

## Snake Bite in Papua New Guinea

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### SUMMARY

Snake bite antivenom prices have risen considerably. In order to optimize the management of snake bite in Papua New Guinea answers to a number of questions are required, and education of health workers and the public needs emphasis.

The venomous snakes of Papua New Guinea (PNG) and Australia belong to the family Hydrophiidae. The land-dwelling members of this family are generally restricted to Australasia, but they are related to the family Elapidae, which includes the Asian cobras. There are 23 species of venomous land snakes in PNG, but only 7 of these are considered dangerous to humans (1). The three most important venomous snakes are the death adder (*Acanthophis antarcticus*), which is widely distributed throughout mainland PNG, and the taipan (*Oxyuranus scutellatus*) and Papuan black snake (*Pseudechis papuanus*), which both seem restricted to the Papuan region. The king brown snake (*Pseudechis australis*) and the eastern brown snake (*Pseudonaja textilis*), while common in Australia, are rare in PNG. The whip snake (*Demansia papuensis*) is thought to be dangerous if a large specimen. The small-eyed snake or white snake (*Micropechis ikaheka*) is found in forest areas, and has reportedly caused a number of deaths in Madang Province including Karkar Island (1,2). Extensive studies by Campbell in the 1960s form the basis of our current understanding of the clinical manifestations and treatment of snake bite in PNG (3-7). However, some areas of uncertainty remain, and the present Health Department expenditure on antivenom is a stimulus for further studies. New immunodiagnostic

techniques will assist in such studies, and should help us better understand the effects of urbanization and deforestation on patterns of snake bite in PNG. The possibility of local antivenom production may be worth future consideration.

The price of an ampoule of polyvalent antivenom has risen 73% from May 1985 to January 1987: Aus \$300 (K212) to Aus \$580 (K367). Over the same twenty-month period black snake antivenom has risen 92%, death adder 88% and taipan 104%. Table 1 shows the antivenom issued within PNG in 1986. At January 1987 prices the cost of the 781 total ampoules for the year would be K243,000.

While in the New Guinea region death adder envenoming probably predominates, the situation in the Papuan area is uncertain. Campbell stated that the Papuan black snake was the commonest venomous snake encountered in Papua (3). However, herpetologists state that Papuan black snakes are now extremely rare. One explanation is that the frog-eating Papuan blacks have succumbed to the introduced venomous cane toad *Bufo marinus*, which secretes poison from neck glands into the snake's mouth (1). Apparently taipans are not partial to frogs. Papuan black snake and taipan bites both cause neurotoxicity. From cases referred to Port Moresby General

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TABLE 1

ANTIVENOMS ISSUED 1986<sup>1</sup>

Centre/Province	Polyvalent	Death adder	Black snake	Taipan	Total
Port Moresby NCD	135	—	8	—	143
Central	91	19	39	16	165
Gulf	37	26	15	8	86
Milne Bay	11	12	10	2	35
Western	45	66	25	20	156
Lae*	30	40	—	—	70
Madang	40	60	1	—	101
Mt Hagen**	—	5	—	—	5
Wewak	—	20	—	—	20
Rabaul	—	—	—	—	—
<b>Total</b>	<b>389</b>	<b>248</b>	<b>98</b>	<b>46</b>	<b>781</b>
Cost per ampoule (Kina) at 1987 prices †	367	231	272	352	
<b>Total cost (Kina) at 1987 prices †</b>	<b>142,763</b>	<b>57,288</b>	<b>26,656</b>	<b>16,192</b>	<b>242,899</b>

<sup>1</sup> Figures provided by Mr M. K. Lee, Supply Officer, Area Medical Store, Konedobu

