

Snakebite and Spiderbite Clinical Management Guidelines 2013 - Third Edition

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Summary Revised clinical resource document which provides information and advise on the management of patients with actual or suspected snakebite or spiderbite, and the appropriate levels, type and location of stored antivenom in NSW health facilities. These are clinical guidelines for best clinical practice which are not mandatory but do provide essential clinical support.

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SNAKEBITE AND SPIDERBITE CLINICAL MANAGEMENT GUIDELINES

PURPOSE

Clinical resource document to advise on the management of patients with actual or suspected snakebite or spiderbite, and the appropriate levels, type and location of stored antivenom in NSW health facilities. These are clinical guidelines for best clinical practice which are not mandatory but do provide essential clinical support.

KEY PRINCIPLES

Determination of antivenom stock requirements is best done at a regional level, either for a whole Local Health District (LHD) or important regions within a Local Health District in collaboration with local Critical Care Clinicians based a review of risks, facilities, past usage and other practical considerations using the following principles:

- Geographic location and degree of isolation
- Local snake and spider distribution
- History of envenoming cases
- Referral role of regional, rural and metropolitan hospitals.

Whilst, the definitive management of snake envenoming can only occur in a hospital with a laboratory that can do an INR/aPTT and there is sufficient nursing care; antivenom treatment can (and should) be given to obviously envenomed patients in smaller hospitals without laboratory services prior to retrieval.

Specifically, the guidelines recommended that at a minimum ALL hospitals in NSW should have:

- One (1) vial of brown snake antivenom
- One (1) vial of tiger snake antivenom
- One (1) vial of polyvalent antivenom should be kept in larger regional and referral hospitals, retrieval services across NSW, and in larger hospitals west of the Great Dividing Range for mulga snake
- Two (2) vials of funnel-web spider antivenom should be kept in all hospitals where the spider occurs.

USE OF THE GUIDELINE

The guidelines should be used as a clinical resource document to assist in the assessment, decision making and clinical management of patients with confirmed or suspected snakebite or spiderbite, and the appropriate levels, type and location of stored antivenom in NSW health facilities.

REVISION HISTORY

Version	Approved by	Amendment notes
March 2014 GL2014_005	Chief Health Officer and Deputy Director General, Population and Public Health	Updated guideline
May 2007 GL2007_006	Director General	New guideline

ATTACHMENTS

1. Snakebite and Spiderbite Clinical Management Guidelines 2013 – Third edition

SNAKEBITE & SPIDERBITE CLINICAL MANAGEMENT GUIDELINES

Third Edition



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Expert review panel

- Associate Professor Geoff Isbister, Senior Research Academic, University of Newcastle and Clinical Toxicologist and Emergency Physician, Calvary Mater Newcastle.
- Professor Nick Buckley, Professor in Medicine, Medical Professorial Unit, POW Hospital Clinical School, University of NSW and Senior Staff Specialist Poisons Information Centre.

Previous contributors (2007 edition)

- Professor Dr Julian White, Consultant Clinical Toxicologist, Women and Children’s Hospital, Adelaide, South Australia. In particular NSW Health would like to acknowledge Professor White’s contribution of images, charts and tables, which are an educational component of these Guidelines.
- Dr Lindsay Murray, Director, NSW Poisons Information Centre, Emergency Physician and Clinical Toxicologist, Sir Charles Gardener General Hospital, Senior Lecturer in Emergency Medicine University of Western Australia.

Review coordination

Mr Bart Cavalletto and Ms Christine Frew, Specialty Service and Technology Evaluation Unit, Health System Planning and Investment Branch, NSW Ministry of Health.

List of Abbreviations

AMRS	Aeromedical and Medical Retrieval Service
AV	Antivenom
aPTT	activated partial thromboplastin time
CK	Creatinine Kinase
CSL	Commonwealth Serum Laboratory
EUC	Electrolytes (Sodium/ Potassium/ Chloride)/ Urea/ Creatinine
FBC	Full Blood Count
FDP	Fibrinogen Degradation Products
FFP	Fresh Frozen Plasma
INR	International Normalised Ratio
LDH	Lactate Dehydrogenase
LHD	Local Health District
LFT	Liver Function Test
NETS	NSW newborn and paediatric Emergency Transport Service
NSAIDS	Non-Steroidal Anti-Inflammatory Drugs
PIB	Pressure Immobilisation Bandage
PIC	Poisons Information Centre
SVDK	Snake Venom Detection Kit
VICC	Venom-Induced Consumption Coagulopathy
WBCT	Whole Blood Clotting Time
WCC	White Cell Count
XDP	Serum Cross-linked Fibrin

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ONE

Introduction

These Guidelines provide information to assist clinicians in the assessment, decision making and clinical management of patients presenting with suspected or confirmed snakebite or spiderbite, for those species normally found in NSW.

This is the 3rd edition of the “New South Wales Snakebite and Spiderbite – Clinical Management Guidelines” which was first released in 1998.

Two key components have undergone significant review:

- The initial part of the guidelines have been simplified and brought in line with the Therapeutic Guidelines advice.
- Antivenom stocks and dosages have been brought in line with current evidence-based clinical practice.

In summary, these guidelines recommend that the decision to stock any antivenom should be based on a review of risks, facilities, past usage and other practical considerations. Determination of antivenom stock requirements is best done at a regional level, either for a whole Local Health District (LHD) or important regions within a Local Health District in collaboration with local Critical Care Clinicians. Whilst, the definitive management of snake envenoming can only occur in a hospital with a laboratory that can do an INR/aPTT and there is sufficient nursing care; antivenom treatment can (and should) be given to obviously envenomed patients in smaller hospitals without laboratory services prior to retrieval.

As such; it is recommended that at a minimum all hospitals in NSW should have:

- One (1) vial of brown snake antivenom.
- One (1) vial of tiger snake antivenom.
- One (1) vial of polyvalent antivenom should be kept in larger regional and referral hospitals, retrieval services across NSW, and in larger hospitals west of the Great Dividing Range for mulga snake.
- Two (2) vials of funnel-web spider antivenom should be kept in all hospitals where the spider occurs.

Two (2) vials of red-back spider antivenom may be kept in any hospital that has the facilities to safely administer antivenom.

Red-back antivenom is sometimes given:

- if there is a history, symptoms and signs consistent with systemic envenoming, and
- severe pain unresponsive to oral analgesics.

However, recent trials show red-back antivenom has a low response rate little better than placebo, and any effect is less than might be achieved with optimal use of standard analgesics.

There are no useful or diagnostic laboratory tests for red-back spider bite.

Appendices contain educational information on first aid management, laboratory studies, antivenom stocking principles, species identification and features of individual snakes. This information is not usually required to guide appropriate management decisions.

In addition, there are references to other resources for further information.

It is essential to obtain early advice on the management of snakebites and spiderbites from the **NSW Poisons Information Centre (13 11 26)**.

TWO

Clinical Advice and Resources

Poisons Information Centre

Advice on the management of snakebite and spiderbites.

Tel. 131126

Medical retrieval

Adults – NSW Aeromedical and Medical Retrieval Service (AMRS) (formerly MRU).

Tel. 1800 650 004

Neonates and children – NSW newborn and paediatric Emergency Transport Service (NETS)

Statewide service providing expert clinical advice, clinical co-ordination, emergency treatment and stabilisation and inter-hospital transport for very sick babies and children up to the age of 16 years. It operates 24 hours a day, 7 days a week.

Tel. 1300 362 500

Clinical and educational resource links

Australian Museum – Spiders and other arachnids

<http://australianmuseum.net.au/Spiders>

Australian Spiders – photos of araneomorphs (modern spiders)

<http://www.xs4all.nl/~ednieuw/australian/Spidaus.html>

Clinical Toxinology Resources

– Women’s and Children’s Hospital Adelaide

<http://www.toxinology.com/>

Agency for Clinical Innovation (Emergency Care Institute)

Clinical tools: envenomation

<http://www.ecinsw.com.au/node/183>

NSW Therapeutic Advisory Group (TAG)

Maintains a list of stock levels of snakebite and spiderbite antivenoms as part of the Life Saving Drugs Register

<http://www.ciap.health.nsw.gov.au/nswtag/documents/publications/other-docs/lsd-current.pdf>

Toxicology and Wilderness Expert Group

Therapeutic guidelines: toxicology and wilderness. Version 2.

Melbourne: Therapeutic Guidelines Limited; 2012

<http://www.tg.org.au/index.php?sectionid=170>

THREE

Snakebite Key Principles

- Snakebite is a potential medical emergency and should always receive high priority assessment and treatment, even if the patient appears initially well.
- Manage cases only in hospitals with laboratory facilities on-site and antivenom. (Point of care pathology testing is not sufficient).
- Admit all cases of probable snakebite for at least 12 hours after the bite to a suitable clinical unit such as an emergency department observation or short stay ward, high dependency unit or other clinical unit where close observation can be maintained. Patients should have serial blood testing (aPTT, INR, CK) and serial neurological examinations.
- Antivenom should be given as soon as there is clear evidence of envenoming. Evidence for systemic envenoming includes history of sudden collapse, venom induced consumption coagulopathy, neurotoxicity, myotoxicity, systemic symptoms and renal impairment.
- Although the definitive care of patients with snake envenoming cannot occur in small hospitals without laboratory facilities or sufficient medical or nursing care to allow close observation, antivenom treatment can be given. In patients with clear evidence of clinical envenoming or sufficient concern by the treating clinicians, antivenom should be administered in smaller hospitals prior to retrieval.
- Each venomous snake group causes a characteristic clinical syndrome which can be used with local geographical distribution information to determine the likely snake. Either one or more monovalent antivenom(s) that cover possible snakes or polyvalent antivenom can be used with equal effect.
- One vial of the correct antivenom is sufficient to neutralise all circulating venom, however recovery may be delayed as many clinical effects of venom are not immediately reversible.
- The majority of snakebites will not result in significant envenoming and will not require antivenom.
- For expert advice on envenoming contact the **NSW Poisons Information Centre (13 11 26)**.

