PAPUAN TAIPAN (*Oxyuranus scutellatus canni*) ENVENOMATION IN RURAL PAPUA NEW GUINEA

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ABSTRACT

Nine cases of serious envenomation following bites by Papuan taipans *Oxyuranus scutellatus canni* are presented. Eight cases were fatal with contributing factors such as delayed presentation with advanced symptoms, incorrect antivenom administration, failure to recognise key indications of envenoming, lack of antivenom, and delayed administration of correct antivenom. The survival of a single patient was greatly assisted by prompt recognition of coagulopathy as a symptom of envenoming, followed by administration of appropriate antivenom and careful patient monitoring using available techniques such as the 20WBCT protocol. A consistent approach to management incorporating strategies such as early recognition of envenomation, prompt pressure immobilisation, precise ongoing clinical assessment and early treatment with appropriate antivenom may improve the prognosis of envenomed patients.

INTRODUCTION

Snakebite in Papua New Guinea is a serious public health problem with localized incidence among the highest of any tropical region in the world[1,2]. Research involving ICU patients at Port Moresby General Hospital (PMGH) found a mean case fatality rate (CFR) of 9.54%, while among children under 10 years the CFR was 15.7%. 39.9% of all cases involved children under the age of 15 years, and these patients accounted for 55.6% of fatalities[3]. The prognosis for patients at rural health centres is sometimes considerably worse with CFR among envenomed patients as high as 60% at some centres (unpublished data).

In two earlier studies envenomation by Papuan taipans *Oxyuranus scutellatus canni*, accounted for 83.2% and 89.0% of snakebite victims admitted to PMGH respectively[1,2]. This species is widely distributed across southern PNG and is the most medically important snake in the country[1-6]. Bites are characterized by rapid development of facial and bulbar paralysis typically in conjunction with coagulopathy, and untreated leads to respiratory failure, cyanosis and death[7-11]. Victims may not respond well to treatment with current antivenoms especially if administration is delayed, and this influences mortality[4,7,9].

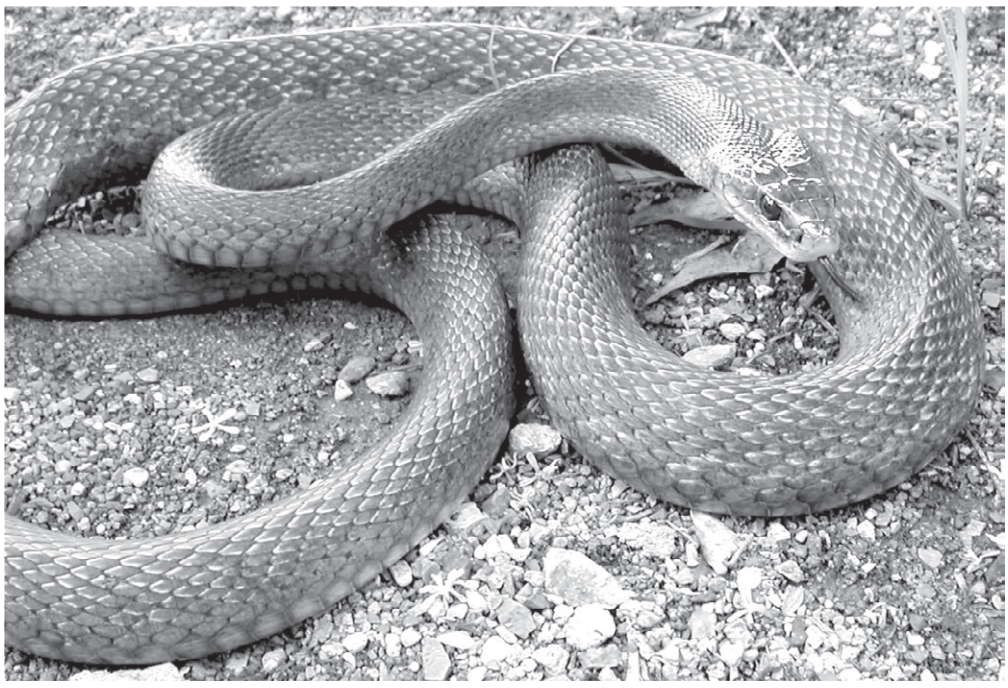

FIGURE 1 PAPUAN TAIPAN *Oxyuranus scutellatus canni* FROM MOREGUINA, CENTRAL PROVINCE, PAPUA NEW GUINEA

