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VENOMOUS SNAKE BITE IN PAPUA AND ITS TREATMENT WITH TRACHEOTOMY, ARTIFICIAL RESPIRATION AND ANTIVENENE*

BY

C. H. CAMPBELL, M.B., M.R.C.P., M.R.A.C.P., D.T.M. & H.

Physician, General Hospital, Port Moresby, Territory of Papua and New Guinea

Death following an elapine snake bite is usually due to the action of the neurotoxins the venom, other toxic factors in the venom being of minor clinical importance. These rotoxins act on the neuromuscular junction (KELLAWAY et al., 1932) and may produce a plete paralysis of all the voluntary muscles. Death is due to respiratory obstruction paralysis of the respiratory muscles or, more usually, to a combination of both these rs (CAMPBELL and YOUNG, 1961). In serious cases the clinical picture thus produced mbles an acute myasthenic crisis.

Severe and potentially fatal paralyses produced by the venom of the common Asiatic ne snake, the cobra, have been reversed in animals (ARTHUS, 1910) and in man (AHUJA SINGH, 1954) by antivenene and cortisone (BENYAJATI et al., 1961). Spontaneous rapid complete recovery within 24 hours has also been repeatedly observed following re and all-but fatal cobra envenomation in animals (ACTON AND KNOWLES, 1914).

KELLAWAY (1932) found "the curarising action" of Australian elapine venoms much difficult to reverse with antivenene and, indeed, he felt that the characteristic feature paralysis produced by lethal doses of elapine venom was that it was irreversible (KELLA- 1932, 1937). Spontaneous recovery of severe paralysis after "sub-lethal" doses of tralian elapine venoms also occurs after several days and is never as rapid as that described cobra envenomation. There has been one report of the rapid recovery of a patient severe paralysis from the bite of an Australian elapine snake after the use of antivenene YVETT and MOLPHY, 1959).

The venomous snakes of Papua all belong to the family Elapidae and include species ch are the same as, or very closely related to mainland Australian species. Papuan ne snakes include two of the most deadly terrestrial snakes that are known to exist, taipan (*Oxyuranus scutellatus canni* Slater) and the death adder (*Acanthophis antarcticus sus* Loveridge), and a third common highly venomous snake, the Papuan black snake *udechis papuanus* Peters and Doria).

In the case of envenomation from these snakes, antivenene given late in the course of paralysis appeared to have no beneficial effect. If, however, the severely paralysed nt was kept alive by means of a tracheotomy, adequate drainage of the bronchi and hea, intravenous fluids and, if necessary, artificial respiration, it was shown that the lysis was not irreversible but lasted for 2 days and then was completely reversible within

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the next 2-4 days (CAMPBELL and YOUNG, 1961). In other words, the behaviour of the paralysis after lethal doses of venom was essentially the same as that following sub-lethal doses of venom.

This paper reports an additional 41 cases of venomous snake bite of varying severity, treated at the Port Moresby General Hospital. In addition, details of 11 of the 15 previously-reported cases (Nos. v-xv incl.) (CAMPBELL and YOUNG, 1961) are included, making a total of 52 indigenous patients admitted with a diagnosis of venomous snake bite between February 15, 1960 and September 30, 1962.

CIRCUMSTANCES OF THE BITE

The patients comprised 44 males and eight females whose ages ranged from 3 to 45 years. Thirty-four were aged between 20-30 years. The admissions were more or less evenly distributed throughout the year. In all but two cases, the patients were bitten while in the Central District of Papua, 38 of the patients coming from within 50 miles of Port Moresby. Two people were bitten in the early evening before 7.0 p.m. All the other patients were bitten in the daylight.

One patient was bitten on the finger, the remainder were bitten on the legs or feet (toe nine, foot 15, ankle eight, heel two and leg 17). In most cases, the bite occurred when the patient was walking along a bush road or track bordered by tall grass, or walking through long grass. The snake was usually not seen until the bite occurred and 12 patients actually trod on the snake before it bit them. Most of the snakes struck very quickly, let go immediately and escaped into the long grass. Often only a fleeting glimpse of the snake was obtained and indeed, eight patients never saw a snake at any time. However, some patients had to kick the snake free of the foot and an occasional patient had to pull the snake off. In only 1 case did the snake bite twice.

Five patients brought in a dead snake. Four of these snakes were quite small (three death adders and one Papuan black snake). The fifth dead snake was a five foot eight inch taipan. Apart from these five cases, the species of snake inflicting the bite remains unknown. Eighteen patients were bitten by large black or brown snakes which may have been taipans or Papuan black snakes.

