RFDS MONOGRAPHS

MANAGEMENT OF SNAKE BITES IN AUSTRALIA AND PAPUA NEW GUINEA

By: Dr Struan Keith Sutherland & Dr James Tibballs

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MANAGEMENT OF SNAKE BITES IN AUSTRALIA AND PAPUA NEW GUINEA

ABOUT THE AUTHORS

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Photo taken by James Braund

Australia's fauna is remarkable for its range of venomous creatures, on land and in the sea and Struan Sutherland was a doyen of medical research in the field of envenomation and the ultimate authority on the medical management of envenomated victims in Australia. Snakes, spiders, ticks, deadly jellyfish and other venomous creatures have always instilled fear into its citizens. The fight against these was lead by the former Commonwealth Serum Laboratories (CSL), with the production of specific antivenoms to the most dangerous snakes, the Red-back Spider, the Paralysis Tick, the Stonefish and the Box-jellyfish. Struan as Head of Immunology at CSL, produced the antivenom against the Funnel-web Spider.

He started work at CSL in 1966 and his initial research involved development of a gammaglobulin for use in immunodeficient children.

His interest in venoms was sparked by the death of a young child after a bite by a Sydney funnel-web spider. In 1967 he re-activated research on an antivenom for the venom of the dreaded Sydney Funnel-web Spider, which curiously amongst mammals affects only primates. This task had defeated others before him and was not until 1980 that an antivenom raised in rabbits, was ready for testing in monkeys in tests conducted with collaborators at the Royal Children's Hospital research laboratory. This was a remarkable scientific achievement, succeeding where many others had failed. Since production of this antivenom, no person has died from the bite of this formidable animal and in many victims the course of illness following a bite has been dramatically shortened. Struan also developed techniques which has made the medical management of envenomation in Australia the best in the World. He was accorded world recognition with his invention of the pressure-immobilisation technique of first-aid. This simple but effective technique revolutionised first-aid management of snake bite and of some other types of envenomation. It made redundant the use of tourniquets and other dangerous first-aid manoeuvres. Similarly, he developed a snake venom detection kit which enabled doctors working at the bedside to ascertain which snake antivenom should be administered to a victim. In recognition of these achievements, he was awarded the AMA prize for medical research and the James Cook medal from the Royal Society of NSW. He also improved medical management of the envenomated victim by succeeding in bringing the subject to the medical curriculum.

MANAGEMENT OF SNAKE BITES IN AUSTRALIA AND PAPUA NEW GUINEA

Struan was a prodigious author. He published over 300 scientific and medical articles, numerous chapters in books and the standard medical textbook on the management of envenomation, Australian Animal Toxins. The second edition of this book, published in 2001, occupied most of his remaining time even when a variant of advancing Parkinson's Disease confined him to home. Indeed, the completion of this book appeared to be his raison d'etre and typified his ability to cope with adversity. He co-authored other books designed for public information including *Venomous Creatures of Australia* and Dangerous Australian Animals and Take Care: *Poisonous Australian Animals*. Struan served the medical fraternity and the public selflessly. He was always available to doctors, or to anybody, to give advice at any hour of the day or night, on management of envenomated victims. This 24-hour advisory service is now continued by members of the Australian Venom Research Unit.

Struan assisted the Royal Flying Doctor Service by writing the initial monograph on the Management of Snake Bites.

By JAMES TIBBALLS

Dr James Tibballs

Associate Professor James Tibballs is an intensivist and anaesthetist and Associate Director of the Intensive Care Unit at the Royal Children's Hospital, Melbourne and Principal Fellow at The Australian Venom Research Unit, Department of Pharmacology of The University of Melbourne. His major research interests are envenomation, resuscitation and poisoning. Since 1980, his major venom research has been the cardiovascular effects of venoms of The Sydney Funnel-Web Spider (Atrax robustus) several Australian and Papua New Guinean snakes (genera Pseudonaja, Notechis, Micropechis), The Box Jellyfish (Chironex fleckeri) and recently those of the jellyfish 'The Irukandji', (Carukia barnesi) and 'The Jimble' (Carybdea rastoni). Much was conducted in collaboration with Struan Sutherland.

He tested the then novel antivenom against The Sydney Funnel-web spider in anaesthetised monkeys before its commercial release and has tested the efficacy of several snake antivenoms in an anaesthetised dog model. Results of the latter redefined the clinical neutralisation dose of the antivenom and established that disseminated intravascular coagulation is the probable cause of sudden collapse after bites from some species.

In anaesthetised piglets he (with co-researchers) has determined that the cardiovascular effects of the venom of The Irukandji (Carukia barnesi) are those of severe systemic and pulmonary hypertension and tachycardia caused by the massive release of endogenous catecholeamines (published in conference abstract, full manuscript in preparation). Similar cardiovascular effects have been observed (unpublished) with the venom of Carybdea rastoni, a similar species of jellyfish.

He is with the late Struan Sutherland, co-author of the standard medical textbook of envenomation in Australia, Australian Animal Toxins, Oxford University Press, 2001

MANAGEMENT OF SNAKE BITES IN AUSTRALIA & PAPUA NEW GUINEA

INTRODUCTION

Australia has numerous species of snakes, including sea snakes. Some are among the most venomous in the World. Unless a snake is known to be non-venomous, a bite from a snake, even a juvenile or small snake, should be regarded as potentially lethal.

Australia is a world leader in the management of snake bite. Antivenoms are available for treatment of bites by the most venomous snakes. Other advances include the development of a safe and effective first aid technique, the pressure-immobilisation bandage, and the use of a bedside Venom Detection Kit.

Over the period 1981 to 1999, there were 2.6 deaths per year from snake bites; a death rate of approximately 0.014/100,000 1. Deaths occurred because of massive envenomation, snake bite occurring in remote locations, and delayed or inadequate antivenom therapy. About 2,000 people are bitten each year, and about 300 of these require treatment with antivenom.

IMPORTANT HERPETOLOGICAL INFORMATION

Geographical distribution of snakes

Although no area in Australia is devoid of snakes, the distribution of the main species (Figure 1) is reasonably well defined and may help in identification of the species responsible for a bite. Sea snakes may be found anywhere around the coast but are rare around southern Australia.

Composition of venoms

The venoms of Australian snakes are complex mixtures of very powerful toxins, usually proteins, that kill prey and aid its digestion. Although individual species or genera of snakes possess characteristic different toxins, the main toxins may be viewed as groups that cause paralysis, coagulopathy, rhabdomyolysis and haemolysis. One toxin may have several different effects. For example Tiger Snake venom has numerous separate neurotoxins of which one not only causes paralysis but also rhabdomyolysis. Neurotoxins act at either or both presynaptic and postsynaptic sites of neuromuscular junctions causing paralysis. Coagulopathy is either procoagulant, caused by prothrombin activators and resulting in disseminated intravascular coagulation and the consumption of clotting factors, or it is anticoagulant. Of the two types, procoagulant coagulopathy is more common but either may cause serious haemorrhage. Rhabdomyolysis of skeletal, and sometimes of cardiac muscle, may cause renal failure as well as generalised loss of muscle mass. Some venoms damage red blood cells and some may cause direct platelet aggregation. The toxins responsible for numerous relatively minor effects of snake venoms have not yet been identified. The major effects are summarised in Table 1.

Untreated, progressive paralysis with respiratory failure is the usual cause of death from snake bite. Bleeding and renal failure (as a complication of rhabdomyolysis, disseminated intravascular coagulation or haemolysis) may also cause death. Some snake venoms are directly nephrotoxic. Occasionally rapid collapse after a bite snake is due to anaphylaxis to venom or due to disseminated intravascular coagulation.

