The Highly and Potentially Dangerous Elapids of Papua New Guinea

The identification, ecology and distribution of venomous species, and the clinical diagnosis and treatment of snakebite

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Introduction

The Second Largest Island in the World

Papua New Guinea constitutes the eastern half of the World's second largest island and it has, in the past, been governed by German, British, Japanese and Australian administrations. The western half of the island, known today as West Papua or Irian Jaya, was formerly a Dutch colony but now constitutes the largest territory in the Republic of Indonesia. Papua New Guinea received its full Independence in 1975 and now comprises nineteen provinces, incorporating several large archipelagoes to the north and east of the mainland, and the National Capital District surrounding the capital, Port Moresby.

The popular view of mainland Papua New Guinea is of a region of impenetrable, and relatively unexplored, montane rainforests clinging precariously to steep, unscalable ridges and peaks with rushing, torrential whitewater rivers gouging their way through the rock and vegetation as they race towards the coasts. Since much of central Papua New Guinea is comprised of a soaring montane backbone, which rises to over 4000m (13,130ft), this description is accurate when applied to the Highland Provinces but large areas of both northern and southern Papua are covered by lowland rainforests, monsoon forests, and vast arid Eucalyptus savanna woodlands which bear more resemblance to the drier areas of neighbouring Australia. There are also extensive freshwater swamps and coastal and estuarine mangrove entanglements. Enormous areas of Papua New Guinea are extremely low-lying and flat, especially the huge expanses of Western Province between the Fly River and the Irian Jaya border which barely rise more than 200m above sea-level. Because this region is subject to extensive seasonal flooding it was necessary to site the administrative centre on the offshore island of Daru. Further east in Central and Milne Bay Provinces the mountains rise almost straight from the sea. There are two major river systems; the Fly-Strickland in the southwest and the Sepik in the northwest.
There are several large archipelagoes to the north and east of the Papua New Guinea mainland the most noteworthy being the large island Provinces of East and West New Britain and New Ireland, the Admiralty Island Group in Manus Province to the north and the Trobriand, D'Entrecasteaux and Louisiade Archipelagoes of Milne Bay Province to the southeast. North Solomons Island Province, containing Bougainville Island, to the far east borders the archipelagoes of the independent Solomon Islands.

The population, comprising mainly Papuans and Melanesians, is believed to number some three million, mostly centred in the Highlands Provinces and the major lowland towns of Port Moresby, Lae, Madang and Rabaul. The country boasts over 700 languages but the main tongues are English, Pidgin, Hiri and Police Motu. Outside the major centres most of the population subsist by slash and burn garden horticulture and hunting and are, therefore, often isolated by many miles from larger villages or main communication routes. Most of the population move about on foot or in small canoes although larger private and Government boats travel along the coasts, between the islands and for considerable distances up the more navigable rivers. An increasing number of settlements are becoming linked by roads, especially around Port Moresby, Madang, Lae, Wewak, Mt Hagen and Popondotta. However, most of these road systems are not interlinked and the major connections between isolated communities remains the extensive air network. Unfortunately international air travel in Papua is fairly expensive.

The climate is equatorial monsoon with distinct wet and dry seasons but the variation in relief brings about localised differences in climate with the central mountainous backbone receiving 225-575cm of rainfall annually compared to the southern coast which lies in a rainshadow zone and receives only 100cm annually and the majority of low-lying Western Province which receives 150cm of rain per year, hence the considerable variations in vegetational cover. Lowland temperatures vary from 70°F to 90°F (21°C - 32.2°C) and relative humidity varies from 70-90%.

A Unique Fauna.

The fauna of New Guinea is ostensibly Australasian sharing many species, genera and families with that huge continent, but numerous taxa of Indonesian or Pacific origin are also represented here together with a unique collection of endemic species and genera. There are no monkeys, no native jungle cats and no vultures. Instead, fantastic birds of paradise, tree kangaroos, flightless cassowaries and spiny egg-laying echidnas are found. Even amongst the herpetofauna several taxa are conspicuous by their absence, namely the venomous solenoglyphous family Viperidae, and the non-venomous/mildly venomous family Colubridae, which are so diverse elsewhere in the tropics, are poorly represented. Other groups have diversified and speciated greatly, in particular the skink lizards, family Scincidae, the pythons, subfamily Pythoninae, and the venomous proteroglyphous family Elapidae. This study concerns this latter family as, although it is well known that Australia is the only country in the world with more species of venomous than non-venomous snakes, it is often not realised that the most venomous species are other than snakes.

Snakebite As A Hazard In New Guinea

Snakebite is mainly as a result of bites from venomous coral snakes, but in Australia it is as a result of bites from the coral snakes. When it is realised that the Australian snakebites, both fatal and non-fatal, are generally from small-eyed coral snakes, it will be seen that many of the small-eyed coral snakes are much more venomous than the species found throughout much of Papua New Guinea. In New Guinea, for example, the deadly small-eyed snake is the Calamaria virgifera, found in the Registro, the central highlands of the country, and the New Guinea small-eyed snake, Calamaria virgifera nigricollis, found in the central highlands of the country, and the New Guinea small-eyed snake, Calamaria virgifera nigricollis, found throughout much of Papua New Guinea. It is now realised that many of the dangerous species of snakes found in Papua New Guinea are much more venomous than the species found throughout much of Papua New Guinea. It is now realised that many of the dangerous species of snakes found in Papua New Guinea are much more venomous than the species found throughout much of Papua New Guinea. It is now realised that many of the dangerous species of snakes found in Papua New Guinea are much more venomous than the species found throughout much of Papua New Guinea. It is now realised that many of the dangerous species of snakes found in Papua New Guinea are much more venomous than the species found throughout much of Papua New Guinea.
and east of the Papua Province and the large island Provinces of the Admiralty Island Group in the north, D'Entrecasteaux and to the southeast, North Island, to the far east of Rabaul. The country is bordered by English, Pidgin, Hiri and population subsist by slash and therefore, often isolated by navigation routes. Most of the through larger private and between the islands and for an increasing number of around Port Moresby, however, most of these road connections between isolated Fortunately internal air

The sweet and dry seasons but differences in climate with the same return of rainfall annually in the lowland savanna/woodland regions of southern Papua, with the notable exception of the ubiquitous Death adders, *Acanthophis* spp., and the elusive rainforest New Guinea small-eyed snake, *Micropolis ikateka*. Death adders are responsible for many serious snakebites in the Ramu River Valley, Madang Province and the small-eyed snake is the cause of the alarming incidence of snakebite accidents on the north coast of Madang Province and on neighbouring Karkar Island. However, many venomous snakebites occur in low-lying southern localities and most of the documented and medically treated bites take place in or around Port Moresby which possesses both a large resident and itinerant human population and a wide variety of suitable habitats for the four species of dangerous elapids known to occur in coastal Central Province. Whilst certain snakes, it is often not realised that New Guinea shares several of Australia's most venomous species and also boasts a few highly venomous endemic species.

Snakebite As A Hazard In Papua New Guinea.

In the majority of the World's tropical regions all elapids are considered a potential threat to human life, whether they are cobras, mambas, kraits or coral snakes, but in Australia and New Guinea the situation is quite different. When it is realised that the Australasian elapids have diversified and speciated widely, to occupy the vacant niches which are otherwise inhabited by the numerous harmless colubrid species throughout the rest of the world, it can be seen that many of the small, secretive, burrowing species of the genera *Toxicocalamus* and *Uenechis*, with their inoffensive manners, small mouths and weak venom, are no more dangerous to man than the tiny non-venomous or only mildly venomous colubrid species which they mirror ecologically such as the Indo-Chinese *Calamaria* or Neotropical *Tantilla*.

Twenty-six species of terrestrial elapids have been recorded from New Guinea and the archipelagoes to the east, including the Solomon Islands. This constitutes 35% of the total known ophiofauna, excluding sea snakes. Only nine species in six genera are considered to be highly dangerous to man — 12% of the recorded Papuan snake fauna. All of these species are confined to the mainland provinces and they will be dealt with individually in the following sections. However, caution is still advised regarding some of the moderately sized insular elapids, with unknown and, therefore, potentially harmful venom. It should be noted that Kinghorn and Kellaway (1943) reported the death adder (*Acanthophis*) from New Britain but the author is unable to locate any further records and de Haas (1950) omits this locality whilst including Indonesian Torres Strait islands. Papuan insular species to be treated with respect include Muller's snake, *Aspidornorphus muelleri* (max. length 640mm), found throughout much of the mainland and the New Britain and New Ireland Provinces, Hediger's snake, *Parapistocalamus hedigeri* (300mm), *Salomella par* (750mm), and the Banded small-eyed snake, *Laveridgelaps elapoides* (800mm), from Bougainville, North Solomons Provinces, and the Solomon Islands. Manus Province has no terrestrial elapids.

Many of the dangerous elapids are confined to the lowland savanna/woodland regions of southern Papua, with the notable exception of the ubiquitous Death adders, *Acanthophis* spp., and the elusive rainforest New Guinea small-eyed snake, *Micropolis ikateka*. Death adders are responsible for many serious snakebites in the Ramu River Valley, Madang Province and the small-eyed snake is the cause of the alarming incidence of snakebite accidents on the north coast of Madang Province and on neighbouring Karkar Island. However, many venomous snakebites occur in low-lying southern localities and most of the documented and medically treated bites take place in or around Port Moresby which possesses both a large resident and itinerant human population and a wide variety of suitable habitats for the four species of dangerous elapids known to occur in coastal Central Province. Whilst certain
areas of Western Province may accommodate more species of highly venomous snakes the population is more scattered and bites are less likely to come to the attention of the authorities. Before a specific antivenom was produced for the death adder 1906 estimated that the Australian mortality rate from this single species was as high as 50%. Even so, notified deaths due to snakebite in Papua during Campbell's time never exceeded eight per year or 1.7/100 000 population.

During a two month visit to Western Province in 1986 the author received reports of ten 'serious' bush snakebites; two of these were confirmed as venomous snakebites. One terminated fatally, possibly due to a combination of venom and the over-exuberant use of knives by villagers to bleed the venom from the patient. This 'first aid' treatment was prevented on the second occasion by an expedition nurse who intervened on the victim's behalf.

Campbell reported that the Papuan blacksnake, *Pseudechis papuanus*, was the main cause of serious snakebites in southern Papua. This somewhat biased assertion was based on identification of the snake by the victim rather than by a qualified person. The Papuan blacksnake seems to instill an almost mythological dread into the people of the Trans-Fly to the point where almost any dark unicolour snake is called a 'Pap black' by Western Province villagers. The author collected a number of battered Papuan blacksnake cadavers which were really harmless colubrid species; *Amphiesma mairii* or *Dendrelaphis punctulatus*. Some Papuans do not differentiate between the blacksnake and the taipan (*Oxyuranus scutellatus*), although Togo villagers believe they are female and male, respectively of the same species (Parker, 1982). It is, therefore, extremely probable that many envenomations by taipans or whipsnakes (*Demansia spp.*), together with symptomless bites from aggressive non-venomous species, such as the diurnal treesnake *Dendrelaphis punctulatus*, will be blamed on the Papuan blacksnake by rural snakebite victims.

It might be true that the Papuan blacksnake was once more common than it is today and, therefore, more deserved its reputation. However, it now seems likely that the situation has changed and this species is extremely rare, if not extinct, in certain parts of its range. In two months in Western Province the author examined 116 snakes but only saw one *Pseudechis papuanus*. The reason for the probable decline of the blacksnake population is most likely to be the introduction of the cane or marine toad, *Bufo marinus*, from South America, into areas of Papua New Guinea and Australia as a biological crop pest control in the 1930's and 1940's. The threats posed by this toad on resident frog-eating snakes, such as the Papuan blacksnake and most other Australasian elapids, with the notable exception of the taipan, are threefold. First, none of the frogs normally preyed upon by these snakes contain any toxins which could harm the snake but bufonid toads, (naturally absent from Australasia), possess a powerful bufotoxin which is at its most potent in *B. marinus*. There are snakes in the Americas which can cope with these toxins but Papuan elapids die rapidly from their effects. Because these toads have no natural enemies they are able to breed in large numbers and invade new habitats without any control on the snake fauna of Papua and themselves. They eat not only the toad itself but also any other species that may be present. Second, the taipan's situation is mirrored in Queensland where the toad has also been introduced, and the taipan is now at least remaining stable, while the Papuan blacksnake and the taipan in mainland Australia (Shine & Archer, 1975; Shine & Horabrook, 1972).

The highly venomous sea snakes (*Laticauda schistosa* and *Echidna schistosa*) which frequent coastal waters. Most are in Papua except by fishermen hauling crab pots, particularly the highly venomous *E. schistosa*, have been recorded in Madang and Fly Rivers. *E. schistosa* has been removed from their aquatic habitats, and *L. laticaudata*, actually deadly gunmetal banded snakes may be found in mangrove swamps and, although their toxicity of their venom may be dangerous. For the purpose of this study, *L. laticaudata* is confined to the population status of the snakes and the author's study.

Parker (1982) also recorded this aquatic snake from southern Queensland as 'normal' with smooth brown skin, a short cylindrical tail but with the possibility of rapid death within minutes. This
species of highly venomous snakes less likely to come to the victim was produced for the Australian mortality rate from snake bite per year or 1.7/100 000 (1986).

1986 the author received 77 of these were confirmed as toxicity due to a combination of venom to bleed the venom on the second revented on the second victim's behalf.

*Psudechis papuanus,* was the causal agent. This somewhat biased the victim rather than by means to instill an almost to the point where almost Western Province villagers. For example, mairii or *Dendrelaphis* between the blacksnake and venomous blacksnakes, believe they are snakes (Parker, 1982). It is, however, important to consult taipans or other less bites from aggressive species, the *Dendrelaphis* blacksnake by rural snakebite people once more common than the peturbation. However, it now this species is extremely rare, if it exists in Western Province the *Psudechis papuanus.* The population is most likely to be a *B. marinus* from South Australia as a biological crop. This toad on resident and most other Australian islands, are threefold. First, these toads contain any toxins but naturally absent from that. It was potent in *B. marinus* that can cope with these toxins because these toads have no numbers and invade new habitats without any control. The second and third effects of the marine toad on the snake fauna of Papua concern the eating habitats of the toads themselves. They eat not only the frogs that the snakes would have otherwise preyed upon, but they also devour the young snakes. Together with habitat destruction, reclamation of land and cutting of woodland which all affect the status of the snakes and their amphibian prey, blacksnake populations are almost certain to decline. The taipan, *Oxyuranus scutellatus,* however, being a strict mammal-eater is not only relatively unaffected by the increasing populations of *B. marinus,* but destruction of woodland for building is likely to attract both the taipan's rodent prey and, therefore, the snake itself. This situation is mirrored in Queensland where taipan numbers have risen, or at least remained stable, whilst anurophagous species have declined (Covacevich & Archer, 1975; Shine & Covacevich, 1983). Taipans appear to be more common than blacksnakes around Port Moresby and on Daru Island, Western Province, where *B. marinus* is well established. However, the status of the toad and the blacksnake in mainland Western Province would make an interesting study.

The small-eyed snake should also be treated with extreme caution. Its habits and the effects of its venom are little understood. It is probably a much more dangerous species than is currently believed, especially in Madang Province and on nearby Karkar Island where the snake is known as the 'white snake', and only its secretive nocturnal nature prevents more human bites. Seven probable fatalities due to this species have been reported (Blasco and Hornbrook, 1972).

The highly venomous seasnakes are extremely common in the surrounding Pacific Ocean. Approximately twenty species have been reported from Papuan coastal waters. Most are inoffensive, totally marine and rarely encountered, except by fishermen hauling in nets. However, some species of seasnakes, particularly the highly venomous common beaked seasnake *Enhydrina schistosa,* have been recorded as travelling for many miles up the larger tidal rivers. *E. schistosa* has been responsible for several serious bites in the Ramu River, Madang Province (Hudson & Fromm, 1986) and the author collected a specimen believed to be *E. schistosa* sixty kilometres inland on the Oriomo River, Western Province. In addition, whilst most seasnakes are helpless when removed from their aquatic environment, the sea kraits, *Laticauda colubrina* and *L. laticaudata,* actually venture onto land to lay their eggs. These black and gunmetal banded snakes may be encountered on coral or rocky beaches or in mangrove swamps and, although they are generally placid and rarely bite, the toxicity of their venom means that they must be considered potentially dangerous. For the purposes of the study discussion of seasnakes will be confined to the population of *E. schistosa* in the Ramu River System.