SYMPTOMS OF ENVENOMATION

The incidence of the various symptoms of envenomation are recorded in Table I. In 17 cases, the symptoms developed within one hour or less of the bite; in eight cases, they developed in from 1-2 hours; in the remainder the symptoms developed within 2 to 12 hours of the bite.

TABLE I. Incidence of symptoms in 52 subjects* of venomous snake bite.

	Vomiting	Headache	Abdominal pain	Visual disturbance	Vomiting or spitting blood	Drowsiness	Pain in groin	Loss of consciousness	Blood-stained urine	Sleep
No. with symptoms	22	19	18	14	11	9	7	4	3	5
No. in which symptoms appeared first	8	13	5	2	3	1	3	4	1	5

(*Four cases without symptoms or signs; one case with mild signs only; Two cases with severe signs but history unobtainable).

Vomiting, headache and abdominal pain were the three commonest initial symptoms. The patient usually vomited two or three times but sometimes as many as nine times. Often the vomitus was blood-stained. Sometimes small amounts of frank blood were vomited

r blood-stained saliva was spat out. The abdominal pain was sometimes severe, particularly n children and occasionally suggested an acute abdomen (CAMPBELL and YOUNG, 1961). he pain was periumbilical, lower or upper abdominal or generalized in distribution.

The patient complained of pain in the lymphatic glands draining the site of the bite, ften within an hour of the bite.

Four patients collapsed soon after the bite occurred and were unconscious for some ime. In seven cases, none of these symptoms occurred and the first symptoms of a venomous e bite were heaviness of the eyelids, blurring of vision, diplopia or drowsiness. These atients often went to sleep and awoke in the early morning or next day with advanced ranial nerve palsies.

There was usually little or no pain in the bitten limb. Only three patients complained f severe pain.

SIGNS OF ENVENOMATION

In only eight cases were puncture wounds visible. Thirty-eight patients had one or everal incisions in the region of the bite, some of which oozed blood freely. In four cases, he local wound was not described and in two cases no definite wound was found (these ases were admitted 20 hours after the bite). Oedema around the wound was usually absent r slight. In two cases it was marked but this was also associated with the prolonged use f a tourniquet.

The earliest sign of envenomation was the appearance of tender, slightly enlarged regional mphatic glands, often within 1-2 hours of the bite. In some patients who complained f abdominal pain, marked abdominal tenderness and even guarding was noted. A slight egree of albuminuria sometimes occurred. In two cases, haemoglobinuria occurred before ny other signs developed.

The commonest objective sign of envenomation was slight ptosis of the eyelids, often ssociated with slight impairment of ocular movements in a vertical or lateral direction nd was present in 39 cases. A nasal voice, difficulty in speaking and swallowing or in pening the mouth or general muscle weakness then appeared.

In the more severe cases, the paralysis became more extensive. The paralysis of the xtrinsic ocular muscles was complete and the eyes were fixed and central in 21 cases. ilateral ptosis with fixed central eyes and pupils that still reacted to light was a characteristic eature of all cases of more than moderate severity.

In the most severe cases, the patient lay as if dead and the only movement that was etectable was an ineffectual twist of the pelvis. The paralysis of the limbs was symmetrical nd complete, the previously-noted asymmetry (CAMPBELL and YOUNG, 1961), being related o the misuse of a tourniquet on the bitten limb. If respiratory obstruction was not relieved, aralysis of the diaphragm progressed rapidly. If this obstruction was relieved and life upported, complete paralysis of the diaphragm usually did not occur for 24-30 hours after he bite. The patients usually remained co-operative and tried to communicate by means f a twist of the pelvis or by raising a finger in response to questions. The clinical picture f the severely paralysed patient has been described previously (CAMPBELL and YOUNG, 961).

No primary circulatory changes were again detected. In one patient (Case No. 13581), dmitted without any obvious respiratory movements, the heart was still beating strongly nd the blood pressure was well maintained. The electrocardiograph was normal when

taken at the height of the paralysis in three severely paralysed patients. Three patients showed some instability of the blood pressure while having artificial respiration, this may have been related to imperfect ventilation of these patients.

One severely paralysed patient (Case No. 8817), who was 24 weeks pregnant, did not miscarry.

The details of the 10 most severe unreported cases are summarized in Table II.