Table 1 Main components and effects of Australian snake venoms

Neurotoxins Presynaptic and postsynaptic neuromuscular blockers cause paralysis. They are present in venoms of all important venomous snakes. The postsynaptic blockers appear to be readily reversed by antivenom whereas the presynaptic blockers are more difficult to reverse, particularly if treatment is delayed. Some presynaptic blockers are also rhabdomyolysins.
Prothrombin activators The venoms of many important species contain proteins that cause disseminated intravascular coagulation with consumption of clotting factors including fibrinogen placing the victim at risk of haemorrhage. Intrinsic fibrin(ogen)lysis generates fibrin(ogen) degradation products.
Anticoagulants The venoms of a relatively small number of dangerous species contain anticoagulants which prevent blood clotting but do not consume clotting factors including fibrinogen.
Rhabdomyolysins Some presynaptic neurotoxins also cause destruction of skeletal and cardiac muscle. Apart from loss of muscle mass and weakness, myoglobinuria and renal failure may also occur.
Haemolysins A few species have venoms which cause haemolysis but this is rarely a serious clinical effect.

Potency of venoms

Some Australian snake venoms are more potent than those of notable snakes in India, Asia, Africa and North America (Table 2) 2. Eleven Australian snakes have venoms that are more potent than the venom of the Indian Cobra (Naja naja) which causes many thousands of deaths each year in India and Asia.

Table 2 Snake venom potencies

Species	LD_{50} in mice	Location
Small-scaled Snake (Oxyuranus microlepidotus)	0.025	Australia
Common Brown Snake (Pseudonaja textilis)	0.053	Australia
Taipan (Oxyuranus scutellatus)	0.099	Australia
Mainland (Eastern) Tiger Snake (Notechis scutatus)	0.118	Australia
Reevesby Tiger Snake (Notechis ater niger)	0.131	Australia
Beaked Sea Snake (Enhydrina schistosa)	0.164	Australia
Western Tiger Snake (Notechis ater occidentalis)	0.194	Australia
Chappell Island Tiger Snake (Notechis ater serventyi)	0.338	Australia
Common Death Adder (Acanthophis antarcticus)	0.400	Australia
Western Brown Snake (Pseudonaja nuchalis)	0.473	Australia
Lowland Copperhead (Austrelaps superbus)	0.560	Australia
Indian Cobra (Naja naja)	0.565	Indo-Asia
Dugite (Pseudonaja affinis)	0.660	Australia
Papuan Black Snake (Pseudechis papuanus)	1.09	Papua New Guinea
Stephen's Banded Snake (Hoplocephalus stephensii)	1.36	Australia
Rough-scaled Snake (Tropidechis carinatus)	1.36	Australia
King Cobra (Ophiophagus hannah)	1.80	Indo-Asia
Blue-bellied Black Snake (Pseudechis guttatus)	2.13	Australia
Collett's Snake (Pseudechis colletti)	2.38	Australia
Mulga (King Brown) Snake (Pseudechis australis)	2.38	Australia
Red-bellied Black Snake (Pseudechis porphyriacus)	2.52	Australia
Small-eyed Snake (Cryptophis nigrescens)	2.67	Australia
Eastern Diamond-back Rattlesnake (Crotalus adamanteus)	11.4	North America
Black Whip Snake (Demansia atra)	>14.2	Australia
Fer-de-Lance (Bothrops atrox)	>27.8	Central & South America

Envenomation

The fangs of most Australian snakes function like hypodermic needles. Although the fangs of the taipan may be up to 11 mm in length those of most other snakes are quite short (Figure 2). Nevertheless they are very effective for injecting venom rapidly and deeply into subcutaneous tissues. The snake may strike a number of times with great speed. Absorption of venom from a bite site is a continuing process.

Snakes may bite but fail to inject venom. Only 20% of patients presenting with 'snake bite' develop clinical envenomation and even when a bite is confirmed envenomation develops in less than 50%.

Figure 2 Comparative sizes of snake fangs (from left to right) – Bitis nasicornis (Rhinoceros Viper, exotic), Taipan, Death Adder, Mainland Tiger Snake, Common Brown Snake (Photo: Peter Mirtschin)

One of the principal difficulties in the management of the person with a snake bite is that the amount of venom injected can be extremely variable (Table 3) - and cannot be measured. This implies that the dose of antivenom cannot be specified with certainty. Not all bites result in the injection of venom.

For the purpose of manufacturing antivenom, venom is obtained by 'milking' (Figure 3) which is a highly artificial situation. The snake is induced to bite through a latex membrane and venom output is maximised by massaging the venom glands. The average yields have been determined for most species and in a number of species is known to exceed the average amount injected at a bite (Table 3).

Table 3 Comparison of venom yields of some Australian snakes

species	average yield on 'milking' (mg)	maximum yield on 'milking' (mg)	average injected on biting mice (mg)
Small-scaled Snake	44	110	17
Common (Eastern Brown) Snake	4#	67	4
Taipan	120	417	21
Mainland Tiger Snake	35	695	14
Death Adder	85	236	nt
Copperhead Snake	26	87	nt
Red Bellied Black Snake	37	94	nt

Derived from references 1-4 and personal communications from John Cann (1986) and Peter Mirtschin (1998). nt not tested. # 8 mg was obtained from Queensland specimens 5.

Figure 3 'Milking' venom from a Taipan (Photo: Peter Mirtschin)

FIRST AID PRESSURE - IMMOBILISATION BANDAGE

Ninety-five per cent of snake bites occur on the arms or legs and hence the pressure-immobilisation technique (Figure 4) is suitable for the majority of cases.1 Bites to the head, neck or trunk are uncommon.

Venom is usually deposited subcutaneously. Research has shown that the application of pressure less than arterial or venous pressure to the bitten area, when combined with immobilisation of the limb (Sutherland's technique), delays the movement of venom to the central circulation 6. The systemic spread of venom is dependent on its absorption by the lymphatics or the small blood vessels 7.

Firm crêpe bandages are ideal for this purpose and should encompass the bite site and as much of the bitten limb as possible, including the digits even if they are distal to the bite site. This aids immobilisation. The pressure should be similar to that employed to bandage a sprained ankle. The aim of the bandage is to compress the lymphatic vessels and the microcirculation. Then further immobilisation of the limb should be achieved by a splint to prevent movement of the entire limb. This prevents muscle contraction that would otherwise aid the flow of lymph. The splint should encompass the joints on either side of the bite site. This effective method of first aid is simple and easily taught. In practical terms, pantyhose or strips of clothing or sheeting and a splint, which can be made of any rigid object, can be applied to the majority of cases. Crêpe bandages or their equivalent are comfortable, do not damage the limb and may be left in place for hours if necessary.

If a snake bite victim reaches hospital in a desperately ill state as a result of inadequate first aid, bandaging of the limb will slow further venom absorption and allow the doctor to deal with the venom that already is in the blood stream. The longer the period between the time that snake venom reaches the circulation and the time that the patient receives antivenom, the greater the amount of antivenom that is required. Thus, first aid may result in a requirement for less antivenom (and a reduced risk of serum sickness). There is some experimental and anecdotal evidence that suggests that some inactivation of venom occurs with the use of the pressure-immobilisation bandage 1,8 but prolonged application has not been subjected to a controlled study.

Bites to the trunk and face.

These are uncommon. It has been suggested that local infiltration of a truncal bite with adrenaline may delay the absorption of venom 9. The injection has to be given within minutes of the victim being bitten. Otherwise, firm pressure at the bite site may delay the spread of venom - a method of application would need to be improvised.