FOUR

Snakebite Guidelines for Assessment and Management

Introduction

Snake envenoming is rare with only a few hundred cases and 1-4 deaths each year in Australia. Suspected snakebite is more common and the observation and exclusion of envenoming in these cases can be done in most rural and regional hospitals if they have on-site laboratory facilities and antivenom. Snake envenoming is potentially life-threatening. As it is uncommon, most medical practitioners have limited experience. However, expert advice to support these guidelines is available at any time through the [NSW Poisons Information Centre](#) on 13 11 26.

Epidemiology

Most bites occur in the warmer months and in regional and rural areas. Brown snakes are the most common cause of severe envenoming and make up the majority of confirmed envenomings in NSW. Antivenoms that cover the important local snakes should be held in most hospitals. Snake collectors may have uncommon snakes in captivity that rarely bite or do not occur in the wild in that part of NSW (e.g. Collett's snake, death adders and taipans) in which case the monovalent antivenom may not be immediately available and polyvalent antivenom should be used. Snake handlers may be reluctant to have antivenom as there is a myth that they are more likely to have a reaction to antivenom. There is no evidence to suggest they are at greater risk of anaphylaxis to antivenom. They are at risk of anaphylaxis to venom and this should be considered in the differential diagnosis.

Clinical effects

In most snake bites, insufficient venom is injected by the snake to cause clinical effects (referred to as a dry bite). Envenoming is indicated by local effects, systemic symptoms (nausea, vomiting, headache, abdominal pain, diarrhoea, diaphoresis), collapse, and other major toxin syndromes (Table 1).

Table 1. Clinical syndromes from Australian snakebite

CLINICAL SYNDROME	CHARACTERISTICS
Sudden collapse	Collapse/syncope that occurs within an hour of the bite and often before patients present to hospital. The collapse is associated with hypotension and loss of consciousness. Spontaneous recovery usually occurs within minutes but in some cases patients have a cardiac arrest or seizure.
Venom-induced consumption coagulopathy (VICC)	<p>Activation of the clotting pathway and consumption of important clotting factors, including fibrinogen, factor V and factor VIII leads to a consumptive coagulopathy. The INR and aPTT are raised or unrecordable, fibrinogen is low or undetectable and very high D-Dimer.</p> <p>Complete or severe VICC is defined as an undetectable fibrinogen, unrecordable INR and aPTT, and very high d-Dimer (10 to 1000 times the assay cut-off).</p> <p>Less severe changes are referred to as Partial VICC (detectable fibrinogen, and INR <3.0)</p>
Neurotoxicity	A descending flaccid paralysis which classically first involves the eye muscles (ptosis, diplopia and blurred vision), followed by bulbar muscles, respiratory muscle paralysis and limb paralysis.
Myotoxicity	Local or generalised myalgia and/or muscle tenderness. The CK is usually normal on admission and then rapidly rises over 24-48 hours (CK ranges from 1000 U/L in mild cases to >100,000 U/L in severe cases).
Anticoagulant coagulopathy	The aPTT is moderately raised (1.5 to 2.5 x normal) with or without a mild elevation in the INR and a normal D-Dimer. This coagulopathy provides a good marker of envenoming by black snakes but is not clinically important.
Thrombotic microangiopathy (TMA)	The presence of intravascular haemolysis on blood film, thrombocytopenia and a rising creatinine which may lead to acute renal failure requiring dialysis.
Systemic symptoms	Non-specific systemic symptoms include nausea, vomiting, abdominal pain, diarrhoea, diaphoresis and headache.

Local effects are not a major feature of bites by Australian snakes and do not indicate severity. Local effects are usually minimal with brown snakes (the most frequently lethal snake), whereas local pain, swelling and bruising are common with black and tiger snake bites. Regional swelling may also occur with these snakes that can cause myotoxicity. Local effects may be the only clinical manifestation of bites from some snakes of minor importance such as whip snakes.

The most important clinical effects from systemic envenoming include coagulopathy, neurotoxicity, myotoxicity and thrombotic microangiopathy (Table 1). The most common serious effect is venom induced consumption coagulopathy (VICC) which accounts for about three-quarters of cases requiring antivenom treatment.

Neurotoxicity and myotoxicity are far less common and take many hours to develop (therefore their low incidence may relate to the early use of antivenom for VICC in many cases). Neurotoxicity manifests as a descending flaccid paralysis where ptosis is the most common early sign. Myotoxicity may not be manifest for 6 to 12 hours post-bite. Thrombotic microangiopathy is always associated with VICC and is characterised by moderate to severe thrombocytopenia, microangiopathic haemolytic anaemia (with fragmented red blood cells) and acute renal impairment/failure.

Anticoagulant 'coagulopathy' occurs with black snake envenoming. However, the elevated aPTT is not associated with important coagulopathy or bleeding. It can be a useful indicator of envenoming to support antivenom use, as black snakes also cause myotoxicity. Renal impairment/failure is most common with brown snakes and is usually associated with thrombotic microangiopathy. Systemic symptoms, including nausea, vomiting, headache, abdominal pain, diarrhoea and diaphoresis (sweating) occur in many cases of snake envenoming, but may at times be difficult to distinguish from anxiety.

Clinical assessment

The key aspects of clinical assessment of a patient with snake bite should include a careful history of the events of the bite and any symptoms or signs that developed in the first hour. A history of an early collapse in a patient bitten by a snake suggests severe envenoming, most likely by a brown snake (Table 2).

Examination for snake envenoming syndromes should focus on:

1. Neurological examination (assessing ptosis, ophthalmoplegia, cranial nerves for bulbar weakness, limb weakness and respiratory muscle weakness).
2. Evidence of bleeding (including from the bite site, cannula site and occult sites of bleeding, such as gastrointestinal, urinary or intracranial).
3. Examination of the bite site is important looking for fang marks, bruising or local necrosis. Draining lymph nodes may be painful and support systemic envenoming.

Note: *Life-threatening scenarios are very unusual and also highly variable in their clinical presentation. Applying PBI has a limited role in hospital – generally only applicable in very small hospitals with no antivenom or lab arranging transfer. i.e. first aid not definitive management.*

Australian snakes cause distinct clinical syndromes

Table 2 provides a summary of the major clinical effects of the important groups of Australian snakes. [Appendix 5](#) provides distribution maps of the most common location of each snake in NSW.

Brown snakes

Brown snakes are the most common cause of severe envenoming in NSW and Australia. They are fast moving, easily alarmed snakes that are rarely kept in captivity. They are widely distributed throughout Australia including both arid and wetter areas, adapt well to human land use and are the most common snake in urban areas and farmlands. Brown snake bites cause minimal local effects and systemic symptoms (often mild) occur in less than half of cases. The major clinical effect is VICC which in about 80% of cases is severe (Table 1). An early cardiovascular collapse is reported in over a third of cases subsequently identified to have severe VICC. Spontaneous recovery is usual over a few minutes after the collapse but a small proportion (<5%) have a cardiac arrest which is fatal if left untreated. Thrombotic microangiopathy occurs most commonly with brown snake envenoming. Major haemorrhage (intracranial or gastrointestinal) occurs in less than 5% of cases. Treatment is with brown snake antivenom.

Tiger snakes

Tiger snakes occur in southern Australia. They are the most common cause of snake envenoming in Victoria and Tasmania, and are responsible for only a small number of cases in NSW. Tiger snake envenoming usually causes local pain and bruising, and systemic symptoms. The major serious toxicity is VICC. Neurotoxicity and myotoxicity occur in 20-30% of envenomings. Collapse, thrombotic microangiopathy and major haemorrhage are rare. Treatment is with tiger snake antivenom.

Rough-scaled snake

Rough-scaled snakes cause similar clinical effects to tiger snakes. They are confined to coastal regions in northern NSW and southern Queensland. The major clinical effect is VICC. Neurotoxicity and myotoxicity appear to be less common than for tiger snakes. Other effects are rare. Treatment is with tiger snake antivenom.

Broad-headed snakes (Hoplocephalus spp.)

This genus includes Stephen's banded snake (*H. stephensii*), the broad headed snake (*H. bungaroides*) and the pale-headed snake (*H. bitorquatus*). These snakes occur in Eastern Australia and the majority of the bites are in snake handlers. They cause VICC without neurotoxicity or myotoxicity, similar to brown snake envenoming. The snake venom detection kit (sVDK) may be positive for tiger but treatment can be with either tiger or brown snake antivenom.

Black snakes

The black snake group includes the red-bellied black snake in eastern coastal Australia, which is a common cause of envenoming in this region and the mulga snake that occurs across Australia except the south and east. Black snakes cause systemic symptoms, anticoagulant coagulopathy and myotoxicity. Collett's snake is a rare black snake (most bites in snake handlers) with a similar clinical picture to other black snakes. Red-bellied black snake is treated with tiger snake antivenom. Mulga and Collett's snake are treated with black snake antivenom or polyvalent antivenom.