PAPUAN TAIPAN (*Oxyuranus scutellatus canni*) ENVENOMATION IN RURAL PAPUA NEW GUINEA

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Oxyuranus scutellatus canni are large (to 3.5 metres) active, diurnal snakes that are frequently encountered by man. They are common inhabitants of village gardens and settlement areas. Base dorsal colour varies from reddish-brown to almost black, but virtually all specimens have a characteristic orange-red vertebral stripe along the backbone (*Figure 1*)[5,6]. A diagnosis of *Oxyuranus scutellatus canni* envenoming should be suspected in all cases where this feature is described, especially if early coagulopathy is evident.

Antivenom is often in critically short supply throughout PNG, and combined with the chronic decline of health care facilities in rural communities; limited training and expertise of health workers; extremely poor transport infrastructure; and communications difficulties, the prognosis for victims of serious envenoming snakebite in remote and rural areas of PNG is generally poor[1-4,12-13]. This paper reports cases of *Oxyuranus scutellatus canni* bites managed in rural health centres, and illustrates the importance of early recognition of clinical indicators of envenoming, careful monitoring of symptoms, and prompt administration of appropriate antivenom in reducing mortality.

METHODS

Representative cases with signs and symptoms consistent with envenoming by *Oxyuranus scutellatus canni* were selected from a large database of cases collected as part of a broad retrospective epidemiological study of snakebite at 14 rural health centres throughout the Central, Gulf and Western Provinces of PNG (PNG MRAC Approval 01.09; JCU HESC H1239/2001). Actual location details have been withheld to protect the anonymity of patients and their families.

CASE REPORTS

Case #1

A woman (18) presented at a rural Health Sub-Centre at 10.45 pm after a snakebite some time earlier. She complained of headache, abdominal pain and vomiting. Bilateral ptosis, tachycardia (120 bpm), drowsiness and limb weakness were noted. Blood pressure (BP) was 100/80 mmHg, with respiration rate (RR) of 40 per minute. An IV was established, tetanus prophylaxis administered and the patient referred by road to Kerema Hospital for further treatment. On arrival at Kerema Hospital at 10.15 am the following morning she was cyanosed (fingertips, toes and lips), gasping for breath, with no radial pulse and no detectable blood pressure. Heart rate (HR) was 56 bpm, pupils were fixed and dilated and there was no response to spoken commands. Suction was used to clear the airway and oxygen administered at 2L/min. Administration of an ampoule of CSL polyvalent antivenom was attempted at 10.25 am, but no vital signs were present and death was pronounced at 10.40 am.

Case #2

A woman (52) presented at a rural Health Sub-Centre at 12.45 pm. She had seen a bandicoot running through grass in front of her, and a "*long, pale black snake*" chasing it had bitten her on the leg. Two bleeding puncture marks were found. A standard 20 minute whole blood clotting time (20WBCT) test at 1.25 pm clotted within 15 minutes. An intravenous line was established and the patient placed under observation. At 9.30 pm bilateral ptosis, dysarthria and diplopia were noted and she was premedicated with 25 mg IV phenergan and 0.25 ml SC adrenaline. Tachycardia (128 bpm) and bulbar paralysis developed, and RR dropped to 13 per minute. Although drowsy, she spat out bloodstained saliva when roused. Infusion of one ampoule of CSL polyvalent antivenom was commenced at 9.45 pm and she was referred by road to a larger health centre. On arrival at 2.30 am, pronounced bilateral ptosis, diplopia, bulbar paralysis and conspicuous bleeding from the gums were observed. Tachycardia (116 bpm) persisted, with BP 130/90, and RR of 24 per minute. She was unresponsive to verbal commands and became comatose. Further premedication was followed by two ampoules of CSL polyvalent antivenom, commencing at 3.00 am. Suction was used to clear airways. At 4.50 am she stopped breathing with no discernible peripheral pulse or heart beat, fixed and dilated pupils, and cold extremities. Death was pronounced at this time.