Parker (1982), also records the presence of an undescribed species of aquatic snake from southern Trans-Fly, Western Province which is said to be 'normal' with smooth brown or yellow-brown scales, enlarged ventrals and a short cylindrical tail but which possesses venom which is believed to cause death within minutes. This snake is considered to be extremely rare and
HIGH RISK AREA.
Central Province species:
Acanthaphis sp.
Demania sp.
Micropechis ibaiko
Oxyruanus scutellatus
Pseudechis papuanus
Pseudonaja textilis?

IRIAN JAYA

GULF
MOROBE
CENTRAL
NORTHERN
MILNE BAY

Indonesia
Papua New Guinea
Australia
HIGH RISK AREA.
Western Province species:
Acanthophis sp.
Damanina sp.
Micropechis ibakaka ?
Oxyuranus scutellatus
Pseudechis australis ?
Pseudechis papuanus
thought only to inhabit the muddy bottoms of small freshwater streams and sago swamps, occasionally emerging onto dry land to bask. With a maximum length of two metres, this snake is thought to have been responsible for the extremely rapid deaths of three young girls who were bathing in the Ouwe Creek near Wipim. Neither Parker, nor this author, has been able to locate a specimen of this snake despite visits to Wipim and interviews with villagers. However, the Ouwe Creek does feed into the Oriomo River, in which this author located a *Hydrophis*, so the possibility of some form of land-locked sea snake being the cause of the deaths cannot be dismissed.

It should, of course, be noted that not all riverine or estuarine snakes are dangerous sea snakes. Two species of harmless file snakes, *Acrochordus* (family Acrochordidae), and five mildly venomous members of the subfamily Homalopsinae (family Colubridae), (Parker 1982; O'Shea 1986), also inhabit these environments.

### History Of Snakebite In Papua New Guinea.

#### The Statistics of Snakebite.

Much of the important early work on snakebite in southern ‘Papua’ concerns cases of elapine snakebite treated at Port Moresby General Hospital and studied by Campbell (1966; 1967; 1969), during the six years period, October 1959 to November 1965, when he was a general physician. Campbell submitted his exhaustive study on the subject as a thesis towards his degree as a Doctor of Medicine at the University of Sydney in 1969 and also published several important papers based on his findings in *The Medical Journal of Australia, Toxicon* and other journals. Other relevant papers in this area include Price and Campbell’s (1979) study of snakebite admissions to PMGH between 1967 and 1971 and Brian and Vince’s (1987) work on snakebites amongst children, aged 2 to 16, admitted to PMGH between 1981 and 1984. In contrast few papers have appeared regarding snake envenomation in northern ‘New Guinea’ other than Blasco and Hornabrook (1972) and Hudson and Pomat’s (1988) survey of snakebite in Madang Province and admissions to Madang General Hospital between 1977 and 1986. These studies of the clinical signs and symptoms of Papua New Guinean snakebite have been drawn on extensively here as this author is a tropical herpetologist with a primary interest in the ecology, taxonomy, distribution and venoms of snakes from a zoological, rather than a medical, standpoint.

Prior to the Second World War few hospitals existed in Papua and snakebite admissions were quite infrequent. The largest single yearly quota for snakebite admissions was six amongst a total of ninety-one admissions to Port Moresby Hospital during 1906-07. Fatalities also rarely featured in the statistics, although undoubtedly they occurred in rural areas. Campbell reports that the *Annual Report of the Territory of Papua,* (which does not include New Guinea north of the mountainous backbone), records three deaths each in the years 1915-16, 1926-27 and 1934-35 and also occasional reports from *The Papuan Villager* during the late 1920’s and early 1930’s. The statistics obtained following World War Two are equally misleading as the *Annual Reports of Papua* records numerous cases which did not result in envenomation. The same heading: ‘Poisonous snakes’ is used which include other causes of fatalities.

Campbell spent a great deal of time studying the statistics and he was finally able to calculate the annual admission and deaths in Papua New Guinea. It must be remembered first, however, that the reports of deaths are largely restricted to the New Guinea districts, which is why they are not represented in Campbell’s data.

Campbell estimated that 2% of the total of 1000 admissions, annually, to hospitals in the Southern Highlands region, had been caused by snakebites. The fate of admissions in the Territory of New Guinea was not recorded, and the fatality rate amongst admissions was approximately 12%. Campbell also reported a similar death rate of approximately 25% in the Northern Territory, where only 10% of admissions died (Parker 1982). Of the 482 suspected snakebites, which were reported over a six years period, Campbell estimated that 15% showed definite signs of envenomation. The other admitted patients were either non-venomous snakes with no envenomation, or those patients who died before they could be admitted to hospital. Of these cases, 175 are believed to be as a result of non-venomous snakes, and Pomat considered 175 admissions as those with definite signs of envenomation recorded. It is important to note that the available medical records are sometimes incomplete and represent less than 25% of the true status of snakebite in rural Papua New Guinea.

In an attempt to understand the true status of snakebite in rural Papua New Guinea, it is necessary to look at the statistics in more detail. A study by Hudson and Pomat considered 111 patients for which case notes were available. 111 patients developed systemic signs of envenomation, of which 100 patients showed positive signs of envenomation and identified as venomous bites. Of the remaining 54 patients...
Papua records numerous snakebites from non-venomous species or bites which did not result in envenomation, together with the serious cases, under the same heading: "Poisoning by Snake Bite", or later as "Effects of Poison" which include other causes of clinical poisoning.

Campbell spent a great deal of time sifting through the available data and statistics and he was finally able to present a series of data for snakebite admission and deaths in Papuan hospitals. This data is summarised here but it must be remembered first, that many snakebite victims never seek medical attention and are, therefore, not included in these statistics, and second, that the New Guinea districts, where at least three dangerous species occur, are not represented in Campbell's data.

Campbell estimated that during the late 1950's and 1960's snakebite, or suspected snakebite, was thought to be responsible for 155 admissions (6.3 per 1000 admissions), annually to Papuan hospitals (Table 1), excluding the hospitals in the Southern Highlands. Snakebites, therefore, accounted for five to ten percent of admissions in outstation hospitals and up to thirty-five percent of admissions to Port Moresby Hospital. Campbell also concluded that the fatality rate amongst admissions to PMGH was 7% and Brian and Vince (1987) reported a similar death rate (7.7%) amongst all children but a much higher rate of mortality (21%) for children under five.

Of 482 suspected snakebite admission to Port Moresby General Hospital over a six years period, Campbell determined that only 123 of these admissions showed definite signs of envenomation. The other cases ranged from possible snakebites with no envenomation to accidents with inanimate objects. Hudson and Pomat considered 175 admissions to Madang General Hospital which were believed to be as a result of possible or definite snakebites but case notes were only available for 129 of these patients and in only 64 of those were symptoms of envenoming recorded. It is, therefore, dangerous to rely too much on the available medical records and statistics although they may be useful as a rough guide. In addition, many indigenous Papuans, in common with many rural peoples throughout the world, place considerable faith in traditional medicines, in preference to 'modern' medical techniques and prefer to remain in their own villages rather than to travel considerable distances to unfamiliar hospitals. Since data is only available for hospital snakebite admissions and deaths the true status of snakebite in rural Papua New Guinea is not known.

In an attempt to understand the statistics of snakebite and be able to predict high risk groups, activities and times of day, Campbell restricted his interest to the 123 patients with unequivocal bites by venomous snakes but Hudson and Pomat considered their entire group of 129 possible and definite snakebites for which case notes were available. Within Campbell's test group 111 patients developed systemic symptoms whilst of 64 of Hudson and Pomat's group showed positive signs of envenoming. Campbell's twelve who showed no signs of envenomation were bitten by snakes which were subsequently killed and identified as venomous. Brian and Vince's study of snakebite amongst children records 63 admissions but case notes were missing for nine patients. Of the remaining 54 patients all but two showed definite signs of envenoming.
TABLE 1
Snakebite Admissions for Papua 1961-67

<table>
<thead>
<tr>
<th>District 1961-67</th>
<th>Snakebite admissions</th>
<th>No. per 1000 admissions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Western</td>
<td>85/10066</td>
<td>8.4</td>
</tr>
<tr>
<td>Gulf</td>
<td>132/11085</td>
<td>11.9</td>
</tr>
<tr>
<td>Central</td>
<td>673/78514</td>
<td>8.6</td>
</tr>
<tr>
<td>Central excl, NCD</td>
<td>182/12774</td>
<td></td>
</tr>
<tr>
<td>Port Moresby NCD</td>
<td>491/65740</td>
<td></td>
</tr>
<tr>
<td>Milne Bay</td>
<td>3/25227</td>
<td>0.1</td>
</tr>
<tr>
<td>Northern</td>
<td>40/23709</td>
<td>1.7</td>
</tr>
<tr>
<td>Total3</td>
<td>933/148601</td>
<td>6.3</td>
</tr>
</tbody>
</table>

1. Complete data for several years from certain hospitals was unavailable.
2. Five to ten seriously envenomated patients transferred annually from outstation hospitals to Port Moresby Hospital are recorded twice.
3. Southern Highlands District was omitted. No highly venomous snakes are known to occur in Southern Highlands.

adapted from Campbell 1969

TABLE 2
Notified Deaths due to Snakebite in Papua 1959-67

<table>
<thead>
<tr>
<th>Year</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>1959-60</td>
<td>8</td>
</tr>
<tr>
<td>1960-61</td>
<td>1</td>
</tr>
<tr>
<td>1961-62</td>
<td>4</td>
</tr>
<tr>
<td>1962-63</td>
<td>2</td>
</tr>
<tr>
<td>1963-64</td>
<td>7</td>
</tr>
<tr>
<td>1964-65</td>
<td>5</td>
</tr>
<tr>
<td>1965-66</td>
<td>3</td>
</tr>
<tr>
<td>1966-67</td>
<td>5</td>
</tr>
<tr>
<td>1959-67</td>
<td>35</td>
</tr>
</tbody>
</table>

adapted from Campbell 1969.

Two of the nine omitted envenoming.

Campbell's study group
one patient each from Western Central Province bites occurred in the majority, seventy persons. Only six bites took place at Brown River and Mt Victoria. When the writer interviewed to be found in the surrounding study concerns patients from referral hospital for Madang.

Campbell discovered that the snakebite victims was fourteen for forty-five and females being additional males aged three also bitten. The highest risk of this group tended to travel north and Vince's juvenile study group female ratio (almost 3:2) similar between the sexes. The high Pomat's Madang sexual ratio is Campbell's Port Moresby ratio.

As would be expected in the Papua elapids, most of Campbell's bites were inflicted during the hours 05.30 and 18.30 and only one bite occurred in the early death adder and small-eyed mainly diurnal or crepuscular in the hottest weather so encouraging that the fear of sorcery and that at home during the night. In major discrepancies to occur in Madang Province and a higher number of nocturna bites were still more frequent in seven cases the time of the apparently nocturnal species human movements during the adders sleeping on forest trees hands or arms resulting from brushwood or log clearance.

In common with other Campbell moves around unshod, mostly...
Two of the nine omitted children subsequently died from snakebite envenoming.

Campbell's study group mostly originated from Central Province with only one patient each from Western, Gulf and Milne Bay Provinces. Most of the 120 Central Province bites occurred along the coastal lowlands of the province with the majority, seventy persons, being bitten within twenty miles of Port Moresby. Only six bites took place at an appreciable distance inland, four between the Brown River and Mt Victoria and two near Tapini in the north of the province. When the writer interviewed locals in Tapini only the death adder was reported to be found in the surrounding mountains and valley. Hudson and Pomat's study concerns patients from throughout the province since MGH is the major referral hospital for Madang town and 27 rural heath centres.

Campbell discovered that the sex ratio of the Port Moresby study group snakebite victims was four males to one female with males between five and forty-five and females between five and thirty-five receiving bites. Two additional males aged three and fifty-five, outside the main age groups, were also bitten. The highest risk group were males aged twenty to twenty five, as this group tended to travel more extensively hunting or looking for work. Brian and Vince's juvenile study group in the same area demonstrated a closer male:female ratio (almost 3:2) since the daily routine of children probably varies little between the sexes. The highest risk group was the under fives. Hudson and Pomat's Madang sexual ratio for all age groups is also much closer than Campbell's Port Moresby ratio (less than 2:1).

As would be expected from a study of the natural history of southern Papuan elapids, most of Campbell's venomous snakebites admitted to PMGH were inflicted during the hours of daylight with 108 bites taking place between 05.30 and 18.30 and only eight bites occurring between 18.30 and 22.00; only one bite occurred in the early morning. With the exception of the secretive death adder and small-eyed snake, the large, dangerous Papuan elapids are mainly diurnal or crepuscular (active at dusk). They only become nocturnal in the hottest weather so encounters are unlikely, especially when it is realised that the fear of sorcery and the dark keeps a large proportion of the population at home during the night. However, the time of envenoming was one of the major discrepancies to occur between the work of Campbell at PNGH and Hudson and Pomat at MGH. Only the death adder and the small-eyed snake occur in Madang Province and since both are nocturnal or crepuscular species a higher number of nocturnal bites would be expected (42 bites, 33%). Diurnal bites were still more frequent with 80 bites (62%) occurring during the day. In seven cases the time of the snakebite was not recorded. Diurnal bites from apparently nocturnal species can be explained by: a) considerably increased human movements during the hours of daylight, b) cryptically patterned death adders sleeping on forest trails used by barefoot travellers, and c) bites to the hands or arms resulting from snakes uncovered during forest or garden brushwood or log clearance.

In common with other countries where most of the indigenous population moves around unshod, most bites (Campbell 112; Hudson & Pomat 105),
occurred on the lower limb with most of those (Campbell 82; Hudson & Pomat 92), on the foot, compared to bites to the upper limb and hand (Campbell 11; Hudson & Pomat 10). Campbell also analysed the circumstances of the bite. Sixty-six bites took place whilst the victim was walking and 28 whilst working, usually in the gardens which are often located some distance from the living quarters, or hunting. Neither series of circumstances is surprising as many paths in lowland Papua New Guinea are fringed, and in places, overhung by dense razor-grass which provides an ideal habitat for the large diurnal elapids such as the taipan or Papuan blacksnake. Gardens too, are often overgrown and debris left lying on the ground for any period of time is likely to become occupied by sheltering snakes. The author received numerous reports of large, fast moving diurnal snakes in these garden habitats. Few bites occur actually within the village confines and the author noted that the Kiwai people of southern Western Province deliberately keep the grass around the houses very short to discourage snakes. Interestingly, few bites amongst the indigenous peoples, only four in Campbell’s study, originate from deliberate attempts to pick up snakes, yet this is the major cause of snakebite in adolescent males in Europe, U.S.A., South Africa and Australia.

Campbell also considered whether snakebite was more likely in any particular month of the year. From his six year study he concluded that victims were admitted to hospital throughout the year but in any particular year a month may go past without a single admission for snakebite. Hudson and Pomat, however, reported almost three times as many snakebite admissions during the wet season (92 October-May) than during the dry season (37 June-September). It is, therefore, difficult to predict months of greatest risk although snakes are generally believed to be more common during the wet season. This last fact is born out from the author’s experiences of catching snakes throughout the Tropics.

The Melanesian Attitude to Snakes and Snakebite.

Snakes, particularly venomous species, feature strongly in the imaginations and stories of the indigenous people of Papua New Guinea. The author encountered several interesting beliefs concerning harmless species such as the wart snake (Acrochordus arafurae), carpet python (Morelia spilota), and brown cat snake (Boiga irregularis), but the snake which has the strongest hold over the villagers of southern Western Province undoubtedly is the ‘Pap blak’. This snake is feared to the point of hysteria if it, or another black coloured snake, is discovered near a village. Snakes are seen as totems, religious deities, protectors of property, signs or omens of impending disaster, reincarnations of dead relatives, avengers of broken taboos and the instruments of sorcery. Some peoples attribute certain snakes, encountered under particular circumstances, with magical powers whilst other people see the snakes merely as the tools of the magic men; the Mega Mega Auri (the man who sends snakes), or Ovedevenar (the black snake man of the Trans-Fly). The author met one such magic man who claimed to be able to call out snakes at will. Other sorcerers are said to turn themselves into snakes to injure the intended victim.

