MOCK UP OF SUBSCRIBER PAGES FOR A SINGLE RECORD
(example is Australian mainland tiger snake)
General Details Section

Notechis scutatus

General Details, Taxonomy and Biology, Venom, Clinical Effects, Treatment, First Aid , Antivenoms

Family: Elapidae
Genus: Notechis
Species: scutatus
Common Names
Easter Tiger Snake , Mainland Tiger Snake

Region
Australia
Countries

Australia

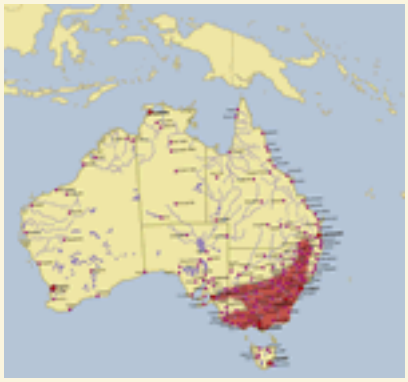
Taxonomy and Biology

General Shape
Moderately large, muscular, robust bodied snake which can grow to more than 2.0 metres. The head is moderately wide, flat and blunt and slightly distinct from the neck. Body scales are smooth and semi-glossy in appearance. Eyes medium in size with round pupils. Capable of flattening entire body when basking or disturbed. Scales around the neck appear like overlapping shields.

Habitat
Found in cool to warm temperate permanent watercourses, swamps and seepage areas on coastal lowlands, inland slopes, plains and ranges including the entire Murray River system. Found in a wide range of habitats including rainforest, dry and wet sclerophyll open forest and woodlands, shrublands, heath and tussock grasslands. Most of the region coincides with pasture and cropping activities. Also found in urban and semi-urban areas.

Habits
Diurnal and crepuscular tending to nocturnal in hot weather. Shelters under large rocks, rotten logs, abandoned burrows and dense matted vegetation, always near permanent water. In cooler, wetter months they tend to move away from water to higher ground. Usually inoffensive and intent on escape if disturbed. If cornered will hold the forebody in a tense but loose curve, head raised slightly and facing directly at an intruder, inflating and deflating the body and hissing loudly. If provoked it will become quite aggressive and strike forcefully.

Prey
Juveniles depend heavily on frogs and tadpoles for survival. Feeds mainly on frogs, tadpoles and mice, but will eat lizards, rats, small birds, eels and fish.



Venom

Average Venom Qty
30 to 70 mg (dry weight), U.S. Dept. Navy (1968) (Ref : R000914).
28 mg (dry weight), Freeman and Kellaway (1934) (Ref : R000686).
35 mg (dry weight of milked venom), Meier and White (1995) (Ref : R000001).
30 to 70 mg (dry weight), Minton (1974) (Ref : R000504).
Preferred LD50 Estimate
0.118 mg / kg sc (mice), Meier and White (1995) (Ref : R000001)
General: Venom Neurotoxins
Pre- & Post-synaptic neurotoxins
General: Venom Myotoxins
Systemic myotoxins present
General: Venom Procoagulants
Prothrombin convertors
General: Venom Anticoagulants
Not present
General: Venom Haemorrhagins
Not present
General: Venom Nephrotoxins
Not present
General: Venom Cardiotoxins
Not present
General: Venom Necrotoxins
Not present
General: Venom Other
Not present or not significant

Clinical Effects

General: Dangerousness
Severe envenoming possible, potentially lethal
General: Rate of Envenoming: 40-60%
General: Untreated Lethality Rate: 40-50%
General: Local Effects
Local pain, swelling & bruising
General: Local Necrosis
Rarely occurs, minor only
General: General Systemic Effects
Variable non-specific effects which may include headache, nausea, vomiting, abdominal pain, diarrhoea, dizziness, collapse or convulsions
General: Neurotoxic Paralysis
Very common, flaccid paralysis is major clinical effect
General: Myotoxicity
Very common, major clinical effect, usually moderate to severe
General: Coagulopathy & Haemorrhages
Very common, coagulopathy is major clinical effect
General: Renal Damage
Recognised complication, usually secondary to myolysis
General: Cardiotoxicity
Unlikely to occur
General: Other
Not likely to occur

First Aid

Description: First aid for bites by Elapid snakes which do not cause significant injury at the bite site (see Comments for partial listing), but which may have the potential to cause significant general (systemic) effects, such as paralysis, muscle damage, or bleeding.
Details
1. After ensuring the patient and onlookers have moved out of range of further strikes by the snake, the bitten person should be reassured and persuaded to lie down and remain still. Many will be terrified, fearing sudden death and, in this mood, they may behave irrationally or even hysterically. The basis for reassurance is the fact that many venomous bites do not result in envenoming, the relatively slow progression to severe envenoming (hours following elapid bites, days following viper bites) and the effectiveness of modern medical treatment.

2. The bite wound should not be tampered with in any way. Wiping it once with a damp cloth to remove surface venom is unlikely to do much harm (or good) but the wound must not be massaged. For Australian snakes only, do not wash or clean the wound in any way, as this may interfere with later venom detection once in a hospital.
3. All rings or other jewellery on the bitten limb, especially on fingers, should be removed, as they may act as tourniquets if oedema develops.
4. If the bite is on a limb, a broad bandage (even torn strips of clothing or pantyhose) should be applied over the bitten area at moderate pressure (as for a sprain; not so tight circulation is impaired), then extended to cover as much of the bitten limb as possible, including fingers or toes, going over the top of clothing rather than risking excessive limb movement by removing clothing. The bitten limb should then be immobilised as effectively as possible using an extemporised splint or sling.
5. If there is any impairment of vital functions, such as problems with respiration, airway, circulation, heart function, these must be supported as a priority. In particular, for bites causing flaccid paralysis, including respiratory paralysis, both airway and respiration may be impaired, requiring urgent and prolonged treatment, which may include the mouth to mask (mouth to mouth) technique of expired air transfer. Seek urgent medical attention.
6. Do not use Tourniquets, cut, suck or scarify the wound or apply chemicals or electric shock.
7. Avoid peroral intake, absolutely no alcohol. No sedatives outside hospital. If there will be considerable delay before reaching medical aid, measured in several hours to days, then give clear fluids by mouth to prevent dehydration.
8. If the offending snake has been killed it should be brought with the patient for identification (only relevant in areas where there are more than one naturally occurring venomous snake species), but be careful to avoid touching the head, as even a dead snake can envenom. No attempt should be made to pursue the snake into the undergrowth as this will risk further bites.
9. The snakebite victim should be transported as quickly and as passively as possible to the nearest place where they can be seen by a medically-trained person (health station, dispensary, clinic or hospital). The bitten limb must not be exercised as muscular contraction will promote systemic absorption of venom. If no motor vehicle or boat is available, the patient can be carried on a stretcher or hurdle, on the pillion or crossbar of a bicycle or on someone's back.
10. Most traditional, and many of the more recently fashionable, first aid measures are useless and potentially dangerous. These include local cauterization, incision, excision, amputation, suction by mouth, vacuum pump or syringe, combined incision and suction ("venom-ex" apparatus), injection or instillation of compounds such as potassium permanganate, phenol (carbolic soap) and trypsin, application of electric shocks or ice (cryotherapy), use of traditional herbal, folk and other remedies including the ingestion of emetic plant products and parts of the snake, multiple incisions, tattooing and so on.

Antivenoms

1. Antivenom Code: SAuCSL10

Antivenom Name: Tiger Snake Antivenom
Manufacturer: CSL Limited
Phone: ++61-3-9389-1624
Toll free: 1800 642 865
Address: 45 Poplar Road
Parkville
Victoria 3052
Country: Australia
Back to top

2. Antivenom Code: SAuCSL12

Antivenom Name: Polyvalent Snake Antivenom (Australia - New Guinea)
Manufacturer: CSL Limited
Phone: ++61-3-9389-1624
Toll free: 1800 642 865
Address: 45 Poplar Road
Parkville
Victoria 3052
Country: Australia

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Taxonomy and Biology Section

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Common Names
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Countries

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Prior Taxonomy - Synonomy

Naja (Hamadryas) scutata, Peters, 1861.
Hoplocephalus fuscus, Steindachner, 1867.
Alecto fasciolata, Jan and Sordelli, 1873.
Notechis scutatus, Boulenger, 1896.
Notechis scutatus scutatus (Peters, 1861).
Notechis scutatus, Cogger, 1983.

Distribution

Australia (Extreme SE Queensland, northern and central New South Wales highlands, southern New South Wales, Victoria, Murray River Valley and extreme SE South Australia).

Family map

Genus map

Species map (can be expanded to 1200 pixels width, to read place names

Appearance and Scalation

Adult Length in Metres: 1.2 m
General Shape
Moderately large, muscular, robust bodied snake which can grow to more than 2.0 metres. The head is moderately wide, flat and blunt and slightly distinct from the neck. Body scales are smooth and semi-glossy in appearance. Eyes medium in size with round pupils. Capable of flattening entire body when basking or disturbed. Scales around the neck appear like overlapping shields.

Head Scales
Head scales typical of genus with 9 large supracephalic shields, frontal shield length about equal to width, internasals present, single preocular in contact with nasal, 2 postoculars, suboculars absent, 6 supralabials (5th and 6th the largest, 3rd and 4th in contact with eye), large lower temporal scale and temporals 1+ 3.
Minimum Mid Body Scale Rows: 17
Modal Mid Body Scale Rows: 17
Maximum Mid Body Scale Rows: 19
Anals: single
Min Ventrals: 140
Max Ventrals: 190
Min Subcaudals: 35
Max Subcaudals: 65
Single / Divided / Mixed: single
Coloration / Markings
Extremely variable. Dorsal surface base colours include grey, brown, olive, green and reddish with distinctive irregular paler crossbanding of similar but paler colour, usually greenish white or grey, brownish white or grey or yellowish brown. Cross banding diminishes in intensity toward the tail. Some specimens are unbanded. Head dorsum usually uniform in colour, similar to dorsal colour, grey or brown.Ventral surface usually yellow or cream.

Notechis scutatus (Easter Tiger Snake) [Original photo copyright © Dr Julian White]

Notechis scutatus (Easter Tiger Snake) [Original photo copyright © Dr Julian White]

References

Cogger H.G. (1996) Reptiles & Amphibians of Australia 5th Edition. Ed. Taylor R., Reed Books Australia, Melbourne.