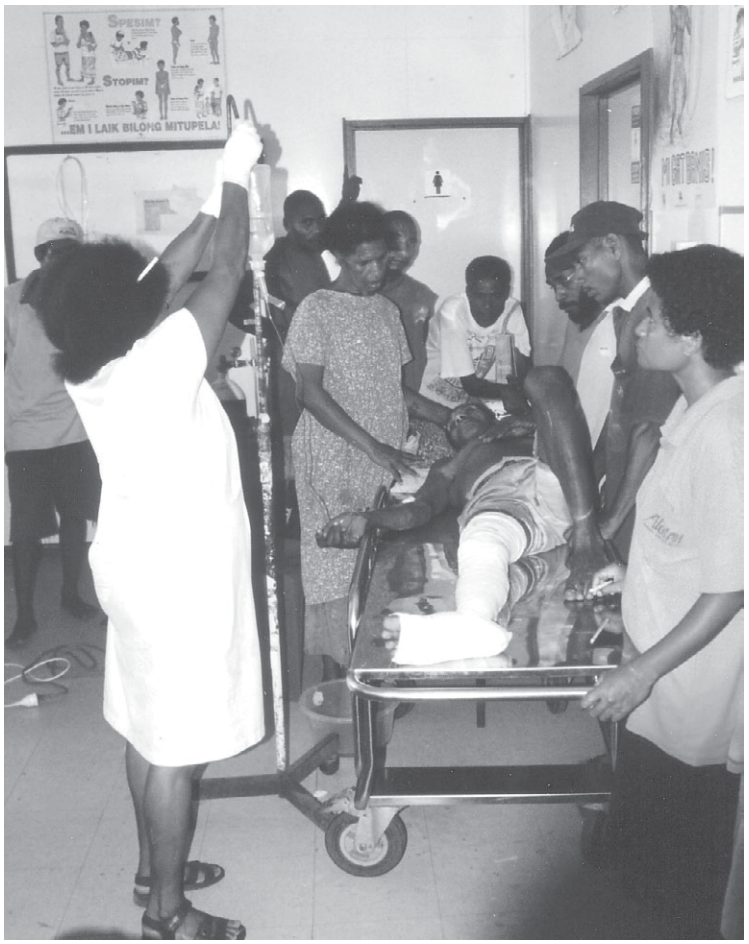

FIGURE 2 FAMILY MEMBERS SURROUNDING MALE (42) PATIENT BEING TREATED FOLLOWING ENVENOMATION BY A PAPUAN TAIPAN *Oxyuranus scutellatus canni* (CASE #3)

Case #3

A male farmer (42) was bitten twice on the left big toe by a *"large black snake with a red stripe on the back"* while walking along a remote rural road at about 1.00 pm. The patient walked several kilometres to a health centre. He was admitted at 3.40 pm, stating that he had vomited blood, had abdominal pain and headache. Four puncture wounds were evident, and the whole leg was wrapped with a firm compression bandage. At 4.00 pm the HR was 88 bpm, RR 32 per minute, and BP of 110/90 mm/Hg. Lymphadenopathy was detected by palpation. Blood taken for a 20WBCT would not clot and he continued to vomit small quantities of bloodstained material.

After standard premedication infusion of one ampoule of CSL polyvalent antivenom diluted in 450 ml normal saline (20 drops/min) commenced at 4.30 pm (*Figure 2*). At 5.00 pm the HR was 92 bpm; BP 90/70 mm/Hg; temperature 38.5°C; and he complained of chills and further abdominal pain. Vomiting of bloodstained liquid continued. At 5.30 pm the BP was 81/69 mm/Hg and on UHF radio advice from physicians at PMGH antivenom infusion was suspended and replaced with 1000 ml Hartmanns solution. Blood taken for a 20WBCT at 6.00 pm failed to clot. HR was 80 bpm; BP 84/70 mm/Hg; temperature 39.0°C; and RR of 32 per minute. An IDC was inserted but only 20 ml of urine was obtained. At 7.00 pm the HR was 96 bpm; BP 90/70mm/Hg; temperature 38.9°C; and RR 36 per minute. 200 mg IV hydrocortisone was given and antivenom infusion reinstated concurrently with the Hartmanns solution.