Snakesbites are viewed as retribution upon that person either himself or a close relation, or Hedura Tauna, to treat the family members are questioned. Should the patient’s condition be hopeless, the first of these is often second a frequent characteristic of Papuan blacksnake bites. Victim at this stage.

This almost fatalistic incantation and the powerful distances involved in reaching the village in the interior, could indicate why medical doctors examine the patient. Campbell suggested that the sorcerer, could delay the delivery of the patient from severe elapine envenomation. The first of these is the Papuan blacksnake, with many experts advising the patient to be treated as quickly as possible.

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Dangerous New Guinea Elapids.

Species of the following genera occur on New Guinea mainland: Acaena, Microphechis, Oxyuranus, and Unichis. To assist readers understand the elapids of New Guinea it is important to note that Ultra calamus is now considered a synonym of with Ultra calamus, is regarded (Makolin & others, 1960). Likewise the New Guinea mainland species is currently synonymised with Parapistocalamus and Salis, the Solomon Islands to the east of the species with most of the species with: Acaena inoffensive with tiny mouth. They they pose no serious threat. A smaller little known species as their larger specimen could have come from Muller’s snake, Aspidelaps, vomiting and sweating (Campbell).
Snakesbites are viewed initially, like any other illness or disease, as a retribution upon that person for the breaking of a strict taboo or ancient law by either himself or a close relative. Attempts are made by the native practitioner, or Hedura Tauna, to treat the bite with local herbs and incantations whilst the family members are questioned to determine the nature of the broken taboo. Should the patient's condition deteriorate, sorcery is believed to be the cause and an attempt to discover the name of the magic man concerned will be made.

If the sorcerer is located he will be offered gifts and requested to reverse the sickness. Various herbal medications and occult practices will be used but if signs such as build up of mucus in the back of the mouth, repeated bite marks or bleeding from mouth or wounds occur, the magic man will consider the case hopeless. The first of these is a symptom of the onset of advanced paralysis, the second a frequent characteristic of taipan bite and the third is a common sign of Papuan blacksnake bite so there would be little chance of survival for the victim at this stage. If death occurs it is always considered to be due to sorcery.

This almost fatalistic approach to snakebite with its reliance on herbs, incantation and the power of the magic men, coupled with the enormous distances involved in reaching hospital and the poor routes of communication in the interior, could indicate that many more snakebite deaths occur than the medical doctors examine or Government officials are able to record. Campbell suggested that as the intervention of the native practitioner, or the sorcerer, could delay modern medical treatment of a patient suffering from severe elapine envenomation, some form of compromise whereby the native practitioner and the medical doctor collaborate over the case would be advisable.

Dangers New Guinea Elapids.

Species of the following terrestrial elapid genera have been recorded on the New Guinea mainland: Acanthophis, Aspidomorphus, Demansia, Glyphodon, Micropsichis, Oxyuranus, Pseudechis, Pseudomaia, Toxicocalamus and Unechis. To assist readers referring to out-dated papers on the subject of the elapids of New Guinea it should be explained that the genus Pseudapistogramacamus is now considered to be a synonymy of Apistogramacamus which, together with Ultrocalamus, is regarded as a subgenus of Toxicocalamus (McDowell, 1969). Likewise the New Guinean members of Denisonia and Suta are currently synonymised with Unechis. In addition the genera Loveridgelaps, Parapistogramacamus and Salomonelaps are known from Bougainville Island and the Solomon Islands to the far east (McDowell, 1969; McCoy, 1980). However, most of the species within these minor genera are small, secretive and inoffensive with tiny mouths incapable of administering a dangerous bite, and they pose no serious threat to man. Care should still be taken, however, with little known species as there always remains the possibility that a bite from a larger specimen could have alarming, if not life threatening effects (ie, a bite from Muller's snake, Aspidomorphus muelleri, has been recorded as causing vomiting and sweating (Campbell, 1969). Although totally aquatic and not strictly elapids, seasnakes of the monotypic genera Enhydrina have been
recorded in freshwater Papuan river systems. As only the underlined genera are considered likely to constitute a threat to human life in Papua New Guinea only species contained within those genera will be considered here. Since *E. schistosa* occurs in large numbers in the Ramu River system where its bite poses a serious potential threat this species has been included whilst exclusively marine species or occasional river invaders have been omitted.

**Highly Dangerous New Guinea Elapids:**

- *Acanthophis antarcticus* and *A. praelongus* (Death Adders);
- *Oxyuranus scutellatus canni* (Papuan Taipan);
- *Pseudechis australis* (Mulga or King Brownsnake);
- *Pseudechis papuanus* (Papuan Blacksnake);
- *Pseudonaja textilis* (Eastern Brownsnake).

**Potentially Dangerous New Guinea Elapids:**

- *Demansia atra* and *D. papuensis* (Papuan Black Whipsnakes)
- *Micropechis ikaheka ikaheka* (Small-eyed or Ikaheka Snake).
- *Enhydrina schistosa* (Common Seasnake)

The taxonomy of the six terrestrial genera is far from simple as the number of recognised New Guinea species and subspecies within these genera varies from six to ten depending on the authority. These problems are summarised below and it is left to the reader to decide on the validity of each issue.

**Taxonomic Problems of Dangerous New Guinea Elapids**

**Acanthophis**

The death adders are easily distinguished from all other snakes within their geographical range, except perhaps from the ground boa (*Candoia aspera*), which also occurs in New Guinea and has a relatively short stout body. However, within the genus *Acanthophis* there are certainly taxonomic problems and many name combinations and couplings have been used in the past; *A. antarcticus antarcticus* and *A. antarcticus pyrrhus* for Australia (Worrel 1963), *A. antarcticus* and *A. pyrrhus* (Cogger 1975; Mirtschin & Davis 1982), *A. laevis* from Western Province, Papua New Guinea (Macleay 1877), *A. antarcticus rugosus* from Merauke, Irian Jaya, and *A. antarcticus antarcticus* (including Macleay's *A. laevis*), from Australia and Papua New Guinea (Loveridge 1948; de Haas, 1950). Storr (1981), records three species for Australia: *A. antarcticus, A. pyrrhus* and *A. praelongus*, with the note that the third of these species also occurs in southern New Guinea. It seems likely that more than one species does occur in New Guinea and this author captured death adders in Western and Central Provinces which certainly appeared to possess different scalation, head shape and degrees of supraocular adornment. It seems quite likely that both *Acanthophis antarcticus* and *A. praelongus* may inhabit New Guinea and only further field work, examination of New Guinea specimens, chromosomal and electrophoretic studies will solve the problem (see key for morphological differences between the two species).

**Demansia**

Formerly all Papuan subspecies of *Demansia* subspecies *D. olivacea* (1968). This second subspecies, considered closely related to *D. olivacea*. The situation with Australian species, *Demansia*, are divided as to whether *D. atra* or *D. papuensis* (Cogger & Whitaker 1982), or possibly *D. antarcticus* (Storr 1978), differentiate on ventral pigmentation, subcaudal scale counts which are considered these character Papuan species. This author which would appear to be

**Micropechis**

Klemmer (1963), considered *i kaheka* to be *Micropechis fasciatus* to Aru Island, accepted view for this species. Northern and southern races accordingly, northern races for the purposes of this study and all Papua, following Klemmer.

**Oxyuranus**

The Australian taipan, *Oxyuranus* from Queensland and Northern considered to represent single species. However, Slater (1967) separate subspecies, *Oxyuranus* keels, the ventral red-orange Papuan specimens are said.

**Pseudechis**

No taxonomic problems for *Pseudechis papuanus*, but the Australian *Pseudechis australis* southwestern Papua, is Worrell (1963), is dubious on its close apparent relations to exist sympatrically. However
ly the underlined genera
life in Papua New Guinea
been considered here. Since
system where it's bite
has been included whilst
ners have been omitted.

(Adders);

Whipsnakes)

ikaheka Snake).

from simple as the number
within these genera varies
problems are summarised
ility of each issue.

Lizards

other snakes within their
boa (Candoia aspera),
atively short stout body. 
are certainly taxonomic
ings have been used in the
psammophis pyrrhus for Australia 
1975; Mirtschin & Davis 
Guinea (Macleay 1877), 
aya, and A.antarcticus 
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and this author captured 
certainly appeared to 
of supraocular adornment. 
us and A.praetongus may 
mination of New Guinea 
ies will solve the problem 
wo species).

Demansia

Formerly all Papuan whipsnakes were recognised as representing either a 
subspecies of Demansia psammophis (Loveridge 1945; Slater 1956), or the 
subspecies Demansia olivacea papuensis (Worrell 1963; Klemmer 1963; Slater 
1968). This second subspecies was eventually elevated to species level but is still 
considered closely related to both Australian species, D.psammophis and 
D.olivacea. The situation is further complicated as Cogger (1975), records the 
Australian species, Demansia atrata, as occurring in southern Papua. Authors 
are divided as to whether Papua is inhabited by one or other of the species, 
D.atra or D.papuensis (Cogger 1975; Scott, Parker & Menzies 1977; Whitaker & 
Whitaker 1982), or possibly both species (Storr 1978; Parker 1982; Golay 1985). 
Storr (1978), differentiates between Australian specimens of these two species 
on ventral pigmentation, head coloration, size and combined ventral and 
subcaudal scale counts but Parker (1972; 1982), reports that McDowell 
considered these characters of dubious value when they were used to define 
Papuan species. This author collected several specimens in Western Province 
which would appear to be D.atra based on Storr's characteristics.

Micropechis

Klemmer (1963), considered the New Guinea populations of Micropechis 
ikaheka to be Micropechis i.ikaheka whilst he confined the subspecies 
M.fasciatus to Aru Island to the southwest of Irian Jaya. This is the generally 
accepted view for this species but Slater (1956; 1968), differentiated between 
northern and southern races on the mainland allocating the subspecies 
accordingly; northern race, M.i.ikaheka, and southern race, M.fasciatus. For 
the purposes of this study only Micropechis i.ikaheka is recognised for New 
Guinea, following Klemmer.

Oxyuranus

The Australian taipan, Oxyuranus scutellatus, is known to occur in 
Queensland and Northern Territory and the New Guinea population was 
considered to represent simply extralirnital distribution of an Australian snake 
species. However, Slater (1954; 1956), described the New Guinea taipan as a 
separate subspecies, Oxyuranus s. canni, based on its more pronounced scale 
keels, the ventral red-orange stripe and possibly also the greater size that 
Papuan specimens are said to attain.

Pseudechis

No taxonomic problems exist regarding the endemic Papuan blacksnake, 
Pseudechis papuanus, but this is certainly appeared to 599
Papua New Guinea

**Key to Provinces Map 1:**

**DISTRIBUTION OF THE DANGEROUS ELAPIDS OF PAPUA NEW GUINEA**

These maps are intended to give a general overview of the ranges of the dangerous Papuan elapids rather than precise locality information. There are numerous areas of questionable or unknown distribution such as Southern Highlands and Gulf Provinces. Further work in these areas would help to clarify the total picture.

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**Pseudonaja textilis** is closely related to the Australian species. Other authors also include it as a synonym of Pseudonaja. (Dawson, 1986). Other authors also synonymize it with Pseudonaja textilis. (Knysna, 1986; Shine, 1987).

**Individual Species Account**

**NEW GUINEA DEATH ELAPID**

**Acanthophis praelongus**

Average/Maximum length: 300-500mm/1000mm

**Description**

a) physique:

An elapid which has otherwise been occupied elsewhere by the species. Highly variable dorsal coloration with broad or narrow alternating bands of yellow and white, black; ventrally either immaculate or yellow tipped; head as body but with white labials, spotted or mottled (Fig. 1)

b) colouration:

Highly variable dorsal coloration with broad or narrow alternating bands of yellow and white, black; ventrally either immaculate or yellow tipped; head as body but with white labials, spotted or mottled.
closely related to the Australian species, \textit{P. colletti} (Mengden, Shine \& Moritz, 1986). Other authors also agree that \textit{P. australis} probably does occur in the Irian Jaya/Papua frontier region (Slater, 1968; Parker, 1982; Mengden \textit{et al} 1986; Shine, 1987).

\textbf{Pseudonaja}

\textit{Pseudonaja textilis} is an Australian species which has subsequently been recorded from a number of northeastern coastal and inland localities in Milne Bay and Northern Provinces (McDowell, 1967 records specimens collected by the Archbold Expeditions of 1953, and both Worrell and Cogger have examined and confirmed identification of \textit{P. textilis} material originating from Papua New Guinea). It is, however, strangely absent from apparently suitable sites in Central and Western Provinces which lie between its disjunct Papuan collection sites and its more familiar continental Australian distribution. It has been suggested by Slater (1968), that these Papuan records originate from specimens or eggs accidentally introduced by Australian military forces during the 1940's. However, the Papuan specimens often appear to be darker (even black) than the usual Australian species and they have a slightly higher maxillary tooth count (12 rather than 9-11, McDowell, 1967).

\textbf{Individual Species Accounts}

\textbf{Highly Dangerous Species:}

\textbf{NEW GUINEA DEATH ADDERS} \textit{Acanthophis antarcticus} Shaw and \textit{Acanthophis praefonatus} Ramsey

\textbf{Average/Maximum length:} 300-500mm/1000mm (1-1\frac{1}{2}ft/3ft)

\textbf{Description}

\textbf{a) physique:}

An elapid which has evolved to fill the niche in Australasia which is occupied elsewhere by the viperids. Characterised by a short, stumpy viper-like body; rough, strongly or weakly keeled, or smooth scales; short tail with comb-like terminal spinal process; broad viper-like head; occasionally raised horn-like supraocular scales and either keeled rugose or smooth scales; relatively long and mobile fangs and vertically elliptical pupil.

\textbf{b) colouration:}

Highly variable dorsally; red, brown or greyish, either uniform or with alternating broad or narrow, pale and dark transverse bands, spotted with black; ventrally either immaculate or spotted with black, tail black white or yellow tipped; head as body, brown or red with darker transverse streaks above with white labials, spotted with black, and white chin, also spotted with black. (Fig. 1)
c) scalation:
21, or rarely 23, rows, midbody; 110-124, (A. antarcticus) 36-50, (A. antarcticus), or paired; anal plate single; lore.

Habitat:
Widespread in monsoon highland, and occasionally in lowland.

Habits:
Commonly encountered, poses a considerable threat in the initial bite the death adder. Manner of the other elapid below the ankle. Considered ovoviviparous.

Dial period of activity:
Nocturnal, sluggish during day.

Prey preferences:
Terrestrial skinks, birds wiggling tail tip to attract prey.

Distribution within PNG:
Wide-spread throughout Milne Bay, Enga and Southern above 1800m in other highlands. Henganofi, Eastern Highlands, Sepik and Markham River. Specimens from New Britain.

Extralimital distribution:
Indonesia from Irian Jaya, Moluccas (de Rooij, 1917; de Rooij, 1917; de Rooij, 1917; de Rooij, 1917; de Rooij, 1917; de Rooij, 1917; de Rooij, 1917; de Rooij, 1917; de Rooij, 1981).

Fang length:
5-8.8mm (Fairley, 1929).

Average/Maximum venom:
42-84.7mg/235.6mg (Fairley, 1929).
c) Scalation:

21, or rarely 23, rows, (A. antarcticus), or 23 rows, (A. praelongus), at midbody; 110-124, (A. antarcticus), or 122-134, (A. praelongus); subcaudals 36-50, (A. antarcticus), or 47-57, (A. praelongus), anteriorly single, posteriorly paired; anal plate single; loreal scale absent; subocular scales present.

Habitat:

Widespread in monsoon and rainforest habitats, both lowland and fairly highland, and occasionally in savanna woodland and upland grassland valleys.

Habits:

Commonly encountered on paths and trails asleep during the day when it poses a considerable threat as it will respond by biting if trodden on. Following the initial bite the death adder will often hang on, not attempting to flee in the manner of the other elapids, so it is frequently killed by its victim. Usually bites, below the ankle. Considered most common at the end of the wet season. Ovoviviparous.

Dial period of activity:

Nocturnal, sluggish during the day.

Prey preferences:

Terrestrial skinks, birds or small mammals and possibly frogs, using wiggling tail tip to attract prey.