Figure 4 Pressure-immobilisation technique of first aid. Reprinted with permission from Sutherland S, Sutherland J (1999), Venomous Australian Creatures, Oxford University Press

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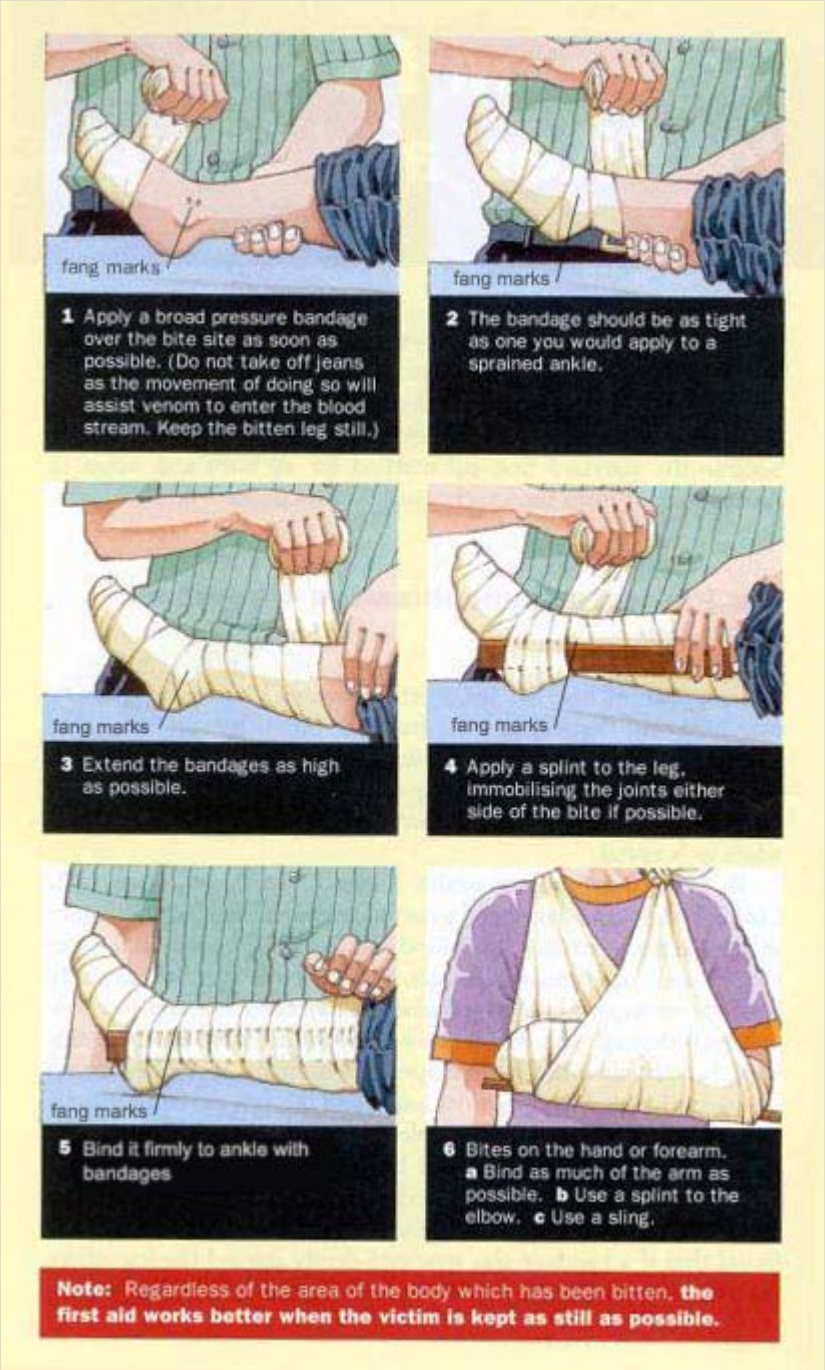


Mistakes in first aid

- The bite site should not be washed because the venom is not absorbed through skin and washing removes venom which could otherwise be used in a Venom Detection Kit test to determine the choice of antivenom.

- An arterial tourniquet should not be applied because it will cause ischaemic damage of tissues and will not be tolerated for a sufficient period.

- The bite site should not be incised, excised or amputated. These measures do not help and may injure important tendons and nerves. Experimental studies have shown that the incision or excision of the bite site removes only a very small amount of venom.

- Application of suction to the bite site does not remove injected venom and may injure tissues 10. It is not recommended and moreover, may remove venom from the skin that could otherwise be used in a Venom Detection Kit (VDK) test to select antivenom.

- Potassium permanganate (Condy's crystals) should not be applied – it is ineffective and injurious to tissues.

CLINICAL MANAGEMENT OF SNAKE BITES

IDENTIFICATION OF SNAKE

Identification of the offending snake allows the selection of the correct monovalent antivenom, as well as anticipation of the particular syndrome that may be produced by injected venom.

Morphology

Although some species, such as Death Adders and Red-bellied Black Snakes have characteristic features, correct identification of a snake by observation of its colour and other physical characteristics can be very difficult. It may be impossible at dusk or at night. It is dangerous to assume the identity of a snake on the basis of a brief glimpse or from an ill-informed amateur description.

Australian snakes can be broadly described as 'black', 'brown' or 'banded'. Unfortunately many different species fit each such description. Moreover, the colouration of a snake may not relate to its descriptive genus. For example, a snake of the Brown Snake genus (Pseudonaja) may not be brown-coloured, nor may a black-coloured snake be from the Black Snake (Pseudechis) genus. Further, many different species are banded and a Tiger Snake may have no bands at all. An un-banded Tiger Snake may be mistaken for a Brown Snake, and a Taipan may be mistaken for a Brown Snake. It is dangerous to assume that a brown-coloured snake is a Brown Snake.

Errors in identification have resulted in incorrect monovalent antivenom selection, contributing to deaths from snake bites. Expert herpetological opinion or reference to an identification guide is required if the snake is to be identified reliably by physical characteristics.

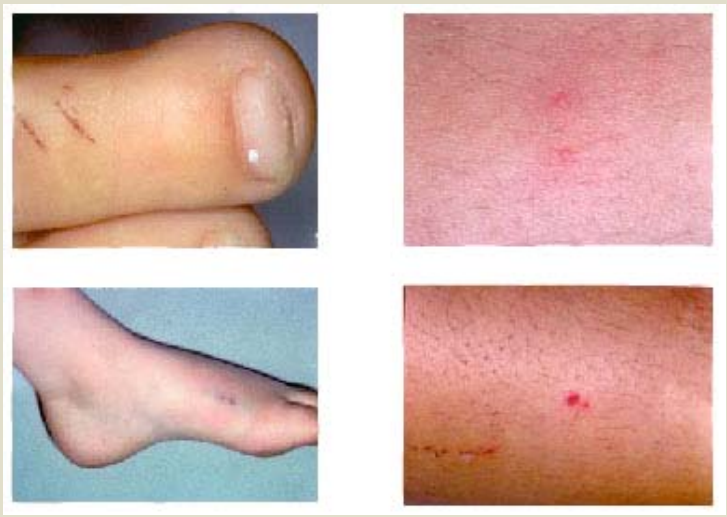
Bite site

Bites by Australian snakes often are relatively painless and the bite may be unnoticed, particularly when the victim has not seen the snake. The appearance of the bite site is variable. Sometimes it shows 'classic' paired or bi-pronged fang marks but frequently the marks will be single, multiple or linear lacerations (Figure 5). Continued bleeding from the bite site may be a feature.

The fine sharp fang marks of Australian snakes combined with very little local reaction to the venom, may render the actual bite site very difficult to find but usually there is mild swelling or bruising. This is in marked contrast to bites by many overseas snakes, where massive local reaction and necrosis is caused by proteolytic enzymes.

For practical purposes the appearance of a bite site cannot be used to identify the snake. Some species from the Black Snake genus are more likely to cause local tissue damage. However, this usually becomes evident long after the crucial decision is needed on the choice of antivenom.

Figure 5. Appearances of Australian snake bite. Top left a 'classic' Common Brown Snake bite with two fang marks about 1 cm apart and little associated trauma or reaction (photo J Tibballs); Top right scratch marks associated with unknown species (photo SK Sutherland); Bottom left bite from unknown Queensland species (photo Prof J Pearn); Bottom right bite from mainland Tiger Snake (photo J Tibballs).



Clinical effects

-- The role of symptoms and signs in the identification of an offending snake is limited because similar effects are produced by many different species.

-- All medically significant genera cause paralysis. It is thus impossible to distinguish snakes on this basis. However, within the Brown Snake genus (Pseudonaja), paralysis is usually only significant with the Common or Eastern Brown Snake (Pseudonaja textilis) and although possible, is not prominent with Pseudonaja nuchalis or Pseudonaja affinis.

-- Paralysis without significant coagulopathy may be caused by Death Adders (Acanthophis spp).