\* Lae supplies Northern, Morobe, Eastern Highlands and Chimbu Provinces

\*\*Mt Hagen supplies Enga, Southern Highlands and Western Highlands Provinces

† Estimated for exchange rate of Aus \$1.00 = K0.63

Hospital it seems likely that taipan bites are sometimes being misdiagnosed as Papuan black bites and being treated initially with monovalent black snake antivenom. The black snake antivenom is ineffective in such cases, and the signs of envenoming progress until polyvalent or taipan antivenom is given. In some areas the villagers do not distinguish between the two species, but call all such snakes black snakes. Some villagers say they are the different sexes of the same species (8). Careful questioning may elicit a description of the taipan's characteristic orange or red streak on the back, and thus avoid the ineffective, expensive and possibly fatal use of black snake antivenom. However, in the majority of envenomed children there is no description of the snake (9). While the three most important snakes in the Papuan region are neurotoxic, the death adder venom does not

have the procoagulant action of the venom of the taipan (4,5). Although the death adder antivenom is considerably cheaper than the polyvalent antivenom, it is at present unsafe to assume that neurological signs but normal clotting time mean a death adder bite. Prospective studies with venom detection assays may help resolve the question of the prevalence of the various snakes' bites, and help define clinical criteria for the use of the cheaper monovalent antivenoms. The role of venom detection kits is also worth consideration (10).

A further area of uncertainty is the appropriate dose of antivenom to administer. One ampoule of antivenom is said to neutralize the average amount of venom injected per bite. With multiple bites, and with severe signs of envenoming (especially neurotoxicity) the standard treatment is to use two ampoules as

initial treatment (11,12). While expensive, this precaution against inadequate neutralizing antivenom is especially important in centres where ventilatory assistance is not possible. However, the adult standard treatment manual says two ampoules should be given "if signs of envenomation are present when the patient is first seen", and 2 further ampoules "if signs of envenomation do not improve 4 hours after the above treatment" (13). While the clotting abnormality of taipan venom is generally reversed with adequate antivenom, the neurotoxicity (pre-synaptic toxins) is probably not reversed but just stopped from progressing (4). With the death adder (post-synaptic toxins) there seems to be a reversal of the neurotoxicity (5). It therefore seems that progression of neurological signs, rather than a lack of reversal, is the indicator of inadequate antivenom administration, especially in taipan bites. We were recently referred an adult patient who had a total of 12 ampoules of polyvalent antivenom (K4408) because the neurological signs were not reversing. Further education of health workers regarding the effects of venoms and the expectations of antivenom therapy should help reduce excessive use of antivenoms. In addition, current prospective studies of snake bite should help better define the indications for use of more than one ampoule of antivenom on admission or subsequently. How often do signs progress despite one ampoule of appropriate antivenom? In taipan bites can the neurological signs progress despite the reversal of the clotting time abnormality?

An important area for education of both health workers and the public is the first-aid management of snake bite. The beneficial effect of the pressure-immobilization technique in Australasian snake bite is well documented (14). The delay in venom absorption with this method can be life saving, and it is now recommended in the PNG paediatric textbooks, but not in the smaller adult or paediatric standard treatment manuals. However, while it is now extensively used in Australia this method of first aid remains virtually unknown in PNG and therefore requires further dissemination. At present around one-third of patients admitted to Port Moresby General Hospital have been treated with local or extensive lacer-

ations, and some have had tourniquets applied to the affected limb — both these dangerous measures have been discredited and need vigorous discouragement.

Finally, prospective studies should help clarify a number of questions concerning clinical aspects of snake bite in PNG. Rhabdomyolysis has been described with Australian taipan bite, but what is its significance in PNG snake bites (15)? What is the relevance of electrocardiograph abnormalities seen in some cases? What is the role of anticholinesterase therapy such as neostigmine (16)? What are the mechanisms involved in the bleeding disturbances? What is the mechanism and diagnostic significance of local inflammation at the bite site? How important are reactions to antivenom, what are the mechanisms involved and can the method of administration be simplified to intravenous injections rather than infusions (17,18)?

In conclusion it is hoped that ongoing studies of snake bite in PNG, combined with continuing education of health workers and the public, will help optimize the management of snake bite and safely minimize the use of expensive antivenoms.

#### *Notes added in proof*

The cost of antivenoms has recently increased further. In March 1988 the cost of an ampoule of polyvalent antivenom was Aus \$879, an increase of 193% from May 1985. At the 1988 prices the total cost of antivenoms issued in 1986 would be K384,168, an increase of 58% over the cost at 1987 prices.

Further information on the small-eyed snake in Papua New Guinea is provided in an article by B.J. Hudson in this issue of the journal (19).

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