Distribution within PNG:

Wide-spread throughout mainland Papua New Guinea excepting Northern, Milne Bay, Enga and Southern Highlands Provinces and extreme altitudes above 1800m in other highland provinces. Small montane race known from Henganofi, Eastern Highlands, and large one metre specimens known from the Sepik and Markham River regions. Kinghorn and Kellaway (1943) report specimens from New Britain but record is doubtful.

Extralimital distribution:


Fang length:

5-8.3mm (Fairley, 1929)

Average/Maximum venom yield:

42-84.7mg/235.6mg\(^\text{A}\) (Fairley & Splatt, 1929)

\(^\text{A}\) = data obtained from Australian specimens
Lethal subcutaneous dose:
- 0.015mg (100gm Guinea pig Campbell, 1969); 0.025-0.15mg/kg (man, Kellaway, 1929)

Toxicity LD50:
- 0.338mg/kg (mouse, Broad, Sutherland & Coulter, 1979)

Toxicity LD100:
- 0.5-0.7mg/kg (mouse s.c., Trehewie, 1971)

Active effects of venom:
Strongly to moderately neurotoxic; weakly haemolytic, possibly anticoagulant (Kellaway, 1929) and cytotoxic. Coagulant factor incomplete prothrombin activator only functioning in presence of factor V (Mebs, 1978). Wounds do not bleed and no clinical haemotoxic effects on blood. Not haemorrhagic. Neurotoxic action, postsynaptic causing peripheral curare-like neuromuscular block (Kellaway, Cherry & Williams, 1932), is reversible. Neurotoxin isolated from *A. antarcticus* (acanthophin A, Sheumack et al, 1979; Sutherland, 1980).

Antiserum/initial dose:

PAPUAN TAIPAN *Oxyuranus scutellatus cannii* (Slater)

Average/Maximum length:
- 1830-2440mm/3355mm (6-8ft/11ft)

Description

a) physique:
A large slender species with elongated 'coffin-shaped' head, distinct from narrow neck; tail long and whip-like; eye moderate-sized with round pupil; supraocular scale over eye sharply shelved giving snake scowling expression.

b) colouration:
Dorsum of body olive, dark brown or dark grey, usually with an orange or pinkish stripe evident in interstitial skin of vertebral scale rows; venter cream to off-white, either immaculate or speckled with orange; head coloured as dorsum or paler, especially in juveniles, snout and labials usually lighter; iris of eye brown. (Fig. 2)

c) scalation:
- 21, or 23, rows at midbody, anteriorly keeled; ventrals 220-250; subcaudals 45-80, all paired; anal plate single; loreal and subocular scales absent.
9]; 0.025-0.15mg/kg (man, liter, 1979)


Slater)

haped' head, distinct from rats; eye-sized with round pupil; make scowling expression.

usually with an orange or yellow cross; scale rows; venter cream to yellow; head coloured as dorsum usually lighter; iris of eye

ular scales absent.

Figure 2
Oxyuranus scutellatus canni
Habitat:
Lowland savanna and savanna woodland.

Habits:
Nervous and retiring but will strike with speed and aggression if startled, threatened or molested, arching the body back, flattening the head and striking forward and upwards, often biting more than once in quick succession. Moves rapidly over the ground, often with head and forepart of body raised. Possesses very acute sense which usually prevent human encounters, but when aroused known for characteristic rapid and multiple "strike and release" bites which may lead to enormous quantities of venom being injected. Commonest during wet season. Oviparous.

Dial period of activity:
Diurnal and crepuscular, but nocturnal in warm weather.

Prey preferences:
Mammals, rats, bandicoots, and possibly ground nesting birds.

Distribution within PNG:
Confined to lowland southern coastal localities in Western, Central, (Kukipi to east of Marshall Lagoon and inland to 1,100ft on Sogeri Plateau), and Milne Bay Provinces. May also occur in Gulf Province although not yet recorded there. Known from mainland Milne Bay Province as far east of Samarai but not recorded from islands of D'Estreecasteaux and Louisiade Archipelagoes. Particularly common on Oriomo Plateau in southern Trans-Fly region, on Daru Island and in the vicinities of Morehead, Lake Murray and Balimo, Western Province and around Port Moresby, NCD, in Central Province.

Extralimital distribution:
Endemic New Guinea subspecies confined to Papua, (although may subsequently be recorded in southeastern Irian Jaya), but close relative, Oxyuranus scutellatus scutellatus, occurs in Queensland and Northern Territory, Australia.

Fang length:
13mm\(^A\) (Kellaway, 1932).

Average/Maximum venom yield:
100-200mg/400mg\(^A\) (Campbell, 1967); 500mg (Trehewie, 1971).

Lethal subcutaneous dose:
0.0025mg\(^A\) (100gm Guinea pig, Morgan 1956); 0.0034mg\(^A\) (25gm mouse); 0.1mg/kg (man, Kellaway, 1929; Morgan, 1956).

Toxicity LD\(_{50}\):
0.002mg/kg (Guinea pig, Morgan, 1956); 0.064mg s.c., Trehewie, 1971a).

Active effects of venom:
Strongly neurotoxic : cytotoxic, myotoxic and coagulant factor complete thrombin in presence or Neurotoxin isolated from the potent terrestrial snake: (Sutherland, 1980) and my

Antiserum/Initial dose:
CSL Monovalent Taipa & Davis, 1982).

Mulga or King Brown Snake:

Average/Maximum length:
2440mm/2745mm (8ft."

Description
a) physique:
A heavy bodied species with round pupil.

b) colouration:
Yellow, red or tan bro cream.

c) scalation:
17 rows at midbody, sn except extreme posterior loreal and subocular scales.

Habitat:
Savanna and savanna woodland in Australia.

Habits:
A slow moving species which, although not as tox
and aggression if startled, swinging the head and striking in quick succession. Moves part of body raised. Possesses counters, but when aroused "bite and release" bites which injected. Commonest during weather.

Habits not yet recorded there.

east of Samarai but not Louisiade Archipelagoes.

Papua, (although may Jaya), but close relative, Queensland and Northern

Toxicity LD50:

0.002mg/kg (Guinea pig, Campbell, 1969); 0.02mg/kg (Guinea pig s.c., Trethewie, 1971a); 0.064mg/kg (mouse, Broad et al, 1979); 0.12mg/kg (mouse s.c., Trethewie, 1971a).

Active effects of venom:

Strongly neurotoxic and coagulant; weakly haemolytic, also possibly cytotoxic, myotoxic and cardiotoxic causing heart failure (Habermehl, 1981). Coagulant factor complete prothrombin activator converting prothrombin to thrombin in presence or absence of factor V (Denson, 1969; Mebs, 1978). Neurotoxin isolated from taipan (taipoxin, Fohlman et al, 1976) is second most potent terrestrial snake neurotoxin known, causing presynaptic blockade (Sutherland, 1980) and myolysis (Harris et al, 1977).

Antiserum/initial dose:

CSL Monovalent Taipan/12 000 units (CSL Med. H/b., 1979; 1985; Mirtschin & Davis, 1982).

Mulga or King Brownsnake Pseudechis australis (Gray)

Average/Maximum length:

2440mm/2745mm (8ft/9ft)

Description

a) physique:

A heavy bodied species with a broad head which is slightly distinct from the neck, especially in large specimens which may have bulbous cheeks; eye small with round pupil.

b) colouration:

Yellow, red or tan brown dorsally both head and body; venter yellowish cream.

c) scalation:

17 rows at midbody, smooth; ventrals 185-225; subcaudals 50-75, all single except extreme posterior few which are paired; anal plate normally divided; loreal and subocular scales absent.

Habitat:

Savanna and savanna woodlands but also in tropical forests and deserts in Australia.

Habits:

A slow moving species but capable of injecting huge quantities of venom which, although not as toxic as that of the eastern P.papuanus, constitutes a
considerable danger. Reputed to be unpredictable and inclined to hold on when it bites this species will flatten its head and strike with rapidity and aggression. Ovoviviparous but may be oviparous in some areas.

**Dial period of activity:**
- Diurnal or crepuscular becoming nocturnal in hot weather and not active during the heat of the day.

**Prey preferences:**
- Small mammals, frogs and reptiles, including other snakes.

**Distribution within PNG:**
- Probably present in Western Province west of the Fly River, especially in the Morehead region near the frontier with Irian Jaya.

**Extralimital distribution:**
- Throughout most of Australia, except extreme southwest, southeast and Tasmania, in a wide variety of habitats. Also from southeastern Irian Jaya in the vicinities of Etna Bay and Merauke (Loveridge, 1948) near frontier with Papua New Guinea.

**Fang length:**?

**Average/Maximum venom yield:**
- 180mg/600mg (Worrell, 1963).

**Lethal subcutaneous dose:**
- 0.16mg A (100 gm Guinea pig, Campbell, 1969).

**Toxicity LD50:**
- 1.91mg/kg A (mouse, Broad et al, 1979).

**Active effects of venom:**

**Antiserum/initial dose:**

---

**PAPUAN BLACKSNAKE**

**Average/Maximum length:**
- 2135mm/2440mm (7)

**Description**

**a) physique:**
- A strong, stout bodied species with tail fairly long; eye small with nostril above.

**b) colouration:**
- Dorsum of body uniformly gunmetal-grey; head as dark as body, usually becoming off-white with black spines.

**c) scalation:**
- 19, or rarely 21, rows of scales at midbody; first 25-45 scales on head and subocular scales absent.

**Habitat:**
- Lowland savanna and forest for damper, swampy ground compared to the taipan.

**Habits:**
- Nervous and inclined to attack. The blacksnake will attack with some aggressiveness other Australo-Papuan species. Oviparous.

**Dial period of activity:**
- Diurnal and crepuscular becoming nocturnal in hot weather and not active during the heat of the day.

**Prey preferences:**
- Small mammals and other reptiles.

**Distribution within PNG:**
- Probably confined to Western Province west of the Fly River, especially in the Morehead region near the frontier with Irian Jaya. Recorded from Yule Island.
PAPUAN BLACKSNAKE *Pseudechis papuanus* Peters & Doria

**Average/Maximum length:**

2135mm/2440mm (7ft/8ft)

**Description**

a) **physique:**

A strong, stout bodied species with a broad, flat head, distinct from neck; tail fairly long; eye small with round pupil; supraocular not distinctly shelled.

b) **colouration:**

Dorsum of body uniformly glossy jet black, rarely brown; venter blue-grey or gunmetal-grey; head as dorsum above but may be lighter on labials; neck yellow to off-white with black specklings. (Fig. 3)

c) **scalation:**

19, or rarely 21, rows at midbody, all smooth; ventrals 221-230; subcaudals 49-63 with first 25-45 single and remainder paired; anal plate divided; loreal and subocular scales absent.

**Habitat:**

Lowland savanna and savanna woodland but showing a greater preference for damper, swampy ground and also extending further into forests than the taipan.

**Habits:**

Nervous and inclined to flee at man's approach but when cornered the blacksnake will attack with power and tenacity apparently unrivalled by any other Australo-Papuan species. More commonly encountered in late dry season. Oviparous.

**Dial period of activity:**

Diurnal and crepuscular but avoiding hottest part of day.

**Prey preferences:**

Small mammals and possibly also frogs and ground nesting birds.

**Distribution within PNG:**

Probably confined to southern coastal lowlands. The species is recorded from the Irian Jaya frontier region and from Morehead, Lake Murray and the Oriomo Plateau, west of the Fly River in Western Province, and from lowland localities around Port Moresby, Marshall Lagoon and Amazon Bay in Central Province. It also occurs along the southern Milne Bay Province but has not been recorded from Northern Province although it may occur in Gulf Provinces. Recorded from Yule Island but not Daru Island. Formerly thought to be much
more common than it is toxic-skinned cane toad, New Guinea).

Extralimital distribution
New Guinea endemic
Prince Frederik Hendrik Island

Fang length:
6.1 mm

Average/Maximum venom:
200 mg/494 mg (Campbell, 1967)

Lethal subcutaneous dose:
0.02 mg (100 gm Guinea)

Toxicity LD₅₀:
?

Active effects of venom:

Antiserum/initial dose:
CSL Monovalent Papuan (1982), Polyvalent Papuan (1982)

EASTERN BROWNSNAKE

Average/Maximum length:
1830 mm/2135 mm (6 ft)

Description
a) physique:
A slender snake with a long, slender body; eye medium-sized.

b) colouration:
Dorsally the body is patterned with crossbands which are creamy-yellow, speckled with cream, lighter throat.
more common than it is today, possibly due to the introduction of the highly toxic-skinned cane toad, *Bufo marinus* (see *Snakebite as a Hazard in Papua New Guinea*).

**Extralimital distribution:**

New Guinea endemic also occurring in Irian Jaya along southern coast to Prince Frederik Hendrik Island.

**Fang length:**

6.1mm

**Average/Maximum venom yield:**

200mg/494mg (Campbell, 1967).

**Lethal subcutaneous dose:**

0.02mg (100gm Guinea pig, Campbell, 1967).

**Toxicity LD₅₀:**

?

**Active effects of venom:**


**Antiserum/initial dose:**

CSL Monovalent Papuan Blacksnake/18 000 units (Mirtschin & Davis, 1982), Polyvalent Papuan/40 000 units (CSL, 1985).

**EASTERN BROWNSNAKE *Pseudonaja textilis* (Dumeril & Bibron)**

**Average/Maximum length:**

1830mm/2135mm (6ft/7ft.)

**Description**

a) physique:

A slender snake with its head barely distinct from the neck; tail moderate length; eye medium-sized with round pupil.

b) colouration:

Dorsally the body is yellow brown, dark brown or black, often with darker crossbands which are particularly evident in juveniles; venter off-white to creamy-yellow, speckled with pink, brown or grey; head coloured as body with lighter throat.
c) scalation:
17 rows at midbody, all smooth; ventrals 185-235; subcaudals 45-75, usually all paired but occasionally with anterior few single; anal plate divided; loreal and subocular scales absent.

Habitat:
Upland grasslands and sandy or rocky heathlands but also in swamplands and cultivated areas.

Habits:
Very fast moving and inclined to flee from human approach but prepared to defend itself vigorously if molested with a raised coiled neck and mouth open wide in readiness for the strike. May strike several times in quick succession from the S-stance. Common in very warm weather. Oviparous.

Dial period of activity:
Usually diurnal.

Prey preferences:
Small lizards and frogs but also small mammals.

Distribution within PNG:
Northeastern Papua New Guinea from Dogura, Cape Vogel and Mori Biri Bay, Milne Bay Province, to Embogo and Popondetta, Northern Province, possibly a human introduction from Australia.

Extralimital distribution:
Eastern Queensland, New South Wales, Victoria and southern South Australia. Also isolated localities in Northern Territory.

Fang length:
2.8mm (Campbell, 1969).

Average/Maximum venom yield:
2-5mg/40-67.2mg\(^A\) (Sutherland, 1983).

Lethal subcutaneous dose:
0.0025mg\(^A\) (100gm Guinea pig, Campbell, 1969).

Toxicity LD\(_{50}\):
0.041mg/kg\(^A\) (mouse, Broad et al, 1979).

Toxicity LD\(_{100}\):
0.25mg/kg (mouse, Trethewie, 1971a).

Active effects of venom:
Strongly coagulant and myotoxic. Coagulant strongly from mucus membranes (\(\text{Cl}^\text{prot}^{\text{thrombin activator}}\) presence or absence of fact.

Antiserum/initial dose:
CSL Monovalent Brown (Davis, 1982); Monovalent ‘b’

Potentially Dangerous Species:
PAPUAN BLACK WHIPS
\(\text{p. papuensis}\) (Macleay)

Average/Maximum length:
658mm/1148mm (2\(D.papuensis\))

Description

a) physique:
- Slender, rapidly moving long and whip-like; eye large.

b) colouration:
- Dorsally olive brown or may be red-brown; interst. posteriorly; head dorsally or yellowish, spotted with circumorbital ring of yel. c) scalation:
- 15 rows at midbody paired; anal plate divided.

Habitat:
Savanna and savanna

Habits:
Probably the fastest rapidly that the eye has specimens which are more season.
Active effects of venom:

Strongly coagulant and neurotoxic; weakly haemolytic and cytotoxic; also myotoxic. Coagulant strongly diffusible causing thrombosis and haemorrhage from mucus membranes (Kellaway, 1938). Coagulant factor of venom complete prothrombin activator causing conversion of prothrombin to thrombin in presence or absence of factor V (Denson, 1969; Mebs, 1978). Strongest known terrestrial snake neurotoxin isolated (textilone, Coulter et al, 1979).

