

## CLINICAL ASPECTS OF SNAKE BITE IN THE PACIFIC AREA

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The venomous snakes of the Australian region all belong to the family Elapidae. They are principally found on the continent of Australia and the Island of New Guinea, both of these land masses having several species in common. The other Pacific Islands, with the exception of the Solomon Islands and Fiji, do not harbour front-fanged venomous snakes. In the south-west Pacific area venomous snake bite is common only in Papua.

The observations which form the basis of this paper were made on a group of 73 patients with varying degrees of envenomation, who were personally studied over a six-year period from October 1959 to November 1965, while I was working as a physician at the General Hospital, Port Moresby, Papua. Some of the data in this paper have been included in earlier papers (CAMPBELL, 1964, 1967; CAMPBELL and YOUNG, 1961).

There are three venomous snakes of particular importance in Papua. The Papuan black snake (*Pseudechis papuanus*), which has a distribution along most of the southern coast of Papua; the highly venomous taipan (*Oxyuranus scutellatus canni*), a distant relative of the cobra, which only occurs in the savanna woodland areas of Papua; and the viper-like death adder (*Acanthophis antarcticus*), which is widely distributed throughout forested regions of the whole Island of New Guinea.

The venoms of these three snakes contain potent neurotoxins. Taipan and Papuan black snake venoms also contain powerful coagulant factors. Papuan black snake venom is the most strongly haemolytic of any Australian snake venom; while taipan venom has only a weak haemolytic action. Haemorrhagin is also possibly present in Papuan black snake venom.

The neurotoxin, which is the killing component of all Australian snake venoms, causes a peripheral neuromuscular block and gives rise to a flaccid muscle paralysis. The coagulant factor produces a defibrination syndrome and incoagulable blood. The action of the haemolysin results in the dramatic sign of haemoglobinuria and lesser degrees of intravascular haemolysis.

The bites of the Australian venomous snakes cause little or no reaction at the site of the bite. Pain is absent or slight. Oedema is more commonly absent but if present is of only mild degree, unless a tourniquet has been used. Inspection of the wound made by an Australian venomous snake will not indicate whether or not venom has been injected and great importance therefore attaches to the presence or absence of symptoms which are indicative of envenomation.

These symptoms fall into three groups:

- (a) The early, non-specific, pre-paralytic symptoms: vomiting, faintness and sweating,

sudden loss of consciousness, headache, pain in the regional lymph nodes, abdominal pain and rarely diarrhoea.

(b) The early symptoms which are associated with the bleeding tendency or the action of the haemolysin: the continued bleeding of the snake bite wound or first aid incisions, the spitting, vomiting or coughing of blood, the passing of blood-stained or black urine.

(c) The symptoms associated with the onset of muscle paralysis, which are usually preceded by one or several symptoms from the previous two groups, but in ten per cent of patients these symptoms are the first manifestation of envenomation. They are: difficulty in seeing, blurred or double vision, difficulty in opening the mouth, difficulty in speaking or in swallowing, and difficulty in getting out of bed after a rest or a night's sleep.

The neurotoxin paralyzes all the voluntary muscles. The most severely affected patients cannot move their limbs or head; cannot open their eyes or mouth; cannot move their eyes or tongue; cannot speak, swallow or cough and they die from respiratory obstruction, due to the accumulation of secretions in the pharynx and lower air passages, or from mechanical respiratory obstruction due to a paralysed tongue or a lax jaw. The respiratory obstruction is invariably associated with respiratory insufficiency, due to a complete paralysis of the chest muscles and a partial or complete paralysis of the diaphragm.

An elapine snake bite poses three basic therapeutic problems:

1. The need to neutralize the venom which is being absorbed, and the hope that the effects of the poison already absorbed may be counteracted by antivenene.
2. The need to relieve respiratory obstruction.
3. The need to deal with respiratory insufficiency.

### *Antivenene*

Large amounts of a specific or polyvalent antivenene must be injected i.v. as soon as there are unequivocal symptoms or signs of envenomation present; or in the absence of these symptoms and signs if a definite bite from an identified venomous snake has occurred.

Injections of antivenene were given to 61 of the 73 patients at the hospital in Port Moresby. Haemoglobinuria, which was present in 11 cases ceased, on the average, within 11 hr of injecting the antivenene (range 3.5–20.5 hr). The coagulation defect commenced to improve within 4 to 8 hr and was almost normal within 48 hr of injecting the antivenene.