Table 2. Summary of features of venomous Australian snakes

SNAKE	COAGULOPATHY	NEUROTOXICITY	MYOTOXICITY	SYSTEMIC SYMPTOMS	THROMBOTIC MICRO-ANGIOPATHY	CARDIOVASCULAR EFFECTS
Brown Snake	VICC	Rare and mild	–	<50%	Uncommon	Collapse (33%) Cardiac arrest (rare)
Tiger snake group						
Tiger snake	VICC	Uncommon	Uncommon	Common	5%	Rare
Rough-scale snake	VICC	Uncommon	Uncommon	Common	<5%	Rare
Black snakes						
Mulga snake	Anti-coagulant	–	Uncommon	Common	–	–
Red-bellied black snake	Anti-coagulant	–	Uncommon	Common	–	–
Hoplocephalus spp.	VICC	–	–	< 50%	<5%	–
Death adder	–	Common	–	Common	–	–
Taipan	VICC	Common	Rare	Common	Rare	Uncommon

Taipans

Taipans occur in northern Australia and although a highly dangerous snake are a rare cause of bites. The major clinical effect is neurotoxicity, which occurs in most cases and VICC which may be less severe than other snakes. Treatment is with taipan antivenom or polyvalent antivenom.

Death adders

Death adders occur across most of mainland Australia (except the south-east i.e. Victoria) and have a distinctive "viper"-like appearance. They usually cause bites when trodden on or near, although half of bites are in snake handlers. They cause neurotoxicity with systemic symptoms. Treatment is with death adder antivenom or polyvalent antivenom.

Other elapids

Whip snakes occur across most of Australia. They generally cause local pain and swelling that may require symptomatic relief in some cases, but non-specific systemic effects are rare. Copperhead snakes (*Austrelaps superbus*) bites are rare, thought to cause neurotoxicity. Treatment is with tiger snake antivenom. Other elapids such as secretive snakes, curl curl snakes are rare and have not been reported to cause major effects.

Investigations

The following investigations are required for diagnosis in suspected snake bite and the treatment of snake envenoming:

- Coagulation studies: A laboratory capable of measuring international normalized ratio (INR) and activated partial thromboplastin time (aPTT) is essential for the management of any snake bite. Point of care devices are inaccurate in snakebite and should not be used. D-dimer and fibrinogen may assist in the diagnosis of VICC but are not essential. The d-Dimer may be elevated 100 to 1000 times normal and remain high for days. Modest increases in d-Dimer should be interpreted with caution, particularly if they are the only evidence for envenoming.
- Full blood count (FBC) including a blood film: A blood film is important in VICC to look for associated red cell fragmentation which would indicate thrombotic microangiopathy. Thrombocytopenia will also occur in thrombotic microangiopathy. A non-specific

leukocytosis and lymphopenia is often associated with systemic envenoming.

- Biochemistry: Serial electrolytes, urea and creatinine (EUC) will detect renal impairment. Creatine kinase will detect myotoxicity, but usually doesn't rise for 6 hours and lags behind the clinical muscle injury by up to 24 hours. Lactate dehydrogenase may indicate haemolysis in thrombotic microangiopathy.

Tests not to be used

Point of care testing (PoCT) for INR or d-Dimer should not be used in the assessment of suspected snake bite patients. There are many cases of false negative INR results in patients with severe VICC. Similarly, the point of care d-Dimer result can be normal with severe VICC. It is important to make sure that small laboratories are not using point of care testing. The whole blood clotting test (or time) is also unreliable. In particular, it is often normal in patients with severe VICC. Assessment of suspected snakebite requires a laboratory that can do an INR. Patients should be transferred ASAP to a hospital that can do this test if it is not available within 2 hours by transporting the specimen.

Snake venom detection kit (sVDK)

The sVDK was designed to determine which of the five major snake groups caused a bite in patients with envenoming to determine which antivenom to use. It should not be used in non-envenomed patients and cannot be used to exclude envenoming. It is best done using a swab from the bite site, although urine can also be used. It should not be used on whole blood or serum. A positive urine test does not indicate systemic envenoming. The sVDK is best done by laboratory staff. A bite site swab can be stored without testing until there is evidence for envenoming. In many cases its use is unnecessary as the geographical location of the bite and clinical and laboratory features indicate which snake group is involved and antivenom can be given based on the clinical diagnosis.

The sVDK has a high false positive rate, particularly in the brown snake well. The sVDK result should only be used in conjunction with the differential diagnosis based around the clinical findings and laboratory tests and with knowledge of snakes occurring in the local geographical region.

First aid

The first aid for a suspected or definite snake bite is a pressure bandage with immobilisation (PBI). The pressure bandage should be a broad (15 cm) elasticised bandage, rather than a crepe bandage. The bandage is applied over the bite site and then distally to proximally covering the whole limb. It should be applied about as tight as that used for a sprained ankle. The limb and whole patient should be immobilised for the first aid to be effective.

The bandage and immobilisation should remain until the patient has been transferred and assessed in hospital. The bandage should only be removed if antivenom is available and after there is no evidence of envenoming based on the admission laboratory tests and clinical examination. If the patient is envenomed the bandage can be removed after antivenom has been administered.

Treatment

Diagnostic process

Examination and investigations should focus on determining if the patient is envenomed and what treatment is required. Most patients present with a history of suspected or confirmed snakebite, but the type of snake and whether or not they are envenomed is not clear. Occasionally, patients present with coagulopathy, following a collapse and no history of a bite.

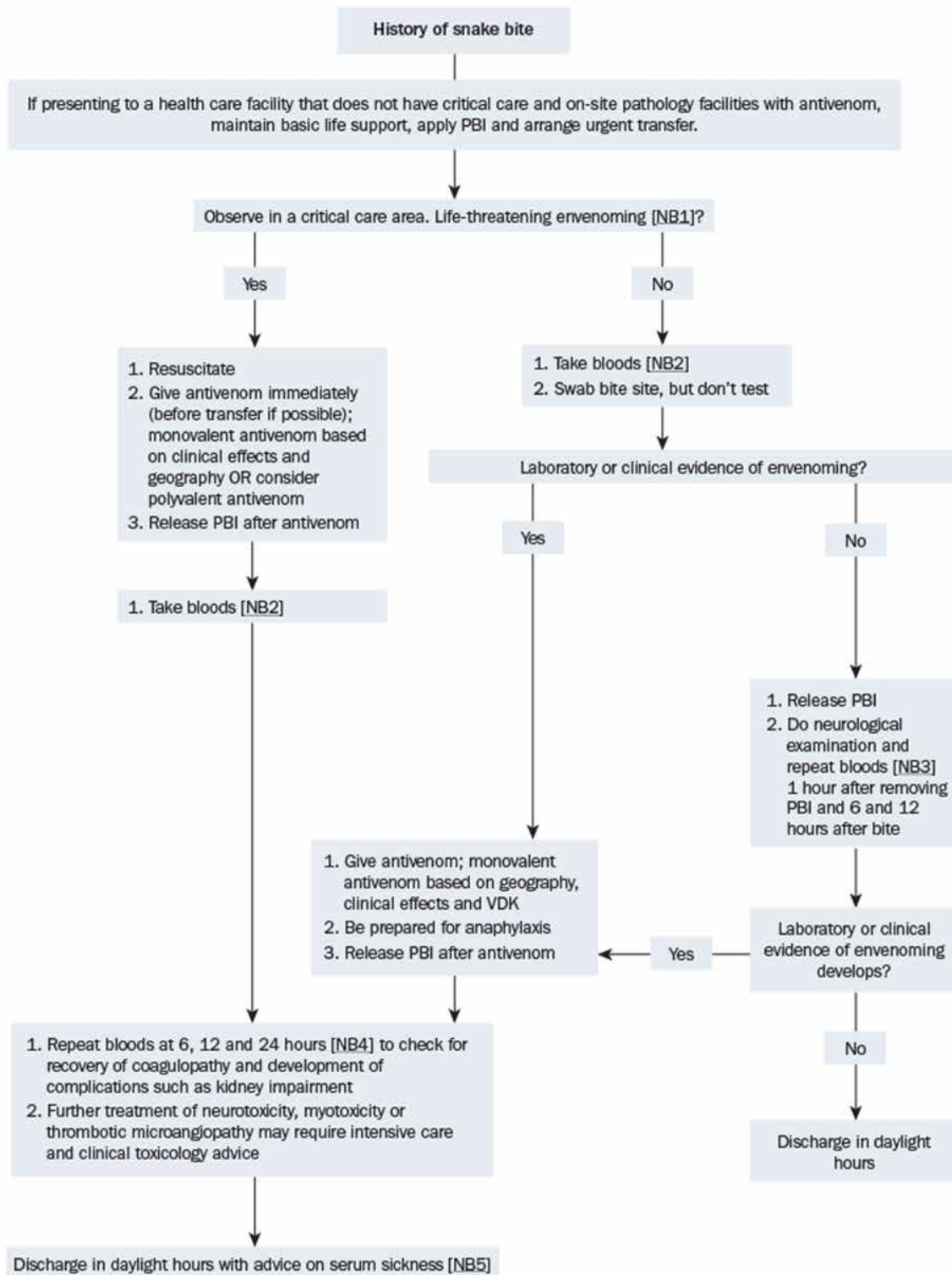
There are just a few logical steps in the diagnosis and treatment of suspected snake bite:

1. Establish whether or not the patient has systemic envenoming.
 - a. Patients can be discharged after 12 hours if there is no evidence of envenoming (see Appendix 7)
2. In patients who have envenoming, determine which type of snake(s) is likely to be involved.
3. Treat with antivenom that covers the likely snake(s) and admit for observation or adjunctive treatment (e.g. mechanical ventilation) of the clinical syndrome from that snake (if required).

The key decisions are shown in a flow chart for patients with suspected snake bite (Figure 1). This covers the process of excluding snake envenoming in asymptomatic patients or patients with minor effects and the diagnosis and treatment of snake envenoming.