Treatment

Antivenene

Antivenene was given intravenously to all patients with signs and symptoms of envenomation and to those without symptoms who brought in the dead snake. At first, the doses of the various antivenenes given were a little haphazard but, over the last 18 months, a standard dose of antivenene was given. Sufficient antivenene was given to neutralize double the average venom yield of each of the three most dangerous snakes, i.e., 12,000 units of death adder, 24,000 units of taipan and 36,000 units of Papuan black snake antivenene. This represents a volume of 180-190 c.c. of serum. In the case of small children, half this volume of serum was given. If the snake was over 3 feet in length, death adder antivenene was not given, as the maximum size of the death adder is 3 feet.

In 39 administrations of serum in this series of cases, 21 patients had some form of reaction to the serum. Eight patients developed urticaria and three, an itchy skin without obvious urticaria. Twelve patients developed a fever which was associated with a rigor in seven cases. The fever lasted about 12 hours. Five patients developed a cough and one, an increase in oral secretions. Three severe reactions occurred—one patient had a slight fall in blood pressure associated with headache, vomiting and urticaria; one involuntarily passed urine and faeces, and also developed a wheeze; one had a profound anaphylactic shock.

In one case, the severe reaction was immediate, but more commonly, the itchy skin and urticaria developed within 10-30 minutes of commencing the injection of the serum. The rigor and fever usually occurred 1-2 hours after the commencement of the injection and usually when the administration of the serum had ceased. Gross proteinuria occurred within an hour of giving the serum and persisted for 12-24 hours.

The results of the antivenene therapy are summarized in Table III.

Tracheotomy

The insertion of an endotracheal tube and the aspiration of endotracheal secretions were sometimes necessary in the ward, before the tracheotomy, as a life-saving measure. The tracheotomy was performed in the theatre under local infiltration anaesthesia. A cuffed rubber tracheotomy tube was inserted, while this was being done, the patient's breathing was assisted if necessary. In six cases, bleeding from the tracheotomy wound was the most troublesome local complication. A steady ooze of blood continued for 24-48 hours and then the blood re-coagulated. No person died as a direct result of the tracheotomy but several patients almost died due to blocked or displaced tracheotomy tubes. Fortunately, the tracheotomy tube is only required for 4-6 days.

Artificial respiration

Artificial respiration was given by means of the manual compression of the re-breathing bag of a small anaesthetic machine, using a closed or semi-closed circuit with carbon dioxide absorption. The artificial respiration was usually carried out by a 2nd or 3rd year nurse who was regularly supervised by experienced sisters and medical officers.

TABLE II. The signs, treatment and results in 10 severe cases of elapine envenomation.

Case Number	Approx. age	Sex	Signs: Paralysis of[1] Eyelids	Eye muscles	Face muscles	Tongue	Palate	Jaw	Neck	Arms and legs	Intercostals.	Diaphragm	Bleeding tendency	Treatment: I.V.I. Antivenene[2] Taipan	Papuan black	Death adder	Tiger	Tracheotomy	Artificial resp. duration (hours)	Estimated death time (hours)	Recovery period (days)
2932	10	F	+ + +	+ + +	+ +	+ + +	+ + +	+ + +	+ + +	+ +	+ + +	+	+	—	18	—	18	Yes	3–4	12	9
3112	12	M	+ +	+ + +	+	+	+ + +	+ +	+ +	+ +	+ + +	—	—	30	9	—	30	Yes	—	30	9
5570	35	M	+ + +	+ + +	+ +	+ + +	+ + +	+ + +	+ + +	+ +	+ + +	+ +	+	30	30	—	30	Yes	39	15	10
5705	7	M	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	+ + +	+ + +	—	12	18	12		Yes	156	9	20
7649	10	M	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	+ + +	+ +	—	18	18	12		Yes	Yes	10	Died
8634	35	M	+ +	+ + +	+	+	+ + +	+ +	+ +	+ +	+	—	—	24	36	12		Yes	—	20	5
8817	30	F	+	+ + +	+	+	+ +	+ +	+	+	+	—	—	24	36	18		Yes	—	26–30	5
9934	35	M	+ + +	+ + +	+ +	+ +	+ + +	+ + +	+ +	+ +	+ + +	+ +	—	24	33	12		Yes	58	14	11
12448	40	M	+ + +	+ + +	+ +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+	24	36	12		Yes	240	8½	20
13581	45	M	+ + +	+ + +	+ +	+ + +	+ + +	+ + +	+ +	+	+ + +	+ + +	+	24	33	12		Yes	37	7	6

[1] + Slight paralysis + + + Complete Paralysis [2] thousands of units.