Ehmann H. (1992) Encyclopedia of Australian Animals. Reptiles. Ed. Strahan R., Angus and Robertson, Pymble, NSW.

White J. (1994) Clinical Toxicology of Snakebite in Australia and New Guinea. In : Handbook of Clinical Toxicology of Animal Venoms and Poisons. Eds. Meier J. and White J., CRC Press Inc., Boca Raton, Florida. pp 595-617.

Mirtschen P.J., Crowe G.R. and Davis R. (1990) Dangerous Snakes of Australia. In : Snakes of Medical Importance (Asia-Pacific Region). Ed. Gopalakrishnakone P. and Chou L.M., National University of Singapore, Singapore. pp 1-174.

Biological Information

Dentition
Front fangs located at anterior end of maxillary bone (proteroglyphous), 4 or 5 maxillary teeth following the fang. Fang length approximately 3.5 mm (varies between 2.0 to 5.5 mm).
Breeding
Mating occurs mainly in spring (September to November) but sometimes in autumn (March to May). Ovoviviparous, usually about 20 to 40 (but as many as 80 has been recorded) young in midsummer to early autumn (January to April).
Habitat
Found in cool to warm temperate permanent watercourses, swamps and seepage areas on coastal lowlands, inland slopes, plains and ranges including the entire Murray River system. Found in a wide range of habitats including rainforest, dry and wet sclerophyll open forest and woodlands, shrublands, heath and tussock grasslands. Most of the region coincides with pasture and cropping activities. Also found in urban and semi-urban areas.

Habits
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Prey
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Venom Section

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Clinical Venom Effects Summary

General: Venom Neurotoxins
Pre- & Post-synaptic neurotoxins
General: Venom Myotoxins
Systemic myotoxins present
General: Venom Procoagulants
Prothrombin convertors
General: Venom Anticoagulants
Not present
General: Venom Haemorrhagins
Not present
General: Venom Nephrotoxins
Not present
General: Venom Cardiotoxins
Not present
General: Venom Necrotoxins
Not present
General: Venom Other
Not present or not significant

Venom Research Summary

Maximum Venom Qty
189 mg (dry weight of milked venom), Meier and White (1995) (Ref : R000001)
Average Venom Qty
30 to 70 mg (dry weight), U.S. Dept. Navy (1968) (Ref : R000914).

28 mg (dry weight), Freeman and Kellaway (1934) (Ref : R000686).

35 mg (dry weight of milked venom), Meier and White (1995) (Ref : R000001).

30 to 70 mg (dry weight), Minton (1974) (Ref : R000504).

Preferred LD50 Estimate
0.118 mg / kg sc (mice), Meier and White (1995) (Ref : R000001)
Other LD50 Estimates
0.04 mg / kg iv (mice), Kellaway (1929) (Ref : R000687).

0.25 mg / kg sc (mice), Kellaway (1929) (Ref : R000687).

0.04 mg / kg ip (mice), U.S. Dept. Navy (1968) (Ref : R000914).

0.118 mg / kg sc (mice, bovine serum albumin), Broad et al (1979) (Ref : R000006).

0.01 mg / kg iv (mice), John and Kaiser (1990) (Ref : R000636).

0.03 mg / kg iv (mice), Minton (1974) (Ref : R000504).

0.04 mg / kg ip (mice), Minton (1974) (Ref : R000504).

0.15 mg / kg sc (mice), Minton (1974) (Ref : R000504).

0.04 (0.035 to 0.045) mg / kg iv (mice), Theakston and Reid (1983) (Ref : R000689).

0.04 (0.035 to 0.045) mg / kg iv (mice), Sanchez et al (1992) (Ref : R000690).

0.005 (0.004 to 0.006) mg / kg icv (mice), Sanchez et al (1992) (Ref : R000690).

NB. icv = intracerebroventricular.

Venom Activity
Presynaptic neurotoxin, Postsynaptic neurotoxin, Procoagulant, Myotoxin

Venom Components
1. Notexin (myotoxin with PLA2 activity, presynaptic neurotoxin).

2. Notechis II-5 (myotoxin with PLA2 activity, presynaptic neurotoxin).

3. Notechis II-1 (non-lethal, non-enzymatic protein homologous with notexin) : Single chain of 119 AA residues and differs from notexin in only a few positions.

4. Toxin 1 and Toxin 2 (postsynaptic neurotoxins) described by Karlsson et al (1972) (Ref : R000028).

5. Scutoxin A and Scutoxin B (Highly toxic PLA2's). Both have Mol. Wts. approx. 13,000 and less basic than either notexin and notechis II-5. Both had specific PLA2 activity of about 136 µmoles fatty acid released / min / mg protein. Francis et al (1991) (Ref : R000647).
Neurotoxins & Channel Toxins
Notexin : Karlsson (1972) (Ref : R000028) found Notexin comprised 6% of crude venom. Harris et al (1973) (Ref : R000032) discovered notexin was presynaptic at the neuromuscular junction. Halpert et al (1975) (Ref : R000030) defined notexin's structure as 119 AA residues in a single peptide chain, crosslinked by 7 disulfide bridges, Mol. Wt 13,574. Cull-Candy et al (1976) (Ref : R000031) found Notexin causes paralysis by inhibition of acetycholine release. Death from notexin was due to respiratory failure. Pluskal (1978) (Ref : R000029) showed notexin had PLA activity and can degranulate mast cells and release vaso-active amines.

Notechis II-5: Karlsson et al (1972) (Ref : R000028) showed PLA activity of notechis II-5 was twice that of Notexin, but only 30% as lethal in mice. Halpert et al (1975) (Ref : R000030) using AA sequences, demonstrated a close relationship between notexin, notechis II-5, porcine pancreatic PLA and PLA isolated from Naja melanoleuca.

Toxin 1 and Toxin 2 : Karlsson et al (1972) (Ref : R000028) are curare-like, fast acting and have Mol. Wt. of 6,000 and 7,000 respectively. Daytner et al (1973) (Ref : R000033) found that effects of these two neurotoxins were easily reversed by antivenom.
Haematological / Haemorrhagins
Jobin et al (1966) (Ref : R000034) showed that Tiger Snake venom exerted its optimal coagulant activity only in the presence of prothrombin, Factor V, a divalent metal (Ca++, Ba++, Sr++ or Mn++) and a phospholipid emulsion. Venom was shown to be capable of activating prothrombin and incapable

of activating Factor X. Harris and MacDonell (1981) (Ref : R000037) report that the PLA2 activity of Notexin is Ca++ dependent and is inhibited by Mg++, Ba++, Sr++, Zn++ and Mn++. Tans et al (1985) (Ref : R000035), isolated the venom activator and determined is Mol. Wt. was 54,000, that it comprised 6% of the crude venom and concluded it had a structure of light and heavy polypeptide chains, held together by one or more disulfide bridges. They found that prothrombin activation by the activator alone was very slow. By adding phospholipid, Ca++ and Factor Va a dramatic increase in reaction rate occurred. The prothrombin activator resembles Factor Xa.

Myotoxins
Notexin : 119 AA residues cross-linked by 7 disulfide bridges, Mol. Wt. 13,578. Halpert and Eaker (1975) (Ref : R000030). Specific activity (25iC, pH 8 in presence of deoxycholate and Ca++) was 840 µequivalents free fatty acids liberated / min / mg protein. Halpert et al (1976) (Ref : R000415). Hood et al (1974) (Ref : R000038) and Sutherland (1977) (Ref : R000039) claimed muscle damage was likely if antivenom administration was delayed. It has been associated with gross elevation of serum enzymes and isoenzymes. Sutherland (1983) (Ref : R000007) showed, using monkeys, an elevation of plasma CK begins 2 hours post sc injection of 120 µg / kg of venom. Harris et al (1978) (Ref : R000036) showed that notexin was also myolytic. Found it produces a necrotising myopathy in skeletal muscle. They showed notexin was a potent PLA2. Harris et al (1978) (Ref : R000036) and Harris and MacDonell (1981) (Ref : R000037) showed that myolytic activity was evident over a wide pH (6.2 to 8.7) and temperature range (0i to 60iC) and was dependent on presence of Ca++ ions. The PLA2 activity of notexin is inhibited by Mg++, Ba++, Sr++, Zn++ and Mn++. Harris and MacDonell (1981) (Ref : R000037). Modification of His 48 with p-bromophenacyl bromide decreased specific activity from 850 to 1.8 µequivalents free fatty acids liberated / min / mg protein, the LD100 iv (mice) increased from 0.025 to 10 mg / kg and significantly decreased myotoxicity. Halpert et al (1976) (Ref : R000415).

Notechis II-5 : Single chain of 119 AA residues differing from notexin in only a few positions. Specific activity was 1390 µequivalents free fatty acids liberated / min / mg protein. Halpert et al (1975) (Ref : R000030). Notechis II-5 had myolytic effects on rat skeletal muscle similar to those of notexin, but was slightly less potent. Harris and Johnson (1978) (Ref : R000036). Lymphopaenia was associated with the inflammatory response after subcutaneous injection into rats. Emslie-Smith and Harris (1989) (Ref : R000416).

Component LD50
Notexin LD50 iv (mice) = 0.017 mg / kg. Cull-Candy et al (1976) (Ref : R000031).

Notexin LD50 iv (mice) = 0.007 mg / kg. Francis et al (1991) (Ref : R000647).

Notechis II-5 LD50 iv (mice) = 0.045 mg / kg. Halpert and Eaker (1976) (Ref : R000080).

Notechis II-5 LD50 iv (mice) = 0.04 mg / kg. Francis et al (1991) (Ref : R000647).

Notechis II-1 LD50 iv (mice) > 20 mg / kg (non lethal in mice). Halpert and Eaker (1976) (Ref : R000081).

Scutoxin A LD50 iv (mice) = 0.006 mg / kg. Francis et al (1991) (Ref : R000647).

Scutoxin B LD50 iv (mice) = 0.006 mg / kg. Francis et al (1991) (Ref : R000647).