All suspected snake bite patients initially require a full set of investigations (INR, aPTT, FBC, EUC, CK) on admission irrespective of whether they are clinically envenomed or are asymptomatic. A few investigations listed (i.e. d-Dimer, fibrinogen and LDH) may not be available but are not essential for the initial assessment of snake bite or uncomplicated envenoming.

Figure 1. Flow chart for the management of snake bite.



NB1: Clinically apparent features of life-threatening envenoming: cardiac arrest, neurotoxicity with risk of respiratory failure, and major haemorrhage (e.g. intracranial, gastrointestinal bleeding).

NB2: Blood tests: coagulation screen (INR, APTT, *D-dimer* and *fibrinogen* may be measured, although are not essential for initial management); full blood count and blood film; EUC, CK, LDH, liver biochemistry

NB3: Serial blood tests in non-envenomed patients: INR, APTT, CK.

NB4: Serial blood tests in envenomed patients: INR, APTT, CK, full blood count, EUC.

NB5: Advise at the time of discharge about the possibility of symptoms of serum sickness (rash, arthritis, fever, etc...) occurring 4 to 14 days after antivenom.

Exclusion of envenoming

The initial presentation and admission bloods diagnose most cases of envenoming. If laboratory studies on admission are normal and there is no clinical evidence of envenoming, the pressure bandage can be removed in a critical care area (where antivenom is available) and the patient observed carefully. Evidence of envenoming will usually develop rapidly within an hour post-removal of the bandage; if it is going to occur. If there is no evidence of envenoming after an hour the patient should be observed for 12 hours after the time of the bite with the following laboratory tests and a careful neurological examination (ptosis, bulbar, respiratory or distal paralysis).

- 1 hour after removal of the bandage: INR, aPTT, CK and neurological examination
- 6 hours post-bite: INR, aPTT, CK and neurological examination
- 12 hours post-bite: INR, aPTT, CK and neurological examination

If the laboratory results are clearly abnormal OR neurotoxicity develops at any time this indicates the patient is envenomed and requires treatment. Testing at 1 hour after removal of the bandage and 6 hours post-bite may roughly coincide and only one set will need to be done. If the 6 hours results are normal the 12 hour laboratory tests can be delayed a few hours until the early morning (rather than calling laboratory staff into the hospital in the middle of the night). At least 97% of envenomed patients are identified within 6 hours with this strategy. For the rare circumstance where PBI is left on for more than 6 hours, final blood tests and neurological examination should be done 6 hours after PBI removal.

A clinical pathway is provided in [Appendix 7](#) for suspected snakebites.

Determining the appropriate antivenom

Determining the type of snake(s) responsible so that appropriate antivenom can be given requires:

1. knowledge of the snakes found in the area;
2. a review of the clinical syndrome defined by the clinical presentation and progression of signs and symptoms, and the admission and subsequent laboratory tests (Table 2); and
3. consideration of the need for sVDK: this can assist where the range of snakes that are possible remains too broad to allow the use of a monovalent antivenom. It should not be used on its own as it is not superior to clinical algorithms.

Identification of the snake should only be done by experts. Snake handlers may be able to assist in the identification of snakes. However, Australian snakes have overlapping shapes, sizes and colours and non-expert identification is generally incorrect.

If there is any doubt about the snakes involved, it may be safer to administer polyvalent antivenom. In many parts of NSW (and Australia) a combination of one (1) vial of tiger snake antivenom and one (1) vial of brown snake antivenom will cover all possible snakebites.

Antivenom treatment

One (1) vial of snake antivenom is required to treat both children and adults for all terrestrial snake envenomings.

There has been a wide range of antivenom doses recommended over the last two decades. However, there is now good *in vitro* and clinical evidence from the Australian Snakebite Project that the dose is one vial for all snake envenomings. Recovery may then be delayed as it is determined by the (lack of) reversibility of the clinical and/or laboratory effects (e.g. synthesis of new clotting factors is required to reverse VICC). Further antivenom will not speed up these processes.

Antivenom should be administered diluted as a slow intravenous infusion over 15 to 30 minutes in normal saline or Hartmann's solution. Dilution will depend on the volume of the antivenom. It will need to be less diluted in young children so large volumes of fluid are not infused. Antivenom must always be administered in a critical care area where anaphylaxis can be treated and resuscitation equipment is available. The reactions are generally not IgE mediated and previous exposure to antivenom is not an important factor to take into consideration. Premedication with adrenaline, antihistamines and corticosteroids is not recommended in Australia.

Cutaneous hypersensitivity reactions occur in 20% of cases of antivenom use. They are more common with larger volume antivenoms. Severe anaphylaxis with hypotension occurs in less than 5% of antivenom administrations in Australia. Serum sickness has been found in about a third of patients given antivenom. Serum sickness is characterized by flu-like symptoms, fever, myalgia, arthralgia and rash occurring 4 to 14 days post antivenom. It can be treated with a short course of prednisolone (25mg mane or BD for 5-7 days). There is no evidence to support the use of prophylactic corticosteroids.

Antivenom should be administered as soon as envenoming is confirmed. This may prevent later development of severe myotoxicity and neurotoxicity. In snakes that cause severe myotoxicity, any indication of envenoming (systemic symptoms, coagulopathy) are an indication for antivenom. Although the CK will later rise from myotoxicity, this rise is too late to be used as a guide for antivenom that might prevent further myotoxicity. VICC develops very rapidly within 1 to 2 hours of a bite. Early antivenom is unable to completely prevent VICC, but if given when VICC has occurred it may prevent other delayed effects from developing. Table 3 provides suggested absolute and relative indications for antivenom.

In most cases one or two monovalent antivenoms can be used based on the local snakes, clinical features and laboratory investigations on admission to hospital. Polyvalent antivenom can be used, however, it has a much larger volume (50ml) and a higher risk of anaphylaxis and serum sickness so should be used only if monovalent antivenom (or a combination of brown and tiger monovalent antivenoms) will not provide coverage. In many parts of coastal NSW, most cases of envenoming are due to brown, tiger, red-bellied black, Hoplocephalus spp. or rough scale snakes. Even if it is not possible to confidently choose between these, administration of one vial each of brown snake and tiger snake antivenom will provide sufficient cover and with a much smaller total volume of antivenom (5 to 10ml), and at a lower cost.

Table 3. Absolute and relative indications for antivenom.

<p>Absolute indications:</p> <ul style="list-style-type: none"> ■ Abnormal INR ■ History of sudden collapse, seizure or cardiac arrest ■ Any evidence of paralysis with ptosis and/or ophthalmoplegia being the earliest signs
<p>Relative indications:</p> <ul style="list-style-type: none"> ■ Systemic symptoms (vomiting, headache, abdominal pain) ■ Abnormal aPTT ■ Leukocytosis ■ CK > 1000U/L

Death adder, mulga/black snake and taipan antivenoms are all large volume (> 10 ml) and all of these snakes account for only a small proportion of severe envenomings in Australia. The administration of polyvalent antivenom is unlikely to be a higher risk than these three large volume monovalent antivenoms and the stocking and use of polyvalent for envenoming of these three snakes rather than keeping three rarely used monovalent antivenoms is recommended. Note that these snake envenomings also have relatively distinct clinical and laboratory features (Table 2).

Although the definitive care of patients with snake envenoming cannot occur in small hospitals without laboratory facilities or sufficient medical or nursing care to allow close observation, antivenom treatment can be given. In patients with clear evidence of clinical envenoming or sufficient concern by the treating clinicians, antivenom should be administered in smaller hospitals prior to retrieval. The decision to give antivenom in such circumstances will depend on the distance to the closest regional or major hospital and should be done in consultation with a clinical toxicologist.

Observation of envenomed patients

All envenomed patients who receive antivenom should be admitted for close observation. Ongoing laboratory testing will monitor the resolution of VICC, and detect thrombotic microangiopathy and myotoxicity. There is no indication for repeat doses of antivenom. Although the specific tests may differ slightly based on the envenoming syndrome, the following laboratory tests should generally be done:

- INR, aPTT, creatinine, creatine kinase and FBC should be done 6 and 12 hours post-antivenom
- INR, aPTT, creatinine and creatine kinase (myotoxic snakes) once to twice daily after the first 24 hours

After VICC, the median time to recovery to an INR<2 is about 15 hours and the INR becomes normal 24 to 36 hours post bite. Most cases of VICC are uncomplicated and patients can be discharged 24 to 48 hours post-bite. However, it is important to monitor the creatinine and platelet count to detect the more severe syndrome of thrombotic microangiopathy in all patients with VICC. Patients with mild neurotoxicity or uncomplicated myotoxicity can be discharged based on resolution of clinical features.

Treatment of complications

Major complications are rare and most often occur in patients with delayed presentations (e.g. severe neuromuscular paralysis requiring intubation and ventilation or rhabdomyolysis with acute renal failure). Major bleeding and thrombotic microangiopathy can occur in any patient with VICC, but is most common with brown snake envenoming. Patients with major complications will require admission to a tertiary hospital that can provide high dependency or intensive care.

1. **Major bleeding:** except for antivenom, the treatment of major bleeding following snake envenoming is the same as for any patient with a severe coagulopathy. Patients may need clotting factor replacement, usually fresh frozen plasma, surgical intervention and supportive care. The use of factor replacement remains unclear in VICC. An RCT of fresh frozen plasma versus nothing within 4 hours of antivenom found that fresh frozen plasma resulted in a more rapid improvement of clotting tests in most patients, but not a decreased length of stay. Fresh frozen plasma within 6 hours of the bite (irrespective of the timing of antivenom) was less likely to be effective. Fresh frozen plasma should be considered in bleeding patients but discussion with a clinical toxicologist is recommended for snake bite patients with major bleeding.
2. **Thrombotic microangiopathy:** no specific treatment (other than antivenom) has been studied. Haemodialysis is sometimes required. There is no evidence to support the use of plasmapheresis (sometimes suggested due to clinical similarities with HUS/TTP).
3. **Rhabdomyolysis and acute renal failure:** Myotoxicity may result in very high creatine kinase (>100,000U/L), but this rarely results in acute renal failure. Treatment is similar to other causes of rhabdomyolysis, with generous fluid therapy and close monitoring for electrolyte imbalances (e.g. hyperkalemia).
4. **Severe neuromuscular paralysis:** Involvement of bulbar or respiratory muscles may require intubation. Patients may require mechanical ventilation for days to weeks.