-- Procoagulative coagulopathy (disseminated intravascular coagulation) with consumption of fibrinogen and other clotting factors is expected after envenomation by species of the Tiger Snake genus (Notechis), Brown Snake genus (Pseudonaja), Taipan genus (Oxyuranus), Rough-scaled Snake (Tropidechis), Hoplocephalus species and the Red-bellied Black Snake (Pseudechis porphyriacus). Prothrombin and activated partial thromboplastin times are prolonged and thrombocytopenia may occur. Fibrin(ogen) degradation products may be generated by intrinsic fibrin(ogen)olysis. Australian snake venoms are not fibrinolytic.

-- Anticoagulative coagulopathy is caused by members of the Black Snake genus (Pseudechis), Copperhead genus (Austrelaps) and Death Adder genus (Acanthophis). Prothrombin time and activated thromboplastin times are prolonged but thrombocytopenia, defibrinogenation and fibrin(ogen)lysis does not occur. Among the Black Snakes, the Red-

Thus paralysis associated with disseminated intravascular coagulation may be caused by a Tiger Snake, Taipan, Brown Snake, Rough-scaled Snake, Hoplocephalus or Red-bellied Black Snake, but simultaneous rhabdomyolysis implies that a bite by a Brown Snake is unlikely. Paralysis associated with anticoagulation may be caused by a Black Snake (other than Red-bellied Black Snake), Copperhead or Death Adder but simultaneous rhabdomyolysis implies that a bite by a Death Adder is unlikely. Paralysis with neither coagulopathy nor rhabdomyolysis may be caused by a Death Adder. This information, although useful, is of limited practical importance. It is essential to administer antivenom as soon as clinically indicated rather than wait until the full syndrome becomes apparent to enable an 'educated clinical guess' in selection of the appropriate antivenom.

VENOM DETECTION KIT (VDK)

The venom detection kit (VDK) has a major role in determining the choice of antivenom. It should be used on every occasion except in those instances when absolutely no doubt exists about the identity of the snake. Since this kit first became available, it has undergone a number of changes to improve its performance. It has improved patient care and it is a unique Australian development (Figure 6).

The VDK is a qualitative in vitro test for detection and identification of snake venom at the bite site, in urine, blood or other tissue. It is an enzyme immunoassay using rabbit antibodies. It is very sensitive, able to detect venom in concentrations as low as 10 ng/mL. On occasions a positive test may be present and the patient may be asymptomatic. It yields a visual result in approximately 25 minutes. A positive result from urine or blood confirms a state of envenomation whereas a positive test from the bite site alone does not. Note that a very high concentration of venom in a sample may overwhelm the test and yield a spuriously negative result. If that possibility exists, a diluted sample should be tested.

The kit has test wells containing antibodies to Tiger, Brown, Black, Death Adder and Taipan venoms, positive and negative controls and a blank. Individual species of snake cannot be identified by the test and several genera may yield a positive result in the same well. For example, a positive result in the Tiger Snake test well may be due to venom from a Tiger Snake, Copperhead Snake, Red-bellied Black Snake, Rough-scaled Snake, or Hoplocephalus species. However, whatever species was responsible for that bite, Tiger Snake antivenom would be the antivenom of choice if antivenom is required on clinical grounds.

A positive result in Death Adder, Taipan or Brown Snake test wells not only indicates that the relevant specific monovalent antivenom should be chosen (if clinically indicated) but also identifies the genus of snake involved. However, species within those genera cannot be identified. A positive result in the Black Snake well indicates that venom from a member of the Black Snake genus (Mulga Snake, Papuan Black Snake, Butler's Black Snake, Red-bellied Black Snake, Blue-bellied Black Snake or Collett's Snake) is present.

Although all could be treated with Black Snake antivenom, only the first three should be treated with Black Snake antivenom (if clinically indicated) because the last three are adequately treated with Tiger Snake antivenom which is of smaller volume and less expensive. The rates of false positive and false negative results of the kit are not known but are generally regarded as low.

Figure 6 The CSL Ltd snake Venom Detection Kit

SYMPTOMS AND SIGNS OF ENVENOMATION

Most, but not all of the symptoms and signs of envenomation can be surmised from a knowledge of the toxins contained in venoms. Not all possible symptoms and signs occur in a particular case and in some cases one symptom or sign may predominate. In other cases they may wax and wane. Perhaps these phenomena are explained by variations in toxin content of venoms of the same species in different geographical areas or by variable absorption of different toxins. The major symptoms and signs and their expected onset are presented in Tables 4 and 5. In massive envenomation or in a child, a critical illness may develop in minutes rather than hours.

Table 4 Progressive onset of symptoms and signs of untreated envenomation

Less than one hour after bite headache nausea, vomiting, abdominal pain transient hypotension associated with confusion or loss of consciousness coagulopathy (laboratory test) regional lymphadenitis
One to three hours after bite paresis/paralysis of cranial nerves e.g. ptosis, double vision, ophthalmoplegia, dysphonia, dysphagia, myopathic facies haemorrhage from mucosal surfaces, needle punctures tachycardia, hypotension tachypnoea, shallow tidal volume
One to three hours after bite paresis/paralysis of cranial nerves e.g. ptosis, double vision, ophthalmoplegia, dysphonia, dysphagia, myopathic facies haemorrhage from mucosal surfaces, needle punctures tachycardia, hypotension tachypnoea, shallow tidal volume
More than three hours after bite paresis/paralysis of truncal and limb muscles paresis/paralysis of respiratory muscles (respiratory failure) peripheral circulatory failure (shock), hypoxaemia, cyanosis rhabdomyolysis dark urine (due to myoglobinuria or haemolysis)

Table 5 Symptoms and signs in 65 people presenting to hospital with snake bite (Prof B. Currie, personal communication 1999)

Symptoms and Signs	%
regional lymphadenitis	77
nausea	72
abdominal pain	52
headache	49
vomiting	48
coagulopathy	42
neurotoxicity	20
myotoxicity	17
early collapse with loss of consciousness	15

The systemic effects of snake venom often commence with headache, nausea, vomiting or abdominal pain. Although these symptoms are protean, they indicate that envenomation has occurred, rather than merely a bite, and they may herald the onset of life-threatening effects. Sometimes sudden and transient hypotension may cause loss of consciousness 1. The cause of hypotension soon after envenomation is obscure but it may be related to disseminated intravascular coagulation causing myocardial ischaemia or pulmonary hypertension 11,12. Brown Snake prothrombin activator gained access to the circulation within minutes after subcutaneous injection in an animal model of envenomation 13. Hypotension may be also secondary to myocardial hypoxaemia that accompanies respiratory failure.

Tender or even painful regional lymph nodes are common. However, they are not per se an indication for antivenom therapy since lymphadenitis also occurs with bites by mildly venomous snakes that do not cause serious systemic illness.

Life-threatening neurological effects are preceded by the onset of cranial nerve palsy or paralysis. Ptosis, blurred or double vision and external ophthalmoplegia should be sought as well as dysphonia, dysphagia and weakness of facial musculature resembling myopathic facies. Progressive neurotoxicity causes weakness of limb and truncal musculature such that the abilities to walk and sit upright are lost. As respiratory muscle strength is compromised, the use of accessory muscles of respiration and tachypnoea become apparent and tidal volume diminishes.

The early signs of cardiovascular involvement are tachycardia and relatively minor ECG abnormalities but these may progress to peripheral circulatory failure (shock) often in association with respiratory failure and sometimes in association with spontaneous haemorrhage from mucosal surfaces. Occasionally intracranial haemorrhage occurs.

In the case of untreated or massive envenomation, rhabdomyolysis may occur after several hours. This usually involves all skeletal musculature and sometimes cardiac muscle. The resultant myoglobinuria may cause renal failure. Direct nephrotoxicity has been suspected in a few cases of Brown Snake bites but this is as yet unproven.

Untreated, the severely envenomated patient presents unconscious, with cardiorespiratory failure. Occasionally, management of envenomation, particularly coagulopathy, is complicated by pre-existing medical or surgical conditions such as warfarin therapy or gastro-intestinal tract ulceration.