No further antivenom was available and extensive efforts to secure transport to transfer the patient to PMGH failed. Heavy rainfall that evening increased the chances of the road being impassable in any event. Hourly observations with an emphasis on evaluating the patient for the development of any signs of neurotoxicity were taken throughout the night, and the 20WBCT was repeated at 2 hour intervals until midnight and then again at 6.00 am the next morning. The compression bandage was removed from the upper leg at 1.00 am, but the lower limb was left firmly bandaged until 9.00 am. Blood remained incoagulable at 8.00 pm and 10.00 pm, but clotted within 11 minutes

at midnight. At 1.00 am the patient was moved to the Ward, and by 6.00 am his fever had subsided to 37.0°C, HR 60 bpm; BP 90/70 mm/Hg; RR of 24 per minute. Blood taken for a 20WBCT at 6.00 am clotted in less than 5 minutes. At 7.00 am he was sitting up in bed proclaiming himself well, asking for food, and wanting to be allowed to return home with family members. Despite slightly hazy vision at distance there were no signs of neurotoxicity. He was discharged home later that afternoon.

Case #4

A man (50) bitten twice on the right foot by a "*Papuan black*", vomited at home, and was admitted to the health centre with incoagulable blood (20WBCT: >15 mins), lymphadenopathy, ptosis, dysarthria, dysphagia and dsypnoea. A compression bandage was applied and after premedication with 25 mg promethazine and 0.5 ml adrenaline he received one ampoule of CSL Black Snake Antivenom diluted in 500 mls 0.9% NaCl. Ptosis, dysphagia, dyspnoea and bulbar paralysis worsened and he was referred to PMGH but died during transport due to asphyxiation.

Case #5

A boy (12) bitten by a "*Papuan black*" on the right leg at 3.30 pm presented at a rural Health Sub-Centre having vomited once, and had a clotting time of 14 minutes by 20WBCT. At 9.00 pm he developed abdominal pain, ptosis and vomited (twice), and was referred to another local health centre due to a lack of transport to take him to PMGH. At the second health centre ptosis, diplopia, dysarthria, dysphagia and excessive pooling of saliva in the pharynx were noted. He was premedicated and given one ampoule of CSL Black Snake Antivenom, but died during subsequent transport by ambulance to PMGH.

Case #6

A teenage boy (16) presented at a Health Sub-Centre at 7.50 pm with a history of snakebite, but with no signs or symptoms. A local traditional remedy ("blackstone") was applied and the patient sent home. At 6.00 am the following morning he was brought back to the Health Sub-Centre with lymphadenopathy, ptosis, diplopia, dysarthria, dysphagia, bulbar paralysis, dyspnoea and incoagulable blood with bleeding from the bite site. He was referred to a larger Health Centre where 0.5 ml adrenaline, 25 mg promethazine and one ampoule of CSL Death Adder Antivenom were infused, however he died during transport to PMGH.

Case #7

A man (26) was found unconscious in the bush and was taken to a rural Health Sub-Centre at 2.00 pm. He was bleeding from the nose and mouth, had pronounced bilateral ptosis, diplopia, dyspnoea, dysphagia and peripheral limb weakness. A 4.5% dextrose infusion was established and he was referred to a larger health centre for further treatment. On admission to this centre at 4.00 pm he was febrile (39.5°C) with HR of 186 bpm and laboured RR of 38 per minute. Blood pressure was not measured. Blood taken for a 20WBCT was incoagulable. Ptosis, dysphagia, and dyspnoea along with bleeding from the bite site, and vomiting of blood stained emesis were noted. 25 mg of IV promethazine and 0.25 ml SC adrenaline followed with one ampoule of CSL polyvalent antivenom, 2 ml IV Crystapen and 0.5 ml IM tetanus toxoid produced no improvement, and he died at 4.55 pm.