Antiserum/initial dose:

CSL Monovalent Brownsnake/1000 units (CSL Med.H/b., 1979; Mirtschin & Davis, 1982); Monovalent Taipan.

Potentially Dangerous Species:

PAPUAN BLACK WHIPSNAKES Demansia atra (Macleay) and Demansia p. papuensis (Macleay)

Average/Maximum length:

<table>
<thead>
<tr>
<th>Species</th>
<th>Average Length</th>
<th>Maximum Length</th>
</tr>
</thead>
<tbody>
<tr>
<td>D. atra</td>
<td>658mm/1148mm</td>
<td>630mm/1515mm</td>
</tr>
<tr>
<td>D. papuensis</td>
<td>630mm/11515mm</td>
<td></td>
</tr>
</tbody>
</table>

Description

a) physique:

Slender, rapidly moving snakes with narrow head distinct from neck; tail long and whip-like; eye large with round pupil.

b) colouration:

Dorsally olive brown or dark brown to black lightening towards tail which may be red-brown; interstitial skin yellow to white; venter blue-grey, darkening posteriorly; head dorsally spotted with dark pigment but labials lighter, white or yellowish, spotted with brown; chin white; eye with brown iris surrounded by circumorbital ring of yellow on preocular and postocular scales. (Fig. 4)

c) scalation:

15 rows at midbody, all smooth; ventrals 160-225; subcaudals 69-105, paired; anal plate divided; loreal and subocular scales absent.

Habitat:

Savanna and savanna woodland but also in cultivated gardens.

Habits:

Probably the fastest snake in New Guinea moving across the ground so rapidly that the eye has difficulty following it. Not an aggressive species but specimens which are molested will bite rapidly. Most common in the drier season.
Dial period of activity:
Diurnal and often seen abroad during the hottest part of the day when other species are not in evidence. Not seen at night. Oviparous.

Prey preferences:
Lizards are possibly frogs and small mammals.

Distribution within PNG:
Differentiating between the New Guinea species of *Demansia* presents considerable difficulties for the taxonomist but representatives of the genus occur throughout the southern savanna lowlands from the extensive grasslands to the west of the Fly River to Balimo in Western Province and also in coastal lowland localities of Central Province. Whether *Demansia* is represented in Gulf Province has yet to be determined.

Extralimital distribution:
*D.atra* occurs in Western Australia, Northern Territory and Queensland, and a subspecies of *D.papuensis, D.papuensis melaena*, has been recently described for Western Australia and Northern Territory. *Demansia spp.* also inhabit the southern savanna of Irian Jaya.

Fang length:

Average/Maximum venom yield:

Lethal subcutaneous dose:

Toxicity LD<sub>50</sub>:
14.2mg/kg (saline, mouse, Broad et al, 1979)

Active effects of venom:
Neurotoxic and possibly haemorrhagic and coagulant (Campbell, 1969).

Antiserum/initial dose:
CSL Monovalent Tiger Snake/3000 units (Mirtschin & Davis, 1982); Monovalent Brownsnake; Polyvalent Papuan.

NEW GUINEA SMALL-EYED OR IKAHEKA SNAKE *Micropechis i. ikaheka* (Lesson)

Average/Maximum length:
1500mm/2000mm (4½ ft/6ft.)
10th part of the day when light. Oviparous.

Demansia presents representatives of the genus from the extensive in Western Province and also. Whether Demansia is determined.

in Territory and Queensland, melaeana, has been recently Territory. Demansia spp. also

Description
a) physique:
Fairly stocky bodied species with relatively short tail; head narrow but distinct from neck; eye small with round pupil.

b) colouration:
Dorsally, body may be yellowish anteriorly with increasingly darker scales edged with cream posteriorly, (northern race), especially recognisable as yellow or white snake in Madang or Karkar Island, or with increasingly more apparent dark crossbands towards the hind part of the body and onto the tail and scattered dark spots on neck, (southern race); tail black with yellow ventral blotches, (northern race), or banded black and brown with cream blotches on belly (southern race); ventrals creamish yellow edged with black, (northern race), or brown, (southern race); head dorsally black or grey with lighter brown or yellow labials, throat and chin.

c) scalation:
15 rows at midbody, all smooth; ventrals 178-223; subcaudals 37-55, all paired; anal divided; loreal and subocular scales absent.

Habitat:
Confined to monsoon and rainforest areas and swamps, but not apparently dry savanna woodlands, from sea level to 15 000m. This species has also been recorded from plantations and commonly encountered under old coconut husks.

Habits:
Generally a secretive semi-fossorial, (burrowing), species inhabiting leaf litter or loose soil and usually only encountered when it ventures onto the surface in a clearing or when uncovered under a decaying log. Reacts with aggression if handled or molested. Most commonly encountered in the drier months. Oviparous.

Dial period of activity:
Both diurnal and nocturnal but usually venturing abroad after dark.

Prey preferences:
Little known but earthworms have been recorded and small burrowing frogs and lizards are also possibilities.

Distribution within PNG:
Widespread throughout mainland Papua New Guinea’s forests but not recorded from Gulf, Southern Highlands, Western Highlands and Enga Provinces. A problem in northern coastal Madang Province and nearby Karkar Island.
Extralimital distribution:
Throughout Irian Jaya including some of its neighbouring islands to the north and west. A second subspecies, *Micropechis i. fasciatus*, is recorded from Aru Island to the southeast of Irian Jaya.

Fang length:
?

Average/Maximum venom yield:
?

Lethal subcutaneous dose:
0.5-1.0mg (25gm mouse, Campbell, 1969).

Toxicity LD₅₀:
?

Active effects of venom:
Highly myotoxic causing myalgia, muscle tenderness, severe neuromuscular paralysis and myoglobinuria (Sutherland, 1983; Hudson & Pomat, 1988). Rhabdomyolysis (Blasco & Hornabrook, 1972; Hudson & Pomat, 1988); oliguria, renal failure and cerebral hypoxia (Sutherland, 1983), minor symptoms of nausea, severe headache and prolonged weakness. Capable of causing unconsciousness and cessation of respiration in just over two hours and death in 1½ hours although the original onset of symptoms may be greatly delayed (Blasco & Hornabrook, 1972).

Antiserum/initial dose:
CSL Monovalent Tiger Snake in large doses (Tscharke in Blasco & Hornabrook, 1972).

COMMON OR BEAKED SEASNAKE *Enhydrina schistosa* (Daudin)

Average/Maximum length:
950-1150mm/1400mm (3-4ft/4ft 8in.)

Description
a) physique:
Body elongate and narrow anteriorly; head small, barely distinct from neck; tail laterally compressed and paddle-like.

b) colouration:
Dorsally blue-grey to grey with dark grey or dark annuli laterally, broadest dorsilaterally, often masked by dark dorsal pigment in adult specimens; venter white.

c) scalation:
49-66 scale at midbody.

Habitat:
Coastal marine but freshwater systems. Not found in freshwater systems. Not found in freshwater systems.

Habits:

Dial period of activity:
Diurnal or nocturnal.

Prey preferences:
Fish.

Distribution within PNG:
Probably throughout PNG system. Recorded for coastal marine and the Ramu River System. A seasnake collected in the Magimbi... believed to be a *Hydrophis*.

Extralimital distribution:
Northwest to the Persian Gulf and Indonesia and south as far as captured in Tonie Sap, Solomon Islands, Philippines.

Fang length:
3mm (Minton & Minton, 1964).

Average/Maximum venom yield:
8.5/79mg (Habermehl, 1981).

Lethal subcutaneous dose:
0.05mg/kg (man, Tretel, 1960); 0.057mg (57µg) (man, Tretel, 1960). (frog) Barne, 1968.

Toxicity LD₅₀:
0.0021-0.0025mg (2.1-2.5µg) (frog) Barma, 1968.
c) scalation:

49-66 scale at midbody, imbricate or subimbricate, weakly keeled; ventrals 239-322; preanals feebly enlarged; characterised by elongate mental shield.

Habitat:

Coastal marine but also estuarine and frequently encountered in freshwater systems. Not found in deep water far from land.

Habits:


Dial period of activity:

Diurnal or nocturnal.

Prey preferences:

Fish.

Distribution within PNG:

Probably throughout Papuan coastal waters and in certain freshwater river system. Recorded for coastal Gulf Province by Whitaker and Whitaker (1982) and the Ramu River System, Madang Province, by Hudson and Fromm (1986). A seasnake collected in the Oriomo River, Western Province, by the author was believed to be a Hydraphis but unfortunately the specimen was lost.

Extralimital distribution:

Northwest to the Persian Gulf and the coast of East Africa, India, Malaysia and Indonesia and south to northern Australia (Smith, 1926). In freshwater captured in Tonlé Sap Lake, Kampuchea (Bourret, 1934). Absent from Solomon Islands, Philippines, southern China and Japan (Bar me, 1968).

Fang length:

3mm (Minton & Minton 1971).

Average/Maximum venom yield:

8.5/79mg (Habermehl, 1981); 15mg (Worrell, 1963)

Lethal subcutaneous dose:

0.05mg/kg (man, Trethewie, 1971a); 1.5mg (man, US Navy, 1962).

Toxicity LD₅₀:

0.0021-0.0025mg (2.1-2.5μg) (20gm mouse dry wt.i.p., Carey & Wright, 1960); 0.057mg (57μg) (rabbit); 0.061mg (61μg) (Guinea pig); 0.02mg (20μg) (frog) Bar me, 1968.
Toxicity LD_{50}:
0.0025mg (2.5μg) (20gm mouse i.v., Barne, 1963); 0.026mg/kg (26μg) (Barne, 1958).

**Active effects of venom:**

**Antiserum/initial dose:**
CSL Seasnake ?

### TABLE 3
Venom Yield and Potency for Papuan Elapids

<table>
<thead>
<tr>
<th>Species</th>
<th>Venom yield mg</th>
<th>LD_{50} mg/kg</th>
<th>LSD_{1} mg/100gm</th>
<th>LSD_{1} mg/25mg</th>
<th>LSD_{1} mg/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Acanthophis antarcticus</em> (&amp; <em>A. praelongus?</em>)</td>
<td>42-85/235</td>
<td>0.338</td>
<td>0.015</td>
<td>0.025-0.15</td>
<td></td>
</tr>
<tr>
<td><em>Demansia atra &amp; D. papuensis</em></td>
<td>?</td>
<td>14.2 (uninfected)</td>
<td>0.5-1.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Micropechis ikaheka</em></td>
<td>?</td>
<td>0.064</td>
<td>0.00025</td>
<td>0.00034</td>
<td>0.1</td>
</tr>
<tr>
<td><em>Oxyuranus scutellatus</em></td>
<td>100-200/400</td>
<td>1.9</td>
<td>0.16</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Pseudechis australis</em></td>
<td>180/600</td>
<td>0.02</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Pseudechis papuensis</em></td>
<td>200/494</td>
<td>0.041</td>
<td>0.0025</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Pseudonaja textilis</em></td>
<td>2-5/67</td>
<td>0.0025</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Enhydrina schistosa</em></td>
<td>8.5/79</td>
<td>0.0025</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. LSD = certain lethal subcutaneous dose
2. saline value not comparable with other values in table

**Clinical Features Of Papuan Elapid Snakebite.**

**Examination of the Wound.**

In cases of true viper or pit viper bites the large fangs, which may measure up to 30mm, frequently leave obvious puncture wounds surrounded by extensive areas of swelling, oedema, discoloration and tenderness. However, the fangs of elapids are much smaller, rarely exceeding 10mm, and many of the accompanying symptoms may be absent. The small puncture wounds made by small fangs may easily close up and, unless accompanied by serum weeping or bleeding, they may become impossible to locate, especially on the bare feet of native people with their tough skins and numerous abrasions, cuts and other day-to-day injuries. If bite or puncture wounds. Lack of venomous species such as the fearsome injury. Puncture wounds. Treble puncture wounds of venomous snakes have a more rapid, and soon be shed. The bite will be on the side or one behind the other. Multiple bites occur, indicated by one, two or more lacerations. Inoculation may be absent. The apparent puncture injuries do not necessarily indicate a venomous bite. These signs and oedema occur in approximately 50% of elapid envenoming (Campbell, 1980). However, continued swelling, spread of snake venom.

**The Early Symptoms.**

The early symptoms of envenoming are non-specific categories as defined by Campbell.

**Non-specific symptoms.**

Non-specific symptoms. Campbell reported that elapid envenoming is vomiting, which occurred in exactly half of his study group of patients. Vomiting of blood, spitting of blood, abdominal pain, diarrhoea, vomiting of blood, spitting of blood, vomiting of blood, spitting of blood and passage of blood.

**Muscular paralysis.**

Muscular paralysis of the tongue, eyes, eyelids, limbs, neck, and abdomen. In addition, vomiting would be less than vomiting with traditional emetic herbs. Vomiting of blood has occurred but this approach...
day-to-day injuries. If bite injuries are identified they may consist of lacerations or puncture wounds. Lacerations may be the result of a bite from a non-venomous species such as a python, a large specimen of which can cause a fearsome injury. Puncture wounds may be single, double, treble or multiple. Treble puncture wounds are common in single bite situations as many venomous snakes have a reserve fang ready to replace an old fang which will soon be shed. The bite will, therefore, consist of two close punctures, side-by-side or one behind the other, together with a third puncture wound a short distance away. Multiple bites from taipans or Papuan blacksnakes will be indicated by one, two or more pairs of equidistant puncture wounds. First aid lacerations will often obscure all puncture wounds. Small ecchymoses around apparent puncture injuries, slight oedema and localised swelling do not necessarily indicate a venomous snakebite as non-venomous bites may produce these signs and oedema may occur as a result of torniquet application. In approximately 50% of elapid bite cases there is often no envenoming (Russell, 1980). However, continued wound bleeding indicates injection of a coagulant snake venom.

### The Early Symptoms.

The early symptoms of elapid snakebite in Papua New Guinea fall into three categories as defined by Campbell (1969);

- **Non-specific symptoms:** vomiting, headache, pain in the lymph nodes, abdominal pain, loss of consciousness, general weakness, visual difficulties, sweating, pallor and diarrhoea.
- **Clinical bleeding symptoms:** bleeding from the gums or gingival sulci, vomiting of blood, spitting or coughing of blood stained sputum, wound bleeding and passage of bloody urine.
- **Muscular paralysis symptoms:** difficulty in moving or operating jaw, tongue, eyes, eyelids, limbs or in swallowing.

### Non-specific symptoms.

Campbell reported that the commonest early symptom of Australasian elapid envenoming is vomiting, or the desire to vomit, adding that it occurred in exactly half of his study group of 68 patients with positive envenoming (31 patients vomited and three demonstrated their desire to vomit) between half an hour and twelve hours after the bite. In 27 patients this was the first symptom which indicated a venomous snakebite and it could, therefore, be a useful indicator that envenoming has taken place. However, vomiting could be caused by other factors such as fear or shock which may easily arise in patients who have been bitten by either venomous or non-venomous species. In addition, vomiting would be expected if the patient had previously been treated using traditional emetic herbal methods. Only if there is no history of this remedy can vomiting be considered important as an early sign of systemic envenoming.

The presence of blood in the vomit may also indicate that a venomous bite has occurred but this appears to be rare; it was recorded in only two of

<table>
<thead>
<tr>
<th>Elapids</th>
<th>LSD(^1) mg/100gm</th>
<th>LSD(^1) mg/25mg</th>
<th>LSD(^1) mg/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Guine pig</td>
<td>0.015</td>
<td>0.025-0.15</td>
<td></td>
</tr>
<tr>
<td>Mouse</td>
<td>0.5-1.0</td>
<td>0.0034</td>
<td>0.1</td>
</tr>
<tr>
<td>Man</td>
<td>0.16</td>
<td>0.02</td>
<td>0.05</td>
</tr>
<tr>
<td>0.0025</td>
<td>0.0025</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Campbell's patients. Bites from the following Papuan species have been observed to cause vomiting in patients; death adder, taipan, Papuan blacksnake, small-eyed snake, eastern brown snake and Muller's snake. This latter species of the genus Aspidomorphus is not included in this study as it is not considered a life-threatening species but bites have occurred and victims have experienced unpleasant symptoms.