Facilities required for hospital admission and indications for retrieval

The management of uncomplicated snake bite can be done in any hospital with basic laboratory facilities, appropriate antivenom stocks and a critical care area where it is possible to monitor for and treat anaphylaxis. Early retrieval or primary retrieval to a large centre is not required. Patients with a suspected snakebite must be transferred to a hospital with laboratory facilities unless a formal INR can be done locally with a result available within 2 hours.

Ongoing care of patients with confirmed systemic envenoming who have been administered antivenom can occur in any hospital where there is close nursing observation, access to a critical care area and after-hours medical support if needed. In large hospitals an emergency department observation or short stay unit is appropriate, or alternatively, an inpatient high dependency unit or other clinical unit where close observation can be maintained. Intensive care is only required for major complications such as neurotoxic respiratory paralysis, thrombotic microangiopathy or severe myotoxicity.

A clinical toxicologist can be contacted for advice on severe envenoming through the Poison Information Centre (13 11 26).

Spiderbite

Guidelines for Assessment and Management

Introduction

Red-back spider envenoming or latrodectism is characterised by severe local or regional pain associated with non-specific systemic symptoms and less commonly autonomic effects. Funnel-web spider envenoming is rare, but causes severe and potentially life-threatening neurotoxicity. There are specific antivenoms for these two syndromes. All other spiders will almost always only cause minor effects and require symptomatic treatment. There are also many other stinging or biting creatures which are briefly covered in the appendices, but for which no guidelines have been developed.

Epidemiology and clinical effects

Spider bites occur more commonly in the warmer months of the year. The majority of funnel-web spider bites have been reported in Sydney, the Central Coast and Newcastle.

Most local effects are a result of simple mechanical trauma from the bite. Pain or discomfort occurs in almost every spider bite and the absence of pain suggests the patient has not been bitten. Most patients will recall the pain of the bite and often see or catch the spider after feeling the pain of the bite. Other local effects are a direct result of the mechanical trauma and include fang marks, bleeding and local erythema. Larger spiders will cause more trauma with bleeding fang marks (big black spiders) compared with minimal evidence of the bite with smaller spiders, such as red-back spiders.

Allergic reactions have not been reported following spider bites. Hypersensitivity reactions after a bite suggests bees, wasps or ants are the culprit. Secondary infection is also rare (< 1%) with spider bites.

Specific features of funnel-web and red back bite are discussed below. Other spiders do not cause severe systemic envenoming.

Clinical assessment

In contrast to snakebite, it is common for people to suspect spiderbite without observing a spider biting. Thus, the initial task is to establish if a spider was seen to bite, whether the culprit was collected and the timing of the signs and symptoms attributed to the bite. In most confirmed cases, the patient will present soon after a definite bite.

Relevant history

The following line of questioning may be useful, with the purpose of identifying if a funnel-web spider or related species needs to be considered

- Was a spider seen to bite (?multiple bites) OR were the circumstances such that a bite might have occurred?
- When did the patient get bitten (elapsed time)?
- Description of spider if possible (colour, size, shape)
- Geographic location that the incident occurred
- Timing and type of first aid and activity after the bite
- Type and timing of symptoms; specifically ask about tingling around lips, tongue fasciculation, excessive lacrimation, salivation, piloerection, hypertension, nausea, dyspnoea (pulmonary oedema), impaired conscious state

There are no specific diagnostic tests for funnel web spider or any other spider envenoming. Examination should include looking for typical features of redback spider and funnel web spider envenoming.

Definite spider bites are then managed in one of three clinical pathways:

1. **Big black spider bites.** This includes potential bites by funnel-web spiders, but also trapdoor spiders, mouse spiders and other mygalomorphs (large, primitive spiders). Distinguishing funnel-web spiders from other mygalomorph spiders should only be done by expert arachnologists. All bites by big black spiders in eastern Australia should be managed as suspected funnel-web spider bites for the first 4 hours after being bitten.
2. **Red-back spiders (*Latrodectus hasseltii*).** The defining feature of red-back spider envenoming is severe pain, which may be associated with non-specific systemic symptoms. It develops gradually and is not life-threatening. Treatment is primarily for symptomatic relief and can include analgesia and/or the red-back spider antivenom. Most people can identify a red-back spider with reasonable accuracy.
3. **Other spiders.** Bites by all other Australian spiders only cause minor effects. If a patient has not been bitten by either a big black spider or red-back spider they can be reassured because no major envenoming will occur.

Some patients present with skin lesions or necrotic ulcers they attribute to a spider bite. Necrotic ulcers have not been reported from confirmed spider bites in Australia. The diagnosis and investigation should be focused on other important causes of ulcers including infection, immunological, inflammatory, vascular and neoplastic aetiologies.

RED-BACK SPIDER BITE

Clinical features and diagnosis

Red-back spider bites are not life-threatening but can result in severe pain and systemic symptoms that can continue for hours to days.

Bites typically occur when putting on shoes left outside, sitting on outdoor furniture, putting on bike helmets, moving things in storerooms or picking up pot plants or other garden items. Most people are able to identify red-back spiders and therefore provide a reliable identification if the spider is seen biting.

Red-back spider envenoming is characterised by pain (localised, radiating and regional), which may be associated with systemic symptoms. The initial bite may only cause mild discomfort or irritation, and sometimes is not even noticed. Local pain increases over an hour or two and may radiate up the limb. Abdominal or chest pain may develop as may pain at other sites. The pain is of an unusual quality, persistent and usually severe enough to interfere with normal activities (work or sleep). The duration of effects vary with only moderate pain for a few hours in some cases to severe persistent pain for 2-5 days.

The bite site is often red and the pathognomic finding is localised diaphoresis (sweating). An obvious bite mark with swelling, inflammation, fang marks or bleeding is uncommon. Non-specific systemic effects such as nausea, vomiting, headache, malaise and lethargy are also common. Rarely, other effects are reported such as neurological manifestations, fever, hypertension and priapism.

Red-back spider envenoming is not life-threatening even to infants and children. No deaths have been reported since the mid-1950s.

In most cases, the diagnosis is made based on the history of a bite by a red-back spider and the clinical effects. The type of pain and the presence of local diaphoresis alone may be sufficient to strongly suggest the diagnosis even if a bite is not confirmed. The diagnosis in children or infants who are unable to give a history may be difficult.

Management

First aid

There is no definitive first aid for latrodectism. Cold packs or alternatively heat packs on the bite area sometimes help diminish pain. A pressure bandage will make the pain worse and is not recommended.

Further management

Red-back spider bite is common but very unlikely to prove lethal, even if untreated. Significant envenoming occurs in about a third of cases, but these patients may have several days of distress.

Those patients who present to hospital with no or minimal symptoms may be allowed home. The patient should be instructed to seek further medical care should pain or other symptoms develop which require treatment.

Antivenom is sometimes given:

- if there is a history, symptoms and signs consistent with systemic envenoming, and
- severe pain unresponsive to oral analgesics.

However, recent trials show antivenom has a low response rate little better than placebo, and any effect is less than might be achieved with optimal use of standard analgesics.

There are no useful or diagnostic laboratory tests for red-back spider bite.

Antivenom treatment

CSL Red Back Spider Antivenom is refined horse IgG F(ab')₂, averaging 1.5mL per vial.

Administration

The preferred route is intravenous (in contrast to manufacturer's product information which states 'This medicine is usually injected into a muscle, or in life-threatening cases may be diluted and given via a vein').

Anaphylaxis to this antivenom is rare.

1. Give 2 vials I.V; dilute the antivenom in 100ml of normal saline and administer over 20mins using a pump. Always have adrenaline and full resuscitation available in case of anaphylaxis, but premedication is not required.
2. If incomplete resolution of symptoms seek expert advice, repeat doses of antivenom are often given but there is no good rationale or evidence to support this and it is not recommended.
3. The dose is the same in adults and children.
4. Pregnancy or lactation are not contraindications to antivenom.

Further management advice can be obtained through the **NSW Poisons Information Centre (13 11 26)**.

FUNNEL WEB SPIDER BITE

Clinical Features and Diagnosis

Funnel web spider bite is potentially rapidly lethal. It should be treated as an acute medical emergency. Specific antivenom is available and life-saving in conjunction with supportive care. Expert advice is always available from the **NSW Poisons Information Centre (13 11 26)**.

First aid

The first aid for Funnel Web Spider bites is the same as for snake bites. Apply a pressure bandage with immobilisation (PBI). The pressure bandage should be a broad (15 cm) elasticised bandage, rather than a crepe bandage. The bandage is applied over the bite site and then distally to proximally covering the whole limb. It should be applied about as tight as that used for a sprained ankle. The limb and whole patient should be immobilised for the first aid to be effective. The bandage and immobilisation should remain until the patient has been transferred and assessed in hospital. The bandage should only be removed if antivenom is available and after there is no evidence of envenoming based on clinical examination. If the patient is envenomed the bandage can be removed after antivenom has been administered.