Crude Venom
Crude venom : Minimum haemorrhagic dose = 42.3 µg / rat, minimum necrotizing dose = 35.5 µg / rat, minimum defibrinogenating dose whole blood clotting = no activity µg / mouse, minimum coagulant dose (human plasma) = 16.5 mg / litre, minimum coagulant dose (fibrinogen) = no activity mg / litre. Theakston and Reid (1983) (Ref : R000689).

Crude venom : Minimum haemorrhagic dose = 58.2 µg / rat, minimum necrotizing dose = 39.3 (37.5 to 42.0) µg / rat, minimum defibrinogenating dose whole blood clotting = no activity µg / mouse, minimum coagulant dose (human plasma) = 15.5 mg / litre, minimum coagulant dose (fibrinogen) = no activity mg / litre. Sanchez et al (1992) (Ref : R000690).

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Clinical Effects Section

Notechis scutatus

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Common Names
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Region
Australia
Countries

Australia

Clinical Venom Effects Summary

General: Dangerousness
Severe envenoming possible, potentially lethal
General: Rate of Envenoming: 40-60%
General: Untreated Lethality Rate: 40-50%
Approx. % Dry Bite: 50%
General: Venom Neurotoxins
Pre- & Post-synaptic neurotoxins
General: Venom Myotoxins
Systemic myotoxins present
General: Venom Procoagulants
Prothrombin convertors
General: Venom Anticoagulants
Not present
General: Venom Haemorrhagins
Not present
General: Venom Nephrotoxins
Not present
General: Venom Cardiotoxins
Not present
General: Venom Necrotoxins
Not present
General: Venom Other
Not present or not significant

Specific Clinical Effects Summary

Clinical Summary
The following applies to bites by tiger snakes in general, not specifically to the species in this record, as bites by all species are similar.

Tiger snake bites have at least a 50% rate of significant envenoming and prior to development of antivenom had a 45% fatality rate. They have small fangs, but bigger than brown snakes, they produce

more venom and the venom is highly toxic. As with brown snakes, clothing and enclosed footwear may reduce the chance of significant envenoming, but the fangs can penetrate at least one layer of normal clothing.

Tiger snake bites are usually noticed, because they are painful and are associated with local mild swelling, bruising, erythema and occasionally superficial necrosis around the bite site. The latter is more likely if either a tourniquet has been used as first aid, or if a PI bandage first aid is left on for many hours. If coagulopathy develops, then local oozing of blood may occur from the bite site.

Systemic envenoming is associated with general symptoms, including headache, nausea, vomiting, abdominal pain, collapse, convulsions (especially in young children). Vomiting and abdominal pain are often predictive of developing systemic envenoming.

The hallmark features of tiger snake bite are coagulopathy, flaccid paralysis, myolysis and renal damage. Not all occur in every case of significant envenoming.

The coagulopathy is defibrination type, with low or absent fibrinogen, gross elevation of FDP/d-dimers, prolonged to grossly prolonged INR/PT and aPTT, but often normal platelet count. The coagulopathy can develop rapidly, with complete defibrination in 15-30 mins after a major bite, if no first aid is used. Major, even lethal bleeding can occur, but spontaneous bleeding is uncommon to rare and bleeding gums are not a feature. Lethal intracranial haemorrhages have occurred, particularly in association with adrenaline premed prior to antivenom treatment, or a concurrent injury to the head. The coagulopathy responds to antivenom therapy, but even without antivenom, will resolve spontaneously after 12-15+ hours. After resolution, FDP/d-dimers are raised for many hours, giving a clue to the prior existence of a coagulopathy.

Flaccid paralysis is caused by both presynaptic and postsynaptic neurotoxins and can result in complete respiratory paralysis over a 12-24 period. First signs of paralysis, notably ptosis and diplopia, are usually seen 1-3 hrs after the bite, but may be delayed much longer than this. Without treatment they may progress through ophthalmoplegia, fixed dilated pupils, poor tongue extrusion, limited mouth opening, dysarthria, dysphagia, drooling, to limb weakness, respiratory weakness, the diaphragm usually being the last to be paralysed, often 18-24+ hrs after the bite. Drooling and inability to protect the airway may force intubation and ventilation much earlier than this. Presynaptic neurotoxins cause paralysis resistant to antivenom, thus once paralysis is established, antivenom will not reverse it, but may prevent much progression. Full paralysis may persist for days, weeks, rarely more than a month. Anticholinesterases are of no benefit in reversing presynaptic paralysis.

The myolysis seen with tiger snake bite, though not present in all cases, can be very severe, with massive skeletal muscle destruction, myoglobinuria, secondary renal failure and hyperkalaemia. The latter can be intractable and lethal. CK levels well in excess of 100,000 IU/l are common. The urine may be muddy brown to black, full of sediment, rather than the classic red of less extreme myoglobinuria . There is usually muscle pain, worse on movement or palpation, and associated muscle weakness. CK levels and muscle pain may not peak until several days after the bite and may take 1-2 weeks to resolve. Muscle weakness from myolysis can continue for weeks and cause emaciation. Secondary renal failure, though not common, is a significant risk, particularly as it will exacerbate effects of hyperkalaemia and so increase the potential for cardiotoxicity.

Fatalities are due to several different mechanisms, including fatal haemorrhages (usually intracranial), hyperkalaemic cardiac toxicity and complications or renal failure. Without treatment, respiratory paralysis will be lethal, but with modern ICU management this is rarely a cause of death.

Detail: Neurotoxicity
Neurotoxicity is the dominant clinical feature & may be moderate to severe flaccid paralysis, pre- & post-synaptic

Detail: Myotoxicity
Myolysis is common & may be moderate to severe, with potential for renal failure & hyperkalaemia

Detail: Coagulopathy
Coagulopathy is a defining feature of envenoming by this species, may be severe, is defibrination type, with potential for major bleeding

Detail: Haemorrhagins
Pathologic bleeding not likely to occur

Detail: Nephrotoxicity
Uncommon, but may be moderate to severe, potential renal failure

Detail: Necrosis
Local necrosis around the bite site is rarely reported and is usually minor

Detail: Other
No other clinically important effects are noted
Detail: Local Effects
The bitten area is usually painful, with mild to moderate swelling, erythema, sometimes with bruising
Detail: Prognosis
Fatal outcome common in untreated severe cases

Special Risk Groups

Pregnancy
Potential risk to foetus from systemic envenoming, similar to risk to mother
Children
Risk to children from local or systemic envenoming is similar in nature to the risk for adults, but a childs smaller body mass implies that the comparitive venom concentration will be higher, so more severe envenoming is likely, the increased risk being inversely proportional to the size of the child.
Elderly
The higher incidence of pre-existing diseases in the elderly and associated frailty place them at higher risk should envenoming occur. The nature and extent of increased risk will largely be determined by the nature and extent of pre-existing disease and by any medications used to treat such disease.

Bite from Notechis scutatus (Easter Tiger Snake) [Original photo copyright © Dr Julian White]

Bite from Notechis scutatus (Easter Tiger Snake) [Original photo copyright © Dr Julian White]

Case Summaries from Literature

Rollison J.W. (1928) Fatal case of tiger-snake (Notechis scutatus) bite. Adelaide Hospital Medical & Scientific Archives, No. 7, pp 18-20.

Male, aged 41, bitten on the left forearm by N. scutatus while handling snake. Used suction and tourniquet as first aid. Within 10 minutes had headache and axillary pain, then nausea, and by 45 minutes, vomiting. presented to hospital several hours later, with developing paralysis, dysarthria, diplopia, ptosis, vomiting of "dark material", and with the bite site swollen, with local ecchymosis. The paralysis progressed with difficulty in swallowing, then respiratory distress and cyanosis, free oozing of blood (coagulopathy) also noted. Finally died in respiratory failure due to paralysis about 20 hours post bite. Blood was found to be unclottable at autopsy.

(Fairley, 1929)
Male, adult, bitten on ankle and hand while handling N. scutatus. Used local incision and tourniquet as first aid. Presented six hours after the bite with a drunken gait, dysarthric, swollen tongue, hyper-reflexic, and no limb paralysis. Bite sites showed purple discolouration. Developed progressive respiratory paralysis without limb paralysis, and died of neurotoxic respiratory failure some 16.1/2 hours post bite. At autopsy the blood was not coagulated, and there were multiple small lung haemorrhages, and ecchymoses around the bite sites.

Tisdall H.T. and Sewell J.E. (1931) Treatment with antivenine twenty-four hours after a bite by a tiger snake. Med. J. Aust., May 16, pp 604-605.

Male, age 43, bitten twice on right middle finger by N. scutatus. Used local incision and tourniquet as first aid. Developed headache, pain in the bitten arm radiating to the axilla, and vomited twice about 3 hours post bite. On the following day vomited further, with continued headache and developed progressive weakness of lower limbs, unsteady gait, blurred vision, and ptosis. Presented to hospital

24 hours post bite, semi-stuperose, with dysarthria, ptosis, unreactive dilated pupils, severe weakness of limbs, numbness of bitten hand. Given tiger snake antivenom. Some 10 hours later noted to have ceased vomiting, but no change in paralysis for several hours, with gradual resolution over several days, the ptosis being present for about 8 days. Developed rash of serum sickness 6 days post antivenom therapy. Full recovery.

(Lloyd, 1932)
Male, age 27, bitten on right index finger by N. scutatus while trying to kill snake. Used incision, suction, and tourniquet as first aid, and "rum enough to make him very drunk". Presented to hospital about 1 hour later, and given tiger snake antivenom (3000 u), with possible collapse, treated with strychnine and adrenaline, and continued use of tourniquet to bitten arm. Vomited several hours later. No neurotoxic signs noted. Developed painful oedematous bite site, and about 6 days later, rash of serum sickness. Full recovery except for stiffness and loss of power in right hand.

Wallace I. (1954) Effects of tiger snake bite. Vic. Naturalist, Vol. 70, pp 227-228.

Male, adult, bitten on right thumb by N. scutatus while handling snake. Used suction only as first aid, as considered glancing trivial bite only. Two minutes later noted "heart began to beat heavily", with "peculiar burning sensation in the mouth". Visiion became blurred, then complete loss of sight, breathing difficulty, palate paralysis, all withing 10 minutes of bite. Presented to hospital and given tiger snake antivenom (3000 u), and within 3 minutes, vision cleared, but still blurred, and breathing easier. Later developed sweating and headache. At 24 hours developed very swollen right hand. Full recovery.

