

## Appendix 3

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### THE PAPUAN BLACK SNAKE (*PSEUDECHIS PAPUANUS*) AND THE EFFECT OF ITS BITE

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THE Papuan black snake (*Pseudechis papuanus* Peters and Doria) is the commonest venomous snake encountered in Papua, bites from this snake being responsible for more hospital admissions than any other snake. The average length of the snake is about 6 ft. but it may grow to 8 ft. The head, neck and back of the snake are jet-black in colour whilst the ventral surface is bluish-grey. The snake is active in the daytime

degrees 10 minutes) and other specimens have been identified from Amazon Bay (longitude east 140 degrees 20 minutes) and the southern shore of Milne Bay (longitude east 150 degrees 30 minutes) (H. G. Cogger, personal communication 1966). Slater (1960a) considered the natural habitat of the snake to be the savanna woodland vegetation. However the snake was found in neighbouring forest

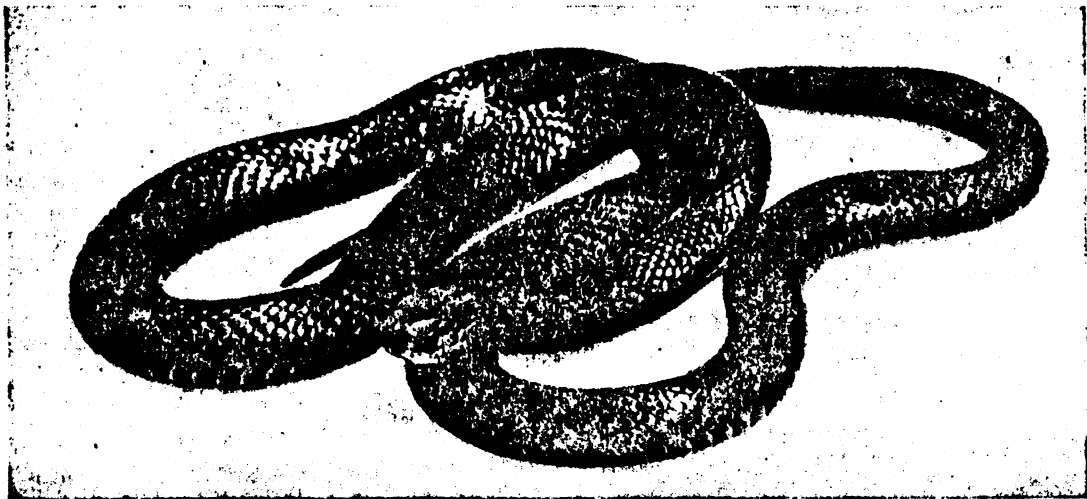


Plate 1.—The Papuan Black Snake—*Pseudechis papuanus*.

[Photo Courtesy R. K. Slater]

and early evening and is said by Slater (1956) 'to fear man', moving away if it encounters him. Nevertheless, if interfered with, it will defend itself with greater strength and determination than any other species known to Slater. In the Mekeo this characteristic ferocity of the snake appears to be recognized by the use of the name '*auguma*'—'to bite again'.

The distribution of the Papuan black snake probably extends along most of the southern coast of Papua from the West New Guinea border to the eastern tip of Papua. K. R. Slater (personal communication 1960a) has collected the snake from the western border of the Fly River to just east of Marshall Lagoon (longitude east 148

and showed a distinct preference for swamp terrain. He thought that the snake would be absent from the coast of the Gulf of Papua but might be present in the foothills behind the Gulf of Papua. The eastern distribution of the snake is certainly well beyond the eastern limits of the area of savanna woodland.

The fangs of the Papuan black snake are small but it has one of the largest venom yields of any Australian snake. The average venom yield obtained at the Commonwealth Serum Laboratories was 200 milligrammes and the maximum venom yield was 494 milligrammes. The venom has about one-eighth the potency of taipan venom. The subcutaneous certainly lethal dose (CLD) for 400 gramme guinea pigs was 0.08 mg. compared to 0.01 mg. for taipan venom (K. R. Slater, personal communication 1960b).

