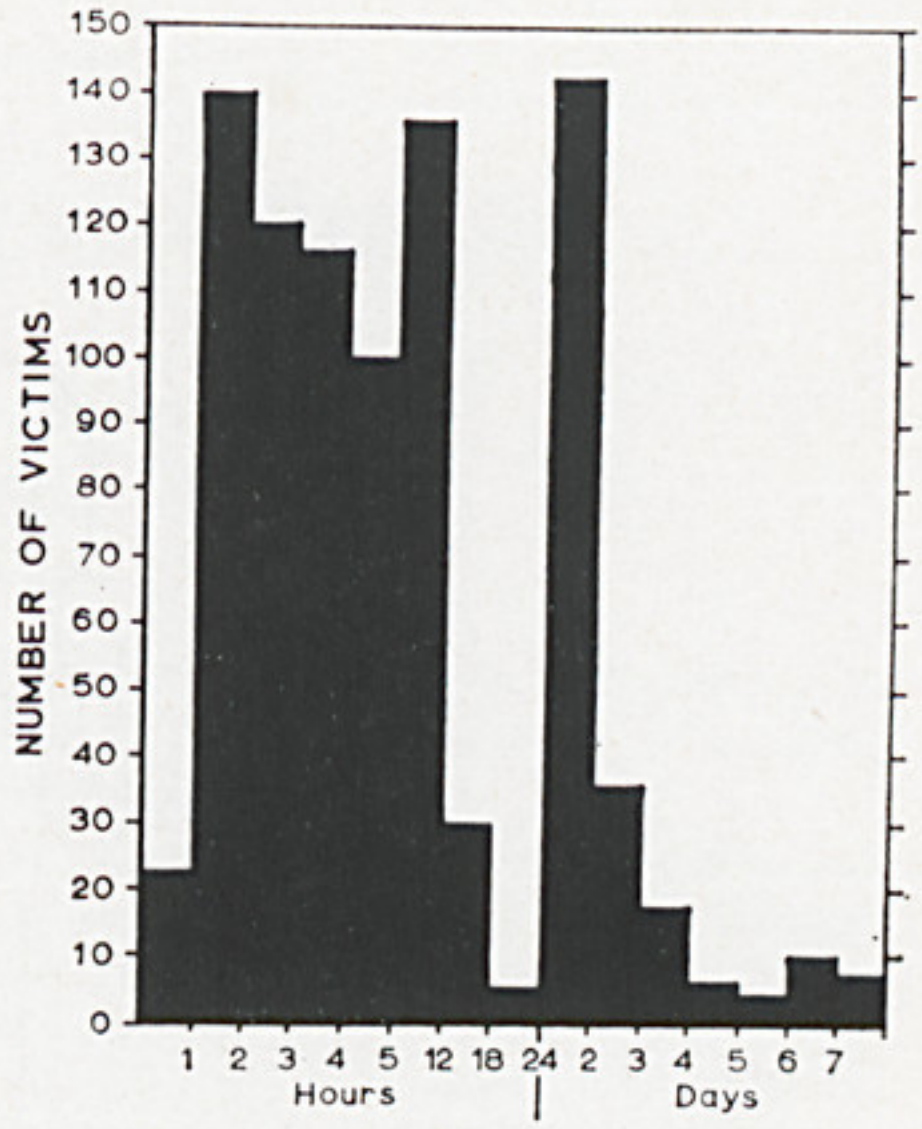

Fig. 1 — Bite: Admission Time. Elapine snakebite victims tended to report in the early hours whereas the very late admissions comprise mainly serious viper bites.