The effect of the antivenene on the course of the muscle paralysis was not so dramatic and may be summarized as follows (Table 1):

TABLE 1. ANTIVENENE EFFECT ON PARALYSIS—RESULTS  
FROM 61 PATIENTS

Effect of antivenene	No.
No change*	12
No progression	16
Paralysis reversed	4
Progression to slight, moderate paralysis	9
Progression to probable death	15
Died	5

\*No progression of paralysis expected and none occurred.

In only 4 patients was the paralysis reversed; while in 9 patients paralysis developed after the injection of antivenene or progressed to a slight or moderate degree. In 15 patients the

paralysis progressed to the point where death would probably have occurred if a tracheostomy had not been performed and artificial respiration carried out.

If reliance was placed on antivenene alone, at least 25 per cent of envenomated patients, who might subsequently be saved, would die.

The side effects of the antivenene therapy are summarized in Table 2.

TABLE 2. INCIDENCE OF THE SIDE EFFECTS OF ANTIVENENE THERAPY

Per cent developing side effects	Side effect
3 (2/61)	Developed anaphylaxis
5 (3/61)	Less severe general reaction
25 (15/61)	Reaction involving skin—itching, urticaria or oedema
13 (8/61)	Febrile reaction alone—rigor, shivering and fever

A total of 46 per cent (28/61) had some side effect from the antivenene but most of these reactions were mild and of little clinical significance; in only 8 per cent (5/61) was the reaction serious.

### *Tracheostomy*

Although the need to relieve respiratory obstruction is listed as the second problem, it is often of primary importance in advanced cases of elapine snake bite. The quick insertion of a cuffed endotracheal tube and the aspiration of pharyngeal and chest secretions may be life-saving measures. First-aid teaching about elapine snake bite must deal with the management of respiratory obstruction, respiratory insufficiency and the correct method of transporting these severely ill patients.

As early as 1883, WALL appreciated the importance of respiratory obstruction in elapine snake bite. He noted that the patient paralysed by cobra venom lay helpless on his back and threatened to be suffocated by saliva running into his paralysed larynx. At autopsy particles of food and medicines could sometimes be found in the air passages. Cushny (CUSHNY and YAGI, 1918) also emphasized that after cobra poisoning in experimental animals, obstruction to breathing occurred from the accumulation of oral secretions in the air passages and that tracheostomy restored respiration by shortening the air passages and increasing the efficacy of feeble thoracic movements.

In this series tracheostomy was carried out on 32 patients. The approximate mean time after the bite at which it was performed was 21 hr, with a range of from 3 to 96 hr. The operation was carried out in the operating theatre under local anaesthesia or more usually under a general (scolene-halothane) anaesthetic. Clinically the chief criterion for tracheostomy was evidence of pooling of secretions in the pharynx. Because of the continued aspiration of oral secretions into the air passages and the necessity to artificially ventilate many of these patients, a cuffed tracheostomy tube was always inserted. Nothing was given by mouth. A broad spectrum antibiotic was given by injection. The tracheostomy tube was left in for an average of 5 days, with a range of 2.25 to 16.0 days.

In no case was death directly due to the performance of the tracheostomy, but intensive nursing care and regular observations are required if these severely paralysed patients are not to die from a complication of the tracheostomy.

### *Artificial respiration*

Paralysis sometimes continued to extend for up to 29 hr after the injection of antivenene and the diaphragm became weaker so that, in addition to tracheostomy, some form of artificial respiration had to be used.

Artificial respiration was given by means of the intermittent compression of the re-breathing bag of a small portable anaesthetic machine using a closed circuit with carbon dioxide absorption.

Seventeen patients required artificial respiration or assisted breathing. Four of these patients died and one did not require artificial respiration after the antivenene had been injected. The remaining 12 patients required artificial respiration for an average of 34 hr, with a range from 8–240 hr.

When patients were kept alive by artificial respiration or tracheostomy, it was found that the paralysis commenced to improve after a period of approximately 48 hr after the bite and had usually completely disappeared within 1 week of the bite. Thus the paralysis produced by elapine snake venom is not irreversible as is commonly taught.

Antivenene, i.v. fluids, tracheostomy and the insertion of a cuffed tracheostomy tube, assisted respiration and intensive nursing care are the basic requirements for the successful treatment of elapine envenomation.

In the treatment of this group of patients blood transfusion (7 patients), injections of fibrinogen (2 patients) and infusions of noradrenalin (7 patients) were also used.

Severe hypotension accompanied the anaphylactic serum reactions or followed episodes of anoxia and preceded death in three of the five fatal cases. The management of this complication requires more investigation and study.

Five of the 73 patients died—a case fatality rate of 7 per cent.

It is believed that the use of tracheostomy and artificial respiration saved at least 25 per cent of the patients, with evidence of poisoning following Papuan snake bite, who would have otherwise died.

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