Case #8

A mother (37) of five was bitten on the right calf by a large "*blacksnake*" at approximately 9.00 am while working in the garden. She walked 3-4 kilometres to the main road but it was not until late afternoon that her husband was finally able to persuade a motorist to take them to a health centre 30 kilometres away. On arrival at 7.20 pm she complained of headache and backache and had a HR of 92 bpm and RR of 24 per minute. A firm compression bandage was applied to the bitten limb. Blood taken for a 20WBCT failed to clot and ptosis, diplopia, dysarthria, dysphagia and dyspnoea were present. 0.25 ml SC adrenaline, 25 mg IV promethazine, 0.5 ml Crystapen, 0.5 ml tetanus toxoid and one ampoule of CSL polyvalent antivenom were administered. At 9.00 pm she was semi-conscious with HR of 98 bpm, BP of 130/90 mmHg and RR of 20 per minute. She became cyanotic and died of asphyxiation at 12.30 am.

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9
Tender lymph nodes			✓	✓		✓			✓
Abdominal pain	✓		✓		✓				
Headache	✓	✓	✓					✓	
Vomiting	✓		✓	✓	✓				
20WBCT > 12 Mins		✓	✓	✓	✓		✓	✓	✓
Bleeding gums/nose		✓					✓		
Bleeding bite site		✓	✓			✓	✓		
Bloodstained vomit or sputum			✓				✓		
Tachycardia	✓	✓					✓	✓	✓
Ptosis	✓	✓		✓	✓	✓	✓	✓	✓
Diplopia		✓			✓	✓	✓	✓	
Dysarthria		✓		✓	✓	✓		✓	✓
Dysphagia	✓	✓		✓	✓	✓	✓	✓	✓
Dyspnoea	✓			✓			✓	✓	
Bulbar Paralysis	✓	✓		✓	✓	✓			
Limb Weakness	✓						✓		
Coma		✓						✓	
Death	✓	✓		✓	✓	✓	✓	✓	✓
Antivenom	PLY	PLY	PLY	BS	BS	DA	PLY	PLY	-
Time given after bite	>12 hrs	> 9 hrs	3 hrs	-	> 6 hrs	> 10 hrs	> 5 hrs	> 10 hrs	-

TABLE 1 SIGNS AND SYMPTOMS OF ENVENOMING IN NINE PATIENTS SUSPECTED OF HAVING BEEN BITTEN BY PAPUAN TAIPANS (*Oxyuranus scutellatus canni*)

Case #9

A girl (10) bitten by an unidentified snake under a village house was admitted to a rural Health Centre at 6.30 pm. She was afebrile with tachycardia (120 bpm), RR of 24 per minute and bilateral ptosis. Blood taken for a 20WBCT failed to clot. No antivenom was available and arrangements were made for transfer to PMGH. At 8.15 pm she was febrile (38.5°C) with HR of 103 bpm, RR of 20 per minute and BP of 90/50 mmHg. Bilateral ptosis, dysarthria, dysphagia and lymphadenopathy were noted. At 8.35 pm the HR was 172 bpm with RR of 30 per minute. The patient died during transfer to PMGH.

DISCUSSION

Rural Health Centres in PNG operate under basic conditions. Many lack reliable power, and only some have solar or gas powered vaccine refrigerators. Medical facilities and equipment are limited and few have either the equipment or training for the resuscitation or ventilation of patients. Most routine pathology functions are unavailable. They are however staffed by dedicated health workers who accept these limitations and consistently endeavour to provide their communities with the best possible medical services. Most are supervised by Health Extension Officers, and while these personnel are not physicians they quickly acquire extensive practical experience in managing medical emergencies.

For rural health workers, snakebite is a serious medical emergency. Eight of the nine cases presented here were fatal. Reported signs and symptoms support the involvement of *Oxyuranus scutellatus canni* as the biting species (*Table 1*). Administration of polyvalent antivenom was delayed by more than five hours in four fatal cases, and three further deaths resulted following administration of other CSL antivenom products. A further death occurred where no antivenom was available. In two cases the administration of antivenom was delayed by more than six hours despite clear evidence of incoagulable blood.