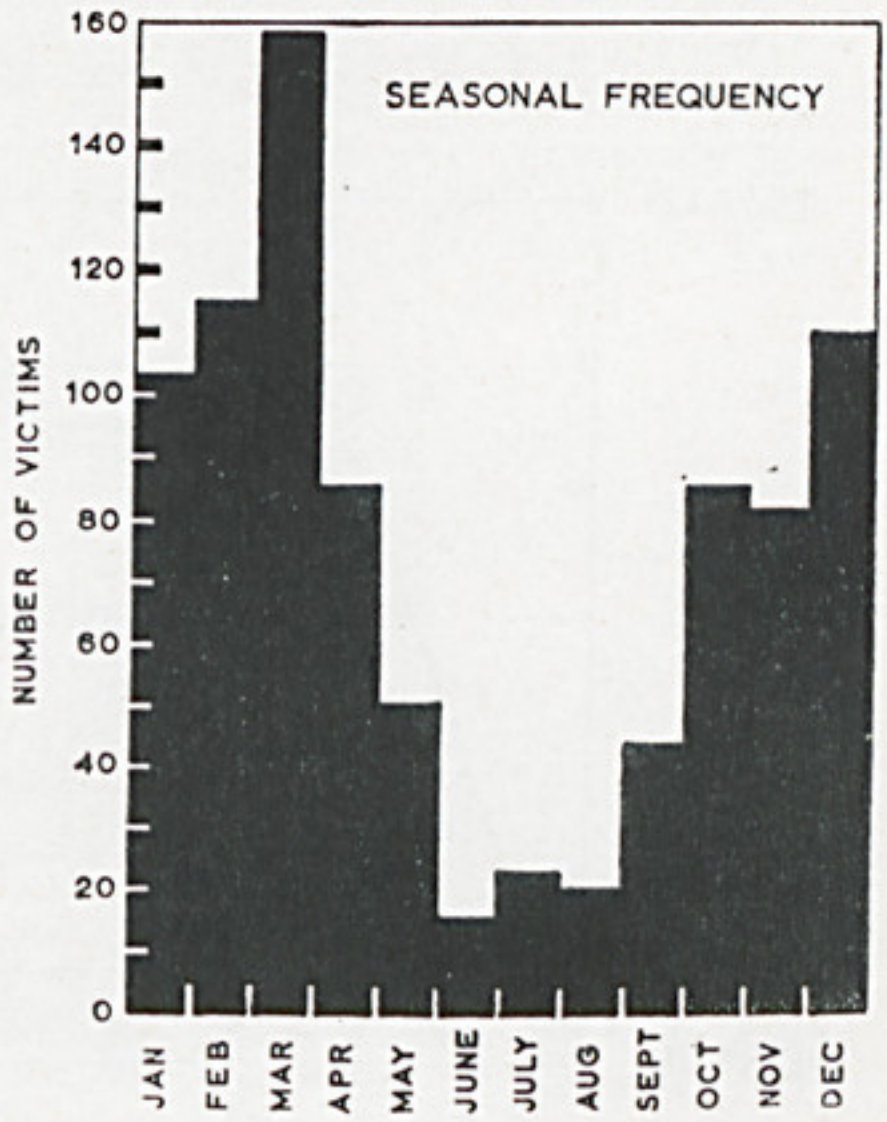

Fig. 3 — Seasonal Incidence of Snakebite. The highest incidence is in the hot humid months when snakes are breeding.