Symons H.S. (1960) Anaphylactic shock and subsequent dementia following the administration of tiger-snake antivenene. Med. J. Aust., December 24, pp 1010-1011.

Male, age 33, bitten on right ankle by N. scutatus while farming. Presented to hospital 2 hours later, no symptoms described, but given slow infusion of tiger snake antivenom (1500 u). Five minutes later the patient became unconscious with shock, severe hypotension, cyanosis, and sweating. Anaphylactic shock was diagnosed and treated with hydrocortisone, adrenaline, fluid load, and oxygen, but while recovery was achieved, the patient was deeply shocked for about 30 minutes. It became apparent later that brain damage had occurred, with complete aphasia, inability to feed himself, and incontinence of faeces and urine, and only partial recovery occurred. No sequelae of tiger snake envenomation were noted.

Hood V.L. and Johnson J.R. (1974) Acute renal failure with myoglobinuria following tiger snake bite. Aust. N.Z. J. Med., Vol. 4, p 415.

Male, age 47, bitten on dorsum of left hand by N. scutatus while handling the snake. Used incision, suction, and tourniquet as first aid. Within 10 minutes felt dizzy, and presented to hospital, where he received tiger snake antivenom (3000 u), without incident. The following day he became confused and disoriented, and complained of aching tender muscles, and then dark urine. By the next day he was oliguric, delirious, with myoglobin in his urine, normal coagulation, raised CPK (4640 U), LDH (3540 U), Potassium (7.7 mEg/1), Creatinine (3.7 mg/100 ml) and Urea (141 mg/100 ml). There was respiratory difficulty, and the patient was intubated and ventilated, and given peritoneal dialysis. Despite this and treatment with resonium A and insulin and glucose, the potassium rose (8.3 uEg/1). Haemodialysis was commenced, with control of hyperkalaemia. Over the next few days the patient also developed atrial fibrillation, managed with digoxin, and severe bilateral broncho- pneumonia, the latter necessitating continued ventilation for 10 days. By the end of this period severe generalised muscle wasting was also apparent, and muscle biopsies showed focal necrotising myopathy. The renal failure entered the diuretic phase at Day 26, and subsequent renal biopsy was consistent with recovery phase of acute tubular necrosis. The weakness due to muscle damage resulted in contractures, partially relieved by physiotherapy. An eventual full recovery of renal and muscle function was achieved.

Campbell C.H. (1977) The Tiger Snake : A review of the toxicology of the venom and the effect of the bite. Herpetofauna (Australasian Affiliation of Herpetological Societies), Vol. 9, No. 1, pp 7-17.

Male, age 42, bitten on the left thumb by N. scutatus, while handling snake. Used incision, suction, and tourniquet as first aid. He rapidly developed sweating, then headache, which became severe, and persisted until the next day. Visual blurring occurred 5 minutes post bite but was short lived. Presented to hospital 30 minutes post bite, then developed nausea and vomiting. No axillary tenderness, abdominal pain, or evidence of paralysis was detected, but the bite site was slightly oedematous, and a

coagulopathy was demonstrated, of the defibrination type. Tiger snake antivenom (9000 u) was given with subsequent resolution of symptoms (except headache) and coagulopathy.

Sutherland S.K. and Coulter A.R. (1977) Three instructive cases of tiger snake (Notechis scutatus) envenomation - and how a radioimunoassay proved the diagnosis. Med. J. Aust., Vol. 2, No. 6, pp 177-180.

Male, age 9, found comatose in a country area, and dead on arrival at hospital. Past history of catching snakes led to suspicion that death was due to snakebite. At autopsy, scratches on lower limbs positive for tiger snake venom (RIA), as were skin samples elsewhere, and particularly high concentrations of venom were found in the para-aortic lymph nodes, the kidneys, and the urine. The urine was dark brown, and contained myoglobin. No comment made on blood coagulation or muscle histology.

Female, age 10, found unconscious in a paddock, presented to hospital as possible head injury or overdose. Skull xrays, CSF normal, but respiration deteriorated requiring assisted ventilation. History from sibling then emerged that she may have had a snakebite. Developed anuria, and residual urine in bladder at admission was dark brown and positive for myoglobin. Serum CPK peaked at 33,200 iu/l. Child then received tiger snake antivenom (9000 u), while urine and blood on sock tested for venom, both proving positive for tiger snake venom (RIA). Required ventilation for 6 days, peritoneal dialysis for 6 days, and subsequent physiotherapy for muscle weakness. Eventual full recovery. No comment made on blood coagulation.

Female, age 7, awoke in early morning with diarrhoea and vomiting, and saying snake had been in her bedroom. Seven and a half hours later she was "very ill, with further diarrhoea and vomiting, associated with apathy, tremors, and blurred vision". By 18 hours post-bite she was stuporous, with dark urine (myoglobin), and was transferred to hospital, where she received polyvalent antivenom (=3000 u tsav), some 26 hours after initially notifying parents. She thereafter made a complete recovery. Tiger snake venom was found in pre-antivenom serum (RIA) and CPK peaked at over 4000 iu/l. No comment made on blood coagulation.

This case was re-reported by Gaynor (1977), who noted the child showed severe ptosis, palatal palsy, dysarthria, and diaphragmatic breathing. The ptosis took 72 hours to resolve. A bite site was apparent on the child's forearm, with scratch marks and a "tender, dark mauve area". Coagulation studies prior to antivenom were normal, and remained so. Pulmonary oedema was evident radiologically at admission.

Frost J. (1980) Tiger snake envenomation. Med. J. Aust., Vol. 3, p 440.

Male, age 4, presented to hospital 15 minutes after alleged tiger snake bite, and noted to be "in extremis", with pallor, central cyanosis, shallow respiration, and masseter spasm. Rapidly given tiger snake antivenom (9000 u), and within 30 minutes child regained consciousness. Two hours later a coagulopathy became apparent, with oozing from the IV site, bite site, two small haematemeses, and eruption of tense haematomas overlying the skull bones. A further dose of antivenom was given (3000 u). Coagulopathy confirmed by laboratory tests, with clear defibrination acutely, and resolution within 18 hours, except fibrinogen, which took 48 hours to reach normal levels. A full recovery was made over 2 days.

Male, age 28, presented 20 minutes after being bitten by a tiger snake above ankle. Over the following 90 minutes developed headache, backache, tender inguinal adenopathy, oozing from IV injection sites and slight haematuria. All symptoms resolved after tiger snake antivenom (12000 u) given. Coagulopathy confirmed with defibrination, and resolution over 24 hours. Skin swab strongly positive for tiger snake venom. Complete recovery.

Frost J. (1981) Tiger snake envenomation. Med. J. Aust., Vol. 2(11), p 579.

Male, age 15, presented 10 minutes after being bitten by N. scutatus on the calf. He developed frontal headache, nausea, vomiting, slowed respiration, drowsiness, ptosis, and muscle twitching, all within 20 minutes of bite. Given tiger snake antivenom (6000 u) with good recovery. However, during this time a compressive bandage and splint were used on the bite site, in total for 24 hours, with resultant local pain and calf muscle spasm, and ultimately an area of local necrosis around the bite site. No significant coagulopathy or myolysis occurred. The patient made a complete recovery, but required skin grafting to the bite site necrotic area.

Murrell G. (1981) The effectiveness of the pressure / immobilisation first aid technique in

the case of a tiger snake bite. Med. J. Aust., Vol. 2, p 295.

(Russell, 1983)
Male, adult, bitten by N. scutatus, developed headache and some muscular weakness 15 minutes post bite, with dysarthria at 30 minutes, and dull abdominal pain. By 45 minutes, had some difficulty breathing, blurred vision, ptosis, and then progressive paresis. All symptoms worsened over next 8 hours, with severe abdominal pain, bright blood in urine and stools. At 30 hours a tracheostomy was performed and artifical ventilation commenced. Coagulopathy noted (not described), also electrolyte imbalances, and a cardiac arrhythmia treated by pacemaker, but no evidence of myoglobinuria. Eventual complete recovery, apart from deficit of smell. No note of antivenom used in treatment.

Male, adult, bitten by N. scutatus, developed headache, dizziness, abdominal pain 30 minutes post bite, then axillary tenderness on bitten side, and mild weakness of that arm at 90 minutes. This progressed to weakness of both upper limbs, with worsening abdominal pain. Antivenom (?tiger snake) given at this time, with resolution of symptoms, though haematuria and coagulopathy noted, the latter lasting about 48 hours.

(Lodge et al, 1988)
Female, age 62, bitten on left hand by N. scutatus while reaching under her bed for cigarettes. Tiger snake venom subsequently found at bite site (ELISA-VDK). She received tiger snake antivenom (9000 u). She developed anuric renal failure, rhabdomyolysis, disseminated intravascular coagulation, haemolysis, and pulmonary oedema. Myoglobin peaked at 1876 u/L, and no myoglobinuria was detected. The renal failure was treated using haemodialysis, but she developed a chest infection and died 6 days post bite. She had pre-existing multiple sclerosis, hypertension, maturity onset diabetes, and chronic pyelonephritis.

(from White, 1987)
Notechis scutatus. Female, age 27. Bitten on third toe of left foot while walking barefoot on a warm evening (see figure 8). Within one hour developed headache, vomiting, local pain in bitten toe and associated oedema. By 9 hours post bite developed ptosis, dysarthria, and oliguria. Given antivenom at this time (1 ampoule polyvalent = 3000 u tiger snake), and no progression of paralysis ensued. Coagulopathy poorly documented. Oliguria responded to fluid load and IV Lasix. Ptosis, mild loss of smell, took several weeks to fully resolve.

(from White, 1987)
Notechis scutatus. Male, age 66. Bitten on index finger of right hand while attempting to hold snake (figure 10). Shortly afterwards felt faint, then collapsed, Promptly treated at local hospital, where given one ampoule tiger snake antivenom (3000 u). Developed only mild pain and swelling at bite site, no evidence of paralysis, but a definite coagulopathy was noted initially, which rapidly responded to antivenom.