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There is no published work on the effect of Papuan black snake venom on the common laboratory animals. The venoms of three other species of the same genus have been studied—the common or redbellied black snake *Pseudechis porphyriacus* (Martin 1895, Kellaway 1930), the king brown snake *Pseudechis australis* Gray (Kellaway and Williams 1929) and the spotted black snake *Pseudechis guttatus* De Vis (Kellaway 1929). These three venoms had a weak neurotoxic action which was shown to be due to a peripheral neuromuscular blocking action in the case of common black snake venom (Kellaway, Cherry and Williams 1932). All three venoms were strongly haemolytic and showed haemorrhagin activity, but there was one important difference between them—the venoms of the common black snake and the spotted black snake had a coagulant action whereas the venom of the king brown snake had a powerful anticoagulant action.

#### PRESENT STUDY.

Over a 19-months period from 1st March, 1964 to 31st October, 1965, 13 patients were admitted to the Port Moresby General Hospital who were thought to have been bitten and poisoned by Papuan black snakes. In the case of only one of these patients was the snake positively identified as a Papuan Black snake (a 5 ft. long specimen). Colour is an unsatisfactory and unreliable means of identifying a snake as a rule but over a seven-year period and despite over 120 admissions for proven venomous snake bite there has only been one other admission in which a Papuan black snake was brought to the hospital, a small 18 in. specimen. However, the red back of the Papuan taipan is a characteristic distinguishing feature so that if a patient with symptoms of snake-poisoning was bitten by a large black snake and the whole of the snake was seen, it has been accepted as a case of Papuan black snake bite.

There were 11 male patients aged 8 to 45 years and two female patients aged 9 and 25 years. All the patients were bitten while it was daylight—10 on the foot, heel or ankle; one on the hand; one on the anterior aspect of the elbow and one on the back of the shoulder. All the patients came from the Central District except one, who was bitten on a plantation at Gadaisu, Orangerie Bay, Milne Bay District (longitude east 149 degrees 45 minutes).

#### Local Wound.

Seven patients had small puncture wounds, varying from two to six in number. One patient had a laceration three millimeters long; one had four gaping lacerated wounds. No definite local wound could be found when one patient was examined 23 hours after the bite. In three patients only first-aid incisions were seen. No oedema was present around the wounds of 11 patients; slight oedema was present in one patient. There was extensive swelling and ecchymoses surrounding the wound on the shoulder. The snake bite wound or the first-aid incisions continued to bleed for some time after the bite in four patients. The wound was usually not painful or only slightly so but the shoulder wound caused severe pain.

#### Symptoms of Poisoning.

Vomiting was the most frequent early symptom (8/13), and occurred as early as 15 minutes after the bite. Seven patients complained of pain in the regional lymphatic glands which persisted for up to 30 hours or more after the bite. Four patients complained of headache which was severe in two. The headache developed within half an hour of the bite and lasted one to several hours. Four patients complained of abdominal pain which was said to be severe by one patient. The abdominal pain lasted one to 18 hours. One patient complained of severe left loin pain. Loss of consciousness for a short period occurred within 15 minutes of the bite in two patients. Two patients vomited blood; two coughed up blood-stained sputum. Four patients, in addition to some of the above symptoms, complained of one of the following symptoms: difficulty in opening the eyes or double vision or inability to speak properly or inability to get out of bed on the morning after the bite.

#### Signs of Poisoning.

The most constant early sign of poisoning was tenderness of the regional lymphatic glands (11/13). In four of the six patients, who were seen within four hours of the bite, the glands appeared to be acutely tender. The glands were slightly enlarged in eight patients. The gland tenderness persisted for up to 30 hours. Oozing of blood from around the teeth was observed in two patients and continued for at least six hours after the antivenene was injected. Three patients had abdominal tenderness which was associated with muscle guarding in one patient. Three out of four patients, who were seen within six hours

the bite and who had evidence of serious poisoning as indicated by a fibrinogen titre of nil, also had haemoglobinuria. (The sera of all four patients showed evidence of haemolysis.) Albuminuria was present in all patients with serious poisoning.

Five patients developed a paralysis of all the voluntary muscles which endangered their life. Lesser degrees of paralysis occurred in four other patients, three of whom had an incomplete ptosis, an incomplete or complete external ocular muscle paralysis and some weakness of the jaw muscles. In addition, one of these three patients had some weakness of the facial, chest and limb muscles. The fourth patient with slight muscle paralysis had trismus and slight weakness of his limbs.