Snake bite sometimes follows alcohol consumption. This can make initial management difficult. Stories of drunks and snakes abound, and a favourite true one is of a drunken man who was bitten by a Brown Snake and so returned the compliment. He arrived in casualty holding a well-chewed snake and promptly collapsed, having succumbed to the combined effects of alcohol and snake venom.

SNAKE BITE IN CHILDREN

Snake bite in young children presents particular problems. Among these are difficulties in establishing a diagnosis when a bite has not been observed by an adult or older play-mate. Any child who reports contact with a snake should be believed. Prompt medical attention is required. The symptoms of early envenomation may pass unsuspected and the signs, particularly cranial nerve effects, are difficult to elicit. Bite marks may be difficult to distinguish from the effects of everyday minor trauma. Lastly, the onset of the syndrome of envenomation is likely to be more rapid and more severe than in adults because of the higher ratio of venom to body mass.

REMOVAL OF THE PRESSURE-IMMOBILISATION BANDAGE

First aid prevents venom gaining access to the circulation and its removal may precipitate rapid collapse. However, a swab of the bite site may be obtained safely by removing the splint temporarily and by cutting a window in the pressure-immobilisation bandage. Thereafter the bandage should be restored and the splint re-applied.

When an asymptomatic snake bite patient reaches hospital with a pressure-immobilisation bandage in place, it should not be removed until antivenom, appropriate staff and equipment have been assembled. If the patient is symptomatic and antivenom is indicated, a bandage should not be removed until after antivenom has been administered. If the patient's condition then deteriorates, a bandage should be reapplied.

ANTIVENOM

Choice

The decision to administer antivenom must be based on clinical criteria of envenomation, not on the results of a VDK test or other information. A positive VDK test establishes the choice of antivenom, not whether it should be given. If the patient is significantly envenomated, antivenom must be administered. There is no other effective treatment. Occasionally envenomation is so mild that spontaneous recovery may occur without antivenom. However, antivenom should only be withheld when it is judged that the risks of possible allergic effects of antivenom would surpass its benefits. If there are no clinical or coagulopathic effects, antivenom should not be administered.

CSL Ltd produces a range of equine monovalent antivenoms against the venoms of the main terrestrial snakes including Tiger Snake, Brown Snake, Taipan, Black Snake and Death Adder. A polyvalent antivenom containing equivalent amounts of all these is also available. It is usually about 50 mL in volume. A sea snake antivenom is produced from horses immunised with Beaked Sea Snake (Enhydrina schistosa) and Tiger Snake venom.

Antivenom should be chosen according to herpetological identification of the snake (Table 6). If this is unknown or doubtful, the antivenom should be chosen according to the result of a VDK test. If neither of these criteria can be fulfilled, and if the situation warrants immediate antivenom therapy, the geographical location should be used as a guide since the distribution of species is reasonably defined (Table 7). For example, in Tasmania, the only important indigenous venomous snakes are Tiger Snakes and Copperhead Snakes. Consequently, only Tiger Snake antivenom is required since Tiger Snake antivenom also neutralises Copperhead venom adequately. In Victoria, the situation is more complicated and two separate antivenoms, Tiger Snake and Brown Snake, are required to neutralise the venoms of all highly venomous indigenous species. Elsewhere in Australia, if the identity of the snake is unknown, polyvalent antivenom is mandatory. However, polyvalent antivenom is more expensive than monovalent preparations and its content of equine protein is significantly greater which predisposes to a greater risk of allergic reaction. Polyvalent antivenom should not be used when a monovalent antivenom could be used instead, i.e. when the identity of the snake is known.

When antivenom is indicated after bites by uncommon snakes, either polyvalent antivenom should be chosen or a monovalent antivenom as indicated by a Venom Detection Kit test. Table 8 lists the species venoms neutralised by available antivenoms.

Administration of the wrong antivenom may endanger the patient's life since antivenom raised against the venom of one species may have little reactivity to another. For example, neutralisation by Mainland Tiger Snake venom of Copperhead venom is 32%, Mulga Snake venom 25%, Death Adder 11%, Taipan venom 8% and Common Brown Snake venom 0.2% 1. Thus Tiger Snake antivenom is virtually useless in the treatment of Common Brown Snake envenomation and only marginally useful against the extremely venomous Taipan, Death adder and Mulga Snake. In contrast, Tiger Snake antivenom is useful for Copperhead bites not only because it has appreciable activity against the venom but also because Copperhead venom is much less potent than the venoms of the other species.

Dose

A number of factors preclude definitive prescription of the dose of antivenom. Foremost is the fact that the amount of venom injected by the snake and its subsequent concentration in the patient's body tissues cannot be determined in usual clinical circumstances. In addition, formulation of the antivenom is based on in vitro studies, not on in vivo clinical conditions. One ampoule of monovalent antivenom is formulated to neutralise in vitro the average yield of venom obtained from 'milking' the specific snake. Although this amount usually exceeds the average amount of venom injected at a bite this should not be assumed to be the in vivo neutralisation dose. Obviously if the amount injected is greater than the average yield by 'milking', one ampoule of antivenom will not be adequate therapy. In severe envenomation, as in multiple bites, a number of ampoules of antivenom will be needed. Furthermore, experiments in animals have shown that the neutralisation dose tested in vivo may be many times more than the in vitro dose 14. Given these difficulties, guidelines for initial doses of antivenom are provided in Table 6. The need for subsequent doses should be guided by the patient's clinical response. After a bite by a species with coagulopathic effects, subsequent doses of antivenom and clotting factors may be titrated against coagulation status. A number of other situations warrant large doses of antivenom. In general, a child requires more antivenom than an adult envenomated by the same amount of venom, and a person in poor general health will likewise require more. Antivenoms manufactured against specific species of snake may have less neutralising ability against other species in the same genus. For example, in the case of envenomation by a Chappell Island Tiger Snake, although Tiger Snake antivenom is the appropriate antivenom, several ampoules are recommended because neutralisation of venom by antivenom is 67% 1. Patients presenting late after envenomation may have toxins bound strongly to target tissues and these may not be dislodged or neutralised easily. Some patients have required mechanical ventilation for many weeks despite adequate amounts of appropriate antivenom.

Administration

Snake antivenoms must be given by the intravenous route. In dire circumstances, if a vein cannot be cannulated, antivenom may be given intraosseously, particularly in young children. The intramuscular route is useless in emergencies as absorption is slow, and there is a large volume of fluid to be administered. required for small children, the dilution may be less to prevent excessive fluid administration.

In emergencies the antivenom may be infused quickly in high concentration. A test dose of antivenom to determine allergy should not be given since it is unreliable and a waste of precious time.

Premedication

The incidence of adverse reactions (5-10%) to antivenom is sufficient to warrant premedication. Adrenaline is effective in reducing the incidence of antivenom-induced reactions and their severity 15. The dose is approximately 0.25 mg for an adult and 0.005 - 0.01 mg/kg for a child given subcutaneously 5-10 minutes before the commencement of antivenom infusion. The routine mode of administration of adrenaline should be the subcutaneous route because it does not cause significant hypertension 15-17. However, in a moribund or critically ill patient when it is essential to administer antivenom as soon as possible, adrenaline may be given intramuscularly or even intravenously in smaller doses. In general, intramuscular and intravenous routes are not recommended since they may induce hypertension which in the presence of venom-induced coagulopathy could cause an intracerebral haemorrhage. Although intracerebral haemorrhage has been recorded in the past in association with premedication, all such cases were accompanied by intravenous adrenaline, none with subcutaneous adrenaline 18.

It is not prudent to forgo premedication and elect to treat anaphylaxis if it occurs. Iatrogenic anaphylaxis has a high mortality despite vigorous and expert resuscitation 19. If there is no adverse reaction to the first ampoule of antivenom, subsequent doses do not need to be preceded by adrenaline.