Notechis scutatus. Female, age 73. Bitten on dorsum of left foot while using outdoor toilet (figure 9). Initial local pain, becoming worse overnight, with some local ecchymosis. Only systemic problems were mild nausea and vomiting at 4 hours, and laboratory evidence of a mild coagulopathy. Successfully treated with polyvalent antivenom (3000 u tiger snake antivenom). No evidence of paralysis or myolysis.

Notechis scutatus. Male, age 33. Bitten on right hand while moving hollow logs. Subsequently developed nausea, vomiting, local pain, oedema and ecchymosis, diarrhoea, drowsiness, mild ptosis, dark urine (?myoglobin) and tender axillary adenopathy. Laboratory evidence of coagulopathy. All problems resolved after adequate antivenom therapy, some 3 ampoules of tiger snake antivenom (9000 u).

White J., Tomkins D., Steven I. and Williams V. (1983-84) Tiger Snake Bite. Records of the Adelaide Children's Hospital, Vol. 3, No. 2, pp 169-173.

Notechis scutatus. Female, age 2. Bitten on left calf twice while stepping out of suspended sand pit, under which the tiger snake had been sheltering (figure 7). Unable to give mother a history of snakebite initially, so that presenting symptom was a grand mal convulsion. Child subsequently developed impaired conscious state, mild paralysis, with ptosis, and a coagulopathy. Treated with one ampoule of tiger snake antivenom (3000 u), and a further ampoule after worsening of ptosis. No further progression of paralysis, and gradual resolution of coagulopathy ensued, but CK rose to 6426 iu/l at 22 hours post bite. Also developed moderate hyponatraemia, and this is thought to

have precipitated a second grand mal convulsion at about 18 hours post bite, complicated by severe aspiration pneumonia, cerebral oedema, and subsequent temporary leucopenia. Full recovery was evident within ten days.

Notechis scutatus. Male, age 22. Bitten on dorsum of first web space, left hand, while trying to feed pet snake. Used tight tourniquet as first aid. Developed severe pain in hand, and subsequently an area of necrosis was delineated which subsequently required skin grafting. Systemically had chest pain, nausea, vomiting, and tender left adenopathy prior to antivenom, and also a brief period of loss of consciousness (but significantly inebriated as well as envenomed). Given one ampoule of tiger snake antivenom (3000u), with resolution of symptoms. Paralysis, myolysis, and coagulopathy not documented.

Penington A. and Johnstone B. (1997) A case of local tissue necrosis following a bite by the Australian tiger snake Notechis scutatus. Aust. N.Z. J. Surg., Vol. 67, No. 6, pp 385-388.

A single case report of a 20 month old boy bitten by a tiger snake (positive ID by venom detection) while walking in bushland in rural Victoria, Australia in 1997 (year case reported, not certain if this was the year bite occurred). Shortly after the bite he collapsed, had an epileptic fit, then recovered consciousness. Several bite marks on the leg and wrist were noted and a PI bandage first aid was applied (probably > 15 mins after the bite). On arrival at a hospital he was drowsy. The first aid was removed about 2 hrs after the bite, followed by marked deterioration in his condition, so he was given 1 ampoule of CSL Tiger Snake Antivenom IV, but developed stridor and required intubation. He was given a further 2 ampoules and FFP. Blood tests around this time showed severe defibrination (INR >10). He was transferred to a major hospital, where the coagulopathy was 'starting to improve', though he was then given a further 2 ampoules of antivenom and more FFP. By the next day he was improved and was extubated. CK at this time was 5650 IU/L. He was also noted to have an area of necrotic skin, about 4x2 cm, around a bite site on his leg. This was debrided and the underlying calf explored, but no compartment syndrome was found. Histology of the excised necrotic area of skin and underlying fat showed thrombosed vessels at the margin and a larger deeper vessel had necrotic walls and was full of thrombus. The defect was successfully skin grafted.

This case adds to the experience with tiger snake bite causing local necrosis around the bite site, though this occurs in a minority of bites, even severe bites.

Nocera A., Gallagher J. and White J. (1998) Severe tiger snake envenomation in a wilderness environment. Med. J. Aust., Vol. 168, No. 2, pp 69-71.

Single case report of a 44 yr old man bitten on the ankle by an unseen animal, later identified as a tiger snake (poisitive ID by venom detection), while bushwalking in an inaccessible area of the Blue Mountains, near Sydney, Australia in 1997. He was not discovered until nearly 16 hrs after the bite and then airlifted to a major hospital, by which time he is severely paralysed (full ptosis, fixed dilated pupils, dysarthric, respiration abdominal), cyanosed, hypotensive (BP 80 systolic), hypothermic (temp. 35.4 C), with coarse creps in both lung bases. Poor weather conditions delayed the retreival process, which took nearly 8 hrs. On arrival at the hospital, about 24 hrs after the bite, he was still hypothermic (35.1 C), BP 106/71, he had frank myoglobinuria, deteriorating renal function (creatinine 200 umol/l), gross myolysis (CK = 287,200 IU/L), but almost normal coagulation (INR = 1.4; aPTT = 38 secs; XDP = 'positive'), potassium normal (4.5 mmol/l). He was given 6 ampoules of CSL Tiger Snake Antivenom, then 2 more ampoules, supplemented by 2 ampoules of CSL Polyvalent Snake Antivenom. Following this there was a slight improvement in pupillary response. It was noted he had swollen forearms and about 6 hrs after arrival he had bilateral decompressive fasciotomies for compartment syndrome. Thereafter there was a slow improvement, but he required ventilation for 13 days and haemodialysis for 32 days, going home 34 days after arrival.

This case is unique for Australian tiger snake bites, because of the development of bilateral upper limb compartment syndromes, presumably associated with the severe myolysis (the bite was to a lower limb). The resolution of the coagulopathy prior to antivenom is typical of tiger snake bite, where coagulopathy starts to resolve after 12-15 hrs post bite, even without antivenom therapy. XDP are a good late marker for the earlier presence of such a coagulopathy. The massive dose of antivenom was given in the hope it would modify progress of the myolysis. This was based on experience with earlier cases. The case report does not detail sequential laboratory findings, but discussion with the authors suggests there was a clear improvement in myolysis following the antivenom. Experience with other cases suggests that without antivenom, the natural history of the myolysis would have been for further worsening, over several days, not the resolution seen in this case.

Tibballs J., Henning R.D., Sutherland S.K., and Kerr A.R. (1991) Fatal cerebral haemorrhage after tiger snake (Notechis scutatus) envenomation. Med. J. Aust., Vol. 154, No. 4, pp 275-276.

Single case report of 11 yr old boy bitten several times on the wrist by a tiger snake (positive ID of snake) in northeast Victoria, Australia in 1989. Inadequate first aid used. Developed nausea within 10 mins of the bite and by 25 mins was drowsy, vomiting, had muscle weakness, abdominal pain, BP 110/70. He was given a premed of IV promethazine 20mg and IV adrenaline 0.25mg, then 1 ampoule each of CSL Tiger Snake and Brown Snake Antivenoms (identity of snake not yet established). During the antivenom treatment his symptoms improved, but shortly after became irritable and aggressive, a reaction blamed on the promethazine. His BP remained stable. He remained unchanged until about 10 hrs after the bite, when his conscious state deteriorated, he had fixed dilated pupils, but able to move all limbs. He had small retinal haemorrhages. He was given 1 ampoule each of tiger and brown snake antivenoms. A third ampoule of tiger snake antivenom was then given, as the snake had now been identified. He showed no improvement with this antivenom and was transferred to a major hospital. On arrival he was not rousable, only the left pupil was fixed dilated and he had multiple petichial haemorrages on his limbs. He had a CT scan of his head which showed an 'extensive haemorrhage in the right frontal region with displacement of the falx to the left of the midline. Aditional smaller haemorrhages were observed in the right posterior lobe and the left frontal lobe.' Coagulation tests at this time showed evidence of a resolving defibrination coagulopathy (Pt = 16 secs; aPTT = 38 secs; fibrinogen = 0.95 g/l; XDP = 16-32 mg/l; platelets 255). There was also myoglobinuria. He had an intracranial pressure of 75mmHg. By 26 hrs after the bite all motor responses had ceased and all brain stem reflexes were absent. Treatment was withdrawn and he then died. Autopsy revealed the CT observed multiple intracerebral haemorrhages and subarachnoid collections of blood. There were no thrombi nor evidence of cerebral infarction. Though the authors do not emphasise the point, subsequent discussion through letters to the editor highlight the likely role of adrenaline premedication in causation of the intracranial bleeding. It should be noted that an IV route was used for the adrenaline for unknown reasons; this route has never been recommended by those who championed the use of adrenaline premedication.

This case was the first recent case to document the dangers of adrenaline premedication for antivenom and though the authors subsequently defended the continued use of adrenaline premed, the result has been a progressive shift away from use of premed for antivenom in Australia and reversal of manufacturers recommendations.

McGarity B.H., Marshall G.P., Loadsman J.A., Carr S.J. and Harper C.G. (1991) Fatal cerebral haemorrhage after tiger snake bite. Med. J. Aust., Vol. 155, No. 1, pp 61-62.

Single case report of 40 yr old man bitten on the heel by a tiger snake (positive ID by venom detection) in the Bathurst region, NSW, Australia. He was initially uncertain if bitten so no first aid was immediately, but later a venous tourniquet was used. When seen at hospital 1 hr after the bite he had nausea, vomiting, was sweaty, pale and confused, BP 120/80. Blood taken shortly after showed a mild coagulopathy (INR = 1.9; aPTT = 54 secs; XDP = 32-128 mg/l; platelets 238). About 2 hrs after the bite he was given a premed of IV hydrocortisone and IV adrenaline followed by 1 ampoule of CSL Tiger Snake Antivenom. There was a transient rise in BP to 195/105 after the adrenaline premed. There was some initial improvement in symptoms, then vomiting recurred and about 75 mins after the antivenom he had a left sided fit followed by left paresis and hemianopia, without retinal haemorrhage evident. A further 2 ampoules of antivenom were given and 4 units of FFP. Coagulation tests at this time showed severe defibrination (INR = 19; aPTT > 300secs). About 60 mins later he lost consciousness and the right pupil dilated, followed by decorticate posture. He was intubated, ventilated, given IV mannitol and dexamethasone, further antivenom and FFP. His coagulopathy now showed evidence of resolution (INR = 2.7; aPTT = 59 secs). However, both pupils then became fixed dilated and a CT head showed a massive right intracerebral haemorrhage. Supportive care was withdrawn the next day, with subsequent death. Autopsy showed a 'massive right intracerebral haemorrhage, with rupture into the right basal ganglia and cerebellum. The brain was oedematous and there was both right tentorial and subfalcial herniation.' The authors conclude that the adrenaline premed was most likely the cause of the haemorrhage, in association with the venom-induced coagulopathy.