Campbell records that headaches were common features of envenoming but that they varied greatly in severity and duration and also that their onset can occur anytime during the first four hours after elapine snakebite. He reports that headache is rare in Melanesian society and, therefore, such a symptom is of more importance in Papua New Guinea than it would be in the West. Envenomation from taipan, Papuan blacksnake, death adder, eastern brown snake and small-eyed snake have all been recorded as causing headaches in Papua New Guinea.

Campbell goes to great lengths to emphasise the extreme importance of these first two symptoms in the diagnosis of envenoming by dangerous elapids in Papua New Guinea or Australia, especially as these may soon disappear or diminish. The danger is that if these symptoms are not recognised as signs of envenoming the patient may go on to develop more severe paralytic symptoms.

Localised pain in the lymph nodes is a common feature of snakebite, particularly the bites of viperid snakes, and it has also been recorded following bites from death adder, taipan and Papuan blacksnakes. In common with vomiting and the onset of headache it may be the first obvious symptom; it usually persists for longer than the other two symptoms, for up to two and a half days even after the administration of antivenom, with varying severity.

Abdominal pain was reported by Campbell. He commented that it could be either localised or generalised, mild or severe and extremely variable in its duration, often remaining after antivenom treatment. Pain in abdominal lymph nodes was also noticed in patients. This symptom was common and severe in envenoming resulting from the Papuan blacksnake but was also recorded from the taipan and eastern brown snake.

Either rapid and sudden unconsciousness or a gradual loss of consciousness, preceded by visual difficulties, may occur following envenoming by the taipan, death adder, Papuan blacksnake and eastern brown snake from a few minutes to one hour after the bite. Periods of unconsciousness have lasted for periods of less than one hour to up to twelve hours some patients have failed to recover consciousness and have subsequently died. The presence of a clammy, cold or sweaty skin and a weak pulse led Campbell to postulate that the venom may have had an effect on the circulatory system which resulted in unconsciousness but he also reports that in some cases the loss of consciousness may be caused by the action of the venom on the brain. Loss of consciousness frequently precedes the dangerous paralytic stage of envenoming which usually becomes apparent when the patient regains consciousness and is unable to move. Blasco and Hornabrook (1972) reported a case of small-eyed snake bite in which the patient became unconscious and suffered respiratory arrest over two hours after the envenoming. Cyanosis of lips and gums and the patient failed to respond and subsequently died.

Drowsiness, often cited as an unreliable sign of envenoming, including the consumption of antivenom, was present and drowsiness, suggested by generalised weakness, may be caused by the paralytic effects of the venom. Those bitten may be 'drowsy', and who went to sleep immediately after the bite and who entered a deep sleep. Convulsions have also occurred in Papua New Guinea but these may be the result of the venom.

The venom of the small-eyed snake is diuretic and diaphoretic and although the taipan and eastern brown snake are highly venomous in Australia, Campbell did not report drowsiness as a symptom of Papuan snakebite.

Clinical bleeding symptoms

Bleeding from the gums was common both in localised and systemic envenoming and was not observed following envenoming by the taipan, death adder, small-eyed snake or eastern brown snake. Patients have not been observed following bites from 'Papuan mulga' (Aspidomorphus melanosoma) which was probably the species that caused haemorrhage and its venom was credited with procoagulant activity in the blood. Subsequent bleeding following envenoming by the Papuan blacksnake and mulga brown snake (Pseudonaja ater) was probably the species of snake responsible for the bleeding. The Papuan blacksnake and mulga or king brown snake (P. ater) have strongly coagulant qualities in their venom and may be the species responsible for bleeding from the fang puncture wounds.
Papuan species have been adder, taipan, Papuan snake and Muller's snake. This included in this study as it is have occurred and victims

Common features of envenoming and also that their onset after elapine snakebite. He society and, therefore, such a guinea than it would be in the snake, death adder, eastern recorded as causing headaches after elapine snakebite. He death adder and, therefore, such a guinea than it would be in the snake, eastern recorded as causing headaches in its venom.

The extreme importance of envenoming by dangerous elapids as these features may soon develop more severe paralytic symptoms. Common feature of snakebite, also been recorded following eastern brownsnake. In common with the first obvious symptom; it symptoms, for up to two and a common, with varying severity.

Campbell commented that it could be extremely variable in its short. Pain in abdominal lymph was common and severe in but was also recorded from emotions or a gradual loss of consciousness from a unconsciousness have lasted hours some patients have died. The presence of a brownness of the patient which resulted in some cases the loss of venom on the brain. Loss of consciousness paralytic stage of when the patient regains

Surnabrook (1972) reported that became unconscious and suffered respiratory arrest within five minutes of admission to hospital, just over two hours after the envenoming. Other early symptoms included vomiting, cyanosis of lips and gums, heainess of limbs and shortness of breath. This patient failed to respond to death adder antivenom or resuscitation and subsequently died.

Drowsiness, often cited as a characteristic of Australian elapid poisoning, is an unreliable sign of envenoming since it may be caused by other factors including the consumption of alcohol or genuine tiredness. However, apparent drowsiness, suggested by general weakness or fatigue and ptosis, may be caused by the paralytic effects of the venom on the muscles. Patients who appeared to be 'drowsy', and who went to sleep, frequently awoke later in a totally paralysed state. Campbell is equally sceptical of the value of irritability as a symptom as it may be as much the product of fear as of the effects of envenoming. Convulsions have also occurred following venomous snakebites in Papua New Guinea but these may be the result of hypotension rather than a direct cause of the venom.

The venom of the small-eyed snake is recorded by Slater as causing diarrhoea, but although this has occurred as a result of other snakebites in Australia, Campbell did not witness it and it does not appear to be a common symptom of Papuan snakebite.

Clinical bleeding symptoms.

Bleeding from the gums and gingival sulci is an important symptom of systemic envenoming and blood stained sputum or vomit has been recorded following envenoming by taipan or eastern brownsnake. These symptoms are not observed following death adder envenoming. Both taipan and brownsnake have strongly coagulant venoms. Campbell reported similar symptoms following bites from "Papuan blacksnares" but it is now thought that the taipan was probably the species responsible. Marshall and Herrmann (1983) deny procoagulant activity in the venoms of both Papuan blacksnake (P. papuanus) and mulga brownsnake (P. australis). Campbell reports that the venom of the mulga or king brownsnake is powerfully anticoagulant but authorities are divided as to whether any of the genus *Pseudechis* possess anticoagulant qualities in their venoms. However, Marshall and Herrmann (1983) reported that three species of interest here; *Pseudechis australis, P. papuanus* and *Acanthophis antarcticus*, did demonstrate the possession of a powerful unknown anticoagulant factor which was neither antithrombin nor fibrinogenolytic. Some snake venoms *in vitro* may be demonstrated to possess both coagulant and anticoagulant properties depending on the concentration of venom tested (Mebs, 1978) and incoagulable blood *in vivo* does not necessarily indicate an anticoagulant venom since coagulant factors will also cause bleeding via a defibrination syndrome. However, the anticoagulant properties of many Australasian venoms are probably of little clinical significance. Regardless of whether the venom is coagulant or anticoagulant positive envenoming can be diagnosed if continued bleeding or serum weeping from the fang puncture wounds or 'first aid' lacerations is observed.
Dark urine may also be observed but it should be tested to determine whether the cause is haemoglobinuria or myoglobinuria. Although rectal bleeding and melaena have been reported following Australian elapid snakebites, neither has been reported from Papua New Guinea.

**Muscular paralysis symptoms.**

Early symptoms of muscle paralysis due to envenoming may manifest as difficulties in moving the eyelids, eyes, jaw, tongue or in swallowing and as a general weakness of the limbs, but it is not indicative of a bite from any particular species of Papuan elapid since the entire group is characterised by the possession of neurotoxins in the venom.

On occasions none of the above symptoms is seen and the first sign of envenoming may be severe peripheral curare-like neuromuscular paralysis of the respiratory and chest muscles. This usually indicates that either a death adder or a small-eyed snake was responsible but occasionally a taipan bite may proceed to neuromuscular paralysis without early symptoms becoming apparent.

In conclusion it is recommended that the first two symptoms, vomiting and headache, should not be ignored or disregarded as they are frequently the earliest features of a potentially serious, life-threatening snakebite. If they are overlooked the patient may subsequently suffer loss of consciousness and paralysis. Continual observation of the patient is essential as many of these symptoms, including the two most important, are only temporary, often of short duration and may be misinterpreted.

**The Clinical Signs.**

**Enlargement and/or tenderness of lymph nodes.**

One or both of these signs can occur within the first few hours after a bite and may endure for up to two days even after administration of antivenom. Although reported as a common feature of 'Papuan blacksnake' bites, tenderness of lymph nodes is not confined to envenoming by that species and may follow bites from taipan or death adder. Lymph node tenderness is due to the action of the lymphatic system in the absorption of venoms with high molecular weight such as those of the vipers and pit-vipers of America, Asia and Africa and many Australian elapids. Campbell (1969), provides details of a lymph node biopsy carried out at the Kanematsu Memorial Institute of Pathology in Sydney Hospital following envenoming from an elapid which was probably a small-eyed snake. He quotes Professor ten Seldam of the Department of Pathology at the University of Western Australia who reports that the severe dilation of the peripheral sinusoids with oedema in the lymph node surrounds and areas of haemorrhage and necrosis within the lymph node, without severe inflammation, indicated that a very toxic substance had entered the lymph node and caused a similar reaction to that seen in lymph node biopsies from two fatal Indonesian snakebites.

Abdominal pain and muscle:

Both tenderness and muscle may be localised following the bite of a taipan, and the tenderness recorded by Campbell following the bite of the effect of the venom in any history of snakebite the possibility of appendicitis.

**Muscular paralysis and the voluntary nervous system.**

Paralysis of the voluntary nervous system is often commencing either at the time of admission to hospital, and which may endure for many hours to become severe. Difficulties in ocular muscle paralysis followed by paralysis of the jaw, tongue, palate and pharynx may place until much later. Papuan elapids bite all times, even in cases of the presence of snakebite.

The early signs of muscle paralysis, which is characterized by bulbar and proximal limb paralysis, acting on the acetylcholine receptors at the neuromuscular junction may result in the impairment of respiratory function.

If the patient is in a resting position the lower jaw to drop back slightly, the tongue may extend his tongue. Speech may be slurred and the flow of saliva indicates that swallowing is a danger of inhalation of saliva.

The next muscles to show evidence of paralysis are those controlling expression, but this may take many hours to occur. A generalised muscle paralysis. In the absence of severe symptoms, the patient may not be able to be completely unconscious, but may still have control over his upper limbs. He may slightly twist the pelvis. Curvature of the spine is a small area surrounding the site of the puncture.

Paralysis of the chest muscles is obviously a serious sign and the patient may be complete.

Respiratory obstruction may result in air entering the lungs from where it cannot be exhaled. Also obstruction of the airway may occur, and it may be necessary to intubate.
would be tested to determine globulinuria. Although rectal bleeding following Australian elapine envenoming may manifest as dark blood or in swallowing and as a haemorrhage is indicative of a bite from any elapid group is characterised by extensive local swelling being seen and the first sign of envenoming may be the neuromuscular paralysis of the tongue. This indicates that either a death adder or occasionally a taipan bite may result in early symptoms becoming apparent.

Both abdominal pain and muscle guarding may manifest as localised muscle guarding may be mild or severe, general or as they are frequently the first symptoms following a bite from the Papuan blacksnake, and possibly also the taipan, and the tenderness in the region of the inguinal ligament was also recorded by Campbell following a death adder bite. The tenderness may be due to the effect of the venom on the abdominal lymphatic system. Without a history of snakebite the patient may be diagnosed as suffering from acute appendicitis.

Abdominal pain and muscle guarding.

Both tenderness and muscle guarding may be mild or severe, general or localised following the bite of the Papuan blacksnake, and possibly also the taipan, and the tenderness in the region of the inguinal ligament was also recorded by Campbell following a death adder bite. The tenderness may be due to the effect of the venom on the abdominal lymphatic system. Without a history of snakebite the patient may be diagnosed as suffering from acute appendicitis.

Muscular paralysis and the neurotoxic effects.

Paralysis of the voluntary muscles is a common sign of elapid envenoming, often commencing either soon after the bite has occurred, or later, after admission to hospital, and continuing for up to thirty hours with increasing severity until it becomes potentially life-threatening. Slight ptosis and other difficulties in ocular muscle movement are usually the first signs of elapid-invoked paralysis followed by visual difficulties and muscular paralysis of the jaw, tongue, palate and pharynx. Complete ptosis, if it occurs, may not take place until much later. Pupil reactions to light can still, however, be observed at all times, even in cases of total eye paralysis.

The early signs of myasthenia gravis may occur. This neuromuscular disorder, which is characterised by the weakness and fatigability of ocular, bulbar and proximal limb muscles, is caused by a decrease in available acetylcholine receptors at the neuromuscular junctions. In severe instances it may result in the impairment of respiratory activity (Drachman, 1987).

If the patient is in a recumbent position jaw muscle paralysis may cause the lower jaw to drop back slightly and it may become impossible for the patient to extend his tongue. Speech deteriorates until it becomes impossible and drooling of saliva indicates that swallowing is also difficult. In a recumbent patient there is a danger of inhalation of saliva.

The next muscles to show signs of paralysis are usually the facial muscle, controlling expression, but complete paralysis of the facial muscles may take many hours to occur. A general weakness of the limbs is an early sign of general muscle paralysis. In the early stages the standard reflex actions can still be registered but later these too will become subdued. Whilst a severely paralysed patient may not be able to sit up, roll over or turn his head, and may even appear moribund, with almost totally closed eyes and expressionless face, he may still have control over movements of his fingers and toes and be able to slightly twist the pelvis. Cutaneous sensation seems to remain intact except for a small area surrounding the actual bite area.

Paralysis of the chest muscles and the muscles of the diaphragm is obviously a serious sign and respiration may cease even before limb paralysis is complete.

Respiratory obstruction may result from oral or chest secretions draining into the lungs from where the patient is unable to cough them up or spit them out. Also obstruction of the airway by the paralysed tongue can be dangerous and it may be necessary to insert an artificial airway. These problems, coupled
with decreased respiratory activity caused by the effect of the venom on the respiratory chest muscles and the diaphragm, eventually result in cyanosis, anoxia, unconsciousness and death due to asphyxia. In most instances of severe paralysis it is necessary to initiate endotracheal intubation or to perform a tracheotomy and remove sputum from the airway. Campbell estimated that most patients would not survive for more than one or two hours beyond the point where a tracheotomy was indicated. Tracheotomies were performed from three to 96 hours after envenoming and the time at which they became necessary could be used as an indicator of the degree of severity of the bite and the probable dosage of venom injected. Campbell reported that although antivenom administered during the early stages of envenoming was frequently successful in reversing or diminishing the degree of muscle paralysis, in the later stages only patients with bites resulting from death adders showed a significant response to antivenom and it would appear that in the cases of Papuan blacksnake or taipan bites, where antivenom is often not capable of reversing paralysis, intubation, tracheotomy and life-support by other means are essential in order to preserve life.

Even so, elapid snake venoms, which contain powerful neurotoxins, are capable of causing extremely powerful, and sometimes irreversible, peripheral neuromuscular blockages. Sutherland (1983) suggests that venoms containing postsynaptic neurotoxins such as the death adders (Acanthophis sp.) probably causes a more rapid onset of paralysis than the venoms with presynaptic components such as the Australian tiger snake (Notechis scutatus). However, postsynaptic neurotoxins are more easily reversed with antivenom than that caused by presynaptic venoms.

**Haemotoxic effects.**

In the section on clinical bleeding symptoms it was noted that both anticoagulant and coagulant properties may be present in the same venom and both may cause the blood to become incoagulable. However, the effects of the anticoagulant factors do not seem to have any serious bearing on the clinical effects and treatment of the envenoming and the dangerous haemotoxic effects of Australo-Papuan elapine snakebite result more from the procoagulant factors of the venom.