A considerable number of Funnel Web Spider bites do not result in significant illness, and do not require antivenom, but all suspected or confirmed funnel web spider bites must be observed for at least 4 hours with routine observations recorded i.e. pulse, blood pressure, respiratory rate and oxygen saturation levels as per local protocols.

The bite is usually painful and fang marks are present in most cases. If systemic envenoming is present, urgently consider antivenom therapy as this may be lifesaving (see [Antivenom Therapy section](#) in these guidelines).

The clinical features arise directly or indirectly from excessive activity of the autonomic and peripheral nervous system are summarised below.

Table 4. Summary of the clinical effects of funnel-web spider envenoming

CLINICAL SYNDROME	CHARACTERISTICS
Autonomic excitation including cholinergic and catecholaminergic effects	<ul style="list-style-type: none"> ■ Generalised diaphoresis and piloerection ■ Hypersalivation, lacrimation ■ Hypertension, bradycardia or tachycardia ■ Miosis or mydriasis
Neuromuscular and sensory excitation	<ul style="list-style-type: none"> ■ Fasciculations – local or generalised; characteristically tongue fasciculation is seen ■ Paraesthesia – local, distal and oral ■ Muscle spasms – local or generalised
Cardiac effects and Pulmonary oedema	<ul style="list-style-type: none"> ■ Non-cardiogenic/neurogenic pulmonary oedema ■ Myocardial injury/stunning leading to <ul style="list-style-type: none"> – Cardiogenic pulmonary oedema (less common than non-cardiogenic) – hypotension
Other severe effects	<ul style="list-style-type: none"> ■ Drowsiness or coma — rare and typically occurs late or in association with severe envenoming ■ Multiorgan failure — occurs late in life-threatening cases if antivenom has not been administered
Non-specific systemic symptoms	<ul style="list-style-type: none"> ■ Agitation/anxiety ■ Abdominal pain ■ Nausea, vomiting ■ Headache

Clinical management of funnel web spider bite

Key toxicological principles for specific treatment

- If the patient arrives without a pressure bandage with immobilisation then apply one immediately
- Do not remove first aid until ready to treat with antivenom
- Establish intravenous access
- If there are any symptoms of systemic envenoming give 2 vials of CSL Funnel Web Spider Antivenom intravenously
- Be prepared to give more antivenom until major symptoms resolve.
- Children require the same dose as adults
- Seek consultation in any patient not responding to initial antivenom.

Supportive care

Respiratory failure is usually due to pulmonary oedema and may require emergency resuscitation and assisted ventilation. Intubation and PEEP may assist in severe cases. Atropine may be useful if cholinergic features are marked and antivenom is not immediately available.

Severe hypertension may occur, and sedation is the most appropriate first line treatment as it will not exacerbate other features of poisoning.

Antivenom administration

- Antivenom for funnel web spider bite should always be given IV with the patient and clinical environment appropriately prepared to manage anaphylaxis should it occur. No pre-medication is required.
- Due to the nature of envenomation by Funnel Web Spiders (i.e. catecholamine storm), anaphylaxis is very unlikely.

Other common spiders that cause minimal effects

There are a number of recognisable groups of spiders that cause minimal effects.

Orb-weaving spiders are common web building spiders and bites cause local pain and redness and often occur from spiders in clothes left out on the washing line overnight. **Huntsman spiders** are recognisable large spiders that are commonly found climbing walls in houses and are feared by many people. Bites from these spiders cause local pain, local bleeding and fang marks due to the size of the spider. **Wolf spiders** are common ground dwelling spiders and cause local pain, fang marks redness and local itchiness in a third of cases.

White-tail spiders are common and found in homes in eastern and southern Australia. These spiders do not cause necrosis and bites cause local pain and redness, and less commonly a persistent red mark and associated itchiness. **Black house spiders** are medium sized spiders found commonly in the corners of windows and doors and have also been blamed for necrotic ulcers. Bites by black house spiders cause local pain and redness.

There are a number of large black spiders that commonly get mistaken for funnel-web spiders. **Trapdoor spiders** look very similar to funnel-web spiders but bites only cause local pain and fang marks. **Mouse spiders** are another big black spider easily mistaken for funnel-web spiders. All bites by these spiders should be initially treated as a suspected funnel-web spider bite.

Clinical management of other common spiders

The treatment of all minor spider bites is reassurance and symptomatic relief of local effects including pain. Tetanus status should be assessed and updated as required for all spider bites.

Necrotic arachnidism and the white-tail spider myth

Necrotic arachnidism has never been confirmed in Australia although there has been significant misinformation in the past about bites by white-tail spiders. Bites by *Loxosceles* spiders (Recluse spiders) can cause cutaneous lesions, but these spiders do not occur in Australia. There is no evidence that any Australian spiders can cause necrotic ulcers, although white-tail spiders, wolf spiders and black house spiders have been blamed for cases of necrotic ulceration.

Prospective studies of definite bites by white-tail spiders, black house spiders and wolf spiders found that none of these spiders cause necrotic lesions. There are numerous reports of cases of misdiagnosed spider bites where an alternate diagnosis has been found, including dermatophytoses, squamous cell carcinoma, staphylococcal infections, pyoderma gangrenosum, cutaneous polyarteritis nodosa, unusual infections and diabetic ulcers. The appropriate investigation of skin lesions attributed to spider bites is included in [Table 5](#).

Table 5. An approach to the investigation and diagnosis of necrotic skin ulcers, presenting as a suspected spider bite

1. Establish whether or not there is a history of spider bite

- IF a clear history of a spider bite (best if spider is caught)
 - Refer to information on definite spider bites
- IF NO history of spider bite
 - Investigation should focus on the ulcer and the provisional diagnosis of a suspected spider bite is not appropriate.

2. Clinical history and examination

- Focus on features suggestive of infection, malignant processes or vasculitis.
- Consider underlying disease processes: diabetes, vascular disease
- Environmental exposure: soil, chemical, infective
- Prescription medications
- History of minor trauma
- Specific historical information about the ulcer (may assist in differentiating some conditions):
 - Painful or painless
 - Duration and time of progression
 - Preceding lesion

3. Investigation

- Skin biopsy:
 - Microbiology (with appropriate transport media): contact microbiology laboratory prior to collecting specimens so that appropriate material and transport conditions are used for organisms such as *Mycobacterium* spp., fungi and unusual bacterial.
 - Histopathology
- Laboratory: other supportive investigations may be important for underlying conditions (autoimmune conditions, vasculitis and pyoderma gangrenosum). These may include, but not be limited to:
 - full blood count
 - coagulation studies
 - biochemistry (including liver and renal function tests)
 - autoimmune screening tests
 - cryoglobulins
 - chest radiography
 - colonoscopy
 - vascular function studies of lower limbs

4. Treatment

- Local wound management
- Appropriate treatment based on established pathology.
- Investigation and treatment of underlying conditions may be important, (pyoderma gangrenosum or a systemic illness such as diabetes)

5. Follow up and monitoring

- Diagnosis: may take weeks or months to become clear.
- Essential that these patients are followed
- Continuing management: coordinated with multiple specialities involved

Source: From Isbister GK and Whyte IM, 2004, Suspected white-tail spider bite and necrotic ulcers, *Internal Medicine Journal* Volume 34, Issue 1-2, 38–44, January 2004

Other Venomous Creatures

Scorpions

Scorpions are common in many parts of rural and urban Australia but live underground or under debris and are active at night, so are rarely encountered by humans. Most stings are from small species (*Lychas*) and usually occur inside and mostly at night. The main clinical manifestation of scorpion stings is severe and immediate local pain. The pain is more severe with smaller scorpions. The pain usually lasts for a few hours. Other local effects include redness, and less commonly numbness and paraesthesia (about 10% of cases). Non-specific systemic symptoms occur in about 10% of cases. Treatment is pain relief, either oral or parenteral analgesia depending on the severity of the pain.

Ticks

Bites from ticks can cause local irritation, allergic reactions or rarely paralysis (some species only). The Australian scrub tick (*Ixodes holocyclus*) produces neurotoxins in its saliva which can cause paralysis. Initially tick bites are painless and the tick is only found when it has become engorged 2 or more days after attaching. They usually bite in moist or vascular areas – the scalp or flexor areas. Local reactions are the most common and resolve without treatment. Tick paralysis mainly occurs in children and develops slowly producing an ascending paralysis. Initially a child may simply present with leg weakness or an ataxic gait. Paralysis will then progress to involve the upper limbs, neck muscles and trunk. Facial and bulbar muscles are then involved, with ptosis, extraocular muscular paralysis and dysarthria. Severe paralysis will involve the respiratory muscles. In less severe cases tick paralysis can cause cranial nerve palsy, most commonly a facial nerve palsy that mimics Bell's Palsy.

Ticks must be removed carefully to prevent leaving the head embedded. There are numerous ways to remove ticks but all focus on removing all parts and not leaving the head. Proprietary tick removal devices are available but fine-toothed forceps are equally effective. Tick paralysis is usually mild and requires no intervention. Paralysis will develop/progress for up to 24 to 48 hours after tick removal. Admission to hospital for serial neurological examination is required. Severe cases will require advanced life support including intubation and ventilation. Tick antivenom is no longer available for human use.

Centipedes

The first pair of centipede legs are modified into biting claws and can cause severe local pain. Most centipede bites cause mild to severe local pain, erythema and oedema. Uncommonly they can cause local itchiness, radiating pain and mild systemic symptoms and rarely severe local effects with extensive swelling and erythema lasting for days. Treatment is symptomatic including analgesia.