Administering the correct antivenom is crucial. CSL Black Snake Antivenom is purchased in large quantities by the PNG Department of Health due in large part to the widespread perception that most snakebites are caused by "*Papuan blacks*". Medical evidence shows that the Papuan black snake (*Pseudechis papuanus*) is responsible for very few cases of snakebites in Central Province[1], and extensive fieldwork throughout southern PNG over the past decade by the senior author and others has only located a few specimens in Western Province. By comparison *Oxyuranus scutellatus canni* are extremely abundant. CSL Black Snake and CSL Death Adder antivenoms are ineffective in treating bites by *Oxyuranus scutellatus canni*. Health workers should be trained to look for and recognise coagulopathy as an indicator for administration of polyvalent antivenom and the acquisition of CSL Black Snake antivenom discontinued until such time as cost effective bedside diagnostic tests become available. CSL Death Adder antivenom should not be used in cases where coagulopathy exists. CSL polyvalent antivenom should be used in all cases of envenomation where the identification of the snake is unconfirmed.

One patient in this series recovered after timely administration of appropriate polyvalent antivenom. Lymphadenopathy, coagulopathy and presumptive identification of *Oxyuranus scutellatus canni* were sufficient to justify immediate antivenom administration in the absence of neurotoxicity, and this decision may have resulted in the rapid resolution of envenoming that was observed. Hypotension observed in this patient may have been due to an early reaction to the polyvalent antivenom however venom from a related species, *Oxyuranus microlepidotus* has been shown to produce hypotension possibly due to endothelium-independent vasodilation[21-22]. Pressure immobilization was also used in this case, and may have delayed absorption of some venom from the bite site, but as the patient walked some distance to obtain medical assistance the benefits are unquantifiable. Compression bandages were used in only three cases and in all instances only after the patient arrived at the health centre. There was no data given on the use of other forms of first aid prior to presentation. Public education regarding first aid for snakebite should be a public health priority given the costs of snakebite to the PNG health system.

Standard practice in many health centres in southern PNG is to wait for development of neurotoxicity before giving antivenom. This may contribute to the poor outcomes seen in *Oxyuranus scutellatus canni* envenomation. A clinically important phospholipase A_2 (taipoxin) in taipan venom produces nerve cell degeneration within as little as one hour post-exposure which may be pronounced after 3-6 hours, affecting up to 70% of nerve terminals within 24 hours, with subsequent reinnervation taking up to 28 days[14]. Previous authors have observed that antivenom poorly reversed the neurotoxic effects of *Oxyuranus scutellatus canni* venom[1,4,9,15-20], and in each of the four fatal cases treated with polyvalent antivenom reported here, neurotoxicity was pronounced prior to administration. In one case the patient received a total of three ampoules of polyvalent antivenom.

Health workers are acutely aware that their decision-making can have very serious consequences for envenomed patients. There are many occasions where despite the best efforts of health workers circumstances beyond their control, such as lack of appropriate antivenom, late patient presentation, equipment deficiencies, lack of transportation and impassable roads conspire to produce poor outcomes. With so many factors weighed against them it is imperative that a consistently appropriate strategy be developed for managing envenomed patients. As can be seen in the cases presented here, delays, errors and inconsistencies in treatment can have lethal consequences.

The cases presented demonstrate the need to develop patient management protocols appropriate to remote settings that emphasize early recognition of systemic symptoms, widespread use of appropriate first aid measures to slow or limit venom absorption, consistent and accurate patient monitoring, and ideally, the earliest possible administration of appropriate antivenom. The use of simple diagnostic markers such as 20WBCT in monitoring patient response to treatment should be considered. A further strategy involving use of manual ventilation using anaesthetic bags by relays of family members requires investigation. Where health resources are limited, simple techniques and strategies that have the potential to offer real improvements in patient outcomes after envenomation should be rigorously explored.