TABLE III. Results of antivenene therapy in 34* subjects of Paupan snake bite.

Case No.	Sex and Age	Interval between bite & antivenene administration	Clinical condition when antivenene administered	Condition after antivenene administration	Case No.	Sex and Age	Interval between bite & antivenene administration	Clinical condition when antivenene administered	Condition after antivenene administration
		hours					hours		
397	F.30	54	Moderate P.	No change	3583	M.22	6½	Slight S.-Minimal P.	Improved
518	M.35	21	Severe P.	No change	5570	M.35	10	Moderate P.	Prog.—> ?D.
866	F.13	48	Slight P.	No change	5705	M.7	6½	Moderate P.	Prog.—> ?D.
1031	M.25	27	Severe P.	Progression	6708	F.8	2	Severe S.-No P.	No progression
1225	M.14	28	Slight P.	No change	7847	M.10	2½	Severe S.-No P.	Slight progression
1317	M.40	16	Slight ptosis only	Improved	8549	M.3	26	Moderate P.	No change
1586	M.25	27	Severe P.	No change	8634	M.35	16	Moderate P.	Prog.—> ?D.
1862	M.25	3½	Severe S.-No P.	No progression	8817	F.30	24	Severe P.	No change
1928	F.20	19	Severe P.	No change	9934	M.40	11	Moderate P.	Prog.—> ?D.
2292	M.17	6	Severe S.-No P.	No progression	10280	M.30	7	Early S.-No P.	Slight progression
2358	F.30	3	Severe S.-No P.	No progression	12129	M.20	2	No. S. or P. Bitten by taipan	? No progression
2483	M.25	9	Severe P.	Prog.—> ?D.	12448	M.40	7	Slight P.	Prog.—> ? D.
2904	M.35	3¾	Symptoms ? P.	No progression	13057	M.35	13	Moderate P.	No change
2932	F.10	4	S.-Slight P.	Prog.—> ? D.	13235	M.41	8	S.-No. P	No progression
3112	M.12	21	Moderate P.	Prog.—> ? D.	13518	M.17	8	S.-No P.	No progression
3232	M.35	16	Moderate P.	Progression	13581	M.45	6	Severe P.	No progression
3501	M.25	49	Slight P.	No change	13855	M.40	3	Severe S.-No P.	No progression

P. = Paralysis. S. = Symptoms. Prog.—> ?D. = Progression to probable death. No change = Progression not expected and did not occur. No progression = Progression expected but did not occur.

(*Antivenene also given to two patients who died and three patients bitten by small snakes).

In the most severe case, complete absence of all respiratory movements was only present for about 6 hours. In two cases, artificial respiration had to be continued for 6-10 days.

One patient developed an atelectasis and pneumonia which responded to tracheal aspiration and postural drainage.

General management

All treatments were recorded and a half-hourly record of the pulse and blood pressure was maintained until the tracheotomy tube was removed. If the patient could not swallow, nothing was given by mouth and intravenous fluids were administered.

The patient usually voided urine spontaneously. Intramuscular injections of paraldehyde were given if he was restless. Half a gramme of intravenous or intramuscular chloramphenicol was given every 6 hours to the most severely paralysed patients. Other antibiotics were sometimes used.

In two of the most severely paralysed patients, noradrenalin infusions had to be used for periods of 12 to 24 hours to treat hypotension which followed episodes of severe anoxia. Three severely paralysed patients each received one injection of intravenous hydrocortisone for acute serum reactions. One patient received 100 mg. hydrocortisone empirically every 6 hours for 4 days.

RECOVERY PHASE

The paralysis of the diaphragm remained complete for up to 6 hours, then weak diaphragmatic movements recommenced. (If larger doses of venom were injected then, presumably, this period of complete diaphragmatic paralysis would last much longer). It took 1-4 days, as a rule, for the diaphragm to recover sufficiently to allow the patient to breath unaided. After the diaphragm, the ocular muscles showed the first sign of recovery as a rule, about 48 hours after the bite the first twitch of ocular movement could be observed. Recovery after this was generally rapid and, within 2-5 days of the first sign of ocular movement, the patient recovered most of his muscle function although it took another week or so before muscle power was normal again.

The recovery times for the severely paralysed patients are recorded in Table IV. The recovery times for the ocular muscles in less severely paralysed patients was of approximately the same order. Observations and records were made several times a day during the first 2-3 days and then twice a day.