APPENDIX 1

Clinical Signs of Snakebite and Envenoming

Paralysis

Blocked transmission at the neuromuscular junction causing skeletal and respiratory muscle flaccid paralysis. Signs include: ptosis (drooping of upper eyelids), diplopia (double vision), ophthalmoplegia (partial or complete paralysis of eye movements), fixed dilated pupils, weakness and respiratory problems.

Figure 2. Ptosis following Tiger Snake Bite



Coagulopathy

Venom induced consumption coagulopathy (VICC) with low fibrinogen, elevated prothrombin ratio (INR) and aPTT. Signs include: bleeding from bite wound, venepunctures, rarely haematemesis and haematuria.

Myolysis

Caused by generalised destruction of skeletal muscle with high serum CK (creatine kinase) and myoglobinuria (red to brown urine testing positive for blood; can be confused with true haematuria). Signs include: muscle movement pain or weakness, and red or brown urine.

Figure 3. Myoglobinuria



Renal damage

Usually secondary to myolysis or thrombotic microangiopathy.

Collapse

Collapse can occur in severe envenoming, most commonly with brown snake. Can be associated with seizures, hypotension and cardiac arrest.

General symptoms

Include headache, nausea, vomiting, abdominal pain, diarrhoea, generalised diaphoresis.

Local symptoms

Vary from minimal to obvious bite marks, local pain, swelling, or bruising. A trivial looking bite site does not mean trivial envenoming.

Figure 4. Local effects of a red-bellied black snake 'fang marks' on finger



Information for Hospital Laboratory Staff

REQUIRED LABORATORY TESTING

1 Coagulation studies:

PT/INR, aPTT, fibrinogen level, d-dimer/FDP, platelet count.

a. Venom induced consumption coagulopathy

(brown snakes, tiger snakes, rough scaled snake, taipans, Hoplocephalus spp).

- i. Characterised by grossly prolonged PT/INR and aPTT, low to undetectable fibrinogen, grossly elevated d-dimer/FDP.
- ii. Do not delay reporting grossly abnormal tests while waiting for other tests.
- iii. Some coagulation machines will have trouble giving results in this situation. Some machines indicate gross prolongation of PT/aPTT or greatly elevated d-dimer/FDP, but fibrinogen “very high”, when there is in fact undetectable fibrinogen.
- iv. Do not delay while trying to get final results from the machine. Similarly, if d-dimer/FDP is elevated, report this first, then go back and determine the actual level, which may take time
- v. In mild cases, PT/INR and aPTT may be normal, fibrinogen level normal or only slightly decreased, but d-dimer/FDP elevated. Repeat testing will be required to confirm envenomation and the decision whether or not to give antivenom will have to be made on clinical grounds.
- vi. It may be simplest to initially do a manual clotting test for the PT/aPTT.

b. Anticoagulation-type coagulopathy (Mulga snakes, Collett’s snake, red belly black snake).

- i. Characterised by mild to grossly prolonged aPTT, often with a normal or marginally elevated PT/INR. The d-Dimer is usually normal.
- ii. Detection of this type of coagulopathy is dependent on the aPTT test that is done and high-phospholipid containing aPTT reagents will have a normal aPTT result compared to low-phospholipid containing reagents.
- iii. Consultation with an experienced laboratory scientist may be required.

2 Full blood count and blood film

(Platelets, Hb, WCC, absolute lymphocyte count, including blood film examination for red cell fragmentation)

- a. Acute systemic envenoming usually causes an elevated WCC, except in brown snake envenoming where an elevated WCC is uncommon.
- b. There may be an associated absolute lymphopenia.
- c. Thrombocytopenia can occur with VICC and is usually associated with renal damage. There will also often be a progressive fall in Hb and platelets, fragmented red cells on the blood film and a later rise in creatinine. These collectively are features of thrombotic microangiopathy.

3 Muscle damage

[creatinine kinase (CK)]

- a. A number of snakes can cause moderate to severe systemic muscle destruction (myolysis) including Mulga snakes, Collett’s snake, Tiger snakes, Rough Scaled snake, Taipans and Black snakes).
- b. This may take 6 to 24 hours to become evident
- c. Significant myolysis is associated with CK levels >1,000 IU/L and can exceed 100,000 IU/L. In severe myolysis acute renal failure and hyperkalaemia can rarely develop.
- d. Urinalysis is often positive for ‘blood’ when there is myoglobinuria. Measurement of serum or urine myoglobin levels does not aid accurate diagnosis and is expensive.

The interpretation of abnormal coagulation studies can be difficult – it is recommended if there are doubts about whether results indicate envenoming expert advice be obtained through the **NSW Poisons Information Centre (13 11 26)**.

Management of Antivenom Reactions

Guidelines for the management of anaphylaxis to antivenom

1 Preparation prior to commencing antivenom

- a. Routine premedication with antihistamines or steroids is not recommended
- b. Dedicate one small bore (18-20 G in adults) IV line to antivenom administration and one large bore I.V. line (16-14 G in adults) for emergency resuscitation.
- c. Prepare 1L Normal Saline (20 ml/kg in children) ready to give by rapid infusion
- d. Prepare adrenaline 1:1000 (1mg in 1 mL) drawn up to a dose of 0.01 mg/kg (max. 0.3 mg, i.e. max 0.3 mL) and label "adrenaline for I.M. injection only (dose in mg)".
- e. Prepare an I.V. infusion of adrenaline 1mg in 100 mL (controlled by infusion pump or syringe driver) ready to attach by a side arm to the resuscitation line. Anti-reflux valves must be attached above the side arm on any other infusions using this I.V., to prevent adrenaline going back up into the other fluid bags. To prevent erroneous administration, do not attach the adrenaline infusion unless it is needed.
- f. Record blood pressures on the other side to the fluid/adrenaline infusion, to avoid pronged cuff inflations and thus extravasation of infusion fluids.

2 Management of a reaction

- a. Most reactions are related to the rate of antivenom infusion, and cause flushing, hypotension and bronchospasm. Some mild reactions resolve with temporary cessation of the antivenom infusion and recommencing it at a slower rate.
- b. Envenomed patients may be severely coagulopathic, so it is important to be cautious when giving adrenaline to avoid blood pressure surges, which might lead to intracerebral haemorrhage.

- c. Initial management of severe reactions (sudden hypotension, bronchospasm):
 - i. Suspend the antivenom infusion.
 - ii. Lie the patient flat (if not already), commence high flow/100% oxygen and support airway/ventilation as required.
 - iii. Rapid infusion of 1L Normal Saline (20 mL/kg in children) over 2-3 minutes.
 - iv. Adrenaline I.M. into the lateral thigh, 0.01 mg/kg to maximum of 0.3 mg (alternatively, those experienced with I.V. adrenaline infusions may proceed directly to this, as below).
 - v. Liaise with toxicology service regarding ongoing management.
- d. For reactions that do not respond to initial management:
 - i. If hypotensive, be prepared to repeat Normal Saline bolus as above
 - ii. Commence I.V. infusion of adrenaline (0.5-1 mL/kg/hour, of 1 mg in 100 mL) and titrate according to response; monitor BP every 3-5 minutes (using the arm opposite to the infusion); beware that as the reaction resolves adrenaline requirements will fall, the blood pressure will rise and the infusion rate will need to be reduced.
 - iii. Consider nebulised salbutamol for bronchospasm, nebulised adrenaline for upper airway obstruction, and I.V. atropine for severe bradycardia.
 - iv. Seek advice urgently from the local/regional ED Consultant and/or ICU Consultant.

APPENDIX 4

NSW Hospital Antivenom Stocks

Antivenom stock recommendations are based on a review of risks, facilities, past usage and other practical considerations. Determination of antivenom stock requirements is best done at a regional level, either for a whole Local Health District (LHD) or important regions within a Local Health District in collaboration with local Critical Care Clinicians. The definitive management of snake envenoming can only occur in a hospital with a laboratory that can do an INR/aPTT and there is sufficient nursing care. However, antivenom treatment can (and should) be given to obviously envenomed patients in smaller hospitals without laboratory services prior to retrieval.

Antivenom stocking in hospitals in NSW is based on the following principles:

- Geographic location and degree of isolation
- Local snake and spider distribution
- History of envenoming cases
- Referral role of regional rural and metropolitan hospitals

Specifically all hospitals in NSW should have:

- One (1) vial of brown snake antivenom.
- One (1) vial of tiger snake antivenom.
- One (1) vial of polyvalent antivenom should be kept in larger regional and referral hospitals, retrieval services across NSW, and in larger hospitals west of the Great Dividing Range for mulga snake.
- Two (2) vials of funnel-web spider antivenom should be kept in all hospitals where the spider occurs.

Two (2) vials of red-back spider antivenom may be kept in any hospital that has the facilities to safely administer antivenom.

Red-back antivenom is sometimes given:

- if there is a history, symptoms and signs consistent with systemic envenoming, and
- severe pain unresponsive to oral analgesics.

However, recent trials show red-back antivenom has a low response rate little better than placebo, and any effect is less than might be achieved with optimal use of standard analgesics.

There are no useful or diagnostic laboratory tests for red- back spider bite.

One vial of the appropriate antivenom is the recommended dose. Brown and tiger snake groups are the most common snakes causing envenoming in NSW. Tiger snake antivenom will also cover red-bellied black snake the other common important snake in NSW. **Therefore all hospitals with antivenom are capable of treating the most common snake bites in their area.** Further stock should be ordered as soon as supplies are used. In many smaller hospitals holding only one vial of brown and tiger antivenom will involve requesting replacement stock from a nearby hospital with greater stocks.

For small country hospitals with limited resources and no access to on-site laboratory testing, the decision to stock any antivenom must be based on distance to a high role delineated hospital and local resources.

Time required to obtain further antivenom stock will obviously be an important consideration for Local Health Districts in determining stock levels for isolated and regional facilities.