Strongly procoagulant venoms such as those of the taipan and the eastern brownsnake are very diffusable and cause a conversion of prothrombin to thrombin resulting in a fibrinogenaemia (Sutherland, 1983). Both of these species possess venoms which are complete prothrombin activators capable of converting prothrombin to thrombin in either plasma deficient in factor V or in normal plasma (Denson; 1969, Mebs, 1978) but the venoms of the death adder and the Australian tiger snake (Notechis scutatus) are incomplete prothrombin activators which cause coagulation only in the presence of factor V (Mebs, 1978). A thrombosis may cause death through extensive haemorrhaging from the mucus membranes (Kellaway, 1938) but the presence of incoagulable blood does not necessarily result in haemorrhagic symptoms. Many coagulant venoms will not cause haemorrhage unless a secondary factor is involved such as a medical history of sto...
The effect of the venom on the patient may eventually result in cyanosis, which in most instances of severe envenoming was frequently accompanied by muscle paralysis, in the death adders showed a apparent that in the cases of death adders is not capable of life-support by other means

The powerful neurotoxins, are sometimes irreversible, peripheral symptoms that venoms containing death adders (Acanthophis sp.) is than the venoms with an tiger snake (Notechis sp.) more easily reversed with

It was noted that both present in the same venom and However, the effects of the poisonous bearing on the clinical dangerous haemotoxic effects are from the procoagulant venom of the taipan and the eastern brownsnake, as a medical history of stomach ulcers or damage to the actual blood vessels through the actions of another venom factor, a haemorrhagin. Haemorrhagins are not generally considered common factors in Australasian snake venoms. Even so, due to the incoagulability of the blood there will be continued bleeding from the fang puncture marks and also from any 'first aid' incisions or tracheotomy wounds.

The defibrination of the blood by a procoagulant venom can also cause symptoms such as bleeding from the gums and coughing and spitting of blood stained sputum which have already been recorded as important early symptoms of venomous snakebite.

Campbell carried out numerous haematological tests, including the now out-dated rabbit anti-fibrin test, (RAF), on patients admitted to Port Moresby Hospital with snakebite envenoming. He found many of the usual blood tests were of limited value in determining the type of snake responsible and the degree of envenoming. However, the simple bedside whole blood clotting test is a quick and useful technique for determining whether the patient has received a bite from a species possessing a coagulant venom.

Campbell also conducted fibrinogen titres to determine the amount of fibrinogen present in the patient’s plasma. He reports that whereas the normal titre for a Caucasoid is 1 in 64, the normal titre for a Melanesian is 1 in 32, the addition of EACA raises the Melanesian titre to that of a Caucasoid but the presence of a coagulant venom such as that of the taipan will reduce the titre to 1 in 16 or even zero.

Prolonged whole blood coagulation and a low fibrinogen titre would suggest that the snake concerned was one of those species with a strongly coagulant venom. Antiserum is usually effective in reversing these effects.

Haemolysis of the blood corpuscles is a further consequence of certain Papuan elapine bites. The venom of the blacksnake, and possibly also the mulga brownsnake, is reported to be strongly haemolytic and it is capable of causing high levels of haemoglobinuria. The taipan is thought to be weakly haemolytic but there has been at least one case of a taipan bite in Papua New Guinea resulting in haemoglobinuria. Death adder and eastern brownsnake venoms are also weakly haemolytic. Antiserum also has a powerful effect in reversing these haemolytic symptoms.

**Proteinuria and Haemoglobinuria.**

Proteinuria may be a consequence of Papuan elapine envenoming but urine samples tested soon after poisoning rarely register any significant level of proteinuria. Similarly, after administration of antivenom the level of any apparent proteinuria may drop off dramatically.

In addition, blacksnake and taipan venoms may have a severely damaging effect on the kidneys if antivenom is delayed or not given. In cases of the late administration of antivenom it is frequently necessary to maintain dialysis due to the renal damage caused by the venom prior to its administration. If antivenom therapy has been greatly delayed the renal damage may be severe and irreversible.
Haemoglobinuria may be evident quite early in the history of envenoming usually before severe paralysis has occurred, and it is normally associated with a high level of proteinuria. Haemoglobinuria is a sure sign that a severe envenoming has taken place and is usually a strong indication of either a Papuan blacksnake or taipan bite. There is also a record of haemoglobinuria from a possible Australian mulga brownsnake bite, (Campbell, 1969), a species which may yet be recorded in Western Province and which is closely related to the Papuan blacksnake, both members of the genus *Pseudechis* (Mengden, Shine & Moritz, 1986). The relationship between haemoglobinuria and anuria causing renal failure is not clear.

The absence of proteinuria or haemoglobinuria does not preclude the possibility that a venomous snakebite has occurred. For example, death adder bites rarely cause proteinuria and never haemoglobinuria. These signs may also be missed if the patient is not seen until some time after antivenom has been administered.

**Myotoxic effects.**

The venom of the little known small-eyed snake is believed to be highly myotoxic causing myalgia, muscle tenderness and severe neuromuscular paralysis. Since small-eyed snake venom does not demonstrate coagulopathy the presence of ‘dark’ urine will indicate myoglobinuria rather than haemoglobinuria (Sutherland, 1983). Hudson and Pomat (1988) report 16 cases of envenoming from Madang Province, 11 of which demonstrated ‘dark’ urine. Six of these patients subsequently suffered renal failure probably caused by the delay between the venomous bite and the administration of antivenom. These 16 cases exhibited signs of myotoxicity or rhabdomyolysis (striated muscle destruction) and since 15 of these bites were from terrestrial snakes and the venom of the only other dangerous elapid in the region, the death adder, is non-myotoxic in its effects it must be considered that these symptoms occurred as a result of small-eyed snake envenoming. In three of the cases the description of a 'long white snake' suggests that the small-eyed snake was responsible. If untreated the consequence of myoglobinuria may be oliguria within 24 hours leading to renal failure and death several days later (Sutherland, 1983). This species has been responsible for several serious human accidents and at least one documented and several other suspected deaths (Blasco & Hornabrook, 1972 and Hudson & Pomat, 1988). Myoglobinuria frequently also causes cerebral hypoxia in severely envenomed patients (Sutherland, 1983).

The venom of the common seasnake (*Enhydrina schistosa*), also causes myotoxic or rhabdomyolytic signs and symptoms, in common with many other hydrophiiids, which may be accompanied by neuromuscular paralysis (Hudson & Fromm, 1986). Muscle tenderness is a very prominent clinical feature of envenoming by *E.schistosa* (Sutherland, 1983) together with myoglobinuria (Reid, 1961). The hyaline lysis and necrosis of the skeletal muscle is a common pathological feature of *E.schistosa* envenoming (Marsden & Reid, 1961). Other signs and symptoms of sea snake envenoming include paresis; aching and muscular stiffness and weal; nasal regurgitation; failing vision; and the severe necrosis of the kidneys where marginally the kidneys of those that are still alive after 48 hours, who are still alive after 48 hours (Barme, 1965).

Cardiovascular effects.

The only Papuan-Australian cardiotoxic is that of the neurotoxic. Whether the venom qualities is unknown although the Papuan blacksnake bite.

The cardiotoxic effects of the presence of a phosphatase in the situation is not clear whether or not it also release adenosine, a hypotension may occur with or without the patient’s heart. This is termed primary hypotension with blueing lips and extremities, almost undetectable respiratory and almost undetectable respiratory and moribund recovery can be frequent.

It is believed that this autopharmacological effect or tissues and cells into the bloodstream, “slow-reacting substance”, differing effects on the heart (Mebs, 1970) and Australia into the plasma, from the action of specific venom enzymes. The effects of the release of stimulation, vasodilation (Silva, 1970). If large quantities of circulation the result will be Bradykinin has a very short half-life and wear off rapidly (Mebs, 1970) responsible for causing autopharmacological effects,

Although the hypotension resembles an anaphylactic response, complete recoveries from
in the history of envenoming it is normally associated with a sure sign that a severe strong indication of either a record of haemoglobinuria (Campbell, 1969), a species and which is closely related to genus Pseudechis (Mengden, haemoglobinuria and anuria which does not preclude the death adder. For example, death adder haemoglobinuria. These signs may also some time after antivenom has been

snake is believed to be highly and severe neuromuscular that demonstrate coagulopathy and myoglobinuria rather than death several days later for several serious human deaths several days later for several serious human deaths (Mengden & Pomat, 1988). Myoglobinuria may be oliguria in the region, the death death several days later for several serious human deaths (Mengden & Pomat, 1988). Myoglobinuria may be oliguria severe from terrestrial snakes which it is believed to be highly and severe neuromuscular strain and which is closely related to genus Pseudechis (Mengden, haemoglobinuria and anuria. For example, death adder haemoglobinuria. These signs may also some time after antivenom has been

snake is believed to be highly and severe neuromuscular strain and which is closely related to genus Pseudechis (Mengden, haemoglobinuria and anuria. For example, death adder haemoglobinuria. These signs may also some time after antivenom has been

Cardiovascular effects.

The only Papuan-Australian snake venom thought to be severely cardiotoxic is that of the mulga or king brownsnake which is only weakly neurotoxic. Whether the venom of the Papuan blacksnake has any cardiotoxic qualities is unknown although Campbell reports normal ECGs from six cases of blacksnake bite.

The cardiotoxic effects of snake venoms are thought to be partially due to the presence of a phosphatase and proteolytic enzymes or a neurotoxin but the situation is not clear with regard to Australian snake carditoxins which also release adenosine, a powerful cardiac depressant, into the heart (Trethewie, 1971 p.89).

Hypotension may occur rapidly in Papuan-Australian elapine bites either with or without the patient losing consciousness or developing convulsions. This is termed primary hypotension and it is signified by pallor, a bloodless face with bluing lips and extremities, cold clammy skin, a slow, weak pulse and almost undetectable respiration. Although the patient may appear quite moribund recovery can be extremely rapid and total.

It is believed that this sudden drop in blood pressure is caused by the autopharmacological effects of the venom on the release of substances from the tissues and cells into the blood. Various venoms cause the release of histamine, "slow-reacting substance", adenyl compounds and anaphylatoxins which have differing effects on the homeostasis of the victim. The venoms of many viperids (Mebs, 1970) and Australian elapids (Warrell, pers.comm.) release bradykinin into the plasma, from the protein precursor bradykininogen, through the action of specific venom enzymes called kinogenases (Rocha e Silva, 1970). The effects of the release of bradykinin into the plasma are smooth muscle stimulation, vasodilatation, increased capillary permeability and pain (Rocha e Silva, 1970). If large quantities of these substances are introduced into the circulation the result will be a rapid drop in blood pressure and 'shock'. Bradykinin has a very short half life and the effects of the primary hypotension wear off rapidly (Mebs, 1978). Other "slow-reacting substances" may also be responsible for causing autopharmacological changes and effects in Australo-Papuan snakebite victims but in many cases their mechanisms are not fully understood.

Although the hypotension caused by Australasian elapine venoms resembles an anaphylactic reaction, Campbell points out that the sudden and complete recoveries from primary hypotension are not typical of anaphylaxis.
Pretreatment with an anti-histamine does not apparently prevent primary hypotension.

If sufficient damage has been done to the circulatory system by either the haemorrhagic qualities or some other factor of the venom a secondary form of hypotension may occur due to fluid loss into the tissues. This is a common feature of viper and pit-viper venoms but the only snakes of interest to this survey which may contain sufficient quantities of haemorrhagin in their venom are members of the genus *Pseudechis* (Campbell, 1969 p.167).

Campbell reported that anoxia was also a major cause of secondary hypotension in severely paralysed patients suffering from "Papuan blacksnares" (possibly taipans) envenoming and correct and intensive nursing procedures could prevent such an occurrence. Even changing the patient's posture in the bed so that a tracheotomy can be performed has resulted in secondary hypotension with fatal results. Pulmonary oedema due to over-transfusion of blood and normal saline has also been responsible for causing death through secondary hypotension in blacksnake bite regardless of the effects of the venom itself.

**Treatment Of Papuan Snakebite.**

This section is only intended as a brief resume of advice proffered by Campbell and Sutherland regarding the treatment of elapid snakebite in Papua New Guinea since the current theories and techniques for the treatment of venomous snakebite will be covered in more detail by another author (see Warrell, this volume). Sutherland (1983), describes in detail the treatment of envenoming in Australo-Papuan elapine snakebite victims.

**First Aid.**

Reassurance of the patient is extremely important, especially in the case of children, and aspirin or alcohol given in extreme moderation may be helpful as a calming influence according to Reid (1980). Possibly a placebo medication could also be considered. Above all the patient should be discouraged from exerting himself. The bitten limb should be elevated, if possible, and the victim should be taken to seek medical help as soon as possible. If should be remembered that Australia and Papua New Guinea contain some of the World's most venomous snakes and that the neurotoxins of some species do not respond well to antivenom once a significant amount of time has elapsed. The identity of the snake is extremely important but frequently overlooked at this stage. If the snake has been killed it should be taken with the patient to hospital. However, further injury should not be risked in a fruitless search for the reptile by either the patient or his companions as some species are capable of delivering several fatal bites. A severely injured and apparently 'dead' snake should not be handled as it may 'come back to life' momentarily and administer a serious bite. Even a decapitated head can deliver a bite should a jaw muscle be stimulated. The body of the reptile should be lifted with a stick into a secure box or glass jar, not a bag which may be sat upon, for the journey to hospital where it should be identified. The snake's body the patient's approximate length, colour, locality and time of day will be noted down accurately and identification of the species in certain regions of Papua New Guinea is of limited value.

Over-exuberant bush treatment of puncture wounds should be avoided and excessive blood loss (blood procoagulant and has cause to tendons (Reid, 1980); delayed obscuring of recognisable sight of blood in children is a tragic outcome. It is unlikely the venom, especially if injected more blood vessels for venenous envenoming to their frequent misuse. A pressure bandage designed to restrict it entirely. However, may be necessary (Reid, 1980) regular short intervals. To splinted with a suitable piece.

The patient should be envenomation as detailed The airway must be kept clear, necessary by tracheotomy patient dispatched to hospital.

Excessive quantities of hazardous tribal magical must be taken to avoid death necessary to work in coll. than risk alienation, rese from the Common available from the Common applicable for Papua New.
apparently prevent primary haemorrhagic shock by either the central nervous system or by the local production of a secondary form of ahaemorrhagin in their venom (J. W. J. 1969 p.167). The major cause of secondary death from “Papuan blacksnake” envenoming is not due to over-transfusion of the patient’s posture in the catheterisation area, which has resulted in secondary death due to over-transfusion of the venom. This is indicated by another author (see below) who has in detail the treatment of this snake in Papua New Guinea.

Over-exuberant bush techniques such as incision and bleeding of the fang puncture wounds should be strongly discouraged as these practices may lead to excessive blood loss (especially in cases where the venom is strongly procoagulant and has caused widespread defibrination); damage to nerves and tendons (Reid, 1980); delayed healing and the possibility of infection and the obscuring of recognisable fang wounds. In addition, wound cutting and the sight of blood in children is likely to lead to intense fright, panic and increased heart beat and circulation which will further increase the possibilities of a tragic outcome. It is unlikely that suction will remove a significant quantity of the venom, especially if injected into tissue, and bleeding probably will open up more blood vessels for venom absorption. Tourniquets are also unpopular due to their frequent misuse. A more acceptable alternative might be a crepe-type pressure bandage designed to slow down circulation in the bitten limb but not restrict it entirely. However, if a large quantity has been injected a tourniquet may be necessary (Reid, 1980) but it is important to relieve the pressure at regular short intervals. To prevent movement in the bitten limb it should be splinted with a suitable piece of wood.

The patient should be observed for the early symptoms of serious envenomation as detailed in the clinical section; vomiting, headache, ptosis etc. The airway must be kept clear of mucus and respiration must be maintained, if necessary by tracheotomy and artificial respiration in very serious cases and the patient dispatched to hospital by the most rapid means possible. Excessive quantities of alcohol, emetic herbal remedies and potentially hazardous tribal magical techniques should be avoided if possible but care must be taken to avoid distrust and infringement of tribal taboos. It may be necessary to work in collaboration with the village tribal magic man rather than risk alienation, resentment and the possible withdrawal of assistance or transportation.

A pamphlet on First Aid for Snakebite in Australia (Sutherland, 1988) is available from the Commonwealth Serum Laboratories, Victoria which is also applicable for Papua New Guinea.

Hospitalisation.

Campbell advises that all suspected venomous snakebite victims should be admitted to hospital and put under regular hourly observations for pulse rate, respiration rate and blood pressure and the presence of the early symptoms of snakebite envenoming. If symptoms are present already or subsequently
appear, the patient should be checked for difficulty in swallowing or breathing. All urine should be tested for protein and blood and the colour noted.