Currie B.J. and Hudson B.J. (1991) Reply to : Fatal cerebral haemorrhage after tiger snake bite. Med. J. Aust., Vol. 155, p 280.

Comment on earlier case, pointing out that increasing experience with adverse effects of adrenaline

premedication prior to antivenom use has caused a change in practice in Darwin; such premedication is no longer used if there is a coagulopathy present or potentially present. The authors also point out the value of a whole blood clotting test in looking for coagulopathy.

Tankel A.S. (2001) Anaphylaxis associated with the same batch of tiger-snake antivenom. Med. J. Aust., Vol. 174, pp 608.

A brief letter to the editor noting 3 cases of snakebite in the Coffs Harbour area, NSW, Australia, with use of CSL Tiger Snake Antivenom, where adverse reaction to the antivenom occurred, questioning if there was a problem batch of antivenom.

Case 1: 67 yr old herpetologist bitten by a broad headed snake, Hoplocephalus bungaroides, who developed a 'significant' coagulopathy, was given tiger snake antivenom, but quickly developed a wheeze, plus was hot, flushed, and required intubation. No note of hypotension.

Case 2: 17 yr old youth bitten by unidentified snake (positive ID by venom detection, for tiger snake; this could include related species in this area), who developed coagulopathy, was given tiger snake antivenom, but developed an urticarial reaction. (No other reaction noted).

Case 3: 18 yr old man bitten by a red bellied black snake, Pseudechis porphyriacus, who developed 'significant systemic envenomation' and was given tiger snake antivenom, preceeded by adrenaline premed, but still developed 'marked urticaria'. (No other effects noted).

The author notes that only the third case had adrenaline premed and this was the least severe case of adverse reaction to the antivenom, though this proves little, as the author's speculation that this was a problem batch of antivenom was not confirmed by subsequent testing.

Maher D.W. (2001) Reply to : Anaphylaxis associated with the same batch of tiger-snake antivenom. Med. J. Aust., Vol. 174, p 608-609.

A reply to the above case reports, pointing out that neither internal CSL testing, nor reports of use of this batch in other hospitals has confirmed any batch quality issues. He further re-enforces the CSL view that adrenaline premed for antivenom is no longer recommended. Further, all reported reactions in the earlier paper were readilly treated and were mostly minor and within the spectrum of known adverse effects of antivenom.

Winkel K.D. (2001) Reply to : Anaphylaxis associated with the same batch of tiger-snake antivenom. Med. J. Aust., Vol. 174, p 609-610.

A further comment on the paper by Tankel, suggesting that adverse reactions to CSL antivenoms still occur and promoting the use of adrenaline premed for antivenom. As the letter is from the Director of the AVRU, a unit established by the champion of adrenaline premed for antivenom, and no substantial evidence is advanced to prove that either reactions to antivenom are common, or adrenaline is actually warranted, it adds little to the discussion.

MOCK UP OF SUBSCRIBER PAGES FOR A SINGLE RECORD (example is Australian mainland tiger snake) Treatment Section

Notechis scutatus

General Details, Taxonomy and Biology, Venom, Clinical Effects, Treatment, First Aid , Antivenoms

Family: Elapidae
Genus: Notechis
Species: scutatus
Common Names
Easter Tiger Snake , Mainland Tiger Snake

Region
Australia
Countries

Australia

Diagnosis: Summary of principle clinical effects

Key Diagnostic Features
Local pain, erythema, bruising + flaccid paralysis, defibrination coagulopathy, myolysis ± renal damage
General: Local Effects
Local pain, swelling & bruising
General: Local Necrosis
Rarely occurs, minor only
General: General Systemic Effects
Variable non-specific effects which may include headache, nausea, vomiting, abdominal pain, diarrhoea, dizziness, collapse or convulsions
General: Neurotoxic Paralysis
Very common, flaccid paralysis is major clinical effect
General: Myotoxicity
Very common, major clinical effect, usually moderate to severe
General: Coagulopathy & Haemorrhages
Very common, coagulopathy is major clinical effect
General: Renal Damage
Recognised complication, usually secondary to myolysis
General: Cardiotoxicity
Unlikely to occur
General: Other
Not likely to occur

Treatment

Treatment Summary
Bites by tiger snakes are characterized by local pain, slight swelling, local bruising, rarely superficial local necrosis around the bite site, and systemic flaccid paralysis (pre- & post-synaptic), myolysis (potentially very severe), defibrination coagulopathy (even without treatment this may resolve over 12-18 hrs after the bite), and secondary renal failure. Probably most bites will develop at least some systemic envenoming, so many cases will require antivenom therapy. Admit all cases of definite or suspected bites.

On presentation, establish a good IV line, commence IV fluids, take bloods for initial tests ('extended coagulation tests' = PT/INR, aPTT, fibrinogen level, FDP/XDP, platelet count; CK, urea, creatinine, WCC, K+), perform venom detection on the bite site (if uncertain of identity of snake). Unless contraindicated by pre-existing medical conditions, give an IV fluid load (1L over 2-3 hrs in an adult; volume determined by weight in a child), then keep well hydrated therafter (100-150 mL/hr in an adult), while carefully watching for fluid overload, monitoring fluid input and output. If a first aid bandage is in place over the bitten limb, leave on until the blood tests, venom detection and full examination are complete and results available. To perform venom detection cut away the bandage immediately over the bite area, to swab for venom. Keep the cut-away bandages, in case they are needed later for venom detection.

If the patient presents with envenoming clearly established, or blood tests show evidence of systemic envenoming (defibrination coagulopathy with elevated PT/INR, aPTT, low or absent fibrinogen, often grossly elevated FDP/XDP; developing flaccid paralysis, even just ptosis; developing myolysis, elevated CK >1,000; secondary renal damage, elevated creatinine & urea), then commence antivenom therapy prior to removal of first aid. Initial dose should be at least 3-4 vials of CSL Tiger Snake AV IV, diluted up to 1:10 (less in children, to avoid volume overload), with adrenaline ready to treat anaphylaxis, should this occur.

Repeat blood tests 3 hrs after completion of initial dose of antivenom therapy, to determine if coagulopathy or myolysis has been arrested (rise in fibrinogen; no further or minimal rise in CK). If the CK has continued to climb dramatically, or the fibrinogen has failed to rise or has fallen further, give further antivenom (usually another 3-4 vials). If the 3hr tests suggest improvement then wait 2-3 hrs and retest. If there is continued improvement, it is likely no further antivenom will be needed. If, however, there has been no improvement or worsening, give further antivenom. The role of antivenom in reversing myolysis is controversial, but given the potential lethality of severe myolysis, further antivenom therapy should always be considered.

For cases where initial blood tests show no sign of either myloysis (CK) or defibrination coagulopathy, and there is no evidence of flaccid paralysis on examination, no antivenom is required at this stage, and first aid should then be removed, but repeat tests 2-3 hrs and 5-6 hrs later and if these show developing envenoming, treat with antivenom, as discussed earlier.

For cases with myolysis, CK may rise to very high levels, >100,000 IU/L. This is accompanied by muscle pain, tenderness and weakness in most cases. There will also be gross myoglobinuria (red to black urine) and the risk of renal failure. Hyperkalaemia can develop secondary to myolysis and is more severe if there is secondary renal failure. It can cause cardiotoxicity and is potentially lethal and may be difficult to treat.

The defibrination coagulopathy is associated with major bleeding, but not commonly. Beware cases with recent trauma or surgery, or who sustain trauma after the bite, particularly a blow to the head, such as caused by a collapse shortly after the bite, as intracranial haemorrhage may develop. However, unlike brown snakes ans taipans, tiger snake defibrination coagulopathy is only short-lived, reversing even without antivenom 12-18 hrs after the bite. For this reason, resolution of coagulopathy cannot be used to guide antivenom dose or effectiveness if >12 hrs have elapsed after the bite.

Flaccid neurotoxic paralysis is a major feature of tiger snake envenoming. Because there are potent presynaptic neurotoxins prominent in the venom, antivenom therapy is very unlikely to reverse paralysis already established. There is no value in continually giving antivenom in the hope of reversing established paralysis, but if paralysis is incomplete, then it is certainly worth giving antivenom to try and prevent progression of paralysis. In this latter situation it is likely that some progression of paralysis will occur over 1-3+ hrs after antivenom, caused by venom already bound to the terminal axon of the neuromuscular junction.

Tiger snake bites commonly develop both local pain and bruising, usually with slight swelling. Though local necrosis is quite uncommon, it is a known complication of tiger snake bites, but is usually limited to the skin immediately around the fang marks. More extensive necrosis occurs if first aid is left on for many hours or if a tourniquet is used as first aid. Secondary infection is uncommon, but if there is a strong suspicion of secondary infection with cellulitis, then antibiotic therapy, initially IV, is required, but in most cases of tiger snake bites, antibiotics are not required. Tetanus immunisation status should always be checked and a booster given when indicated, but not until any coagulopathy has been reversed.