Storage and handling of antivenom

- When transporting antivenom (or venom detection kits) it is essential that the “cold chain” be maintained, as any significant rise in temperature may adversely affect the potency of antivenom.
- Equally, freezing of antivenom may also adversely affect potency.
- Use of a simple icebox or if available, car fridge, is usually the most appropriate means of maintaining temperature during transport.
- Keep each vial of antivenom in its original box.
- It is recommended that stocks be rotated between hospitals with few presentations and those with higher numbers to reduce expiration of stock. This could be facilitated through the Local Health District Drug/Pharmacy Service.

The NSW Therapeutic Advisory Group (TAG) maintains a list of stock levels of snakebite and spiderbite antivenoms in NSW public hospitals [in Part 4 of the Life Saving Drugs Register (LSDR)] based on advice provided by NSW Ministry of Health within these guidelines. This list is updated annually and includes contact numbers for each hospital and the location of stock in each hospital which may be useful in locating stock when required in emergency situations. Stock levels listed however will not necessarily be actually held at all times. For more detailed information about actual stock holdings at a particular time, and access to stock in emergency situations, it is strongly recommended that contact is made with individual hospitals.

APPENDIX 5

Distribution Maps of Snakes

Legend: GREEN shading denotes approximate distribution of snakes in all maps

Brown snake group



Common brown snake (*Pseudonaja textilis*)



Juvenile brown snake



Ringed brown snake (*Pseudonaja modesta*)



Tiger snake group



Common banded tiger snake (*Notechis scutatus*)



Brown un-banded phase of the common tiger snake

Rough scaled snake



Rough scaled snake (*Tropidechis carinatus*)



Broad headed snake (*Genus hoplocephalus spp.*)



Broad headed snake (*Hoplocephalus bungaroides*)



Stephen banded snake (*Hoplocephalus stephensi*)



Pale headed snake (*Hoplocephalus bitorquatus*)



Black snake group



Mulga snake (*Pseudechis australis*)



Red bellied black snake (*Pseudechis porphyriacus*)



Black snake group *cont'd*



Spotted colour phase of the blue bellied black snake (*Pseudechis guttatus*)



Black colour phase of the blue bellied black snake (*Pseudechis guttatus*)

Death adder



Death adder (Genus *acanthophis*)



Taipan



Inland taipan (*Oxyuranus microlepidotus*)



Snakebite Observation Chart

Note: To be used in conjunction with Standard Adult General Observation ([SAGO](#)) chart which is part of the [Between the Flags Program](#)

Management Guidelines for Snakebite & Spiderbite in N.S.W.

Snakebite Observation Chart

Patient Surname: _____ Date of bite: _____
 Forename: _____ Time of bite: _____
 Date of birth: _____ Type of snake: _____
 UR number: _____ Number of bites: _____

Date: _____
 Time: _____
 Time after bite: _____

GENERAL:

Pulse rate: _____
 Blood pressure: _____
 Temperature: _____

SPECIFIC:

Regional lymph node tenderness: _____
 Local bite site pain: _____
 Bite site swelling: _____
 Headache: _____
 Nausea: _____
 Vomitting: _____
 Abdominal pain: _____

PARALYTIC SIGNS:

Ptosis: _____
 Ophthalmoplegia: _____
 Fixed dilated pupils: _____
 Dysarthria: _____
 Dysphagia: _____
 Tongue protrusion: _____
 Limb weakness: _____
 Respiratory weakness: _____
 Peak flow rate: _____

MYOLYTIC SIGNS:

Muscle pain: _____
 Myoglobinuria: _____

COAGULOPATHY SIGNS:

Persistent blood ooze: _____
 Haematuria: _____
 Active bleeding: _____

RENAL:

Urine output: _____

LABORATORY KEY TESTS:

INR/prothrombin time _____
 aPTT _____
 Fibrinogen _____
 XDP/FDP _____
 Platelet count _____
 CK _____
 Creatinine _____
 Urea _____
 K⁺ _____

ANTIVENOM:

Type/amount/time: _____

 Reaction: _____

APPENDIX 7

Snakebite Clinical Pathway

All cases of suspected or confirmed snakebite should be observed with serial blood testing for 12 hours to exclude severe envenoming using the following pathway.

Date _____ MRN: _____

Initial for YES

INTERVENTION /OUTCOME	INITIAL
Patient presented at _____hrs. Pressure bandage in situ.*	
Pathology taken on admission for: Coagulation tests (INR, aPTT, quantitative d-dimer), FBC,UEC,CK,VDK**	
Pathology results reviewed within one hour and are within normal limits. The patient has no signs of neurotoxicity (ptosis, bulbar, respiratory or distal paralysis)** IF pathology results are abnormal, OR neurotoxicity develops, exit pathway, call Poisons Information Centre (13 11 26)	
Remove pressure bandage and immobilisation; observe for any clinical evidence of envenoming.	
Repeat bloods 1 hour post- bandage removal: Coagulation tests (INR, aPTT) and CK	
Pathology results reviewed and are within normal limits. The patient has no signs of neurotoxicity (ptosis, bulbar, respiratory or distal paralysis)** IF pathology results are abnormal OR neurotoxicity develops, exit pathway, call Poisons Information Centre (13 11 26)	
Repeat bloods 6 hours post-bite (unless already >6 hours): Coagulation tests (INR, aPTT) and CK	
Pathology results reviewed and are within normal limits. The patient has no signs of neurotoxicity. IF pathology results are abnormal OR neurotoxicity develops, exit pathway, call Poisons Information Centre (13 11 26)	
Final Bloods at 12 hours post-bite: Coagulation tests (INR, aPTT) and CK.	
Pathology results reviewed and are within normal limits. The patient has no signs of neurotoxicity. Patient discharged in daylight hours IF pathology results are abnormal OR neurotoxicity develops, exit pathway, call Poisons Information Centre (13 11 26)	
*A bite swab should be collected and stored, test if there are any signs of envenoming #Patients with a suspected snakebite must be transferred to a hospital with laboratory facilities unless a formal INR can be done locally with a result available within 2 hours ** Neurotoxicity can be subtle and it is important to include both looking for ptosis and assessing for fatigue (eyelid droop from failure to maintain an upward gaze)	

APPENDIX 8

Recommended Further Reading

The following books are useful sources of further information and are not listed in order of importance. There are many papers published on aspects on envenoming, relevant to Australia, which may be found in a variety of journals or via Index Medicus. Of particular note is The Medical Journal of Australia for clinical information and Toxicon for venom research.

BUCKLEY, N., WHYTE, I. (2012) *Antivenom Update*. Australian Prescriber; 2012: 35 (5) pp.152 -5.

COGGER, H.G. (2000) *Reptiles and Amphibians of Australia*, 6th edition. Sydney: Reed New Holland. The most comprehensive textbook on the taxonomy of Australian venomous snakes (and other reptiles). Contains comprehensive identification keys.

EHMANN, H. (1992) *Encyclopedia of Australian Animals: Reptiles*. Sydney: Angus and Robertson. A comprehensive book on the biology and taxonomy of venomous snakes (and other reptiles).

IRELAND, G., BROWN, S., BUCKLEY B., STORMER, J., CURRIE, B., WHITE, J., SPAIN, D., and ISBISTER, G (2010) 'Changes in serial laboratory test results in snakebite patients: when can we safely exclude envenoming?' *Medical Journal Australia*; 2010:193, pp. 285–290.

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ISBISTER G.K. AND WHYTE I.M.,(2004), 'Suspected white-tail spider bite and necrotic ulcers' *Internal Medicine Journal*; Jan- Feb; 34,(1-2), pp.38–44.

ISBISTER G.K, PAGE C.B, MCCOUBRIE D, GREENE S, BROWN S.G.A, BUCKLEY N.A.,(2013) 'Treatment of snake bite in Australia: a practical approach to diagnosis and treatment'. *Medical Journal Australia*, 2013: 199(11), pp. 763-768.

Glossary of Terms

Anaphylaxis

An immediate and severe allergic reaction to a substance (e.g. venom, food or drugs). Symptoms of anaphylaxis include breathing difficulty, loss of consciousness and a drop in blood pressure. This condition can be fatal and requires immediate medical attention.

Antivenom

Antivenom (or antivenin, or antivenene) is a biological product used in the treatment of venomous bites. It is created by injecting a small amount of the targeted venom into an animal such as horses, sheep, goats, or rabbits; the subject animal will suffer an immune response to the venom, producing antibodies against the venom's active molecules which can then be harvested from the animal's blood and used to treat envenomation in others.

Coagulopathy

A disorder that prevents normal clotting of the blood.

D-dimer

D-dimer is an end product derived from plasmin-mediated degradation of cross-linked fibrin clots. D-dimer measurement has proved to be a sensitive marker for the evaluation of disseminated intravascular coagulation (DIC).

Defibrination

The loss of fibrin from blood.

Envenoming

The envenoming mechanism refers to venom actually being injected as a result of a bite/sting.

Monovalent

An antivenom that is specific for a single snake venom type.

Myoglobin

A protein found in muscles. Myoglobin is released from damaged muscle tissue (rhabdomyolysis). The released myoglobin is filtered by the kidneys but is toxic to the renal tubular epithelium and so may cause acute renal failure.

Polyvalent

An antivenom that is active against multiple snake venom types.

Priapism

Is a persistent and often painful penile erection that lasts for several hours (i.e. 4-6 hours) which is considered a medical emergency.

Venom

A liquid injected by one animal into another, which causes pain or destruction of body tissue. Some venoms are lethal, depending on the animal (or person) being injected. Venom can be injected by either stingers, fangs or spines.