RESULTS

Of the 41 new cases reported, the 10 who were severely paralysed would have undoubtedly died without the performance of a tracheotomy and the use of artificial respiration. Nine of these patients recovered completely, one died. This patient became anoxic during the performance of the tracheotomy and then hypotensive. There was considerable delay before effective resussitative measures were instituted and irreversible hypotension occurred. One of the previously reported patients died, making two deaths in the 52 cases. The two patients who died were not the most severely affected cases.

The performance of a tracheotomy and the use of artificial respiration reduced the mortality rate from a possible 29 per cent. to 4 per cent. Antivenene may have prevented serious paralysis developing in another nine cases (17 per cent.).

Discussion

The pain in the regional lymphatic glands and the tender regional lymphadenopathy is a well-documented sign and symptom of a viper bite (Efrati and Reif, 1953), but is not commonly recognized as an important early symptom and sign of a venomous elapine snake bite.

Table IV. The Recovery times (from the time of the bite) for the extrinsic ocular muscles and other movements following elapine envenomation.

Case number	Extrinsic ocular muscles		Normal tongue movements	Ability to swallow normally	Sit up unaided
	First sign of recovery hours	Complete recovery days hours	days hours	days hours	days hours
2932	48	4 12	4 —	5 —	7 —
3112	52	4 —	2 22	3 22	5 10
5570	44	3 12	2 18	4 4	5 18
5705	44½	3 3	4 18	5 6	7 —
8634	50	3 —	3 —	3 12	3 —
8817	48	3 6	3 6	3 22	3 22
9934		Details	Not	Recorded	7 —
12448	47	3 12	3 6	5 —	12 —
13581	66	4 —	3 6	3 12	4 12

The symptoms—headache, vomiting, loss of consciousness, pain in the regional lymphatic glands, the spitting or vomiting of blood, abdominal pain and blood-stained urine are very important because they occur usually before the paralysis develops and at a time when, if their significance is recognized and antivenene is given, it will be of value. Unless some such dramatic symptoms as the above occur, the early signs of envenomation may pass undetected. The ptosis and ocular palsy and bulbar paralysis may not be recognized until a late stage in their development.

Owing to the great variability in the amount of venom injected by snakes (Reid, 1956), it is impossible ever to know if serum therapy has been of value at all unless some dramatic reversal of severe paralysis occurs. In only two cases did any reversal of paralysis occur after the injection of antivenene. Both patients had minimal signs, namely a slight ptosis.

In nine patients with severe early symptoms and signs of envenomation, severe paralysis did not occur after the antivenene was given. Antivenene was thought to have been of definite value even though given up to 8 hours after the bite.

Case No. 13855: a male aged 40 years was bitten in the right heel at 9 a.m. One hour after the bite he developed central abdominal pain and pain in the right groin, and started to spit out blood-stained saliva. He was admitted to hospital at 11.30 a.m. On examination, two minute bleeding points were seen one cm. apart on the R. heel. There were tender, slightly enlarged right inguinal glands present and some central abdominal tenderness. Antivenene was given between 12.5 and

12.55 p.m. The abdominal pain and pain in the right groin persisted up until 9.0 p.m. but the next morning the patient was quite recovered.

One patient with similar early signs and symptoms of envenomation who inadvertently did not receive any antivenene did not develop any paralysis. In view of this, one cannot be certain that antivenene was of value in the other nine cases.

Once significant paralysis had developed, antivenene did not appear to be of any value even though it was given as early as 4 hours after the bite. The recovery rate of the paralysis was much the same in all severely and moderately paralysed patients whether they received antivenene or not and irrespective of the different dosages and types of antivenene given. The good results obtained in the severely paralysed patients could not be attributed to the antivenene and no obvious benefit followed the use of antivenene in such cases. Quite often the paralysis continued to progress.

Case No. 5705: a male, aged 7 years, was bitten on the right leg at 6.0 p.m. He developed heaviness of the eyes and abdominal pain. He was admitted at 8.0 p.m. On examination, a partial ptosis and marked restriction of eye movements was present. He could still sit up unaided. Tender slightly enlarged lymph glands were present in the right groin. He was acutely tender in the right hypochondrium and some guarding of the abdominal muscles was also present. He also had haemoglobinuria. Between 8.30 and 9.45 p.m. antivenene was given intravenously but the paralysis continued to progress. A tracheotomy was required by 10 p.m. and his breathing had to be assisted. Complete cessation of diaphragmatic movement occurred next day. He subsequently recovered completely.