#### Laboratory Findings.

In the more severely affected cases the whole blood coagulation time was prolonged longer than one hour and as no clot formed in the test-tubes the prothrombin time, Fearnley fibrinolysis time and euglobulin lysis time could not be estimated; the rabbit anti-fibrin test, a flocculation test for human fibrin degradation products (Ferreira and Murat 1963), was strongly positive; the fibrinogen titre (Sharp *et al.* 1958) was nil and was unaltered by the addition of epsilon aminocaproic acid. There was only slight transitory fibrinolytic activity when the patient's serum was tested on heated and unheated human fibrin plates. The bleeding time was sometimes prolonged.

The platelet count was reduced in some cases but never below 200,000/cmm. The erythrocyte sedimentation rate (Wintrobe) was one millimetre in three of the four serious cases, who were seen within four hours of the bite, and a high neutrophil leucocytosis (45,200/cmm) was present in one of these cases.

Some of the clinical and laboratory findings are summarized in *Table 1*.

#### DISCUSSION.

The Papuan black snake would appear to be naturally more aggressive than Slater suggests. Papuans relate this more aggressive behaviour to breeding seasons. Some patients' histories substantiate the view that the snake then makes unprovoked attacks. Two patients in the present study saw the snake which made towards them. The boy bitten on the shoulder was bending over sharpening a stick in front of a 3 ft. high mound. Usually the snake bites, lets go and rapidly moves off. Occasionally the snake has wound itself round the legs and has to be kicked free

or pulled off. Snake bite in Papua is an occupational hazard of gardening, the patients often being bitten on the way or while working in the gardens.

The early preparalytic symptoms and signs of snake poisoning—vomiting, pain in and tenderness of the regional lymph nodes, abdominal pain, headache—appear to be more severe after, and a more constant feature of, Papuan black snake bites.

The extensive ecchymoses and swelling around the shoulder bite wound was the first time in eight years that such a marked reaction had been seen around a Papuan snake bite wound. Occasionally a tiny ecchymosis surrounds the minute puncture wound but usually there is little or no reaction around a wound on the limbs. As the venom contains a powerful coagulant and haemolysin and probably haemorrhagin as well, it is puzzling why this should be so, because extensive local oedema and haemorrhages were noted in laboratory animals at the site of the injection of the three other *Pseudechis* species venoms.

Clinically the venom appears to have a very powerful neurotoxic action. In patients treated in hospital, after the administration of anti-venom, the most likely cause of death would be due to the action of the neurotoxin in the venom. The venom produces a generalized peripheral muscular paralysis which would lead to death from respiratory obstruction and, or, respiratory insufficiency. There is a significant latent period, dependent in part on the dose of venom injected, between the time of bite and the subsequent development of paralysis. Even after sufficient venom has been absorbed to produce incoagulable blood there is still a latent period before paralysis develops. The paralysis is usually first evident in the muscles of the upper eyelids, the extrinsic ocular muscles or the muscles of the tongue and jaw and progresses slowly or rapidly to involve other muscle groups, depending on the dose of venom injected. The last muscle to be completely paralysed is the diaphragm. None of the patients in this study developed a complete diaphragmatic paralysis.

The patient's blood is incoagulable after a serious Papuan black snake bite. The local wound produced by the snake or by a first-aid worker may continue to bleed. The continued oozing of blood from small puncture wounds, the oozing of blood from around the teeth, the coughing of blood-stained sputum, and the

Table 1.—Summary of some clinical and laboratory details concerning 15 patients bitten by Papuan Black Snakes.