The aims of the physician should be to:

a. neutralise the effects of the venom using antivenom,
b. relieve respiratory obstruction and maintain an airway, by endotracheal intubation or tracheotomy if necessary,
c. deal with respiratory insufficiency, by artificial respiration if necessary.

If the signs and symptoms of envenoming occur the patient should be treated with antivenom following the recommendations of the manufacturers. Antivenom for use in Papua New Guinea is produced by the Commonwealth Serum Laboratories in Victoria, Australia. Depending on the identity of the snake the practitioner can administer Monospecific Death Adder, Taipan, Blacksnake or Brownsnake antivenoms. In cases where the snake was not positively identified, a Polyspecific Papuan antivenom is available. Tiger snake antivenom is also, reportedly, effective in the treatment of certain Papuan snakebites. CSL also developed a Venom Detection Kit (chap.5, Sutherland, 1983) which was intended to make it possible to identify the snake species responsible for an envenoming in a very short time and allow the use of the correct monospecific antivenom. The kits comprised a series of colour coded capillary tubes attached to a fine syringe. Each section was coated with a specific antivenom and the venom's identity was indicated by a colour change in the section coated with antivenom specific to that species, or group of related species. However, they have not proved to be very successful due to the occurrence of paraspecific reactions and false positive results (Warrell, pers. comm.) and whether such a finely-tuned technique, if fully reliable, would be of much value in a small Papuan clinic must also be queried.

The elapsed time between the snakebite and the administration of antivenom is extremely important in the treatment of Papuan elapid bites. It is reported that cobra bites respond well to antivenom even if administered extremely late in the treatment, when the patient is moribund and lifeless (Ahuja & Singh, 1954) and it seems likely that the effects of death adder venom may respond similarly (Campbell, 1967). However, taipan and blacksnake venoms are very different and late administration of the either polyspecific or monospecific antivenom frequently has little effect on the neurotoxic component of the venom although it may reverse the effects of the haemolytic and procoagulant venom factors. The recommended antivenom dose is intended to neutralise the average venom yield of the species concerned. In cases of taipan or blacksnake envenomation, where multiple bites are not uncommon, Campbell (1967), suggests that up to ten times the recommended dose may be required to even stand a chance of reversing paralysis, especially if antivenom is not administered until a considerable time after the occurrence of the bite.

The Tensilon (edrophonium) test for myasthenia gravis, caused by a decrease in acetylcholine receptors at the neuromuscular junctions, is useful in determining whether skeletal muscle weakness and general fatigue are due to snakebite envenoming with a neurotoxic venom. A bolus of 10mg of
in swallowing or breathing, and the colour noted.

if the airway is not patent, by endotracheal intubation or by needle aspiration if necessary.

if death should occur the patient should be transported with the colour noted. The Commonweal th Department of Health can provide the identity of the species responsible. The Colour Kit (chap.5, Sutherland, 1987) can be used to identify the snake species concerned. In some instances, or if the patient is moribund and lifeless, a series of colour coded plastic patches were coated with a product that, when activated by a colour change due to the haemolytic effects of the venom, allows the species concerned to be queried.

If the administration of antivenom is delayed, the chance of anaphylaxis caused by the intravenous introduction of antivenom is significant. An antivenom dose of 1-2mg (Clarke, 1987) should be followed by slow direct pulse injection (Malasit et al, 1986), or by slow infusion over thirty minutes, together with a glucose or saline solution of equal volume, via an infusion giving set. In case of anaphylaxis, or other allergic reaction, it is advisable for the practitioner to have adrenaline, (0.5ml of 1:1000 adrenaline), and the necessary equipment for emergency resuscitation standing by.

Adrenaline is injected intravenously following a test dose of 0.01mg. A rapid improvement lasting 2-3 minutes indicates a positive result but the test should also be performed using a control substance such as normal saline to eliminate error. Care should also be taken when conducting the Tension test as it may cause bronchial obstruction and syncope (Clarke, 1987). Facilities for resuscitation should therefore be available. Considerable success has been reported for this technique in determining neurotoxic snake bite envenoming by Australian elapids (Warrell, pers. comm.), i.e. in foals bitten by Eastern brownsnakes, Pseudonaja textilis, and in humans receiving bites from Philippine cobras, Naja naja philippinensis, (Watt et al, 1986).

Blood gases can be estimated regularly in large hospitals to test for respiratory insufficiency caused by ongoing neuromuscular paralysis but in smaller field clinics adequate results can be obtained using peak flow metres to measure vital capacity and expiration.

A bolus of 10mg of edrophonium, an anticholinesterase, is injected intravenously following a test dose of 1-2mg (Clarke, 1987). A rapid improvement lasting 2-3 minutes indicates a positive result but the test should also be performed using a control substance such as normal saline to eliminate error. Care should also be taken when conducting the Tension test as it may cause bronchial obstruction and syncope (Clarke, 1987). Facilities for resuscitation should therefore be available. Considerable success has been reported for this technique in determining neurotoxic snake bite envenoming by Australian elapids (Warrell, pers. comm.), i.e. in foals bitten by Eastern brownsnakes, Pseudonaja textilis, and in humans receiving bites from Philippine cobras, Naja naja philippinensis, (Watt et al, 1986).

Blood gases can be estimated regularly in large hospitals to test for respiratory insufficiency caused by ongoing neuromuscular paralysis but in smaller field clinics adequate results can be obtained using peak flow metres to measure vital capacity and expiration.

To reduce the chance of anaphylaxis caused by the intravenous introduction of antivenom it should be administered either by slow direct pulse injection (Malasit et al, 1986), or by slow infusion over thirty minutes, together with a glucose or saline solution of equal volume, via an infusion giving set. In case of anaphylaxis, or other allergic reaction, it is advisable for the practitioner to have adrenaline, (0.5ml of 1:1000 adrenaline), and the necessary equipment for emergency resuscitation standing by.

Allergic reactions may vary from severe and sudden anaphylaxis to abdominal pain, lasting and widespread urticaria or shivering and a rise in body temperature. In the long term serum sickness may occur up to nine days after antivenom treatment and administration of steroids may be necessary. Following the administration of antivenom the patient should continue to be observed as antivenom may not immediately arrest the onset of life-threatening paralysis, particularly in the case of taipan or blacksnake bites. If partial paralysis of the chest, palate or pharynx occurs the patient should be nursed on one side and observed closely in case endotracheal intubation or a tracheotomy is required. A build up of oral secretions in the pharynx will indicate that immediate action is necessary. Campbell estimated that if reliance was placed solely on administration of antivenom at least 25% of patients, who could have been saved if adequate and unimpeded respiration was maintained, would die. Following the operation, close and constant nursing and monitoring for blockage of the airway will also be necessary. In serious cases of paralysis artificial respiration may become essential and the need for continual and skilled nursing and medical supervision cannot be over emphasised. Eventually, under these conditions, even severe paralysis should diminish although this may take up to seven days of intensive care.

Campbell gravis, caused by a neurotoxic snake bite, is useful in detecting the presence of curare. A bolus of 10mg of
Appendix One:
Key to Papuan Snake Families.

Five of the World's ten families of snakes are represented in the herpetofauna of New Guinea. The taxonomic position and relationships of highly venomous sea snakes and the dangerous land snakes of Australasia have long been a cause of controversy. Smith, Smith and Sawin, (1977) confined the usage of the family name Elapidae to the terrestrial proteroglyphous snakes of Africa, Asia and America and the amphibious sea kraits of the genus *Laticauda* together termed "Palatine Erectors". The true sea snakes and all terrestrial Australasian proteroglyphs received a separate family, the Hydrophiidae, the "Palatine Draggers". Although this classification has found many followers (Golay, 1985) it is perhaps misleading in that it suggests a strong relationship between Smith *et al*’s terrestrial Elapids and *Laticauda* which has not been proven (McCarthy, 1985).

1a. Tail enlarged and laterally compressed, paddle-shaped or body banded blue and black.

**Marine Elapidae (Laticaudinae, sea kraits, and Hydrophiinae, sea snakes)**

1b. Tail not compressed into paddle-shape, usually cylindrical.

2a. Ventral scales not enlarged, similar in size to dorsal scales.

2b. Ventral scales broader than long, at least three times width of dorsal scales.

3a. Less than forty scales at midbody, all smooth; eyes vestigial and often barely visible.

**Typhlopidae (blind snakes)**

3b. In excess of eighty scales at midbody, rough and tubucular; skin very loose; eyes well developed.

**Acrochordidae (wart or file snakes)**

4a. In excess of thirty midbody scale rows; heat sensitive labial pits usually present.

**Boidae (Pythoninae — pythons)**

4b. Less than thirty scale rows at midbody.

5a. One or more loreal scales present between nasal and preocular, if loreal scale absent, then 23 or more scales at midbody and divided anal plate.

**Colubridae (Colubrinae, Natricinae and Homalopsinae — typical snakes)**

5b. Loreal scale absent, anal entire if 23 scale rows at midbody.

**Terrestrial Elapidae (Elapinae)**
are represented in the position and relationships of snakes of Australasia have been established. Sawin, (1977) confined the proteroglyphous snakes of the genus *Laticauda* to the terrestial Hydrophiidae, the has found many followers suggests a strong relationship *Laticauda* which has not been

**Hydrophiiniae, sea snakes**
- cylindrical.
- snout blunt.
- loreal scales.
- three times width of dorsal scale.
- scales vestigal and often bare.

**Typhlopidae (blind snakes)**
- tubular; skin very loose;
- sensitive labial pits usually

**Dipsadidae (wart or file snakes)**
- loreal.

**Pythoninae — pythons**
- loreal and preocular, if loreal scale divided.
- divided anal plate.

**Elapinae — typical snakes**
- loreal.

**Vesper Elapidae (Elapinae)**
Appendix Two:

Key to the Dangerous Papuan Elapids

1a. Body short, stout, viper-like with short tail; total length less than one metre; head angular and viper-like and very distinct from neck; two or three subocular scales present separating supralabials from orbit; comb-like spine on tip of tail; scales of body and head smooth or rugose; scale rows at midbody 21 or 23; anal plate entire; subcaudals anteriorly single, posteriorly paired; supralabials number six or seven with temporolabial contacting or failing to contact lip; eight infralabials; pupil of eye vertically elliptical.

2b. Scutes of head moderately rugose; anterior dorsals usually strongly keeled; supralabials not boldly patterned; free edge of supraocular often raised; dorsal scale rows fewer on neck than at midbody.

Genus Acanthophis

2a. Scutes of head smooth or weakly rugose; anterior dorsals weakly keeled or smooth, supralabials boldly patterned with black, or dark brown, and white; free edge of supraocular not usually raised; dorsal scale rows usually not fewer on neck than at midbody.

Acanthophis antarcticus

2b. Scutes of head moderately rugose; anterior dorsals usually strongly keeled; supralabials not boldly patterned; free edge of supraocular often raised; dorsal scale rows fewer on neck than at midbody.

Acanthophis praelongus

3a. Total length less than one metre; always six infralabials; usually 15 scale rows at midbody; occasionally 13 or 17, (17 rows on Fergusson and Woodlark Islands, Milne Bay Prov., only); subcaudals all or mostly paired, (if all single first subcaudal always divided into pair, Rossel Island, Milne Bay Prov., only); anal plate usually divided but if entire, then supralabials either number four or five with second and third entering orbit; otherwise supralabials usually number six with third and fourth entering orbit; temporolabial scale absent.

Genus Toxicocalamus (9 species)

3b. Total length varies from less than one metre to over three metres; always seven infralabials; 15 or more scale rows at midbody; subcaudals all single, all paired or combined single and paired; anal plate divided or entire; supralabials usually number six or seven, usually with third and fourth entering orbit; temporolabial scale present or absent.

4a. Total length less than two metres; scale rows at midbody number 15. 5

4b. Total length from less than one metre to over three metres; scale rows at midbody number 17 or more.

5a. Small snakes, total length less than 0.5 metre; anal plate entire; subcaudals single; pupil of eye round.

Genus Unechis (1-2 species)

5b. Small to medium sized; subcaudals all paired.

6a. Eye very small with body banded or pro rows not reducing in size.

6b. Eye medium to large; dorsum either scale rows reducing or not.

7a. Midbody scale rows usually 17 or more; subcaudals number 24 or more; rostrals absent; eye or barely discernible.

7b. Midbody scale rows not usually 17 or more; subcaudals less than 24.

8a. Ventrals and subcaudals both paired.

8b. Ventrals and subcaudals both single.

9a. Scale rows at midbody 17 or more.

9b. Scale rows at midbody number 15.

10a. Small to medium sized; prefrontal and supralabial in contact with preocular; temporo-labial present and contacting prefrontal.

10b. Medium to large sized; prefrontal and supralabial in contact with preocular; temporo-labial absent or barely discernible.

11a. Subcaudals either totally or mostly single.

11b. Subcaudals mostly or all paired.

12a. Scale rows at midbody variation of black and brown spots.

12b. Scale rows usually 15; colouration usually 12 spots.

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Small to medium snakes, total length up to two metres; anal plate divided; subcaudals all paired; pupil of eye round or vertically elliptical. 6

6a. Eye very small with round pupils; total length up to 2 metres; dorsum of body banded or progressively darker towards posterior; midbody scale rows not reducing in number towards cloaca. *Micropelphis ikaheka*

6b. Eye medium large with elliptical or round pupil; total length up to 1½ metres; dorsum either immaculate, mottled, flecked or striped; midbody scale rows reducing or not reducing in number towards cloaca. 7

7a. Midbody scale rows not reducing in number towards cloaca, 15-15; subcaudals number forty or less; body and tail not elongate; canthus rostralis absent; eye medium with vertically elliptical pupil, either obvious or barely discernible; total length less than 0.7 metres. 8

7b. Eye medium to large with elliptical or round pupil; total length up to 1½ metres; dorsum either immaculate, mottled, flecked or striped; midbody scale rows reducing or not reducing in number towards cloaca. 7

8a. Ventrals and subcaudals number 257-270; head unspotted. *Demansia atra*

8b. Ventrals and subcaudals number 281-322; head pale brown with darker brown spots. *Demansia papuensis*

9a. Scale rows at midbody number 17. 10

9b. Scale rows at midbody number more than 17. 12

10a. Small to medium sized snake, total length less than one metre; broad contact of prefrontal with second supralabial prevents contact of nasal with preocular; temporolabial present; subcaudals all paired; dorsum grey to gunmetal, occasionally with lighter band across nape of neck. *Pseudonaja textilis*

10b. Medium to large snakes, total length up to 2½ metres; contact between prefrontal and supralabials confined to point contact, not preventing point contact between nasal and preocular; temporolabial absent or present; subcaudals usually combined single and paired; dorsum usually dark or light brown without visible nape markings. *Pseudonaja australis*

11a. Subcaudals either totally paired or mostly paired with few anterior single subcaudals; temporolabial absent moderately slender body; eye medium sized. *Pseudonaja australis*

11b. Subcaudals mostly single with paired subcaudals posteriorly; temporolabial absent; heavy stout body; eye small. *Pseudonaja textilis*

12a. Scale rows at midbody usually 19 rarely 21, all smooth and glossy; dorsal colouration deep black; supralocular not protruding over eye; anal plate divided; subcaudals mostly single with paired subcaudals posteriorly; heavy stout body; eye small. *Pseudonaja australis*

12b. Scale rows usually 21, occasionally 23, keeled anteriorly, not glossy; dorsal colouration usually brown with lighter head and red vertebral stripe; supralocular protruding as distinct shelf over eye; anal plate entire; subcaudals all paired; moderately slender body; eye large. *Oxyuranus scutellatus*
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Introduction

The preceding chapter dealt with the importance of snake bite in Asia-Pacific Region. The most striking are the data from Sri Lanka, where the incidence of one death per 100,000 population per year is more than six per 100,000 per year in some rural areas. Despite the evident importance of snake bite as a cause of death (Gajdusek 1980) and recent figures suggest an increase in deaths per year in Maharastria, India, the evidence on which to review the therapeutic controversy is still imperfect.

First aid treatment

All patients bitten by snakes should be transported promptly to medical staff. First aid is the emergency treatment by others at the scene of the bite. It must not be delayed.

1. To deliver patients as quickly as possible to medical staff.
2. To delay the evolution of the local symptoms in a place where they can be treated later.
3. To alleviate severe early symptoms.

First aid treatment must be given to the vast majority of cases, those that are not to be medically trained.