Although traditionally used in Australia, premedication with promethazine was ineffective in a prospective randomised controlled trial in Brazil 20. It may also cause obtundation and hypotension both of which may exacerbate or confound the state of envenomation. It is not recommended. Other drugs such as steroids and aminophylline are also not useful in preventing anaphylaxis. They are unproven, and their onsets of action too slow. However steroids are useful in the secondary treatment of anaphylaxis and for the treatment of serum sickness.

Adverse reactions

Antivenom infusion should always be administered in a location equipped and staffed by personnel capable of managing anaphylaxis. Adrenaline should be available and ready for use. The principal signs of anaphylaxis are hypotension, tachycardia, bronchospasm, urticaria, angioneurotic oedema of the upper airway, and abdominal pain.

The immediate initial treatment consists of:

--cessation of antivenom administration

--adrenaline intramuscularly, approximately 0.25-1.00 mg for adults and 10 ug/kg for children

--intravenous fluid 20 mL/kg

--oxygen by mask

--inhaled salbutamol or adrenaline

Further doses of drugs and intravenous fluid, endotracheal intubation, mechanical ventilation may be required. Steroid, aminophylline and antihistamine may also be useful particularly after resuscitation when the patient, admittedly in a hapless situation, must continue to receive antivenom.

Less severe immediate adverse reactions including headache, chest discomfort, fine rash, arthralgia, myalgia, nausea, abdominal pain, vomiting, and pyrexia. These manifestations may be managed by temporary cessation of the infusion and administration of steroids and an antihistamine. Then the infusion may be recommenced.

Serum sickness, a delayed hypersensitivity reaction, should be anticipated. Patients should be warned of the symptoms and signs which usually appear several days to two weeks after antivenom administration. Severity may range from a faint rash and pyrexia to serious multi-system disease including lymphadenitis, polyarthralgia, urticaria, nephritis, neuropathy and vasculitis. The incidence of serum sickness is greater with multiple doses of monovalent antivenom or polyvalent antivenom. In these circumstances prophylactic steroids should be given over several days, commencing with antivenom treatment. In established serum sickness, a course of steroids, e.g. prednisolone 1 mg/kg per day in divided doses may be required for several weeks.

Table 6 Antivenom and initial dosages when snake identified

Snake	Antivenom	Dose (units)
Common Brown Snake	Brown Snake	4000-6000
Chappell Island Tiger Snake	Tiger Snake	12,000
Copperheads	Tiger Snake	3000-6000
Death Adders	Death Adder	6000
Dugite	Brown Snake	4000-6000
Gwardar	Brown Snake	4000-6000
Mulga (King Brown) Snake	Black Snake	18,000
Papuan Black Snake	Black Snake	18,000
Red-bellied Black Snake	Tiger Snake	3000
	or Black Snake*	18,000
Rough-scaled (Clarence River) Snake	Tiger Snake	3000
Sea snakes	Sea Snake	1000
	or Tiger Snake	3000
Small-scaled Snake	Taipan	12,000
Taipans	Taipan	12,000
Tasmanian Tiger Snake	Tiger Snake	6000
Tiger Snake (mainland)	Tiger Snake	3000

* smaller protein mass tiger snake antivenom preferable

Antivenom units per ampoule: Brown Snake 1000; Tiger Snake 3000; Black Snake 18,000; Taipan 12,000; Death Adder 6000; polyvalent 40,000.

If the patient is critically-ill on presentation, greater amounts should be given initially.

Additional antivenom may be required in the course of management since absorption of venom may be delayed.

Table 7 Antivenom and initial dosages when identity of snake uncertain

Location	Antivenom	Dose (units)
Tasmania	Tiger snake	6000
Victoria	Tiger snake	3000
	and Brown Snake	4000-6000
New South Wales & ACT	Polyvalent	40,000
Queensland	Polyvalent	40,000
South Australia	Polyvalent	40,000
Western Australia	Polyvalent	40,000
Northern Territory	Polyvalent	40,000
Papua New Guinea	Polyvalent	40,000

Table 8 List of species venom neutralised by antivenoms (adapted from Sutherland SK, Tibballs J. 2001)

Species	Antivenom
Mainland (Eastern) Tiger Snake **Black Tiger Snakes** *Tasmanian Tiger Snake * King Island Tiger Snake *Western Tiger Snake *Krefft's Tiger Snake *Peninsula Tiger Snake *Chappell Island Tiger Snake **Copperhead snakes** *Lowland Copperhead *Highland (Alpine) Copperhead *Pygmy (Adelaide Hills) Copperhead **Black snakes** *Red-bellied Black Snake *Blue-bellied (Spotted) Black Snake *Collett's Snake Rough-scaled (Clarence River) Snake **Small-eyed Snake** **Genus Hoplocephalus** **Broad-headed (Yellow-spotted) Snake** **Stephens' Banded Snake** **Pale-headed Snake** **Bandy-bandy** **Curl (Myall) Snake** **De Vis' Snake Ornamental Snake** **Black Whip Snakes** **Yellow-faced Whip Snake**	**Tiger Snake or Polyvalent**

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Species	Antivenom
Common (Eastern) Brown Snake **Western Brown Snake (Gwardar)** **Dugite** **Speckled Brown Snake** **Five-ringed Brown Snake** **Peninsula Brown Snake** **Ingram's Brown Snake Tanner's Brown Snake**	**Brown Snake or Polyvalent**
Iga (King Brown) Snake **Butler's (Yellow-bellied) Black Snake Papuan Black Snake**	**Black Snake or Polyvalent**
Death Adders *Common Death Adder *Desert Death Adder *Northern Death Adder *Pilbara Death Adder *Other Death Adders, including Papua New Guinean and Irian Jayan **Bardick**	**Death Adder or Polyvalent**
Taipan (Coastal Taipan) **Small-scaled Snake (Fierce Snake, Inland Taipan)** **Papuan Taipan**	**Taipan or Polyvalent**
All marine species	**Sea Snake (Enhydrina schistosa) or Tiger Snake**

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Species	Antivenom
Grey Snake	Polyvalent or
Black-bellied Swamp (Marsh) Snake	Monovalent according to Venom
Golden-crowned Snake	Detection Kit test
White-lipped Snake	
Brown-headed Snake	
False Mulga Snake	

* Use monovalent whenever possible

PRACTICAL MANAGEMENT by SEVERITY of ENVENOMATION

From a practical point of view, one of three clinical situations – determined by severity of envenomation - is usually encountered after snake bite. A plan of management for each of these is summarised in Figure 7.

1. If the patient is critically ill

*The priorities in management are:

*Resuscitation. Treat hypoventilation with mechanical ventilation and oxygen and restore blood pressure with intravenous fluids and inotropic agents as needed

*Application of a pressure-immobilisation bandage

*Administration of antivenom on a contingency basis according to geographical location if identity of snake is unknown or doubtful.

*Performance of investigations.

*The patient should be managed in an intensive care environment.

2. If the patient is envenomated but not critically ill

More time is available to identify the snake and to administer specific monovalent antivenom. A pressure-immobilisation bandage should be applied if not already in place, and not removed until antivenom has been administered. The patient should be managed in an intensive care environment whenever possible.

The majority of patients in this group are admitted to hospital within two hours of the bite. Usually their condition is not critical at this stage, and a rational clinical assessment can be made. In the days before antivenoms were available, it was unusual for an adult to die in less than 14 hours after a bite and at least two out of three fatal cases survived more than seven hours after the bite.

3. If the patient is not envenomated, despite a bite or possible bite

The patient should be admitted to hospital or emergency department. Adults should be observed for at least several hours and children for 12 hours. Regular examination, at least hourly, for symptoms and signs of envenomation is required. This includes neurological observations normally accorded to a patient with a head injury. Occasionally, the syndrome of envenomation is very slow in onset, occurring over hours with a symptom free initial period.

A test of coagulation should always be performed. If coagulopathy is present, specific monovalent antivenom should be administered after identification of the species or as indicated by a VDK test. If only a mild coagulopathy is present it may be acceptable to withhold antivenom in the hope of spontaneous resolution but coagulation should be checked at intervals and the patient monitored until coagulation is normal. On discharge, the patient or relatives of a child should be instructed about late onset of envenomation.