Hospital Number	Sex—Approx. Age (Years)	Interval between Bite and Admission (Hours)	Coagulation Time (1) (Lee and White) (Minutes)	Fibrinogen Titre (Normal 1/32)	Haemolysis	Clinical Bleeding Tendency (2) (3)	Paralysis	Treatment and Response (2)
27130	M 10	0.25	7	‡	—	Sputum sl. blood-stained.	Slight ....	Slight progression after antivenene; recovered.
26580	F 9	0.5	30	NIL	Haemoglobinuria	Bleeding from mouth, bite wounds	Severe generalized ....	Progression after antivenene; required tracheotomy and artificial respiration; recovered.
30202	M 25	0.5	60	NIL	+	Bleeding from around teeth and incised wounds	None ....	No progression after antivenene.
24281	M 30	2.0	60	NIL	Haemoglobinuria	None evident ....	Slight ....	Slight progression after antivenene.
28643	M 25	2.0	4.17	1/32	—	None evident ....	None ....	No progression after antivenene.
28868	M 26	3.0	50	NIL	Haemoglobinuria	Bleeding from puncture wounds.	None ....	No progression after antivenene.
29314	M 8	6.5	10	‡	—	Vomited blood	Severe generalized ....	Antivenene at O.S.; further progression after more antivenene; tracheotomy required; recovered.
30254	M 25	9.0	5.25	1/32	—	None evident ....	None ....	Antivenene at O.S.; none at hospital; no progression; recovered.
26601	M 45	20.0	3.5	‡	—	None evident ....	Severe generalized ....	No change after antivenene; tracheotomy required; recovered.
35138	M 35	22.0	6.5	‡	—	Vomited, spat blood at O.S.	Moderate ....	Antivenene at O.S.; none at hospital; no change; gradual recovery.
30386	F 25	23.0	8.5	1/16	+	Coughed up blood at O.S.	Severe generalized ....	No antivenene at hospital; required tracheotomy; recovered.
33122	M 40	25.0	7.5	1/32	Haemoglobinuria	None evident ....	Slight ....	Antivenene at O.S.; no change after more antivenene; anuric 14 days; recovered.
36371	M 10	27.0	6.5	1/16	—	Vomited blood p.w. bleeding at O.S.	Severe generalized ....	Antivenene at O.S. progression following it; tracheotomy required.

(1) normal coagulation time for Papuans is 3 to 6 minutes. (2) O.S. = out station. (3) p.w. = puncture wounds.

vomiting of blood-stained vomitus are another group of very important early signs of a serious Papuan black snake bite, present before the neurotoxic symptoms and signs become evident. Even after the injection of antivenene troublesome bleeding may continue from a tracheotomy wound for 24-36 hours. Papuan black snake venom appears to have a strong coagulant action but the nature of the coagulant action has not been investigated *in vitro*. A secondary afibrinogenemia renders the blood incoagulable.

As all four patients with serious poisoning, who were seen within a few hours of the bite, had evidence of haemolysis, Papuan black snake venom must be regarded as being strongly haemolytic. One patient (Hospital No. 33122), who was passing heavily blood-stained urine on an outstation for 12 hours, was anuric on admission and recovered after repeated peritoneal dialyses for 11 days. As haemoglobinuria occurs before any signs of paralysis are present, it is a most important early and dramatic sign of serious poisoning. Haemoglobinuria is an uncommon and inconstant sign of Papuan taipan bite and does not occur after a death adder bite. If haemoglobinuria is present after a snake bite in Papua the patient has most probably been bitten by a Papuan black snake.

Papuan black snake venom probably also contains significant amounts of haemorrhagin, the toxic factor in snake venom which damages the walls of small blood vessels. The evidence for this is indirect and tenuous:—(a) The oozing of blood from around the teeth, the spitting, coughing and vomiting of blood are more common features of serious Papuan black snake bites than taipan bites. This possibly means that, apart from the afibrinogenemia which occurs after taipan and Papuan black snake bites, there is also some damage to the walls of small blood vessels in the case of Papuan black snake bites; and (b) in an autopsy on a patient, who died from a Papuan black snake bite, numerous small haemorrhages were present in the viscera (B. Todd, personal communication 1959).

When serious poisoning was present, as indicated by the presence of incoagulable blood or haemoglobinuria, antivenene in some cases prevented the development of paralysis, in other cases it probably lessened the extent and severity of the subsequent paralysis. In several patients antivenene did not prevent the development of a life-threatening paralysis. The paralysis pro-

duced by Papuan black snake venom was never reversed by specific or polyvalent antivenene. The coagulation defect commenced to improve within five hours of giving the antivenene and was normal within 48 hours. The haemoglobinuria ceased soon after the antivenene was administered (Campbell 1967).

#### SUMMARY.

1. The clinical and some of the laboratory findings in 13 patients who were thought to have been bitten by Papuan black snakes are summarized.

2. The venom of the Papuan black snake is strongly haemolytic and coagulant in its action. The venom also contains a potent neurotoxin and probably haemorrhagin as well.

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