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REFERENCES

1. LALLOO DG, TREVETT AJ, SAWERI A, *et al.* The epidemiology of snakebite in Central Province and National Capital District, Papua New Guinea. *Trans Roy Soc Trop Med Hyg.* 1995 89:178-182.
2. CURRIE BJ, SUTHERLAND SK, HUDSON BJ, SMITH AM An epidemiological study of snake bite envenomation in Papua New Guinea. *MJA.* 1991 154(4): 266-8.
3. WILLIAMS DJ, KEVAU IH, HIAWALYER GW, *et al.* Analysis of Intensive Care Unit admissions for treatment of serious snakebite at Port Moresby General Hospital. 6th Asia Pacific IST Congress, Cairns Australia 2002.
4. TREVETT AJ, LALLOO DG, NWOKOLO NC, *et al.* The efficacy of antivenom in the treatment of bites by the Papuan taipan (*Oxyuranus scutellatus canni*) *Trans Roy Soc Trop Med Hyg.* 1995 89:322-25.
5. SLATER KR On the New Guinea taipan *Mem Nat Mus* Melb. 1956 30(1):201-5.
6. O'SHEA MT A guide to the snakes of Papua New Guinea. Port Moresby (PNG). Independent Publishing, 1996.
7. CAMPBELL CH. The taipan (*Oxyuranus scutellatus*) and the effect of its bite. *MJA.*. 1967 1(15):735-9
8. LALLOO DG, HUTTON R, BLACK J, *et al.* Mechanisms of coagulopathy following envenoming by the Papua New Guinean Taipan. *Toxicon.* 1993 31(8):937.
9. LALLOO DG, TREVETT AJ, KORINIHONA A, *et al.* Snake bites by the Papuan taipan (*Oxyuranus scutellatus canni*): Paralysis, hemostatic and electrocardiographic abnormalities, and effects of antivenom *Am J Trop Med Hyg* 1995 52(6):525-31.
10. LALLOO DG, TREVETT AJ, NWOKOLO N, *et al.* Electrocardiographic abnormalities in patients bitten by taipans (*Oxyuranus scutellatus canni*) and other elapid snakes in Papua New Guinea *Trans Roy Soc Trop Med Hyg.* 1997 91(1):53-6.
11. TREVETT AJ, LALLOO DG, NWOKOLO NC, *et al.* Electrophysiological findings in patients envenomed following the bite of a Papuan taipan (*Oxyuranus scutellatus canni*). *Trans R Soc Trop Med Hyg* 1995 Jul-Aug 89(4):415-7
12. CHENG AC, RATCLIFF A, ADHIKARI P. Snake anti-venom in Papua New Guinea. *Fellowship Affairs* 2000 19:26.
13. CHENG AC, WINKEL KD. Snakebite and antivenoms in the Asia-Pacific: *wokabaut wantaim*, *raka hebou* ("walking together") *MJA* 2001 175:648-651
14. HARRIS JB, GRUBB BD, MALTIN CA, DIXON R The neurotoxicity of the venom phospholipases A2, notexin and taipoxin. *Exp Neurol.* 2000 161:517-526.
15. CAMPBELL CH, YOUNG LN. The symptomatology, clinical course and successful treatment of Papuan elapine snake envenomation. *MJA.* 1961 478-486.
16. CAMPBELL CH. Antivenene in the treatment of Australian and Papuan snake bite. *MJA.* 1967 2(3):106-10.
17. CAMPBELL CH. Clinical aspects of snake bite in the Pacific area. *Toxicon* 1969 7(1):25-8.
18. TREVETT AJ, LALLOO DG, NWOKOLO NC, *et al.* Analysis of referral letters to assess the management of poisonous snake bite in rural Papua New Guinea. *Trans Roy Soc Trop Med Hyg.* 1994 88:572-74.
19. TREVETT AJ, LALLOO DG, NWOKOLO NC, *et al.* Failure of 3,4-diaminopyridine and edrophonium to produce significant clinical benefit in neurotoxicity following the bite of Papuan taipan (*Oxyuranus scutellatus canni*) *Trans Roy Soc Trop Med & Hyg.* 1995 89:444-46.
20. WARRELL DA, LALLOO DG, TREVETT AJ, *et al.* Snake bites by taipans in Papua New Guinea: Severe neurotoxicity and haemostatic dysfunction; limited efficacy of specific antivenom. *Toxicon.* 1992 31(1):264.
21. BELL KL, SUTHERLAND SK, HODGSON WC Some pharmacological studies of venomfrom the inland taipan (*Oxyuranus microlepidotus*) *Toxicon* 1998 36(1):63-74.
22. BELL KL, KEMP BK, MCPHERSON GA, HODGSON WC The smooth muscle relaxant effects of venom from the inland taipan (*Oxyuranus microlepidotus*) *Toxicon* 1999 37(1):229-231.