1. Resuscitate (treat respiratory failure and shock)

Be prepared to intubate and mechanically ventilate. Administer intravenous fluids. Admit to intensive care.

2. Apply pressure-immobilisation bandage Do not remove if already applied (b).

3. Give antivenom intravenously (c,d,e,f,g)

*Give monovalent if species reliably known or appropriate antivenom indicated by venom detection test.

*In critically ill victim, don't wait for venom test result or if species unidentified - give according to location:

Victoria - Brown and Tiger Snake

Tasmania - Tiger Snake

Other Australian states and territories, Papua New Guinea - polyvalent.

*Titrate antivenom against clinical and coagulation status (NB: Death Adders don't cause significant coagulopathy).

4. Perform investigations

Bite site swab for venom detection. (First aid bandage may be cut to expose bite site, and then reinforced)

Blood for venom detection, coagulation, type and cross-match blood (if bleeding), fibrin degradation products, full blood examination, enzymes, electrolytes, urea, creatinine

5. Examine frequently to detect slow onset of paralysis (h), coagulopathy, rhabdomyolysis and renal failure.

Dangers and mistakes in management

a. Fang marks not visible to naked eye. Sometimes seen for first time at post mortem!

b. Premature release of bandage may result in sudden systemic envenomation. Leave in situ until victim reaches full medical facilities. If clinically envenomated, remove only after antivenom given.

c. Erroneous identification of snake may cause wrong antivenom to be given. If any doubt, treat as unidentified.

d. Antivenom without premedication. Anaphylaxis is not rare and may not respond to treatment.

e. Insufficient antivenom. Titrate dose against clinical and coagulation status.

f. Blood and coagulation factors (fresh frozen plasma, cryoprecipitate) not preceded by antivenom will worsen coagulopathy.

g. Antivenom given without clinical or laboratory evidence of envenomation.

h. Delayed onset of paralysis may be missed. Patient must be examined at least hourly.

INVESTIGATIONS and MONITORING

Access to laboratory services is highly desirable. A test of coagulation is essential.

Bite site
A swab for venom testing should be collected. It has the highest likelihood of detecting venom provided bite site has not been washed. The site may be squeezed to yield venom if it has been washed.

Urine
Test the urine for venom. This may be present when venom in blood has been bound by antivenom and is therefore undetectable. Urine should also be tested for blood and protein. If the urine is pigmented, whether this is haemoglobinuria or myoglobinuria should be determined (This is not possible with simple ward tests). Urine output should be recorded.

Blood and Serum

*Coagulation tests should include prothrombin time, activated partial thromboplastin time, serum fibrinogen and fibrin degradation products. If these cannot be performed, a bedside 20 minute whole blood clotting or bleeding time would suffice.

*A full blood examination should be done for haemoglobin level, haematocrit, evidence of haemolysis and the platelet count. A mild elevation in white cell count is expected.

*Serum electrolytes, urea and creatinine are necessary if rhabdomyolysis and renal insufficiency occurs. Creatine phosphokinase should be measured and if possible its skeletal and cardiac isoenzymes. Troponin estimations are also useful.

*Blood grouping and cross matching are necessary if haemorrhage occurs.

*Arterial blood gases are useful to guide management of respiratory failure

Electrocardiogram

Monitoring is important. Sinus tachycardia, ectopy and ST segment and T-wave changes are not uncommon. These effects may be the result of venom toxins, electrolyte disturbances caused by rhabdomyolysis or renal failure, or secondary to myocardial ischaemia or hypotension induced by disseminated intravascular coagulation.

Pulse oximetry

This is useful to warn of impending respiratory failure in the spontaneously breathing patient and as an aid to monitoring gas exchange in the mechanically ventilated patient.

SECONDARY MANAGEMENT

Coagulation factors and blood transfusion

Coagulation factors are required if haemorrhage is present. Although coagulopathy without haemorrhage often resolves after several doses of antivenom it should be remembered that antivenom per se does not restore coagulation - it only permits newly released or manufactured coagulation factors to act unopposed by venom. Restitution of normal levels of clotting factors may take many hours. If normal coagulation is not restored after several doses of antivenom over several hours, it is prudent to administer fresh frozen plasma and to repeat coagulation studies at intervals. Antivenom, to ensure neutralise venom prothrombin activator, should be given before coagulation factors are administered. Platelets may be required. Whole blood is rarely indicated except after significant haemorrhage.

Intravenous fluids, rhabdomyolysis and renal protection

After acute resuscitation, intravenous fluids should be administered to prevent tubular necrosis as a consequence of rhabdomyolysis. Fluids should be given in sufficient volume to maintain urine output at about 40 mL/kg/day in an adult and 1-2 mL/kg/hr in a child. Frusemide, mannitol and/or an inotropic agent, such as dopamine may be needed to ensure renal perfusion and urine output. Life-threatening hyperkalaemia and hypocalcaemia may develop with rhabdomyolysis. Hyperkalaemia may require urgent treatment with sodium bicarbonate, calcium chloride/gluconate, or glucose and insulin. Haemofiltration or dialysis may be required.

Heparin

Although this anticoagulant has prevented the action of prothrombin activators in an animal model of envenomation, it does not improve established disseminated intravascular coagulation 14. It is not recommended. Emphasis should be on neutralising venom with antivenom.

Analgesia and sedation

Australian snake bite does not cause severe pain. However, sedation is required for the mechanically ventilated venom-paralysed patient.

Care of the bite site

Usually no specific care other than routine wound care is required. Occasionally the site may ulcerate or necrose particularly when a first aid bandage has been in place for a considerable time or when the bite was from a member of the Black Snake genus.

Other drugs

Antibiotics are not routinely required but should be given if the wound appears infected. Sea snake bites may cause gram negative infections. Tetanus prophylaxis should be reviewed.

MISTAKES in MANAGEMENT OF SNAKE BITE

Lack of first aid

Application of a pressure-immobilisation bandage is important by first aiders in the field, by ambulance officers before and during transport and by medical/nursing staff in hospitals. It is often forgotten or badly applied by hospital staff even when the patient has been obviously envenomated. Although prolonged use of first aid may have a beneficial effect, it may be removed when the asymptomatic patient reaches definitive care.

Inadequate observation

Regular observations as for a head injury must be made and staff who are not familiar with signs and symptoms of snake bite should be instructed in their recognition. Patients have developed neurological signs that were not recognised in hospital or have returned to hospital desperately ill after being sent home too early.

Failure to diagnose envenomation or appreciate the danger

Failure to make the diagnosis in the case of unobserved snake bite may lead to a tragic outcome. The onset of neurological symptoms and signs after outdoor activity should prompt a consideration of snake bite. No other neurological illness accompanied by coagulopathy is likely. The onset of rhabdomyolysis in the absence of hyperthermia should likewise prompt the diagnosis. No other venomous creature causes a combination of neurotoxicity, coagulopathy and rhabdomyolysis. Every snake bite should be regarded as potentially lethal, even bites by juvenile snakes.

Mistakes with antivenom therapy

*No antivenom when clearly indicated. Antivenom is indicated for all envenomated patients unless the effects of envenomation are so mild that the risks of anaphylaxis and serum sickness possible with antivenom outweighs its benefits.

*Inadequate quantity of antivenom. Titrate the dose against the clinical and coagulation status.

*Wrong antivenom. Accurately determine the species involved. If not possible give monovalent antivenom(s) on a geographical basis or use polyvalent antivenom.

*Lack of premedication. The incidence of adverse reactions is considerable.

*Lack of preparedness and ability to treat anaphylaxis. Treatment of anaphylaxis may be difficult and prolonged. It does not always respond to medical therapy.

*Inadequate or infrequent laboratory tests. Tests should be performed regularly, interpreted quickly and treated promptly to counter venom effects and its complications. Serial coagulation tests and tests of renal function are especially important. Remember that absorption of venom from the bite site is a continuing process and that management must anticipate unabsorbed venom.