Tracheotomy is undoubtedly of great value in the treatment of severe cases of elapine snake envenomation and the indications for its use in this condition are similar to those laid down for its use in a wide variety of other clinical states characterized by mechanical and secretional ventilatory obstruction (NELSON, 1958). Tracheotomy was performed in 16 of the 52 cases with only one death in the tracheotomized patients.

Any drug which can shorten the period during which "medical brinkmanship" has to be practised is worthy of a trial. BENYAJATI et al. (1961) noted that, after the injection hydrocortisone, patients suffering from paralysis due to cobra envenomation often improved more rapidly than they would have done normally and regular administration of hydrocortisone often brought about dramatic improvement.

Because of this favourable report, 400 mg. of hydrocortisone per day was given intravenously empirically to one severely paralysed patient (Case No. 13581). This patient recovered slightly quicker than usual although the recovery time of his extrinsic ocular muscles was slower than normal (this is the best muscle group for the objective measurement of muscle recovery). The three patients who received one injection of hydrocortisone showed no obvious benefit from it. However, cortisone should be tried further before it is rejected as being of no value in Papuan elapine envenomation.

The extrinsic ocular muscles and the elevators of the upper eye lid are the most susceptible muscles to the action of elapine venom. A complete paralysis of the extrinsic ocular muscles is produced by much smaller amounts of venom than a lethal dose. The most resistant muscle to the action of the venom is the diaphragm. The dose of venom required to paralyse the diaphragm completely is probably several times that required to paralyse the ocular muscles completely.

In the most severely paralysed patient reported here, (Case No. 13581), the estimated death time would have been approximately 7 hours. It is reasonable to suppose that this patient had received at least two and possibly three, human lethal doses of venom (ACTON and KNOWLES, 1921). If his diaphragm received two to three paralysing doses (actually a lethal dose is probably less than a complete diaphragm-paralysing dose, but this does not

affect the argument) his ocular muscles must have received four to six times their paralysing dose. In this case the ocular muscles recovered completely despite this relatively large dose of venom.

It seems reasonable to infer therefore, that if a patient received four to six human lethal doses of venom (doses greater than six lethal doses would be very rare) his diaphragm, like his ocular muscles, should still recover completely. KELLAWAY et al., 1932, showed that even after 7-20 intravenous lethal doses of death-adder venom, strong respiratory impulses continued to pass down the phrenic nerve to the paralysed diaphragm of an artificially ventilated rabbit for 1-2 hours.

The failure of animal experiments to show that the "curarising effect" of the venom on the diaphragm was reversible (KELLAWAY, 1932; SILBERBERG, 1954) can in part be explained by the technical difficulties involved in keeping small tracheotomized and artificially ventilated animals (rabbits) alive for any length of time.

However, the irreversible paralysis was also thought to be due to the fact that snake venoms, apart from their action at the motor-end plate, have a direct paralysing effect on the muscle cells which could not be reversed by antivenene and this led to a complete loss of muscle irritability (KELLAWAY, 1937). There is no clinical evidence that such irreversible changes in muscle ever occur following the natural bite of Papuan elapine snakes. Although there is *in vitro* experimental evidence for this direct effect of the venom on muscle cells (HOUSSAY et al., 1922; KELLAWAY, 1937) the loss of muscle irritability in animal experiments could also be explained by such factors as continued over- or under-ventilation of the animals.

The 10 serious cases reported in this paper are, on the whole, more severe than those reported previously. The estimated death time was 20 hours or less in eight of the 10 cases. This tends to emphasize further the previous conclusion (CAMPBELL and YOUNG, 1961) that the paralysis due to elapine envenomation should always prove reversible, provided that no other toxic factor in the venom is in high enough concentration to cause other serious effects and this seems an unlikely happening.

Conclusions based on the treatment of such highly venomous snakes as the Papuan elapine snakes probably should be applicable to poisoning by other elapine snakes for their action is essentially the same (KELLAWAY et al., 1932) and taipan and death adder venom are more toxic than cobra venom (FAIRLEY, 1929; MORGAN, 1956).

SUMMARY

1) An additional 41 cases of venomous snake bite treated at the Port Moresby General Hospital are reported. The details of the 10 most severe cases are tabulated.

2) The clinical findings in these cases and in 11 previously reported cases are reviewed.

3) The performance of tracheotomy and the use of artificial respiration reduced the mortality rate from a possible 29 per cent. to 4 per cent. Antivenene may have benefited another 17 per cent.

4) The complete reversibility of the muscular paralysis produced by lethal doses of elapine venom was again demonstrated.

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