SITE OF SNAKE BITE

7 years 1957-63 Natal

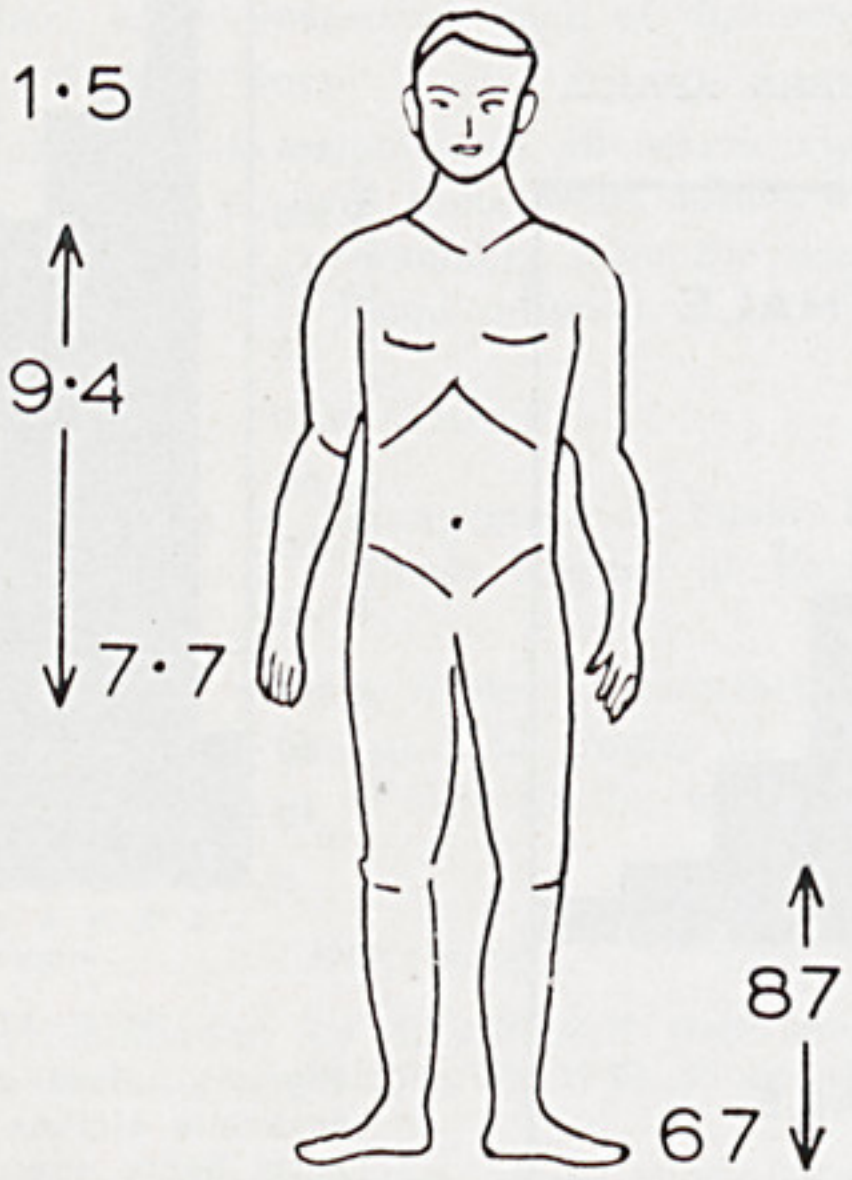

Fig. 4 — Situation of the Bite. The figures represent percentages.

SNAKE BITE — THE MARCH & RECOVERY OF SEVERE CASES

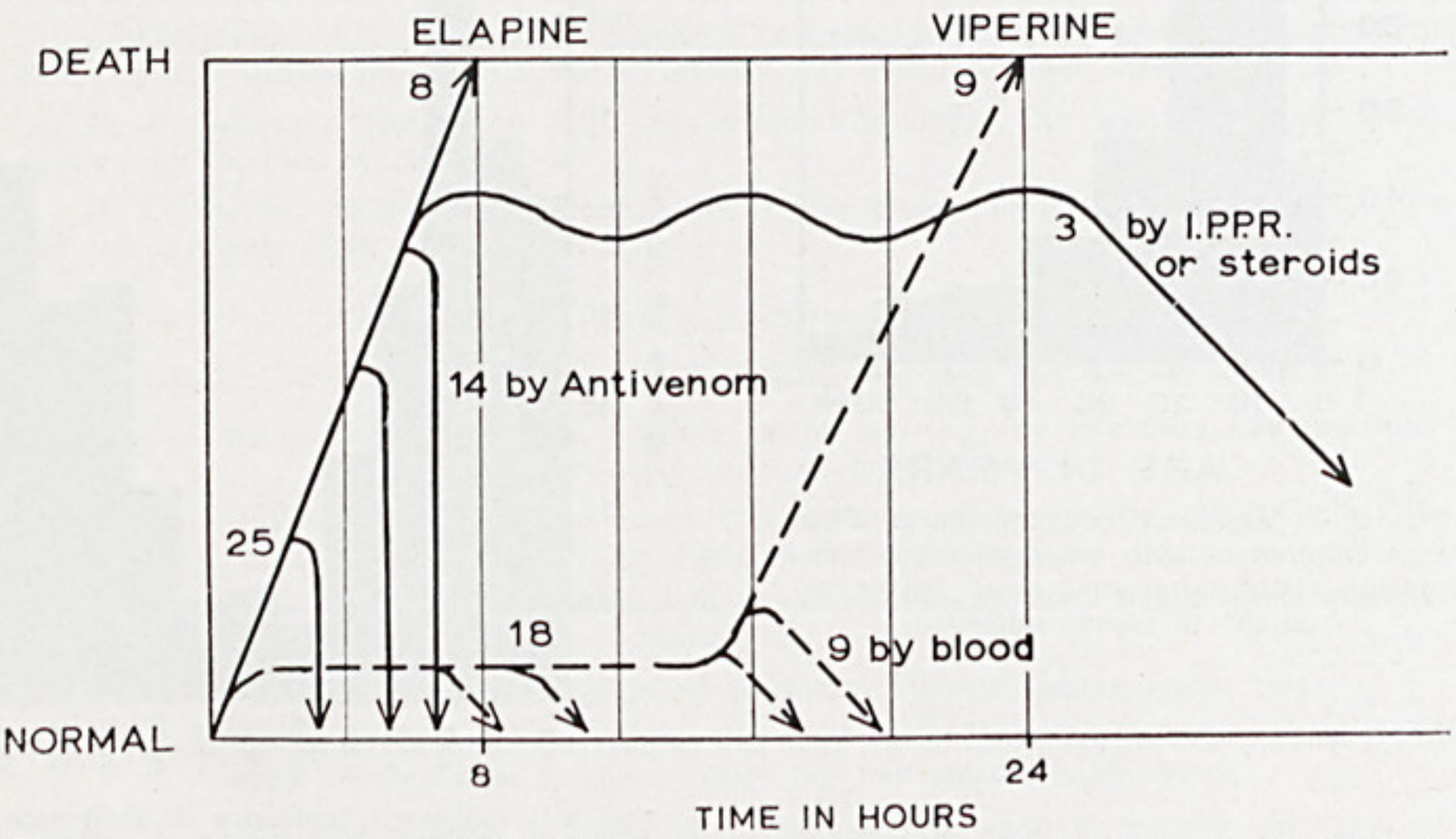

Fig. 5 — A graphic record of the march to death or recovery in severe cases of identified elapine and viperine snakebite.