LONG-TERM EFFECTS OF SNAKE BITE

After appropriate and adequate treatment, recovery is expected. However, it may be slow taking many weeks or months, particularly when a critical illness, neurotoxicity and rhabdomyolysis had been established on delayed presentation. Isolated neurological or ophthalmic signs may persist. Long-term loss of taste or smell occurs occasionally. Serum sickness may also occur.

SNAKE BITES IN REMOTE AREAS

Advice on the measures to be taken to deal with snake-bites in remote areas is often requested by bush-walking groups, school parties, and organisations with staff members working in isolated areas. Complex hypothetical situations can be presented with a request for clear guidelines. It may not be possible to provide these. For example, a lone man without transport and 150 km from help who receives multiple bites from a 2 metre taipan will be in the same hapless situation as if he had suffered a severe gunshot wound or had suffered a massive coronary occlusion.

Since most cases of snake bite are the result of careless and inappropriate human behaviour, observance of the fundamental rules to avoid snake bite are emphasised.

The pressure-immobilisation technique of first aid will improve the prognosis in cases of snake bite that have occurred some distance from medical care. However, a lone individual who is bitten by a snake is in a difficult predicament. All that can be done is to apply pressure to the bitten area and then set off for help. Since an essential part of the first aid is immobilisation of the limb, such measures will be only partially successful. However, severe envenomation only occurs in 10% of cases and death is unlikely to occur in less than 14 hours in adults.

When others are present, it is essential that transport be brought to the victim rather than vice versa. If only one other person is present, this person should leave to arrange transport once first aid measures have been applied. In difficult terrain, a rescue party may be needed. The sooner it is organised, the better. Parties which venture into remote areas should take a radio transmitter or satellite telephone to seek help in emergencies such as snake bite.

Doctors who are involved in recreational activities in remote areas often contact the Commonwealth Serum Laboratories Ltd or Australian Venom Research Unit (AVRU) for advice on whether to take antivenom with them as a safeguard. This is usually discouraged. Enforcement of snake bite avoidance rules are encouraged as well as knowledge of first aid. A pamphlet describing first aid is available from AVRU. Not carrying antivenom is a calculated risk, that weighs the chances of a snake bite against factors such as its cost, storage requirements and difficulties of administration by untrained personnel in adverse circumstances.

As a general rule, antivenom is best administered in hospital, where facilities exist to cope with any problems that may develop. Sometimes, a large camp of workers will be employed in an area of very high snake infestation, in which case it may be wise to hold antivenoms for administration by paramedical personnel who should receive instruction in snake bite management. The Royal Flying Doctor Service carries antivenom in its aircraft when the Service is called to attend snake bite victims in remote areas.

MANAGEMENT BY RURAL AND REMOTE HEALTH SERVICES

Each health service should define its level of care in the management of snake bites. For example, in an isolated nursing post, first aid measures, urgent medical consultation and preparation for transport are required. In larger rural and remote hospitals with supplies of antivenom, facilities for resuscitation and short-term ventilation, and with appropriately skilled medical staff, decisions regarding the transport of patients to major centres and timing must be made. These decisions should be made in consultation with specialist staff at the nearest major referral centre and aero-medical (or ambulance) service.

SEASNAKE BITES

There are many species of sea snakes found in the warmer coastal waters of Australia. Some are known to produce very toxic venoms that cause widespread damage to skeletal muscle with consequent myoglobinuria, neuromuscular paralysis or direct renal damage. The venoms of lesser known species have not been researched. The principles of treatment are essentially the same as for envenomation by terrestrial snakes. The venoms of significant species are neutralised with CSL Ltd Sea Snake (Enhydrina shistosa) antivenom. If that preparation is not available, Tiger Snake or polyvalent antivenom should be used. Sea snakes are rarely aggressive and bites are uncommon. No deaths have been recorded from bites in Australian waters.

UNCOMMON AND EXOTIC SNAKE BITES

Usually these occur in zoo personnel, herpetologists and amateur collectors who catch, maintain or breed uncommon Australian snakes, or who import or breed exotic (overseas) snakes. Occasionally, a bite from an uncommon or lesser Australian snake occurs among the general public. There are no specific antivenoms to the venoms of uncommon Australian snakes but neutralisation is provided by polyvalent antivenom or by monovalent antivenom listed in Table 8 or as indicated by the Venom Detection Kit.

Venomous exotic snakes should only be kept by licensed organisations but there is little doubt that such snakes are kept by some Australian amateur collectors. Whether the snakes have been imported legally or illegally or bred in captivity, bites by exotic snakes pose special medical problems. Among these are the lack of medical knowledge and experience to treat envenomation in Australia, and the lack of antivenoms 21.

Limited exotic antivenoms are maintained by

Venom Supplies Ltd, Tanunda, South Australia (tel 08 8563 0001),

Royal Melbourne Hospital (tel, 03 9342 7000)

Royal Adelaide Hospital (tel 08 8223 4000)

Australian Reptile Park (tel 02 4340 1022)

Taronga Zoo (Mosman, tel 02 9969 2777)

SNAKE BITE IN PAPUA NEW GUINEA

Snake bite is a frequent occurrence in Papua New Guinea with an incidence on the south-west coast and surrounding the capital Port Moresby of 215/100,000 population. The mortality rate is 500-600 times greater than that for Australia and exceeded in few other areas in the world 22.

The highly venomous and potentially dangerous species of snakes are the same or closely related to species in Australia 23. These include Death Adders, Papuan Taipan, Mulga Snake, Papuan Black Snake, Common Brown Snake, Papuan Whip Snakes, Beaked Sea Snake and the (Papuan) Small-eyed Snake (Micropechis ikaheka ikaheka). The last named species is not found in Australia and is unrelated to the Australian Small-eyed Snake (Cryptophis nigrescens). Its venom causes neurotoxicity, rhabdomyolysis, anticoagulopathy and haemolysis and envenomation can be treated with CSL Ltd polyvalent antivenom 24.

The principles of management of snake bite in New Guinea are identical to that described for Australia. The high mortality rate with snake bite is multifactorial including a reluctance to seek medical care among the indigenous population, difficulties with transport, a critical shortage of antivenom, inadequate intensive care facilities and lack of Venom Detection Kits. Death is commonly due to respiratory failure.

SNAKE BITE IN DOMESTIC ANIMALS

The principles of treatment are the same as for humans but the cost of treatment becomes a very significant factor as it has to be met by the owner of the animal. Horses are very susceptible to snake venom; dogs are moderately susceptible whereas cats are more resistant

PREVENTION OF SNAKE BITE

Snakes are shy creatures which avoid human contact and usually flee when disturbed. Incidentally, the swiftest snake has a maximum speed on level ground of 7 km/hr but only for short distances. Although snakes have an excellent sense of smell, their vision is not good and often they will not notice anything until it moves. Snakes have a very rudimentary hearing apparatus and probably cannot detect airborne sounds but they can detect the approach of humans by the vibrations transmitted through their bodies from the ground.

Snake bite is often 'accidental'. A snake is trodden upon or suddenly disturbed by an unwary human. All too often however, bites occur when humans deliberately interfere with snakes or handle them. The herpetologist or snake collector is at special risk. Not only do such persons invariably sustain bites in the course of their work or hobby 25, but they are also at risk of developing allergic reactions to the venoms and to the antivenoms used in their treatment. An inexperienced person attacking a snake with a stick may be surprised by the speed of the snake's response. Snakes should be left alone unless they are near playgrounds or houses in which case they should be re-located by a registered snake catcher or killed by an experienced adult. Stout footwear, thick socks or gaiters, and clothing give considerable protection against fang penetration on the lower limb. Loose fitting clothing may render the target less obvious. Hands should never be put into hollow logs or thick grass without prior inspection. Snakes will be attracted to sheds and barns if they are not kept free of rats and mice. Many cases of snake bite occur on warm summer nights. A torch should always be used at night when moving around riverbanks, camps or farmhouses. Snake bites may occur when grass has not been cut in playgrounds or vacant allotments. Children should be discouraged from collecting snakes as a hobby because the consequences could be disastrous.

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