General Approach to Management

All cases should be treated as urgent & potentially lethal. Rapid assessment & commencement of

treatment including appropriate antivenom (if indicated & available) is mandatory. Admit all cases.
Simplified Treatment Flow Chart:
Click here for a Simplified Treatment Flow Chart.
Immediate Effects Management
Establish IV line, IV fluids, secure airway, maintain respiration if imperilled, treat hypotension (if present), do not clean wound until venom detection performed
Important Laboratory Test
Coagulation (WBCT or PT/INR, aPTT, fibrinogen, FDP/XDP); FBC/CBP (platelets, Hb, WCC, absolute lymphocyte count); renal function (creatinine, urea); CK; electrolytes (K, Na, Cl, Glucose).
Local Effects Management
Do not clean wound
General Systemic Effects Management
Symtomatic care + IV fluids (as required)
Neurotoxic Paralytic Effects Management
Support respiration, protect airway, intubate & ventillate if required, give appropriate antivenom at earliest sign of paralysis
Neurotoxic Excitatory Effects Management
Excitatory neurotoxic effects not likely
Neurotoxic Other Effects Management
Psychological support of paralysed patient
Myotoxic Effects Management
Ensure good renal flow (adequate IV fluids), consider appropriate antivenom, consider alkalinising urine, watch for secondary renal damage & hyperkalaemia
Haematologic Effects Management
Haematologic effects not likely
Haematologic Other Effects Management
Not applicable
Renal Effects Management
Monitor renal function & output, consider IV fluid load, treat renal failure as required
Cardiotoxin Effects Management
Monitor for secondary cardiotoxicity due to hyperkalaemia induced by myolysis
Necrotoxin Effects Management
Local necrosis unlikely
Other Specific Effects Management
Ensure tetanus immunisation, after any coagulopathy reversed
Other Issues in Treatment
Not applicable
Antivenom Therapy
Antivenom is the key treatment for systemic envenoming. Multiple doses may be required.
Antivenom Dosage
Refer to dosage schedule for relevant antivenoms listed on this site
Antivenom Reactions
Full range possible; see relevant section in antivenom pages
Adverse AV Reaction Management
Cease antivenom, resuscitate, adrenaline, IV fluids, then cautiously restart antivenom
Follow Up
Follow up all cases given antivenom therapy, checking particularly for serum sickness

Principle Laboratory Findings

WBCT: Prolonged
PT / INR: Grossly prolonged
aPTT: Grossly prolonged
Fibrinogen: Defibrination
FDP/XDP: Grossly elevated
Platelets: Most likely normal
CK: Potentially grossly elevated
Creatinine: Secondary renal failure
K++: Potential secondary elevation
WCC: Leukocytosis
Abs. Lymph: Lymphopenia
Urea: Secondary renal failure
Hb: Most likely normal
APaO2: Potentially low secondary to paralytic respiratory failure

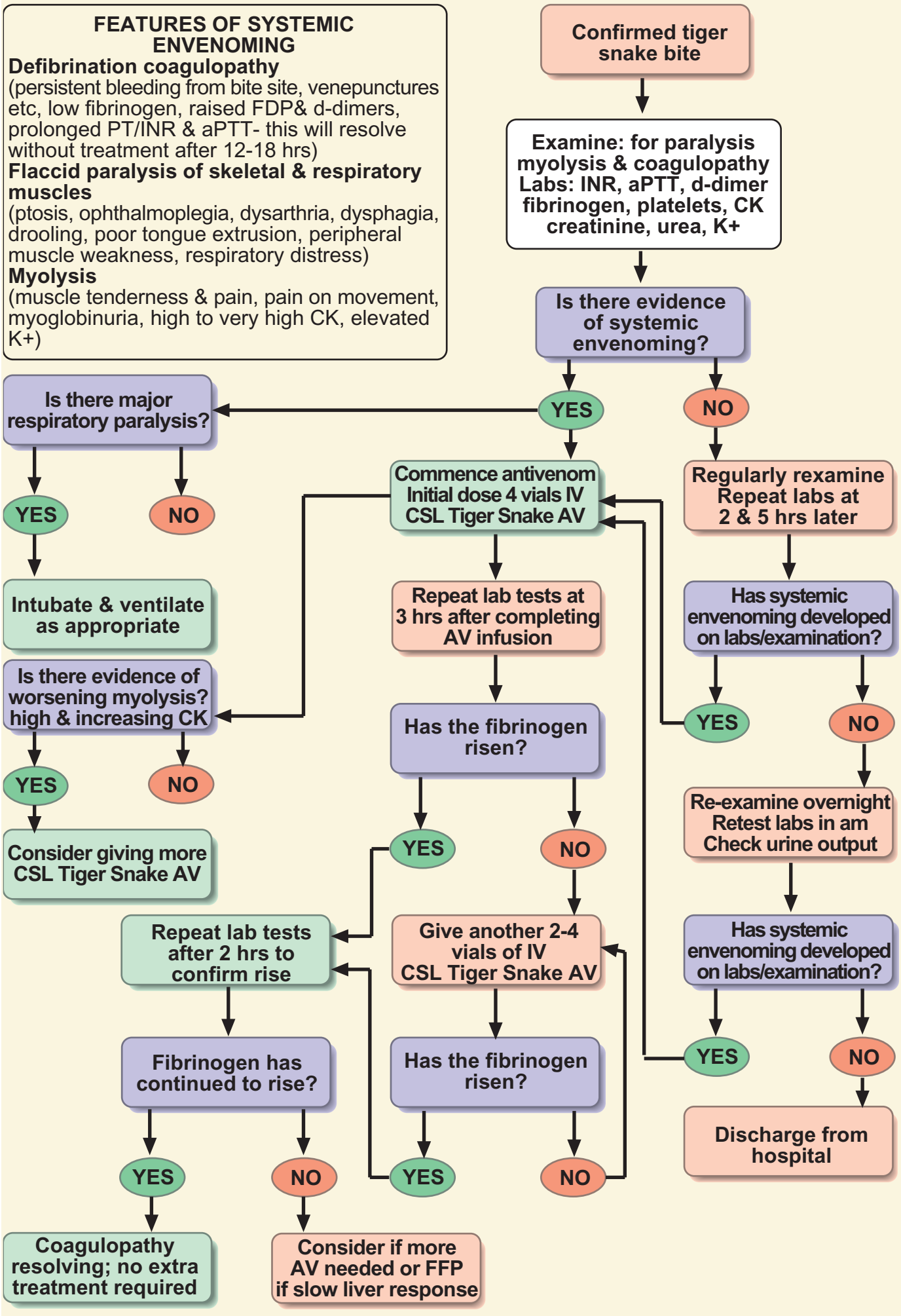
CLINICAL DECISION TREE
Australian tiger snakes - Genus *Notechis*

FEATURES OF SYSTEMIC ENVENOMING

Defibrination coagulopathy
(persistent bleeding from bite site, venepunctures etc, low fibrinogen, raised FDP& d-dimers, prolonged PT/INR & aPTT- this will resolve without treatment after 12-18 hrs)

Flaccid paralysis of skeletal & respiratory muscles
(ptosis, ophthalmoplegia, dysarthria, dysphagia, drooling, poor tongue extrusion, peripheral muscle weakness, respiratory distress)

Myolysis
(muscle tenderness & pain, pain on movement, myoglobinuria, high to very high CK, elevated K+)



MOCK UP OF SUBSCRIBER PAGES FOR A SINGLE RECORD (example is Australian mainland tiger snake) First Aid Section

Notechis scutatus

General Details, Taxonomy and Biology, Venom, Clinical Effects, Treatment, First Aid , Antivenoms

Family: Elapidae
Genus: Notechis
Species: scutatus
Common Names
Easter Tiger Snake , Mainland Tiger Snake

Region
Australia
Countries

Australia

First Aid

Description: First aid for bites by Elapid snakes which do not cause significant injury at the bite site (see Comments for partial listing), but which may have the potential to cause significant general (systemic) effects, such as paralysis, muscle damage, or bleeding.

Details: 1. After ensuring the patient and onlookers have moved out of range of further strikes by the snake, the bitten person should be reassured and persuaded to lie down and remain still. Many will be terrified, fearing sudden death and, in this mood, they may behave irrationally or even hysterically. The basis for reassurance is the fact that many venomous bites do not result in envenoming, the relatively slow progression to severe envenoming (hours following elapid bites, days following viper bites) and the effectiveness of modern medical treatment.
2. The bite wound should not be tampered with in any way. Wiping it once with a damp cloth to remove surface venom is unlikely to do much harm (or good) but the wound must not be massaged. For Australian snakes only, do not wash or clean the wound in any way, as this may interfere with later venom detection once in a hospital.
3. All rings or other jewellery on the bitten limb, especially on fingers, should be removed, as they may act as tourniquets if oedema develops.
4. If the bite is on a limb, a broad bandage (even torn strips of clothing or pantyhose) should be applied over the bitten area at moderate pressure (as for a sprain; not so tight circulation is impaired), then extended to cover as much of the bitten limb as possible, including fingers or toes, going over the top of clothing rather than risking excessive limb movement by removing clothing. The bitten limb should then be immobilised as effectively as possible using an extemporised splint or sling.
5. If there is any impairment of vital functions, such as problems with respiration, airway, circulation, heart function, these must be supported as a priority. In particular, for bites causing flaccid paralysis, including respiratory paralysis, both airway and respiration may be impaired, requiring urgent and prolonged treatment, which may include the mouth to mask (mouth to mouth) technique of expired air transfer. Seek urgent medical attention.
6. Do not use Tourniquets, cut, suck or scarify the wound or apply chemicals or electric shock.
7. Avoid peroral intake, absolutely no alcohol. No sedatives outside hospital. If there will be considerable delay before reaching medical aid, measured in several hours to days, then give clear fluids by mouth to prevent dehydration.
8. If the offending snake has been killed it should be brought with the patient for identification (only

relevant in areas where there are more than one naturally occurring venomous snake species), but be careful to avoid touching the head, as even a dead snake can envenom. No attempt should be made to pursue the snake into the undergrowth as this will risk further bites.
9. The snakebite victim should be transported as quickly and as passively as possible to the nearest place where they can be seen by a medically-trained person (health station, dispensary, clinic or hospital). The bitten limb must not be exercised as muscular contraction will promote systemic absorption of venom. If no motor vehicle or boat is available, the patient can be carried on a stretcher or hurdle, on the pillion or crossbar of a bicycle or on someone's back.
10. Most traditional, and many of the more recently fashionable, first aid measures are useless and potentially dangerous. These include local cauterization, incision, excision, amputation, suction by mouth, vacuum pump or syringe, combined incision and suction ("venom-ex" apparatus), injection or instillation of compounds such as potassium permanganate, phenol (carbolic soap) and trypsin, application of electric shocks or ice (cryotherapy), use of traditional herbal, folk and other remedies including the ingestion of emetic plant products and parts of the snake, multiple incisions, tattooing and so on.

Comments: Snakes included in this group are Australian Elapid snakes (especially species in the following Genera; Acanthophis, Austrelaps, Hoplocephalus, Notechis, Oxyuranus, Pseudechis, Pseudonaja, Tropidechis), sea snakes, kraits (Bungarus spp.), coral snakes (from Americas and Asia; Micrurus, Micruroides, Maticora, Calliophis), mambas (Dendroaspis spp.), shield nose snakes (Aspidelaps spp.), African garter snakes (Elapsoidea spp.), selected cobras, such as Philippines cobra (Naja philippinensis), forest cobra (Naja melanoleuca), Egyptian cobra (Naja haje), Cape cobra (Naja nivea), water cobra (Boulengerina spp.), oxus cobra (Naja oxiana), black desert cobra (Walterinnesia aegyptia). King cobras (Ophiophagus hannah), though capable of causing local tissue injury at the bite site, are likely to cause paralysis which may prove lethal if untreated, therefore these snakes should also be included in the above grouping.

MOCK UP OF SUBSCRIBER PAGES FOR A SINGLE RECORD (example is Australian mainland tiger snake) Antivenoms Section

Notechis scutatus

General Details, Taxonomy and Biology, Venom, Clinical Effects, Treatment, First Aid , Antivenoms

Family: Elapidae
Genus: Notechis
Species: scutatus
Common Names
Easter Tiger Snake , Mainland Tiger Snake

Region
Australia
Countries

Australia

1. Antivenom Code: SAuCSL10

Antivenom Details

Antivenom Name: Tiger Snake Antivenom
Antivenom Type: Snake (SN)
Immunisation Host: Horse serum
Product Description: Salt precipitated, pepsin digested.
Liquid final product 7.4 to 11.9 ml.
3000 units / ampoule.
Dialysed and ultra-filtered to a final concentration of 170 g / l protein.
1 unit antivenom neutralizes 0.01 mg venom in vitro.
Source Species: Notechis scutatus
Coverage Species: Austrelaps superba , Austrelaps ramsayi , Notechis ater , Notechis scutatus , Pseudechis colletti , Pseudechis guttatus , Pseudechis porphyriacus , Tropidechis carinatus
Administration Route: i.v.
Volume: 10 ml (approx.)
Initial Dose (Mnfr): Depends upon severity.
Local Cost: A$230.00 / 3,000 units (2000).
Storage Life: 3 years, Expiry date noted on vial.
Languages on Label: English
Comments: Sometimes used for sea snake bites if specific CSL Sea snake antivenom is unavailable.

Antivenom Manufacturer

Manufacturer: CSL Limited
Address: 45 Poplar Road
Parkville
Victoria 3052
Country: Australia
Phone: ++61-3-9389-1624
Toll free: 1800 642 865
Fax: ++61-3-9389-1160
Telex: AA32789

E-mail: pharminf@csl.com.au
Web Site: www.csl.com.au
Status: Active manufacturer & supplier
Contact: Peter Hobbs
Medical Affairs Manager
45 Poplar Rd, Parkville, VIC 3052
Ph: 03-9389-191
Fax: 03-9389-1160
peter_hobbs@cls.com.au
Medical Expert: Assoc. Prof. Julian White
Head of Toxinology Department
Women's and Children's Hospital
72 King William Street, North Adelaide,
SA 5006
Ph: 08-8204-7436; Fax: 08-8204-6049
toxinaus@wch.sa.gov.au
mobile: 0419-825-029
Number of AV: 12
AV Code Nos.: MAuCSL01 ; MAuCSL02 ; MAuCSL03
IAuCSL04 ; IAuCSL05 ; IAuCSL06
SAuCSL07 ; SAuCSL08 ; SAuCSL09
SAuCSL10 ; SAuCSL11 ; SAuCSL12
Antivenoms made by this company: Box jellyfish antivenom (Sheep) [Chironex fleckeri , Chiropsalmus quadrigatus]

Stonefish antivenom (Horse) [Synanceja trachynis , Synanceja species]

Sea snake antivenom (Horse) [Notechis scutatus , Enhydrina schistosa , Aipysurus laevis , Astrotia stokesii , Hydrophis species , Laticauda laticaudata , Laticauda semifasciata , Lapemis hardwickii , Hydrophis gracilis , Pelamis platurus , Thalassophina viperina. Also covers Austrelaps superbus , Pseudechis porphyriacus , Tropidechis carinatus.]

Funnel-web spider antivenom (Horse) [Atrax robustus , Atrax species , Hadronyche species]

Red-backed spider antivenom (Horse) [Latrodectus hasselti , Latrodectus species.]

Tick antivenom (Dog) [Ixodes holocyclus]

Death Adder Antivenom (Horse) [Acanthophis antarcticus, Acanthophis pyrrhus , Acanthophis praelongus]

Taipan Antivenom (Horse) [Oxyuranus microlepidota , Acanthophis antarcticus , Pseudechis australis , Notechis scutatus , Pseudonaja textilis , Oxyuranus scutellatus]

Black Snake Antivenom (Horse) [Pseudechis australis , Pseudechis colletti , Pseudechis guttatus , Pseudechis porphyriacus]

Tiger Snake Antivenom (Horse) [Austrelaps superba , Austrelaps ramsayi , Notechis ater , Notechis scutatus , Pseudechis colletti , Pseudechis guttatus , Pseudechis porphyriacus , Tropidechis carinatus]

Brown Snake Antivenom (Horse) [Pseudonaja affinis , Pseudonaja nuchalis , Pseudonaja textilis]

Polyvalent Snake Antivenom (Australia - New Guinea) (Horse) [Acanthophis antarcticus, Austrelaps superba , Notechis scutatus , Oxyuranus microlepidotis , Oxyuranus scutellatus , Pseudechis papuanus, Pseudechis australis , Pseudonaja affinis , Pseudonaja nuchalis , Pseudonaja textilis]

2. Antivenom Code: SAuCSL12

Antivenom Details
Antivenom Name: Polyvalent Snake Antivenom (Australia - New Guinea)
Antivenom Type: Snake (SN)

Immunisation Host: Horse serum
Product Description: Salt precipitated, pepsin digested.
Liquid final product 48.3 to 51.5 ml.
3000 units / ampoule.
Dialysed and ultra-filtered to a final concentration of 170 g / l protein.
1 unit antivenom neutralizes 0.01 mg venom in vitro.
Source Species: Oxyuranus scutellatus , Acanthophis antarcticus , Notechis scutatus , Pseudechis australis , Pseudonaja textilis
Coverage Species: Acanthophis antarcticus, Austrelaps superba , Notechis scutatus , Oxyuranus microlepidotis , Oxyuranus scutellatus , Pseudechis papuanus, Pseudechis australis , Pseudonaja affinis , Pseudonaja nuchalis , Pseudonaja textilis
Administration Route: i.v.
Volume: 50 ml (approx.)
Initial Dose (Mnfr): Taipan 12000 units, King Brown 18000 units, Tiger 3000 units, Death Adder 6000 units and Brown 1000 units.
Local Cost: A$1270.00 / 40,000 units (2000).
Storage Life: 3 years, Expiry date noted on vial.
Languages on Label: English

Antivenom Manufacturer

Manufacturer: CSL Limited
Address: 45 Poplar Road
Parkville
Victoria 3052
Country: Australia
Phone: ++61-3-9389-1624
Toll free: 1800 642 865
Fax: ++61-3-9389-1160
Telex: AA32789
E-mail: pharminf@csl.com.au
Web Site: www.csl.com.au
Status: Active manufacturer & supplier
Contact: Peter Hobbs
Medical Affairs Manager
45 Poplar Rd, Parkville, VIC 3052
Ph: 03-9389-191
Fax: 03-9389-1160
peter_hobbs@cls.com.au
Medical Expert: Assoc. Prof. Julian White
Head of Toxinology Department
Women’s and Children’s Hospital
72 King William Street, North Adelaide,
SA 5006
Ph: 08-8204-7436; Fax: 08-8204-6049
toxinaus@wch.sa.gov.au
mobile: 0419-825-029
Number of AV: 12
AV Code Nos.: MAuCSL01 ; MAuCSL02 ; MAuCSL03
IAuCSL04 ; IAuCSL05 ; IAuCSL06
SAuCSL07 ; SAuCSL08 ; SAuCSL09
SAuCSL10 ; SAuCSL11 ; SAuCSL12
Antivenoms made by this company: Box jellyfish antivenom (Sheep) [Chironex fleckeri , Chiropsalmus quadrigatus]

Stonefish antivenom (Horse) [Synanceja trachynis , Synanceja species]

Sea snake antivenom (Horse) [Notechis scutatus , Enhydrina schistosa , Aipysurus laevis , Astrotia stokesii , Hydrophis species , Laticauda laticaudata , Laticauda semifasciata , Lapemis hardwickii , Hydrophis gracilis , Pelamis platurus , Thalassophina viperina. Also covers Austrelaps superbus , Pseudechis porphyriacus , Tropidechis carinatus.]

Funnel-web spider antivenom (Horse) [Atrax robustus , Atrax species , Hadronyche species]

Red-backed spider antivenom (Horse) [Latrodectus hasselti , Latrodectus species.]

Tick antivenom (Dog) [Ixodes holocyclus]

Death Adder Antivenom (Horse) [Acanthophis antarcticus, Acanthophis pyrrhus , Acanthophis praelongus]

Taipan Antivenom (Horse) [Oxyuranus microlepidota , Acanthophis antarcticus , Pseudechis australis , Notechis scutatus , Pseudonaja textilis , Oxyuranus scutellatus]

Black Snake Antivenom (Horse) [Pseudechis australis , Pseudechis colletti , Pseudechis guttatus , Pseudechis porphyriacus]

Tiger Snake Antivenom (Horse) [Austrelaps superba , Austrelaps ramsayi , Notechis ater , Notechis scutatus , Pseudechis colletti , Pseudechis guttatus , Pseudechis porphyriacus , Tropidechis carinatus]

Brown Snake Antivenom (Horse) [Pseudonaja affinis , Pseudonaja nuchalis , Pseudonaja textilis]

Polyvalent Snake Antivenom (Australia - New Guinea) (Horse) [Acanthophis antarcticus, Austrelaps superba , Notechis scutatus , Oxyuranus microlepidotis , Oxyuranus scutellatus , Pseudechis papuanus, Pseudechis australis , Pseudonaja affinis , Pseudonaja nuchalis , Pseudonaja textilis]