

Guideline

Emergency Management of Snakebites in Children

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Author/custodian	Director, Paediatric Emergency Department			Review date	15/02/2025
Supersedes	1.0				
Applicable to	All CHQ staff involved in the care and emergency management of children with suspected snakebite				
Authorisation	Executive Director Clinical Services				

Purpose

This guideline has been developed to provide an approach for medical officers involved in the emergency investigation and management of snakebites and potential snakebites in children.

Scope

This guideline applies to all staff involved in the care of children who have sustained or potentially sustained a snakebite.

Related documents

Forms, templates and appendices

- [Snake bite Clinical pathway record](#)

Introduction

There are approximately 3000 snake bites annually in Australia, most occurring in regional and remote areas. While not all snakes are venomous, correct identification of the snake is unreliable. Therefore, all snakebites should be considered potentially life threatening, assessed rapidly and have indicated treatment commenced promptly. Assessment for snakebites should be done in centres supported by 24-hour laboratory services where antivenom is available and trained medical staff are available to provide treatment.

Given the low frequency of snakebite envenomation most medical staff will have limited experience in the treatment. This guideline will help aid emergency medical and nursing staff in the assessment, investigation and management of children with a suspected snakebite.

First Aid



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Snake bites can cause direct myotoxicity. If the patient is collapsed or is in cardiac arrest, manage as per Advanced Life Support principles. If the patient has a history of collapse, early discussion with toxicology is warranted.

A pressure bandage with immobilisation (PBI) is recommended as first aid for all suspected snakebites. The bandage should be broad (ideally 15cm) and preferably elasticated rather than crepe¹. A PBI should not be applied more than 4 hours after the bite has occurred as it is unlikely to be effective¹.

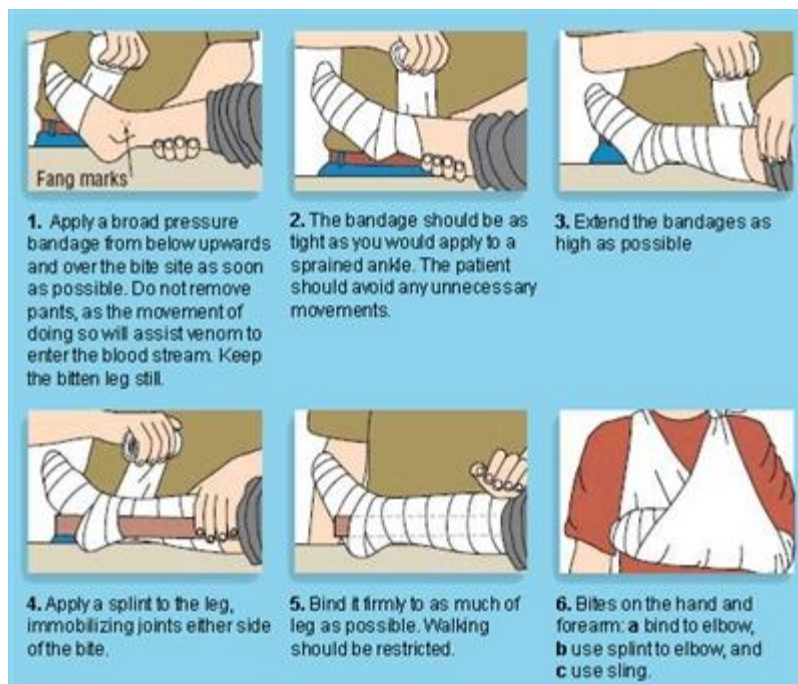


Figure 1: Pressure Immobilisation. Taken from NSW Poisons Information Centre

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The PBI should only be removed if initial exam and investigations do not suggest envenomation and the patient is in a facility with antivenom available.

In patients with confirmed envenomation, the PBI should be removed only after antivenom administration.

Careful observation of the patient is required for at least one hour following the removal of the PBI as rare cases of delayed envenomation following the application of early PBI have been reported¹.

Assessment

Envenomation can manifest in localised symptoms, systemic symptoms (nausea, vomiting, headaches, abdominal pain, and diaphoresis), collapse and major toxin syndromes ([Table 1](#)). Local pain, swelling and bruising may occur.

In Australia, approximately two deaths occur annually secondary to snake envenomation. These are most often following brown snake envenomation with early collapse and cardiac arrest.

History:

- Circumstances of bite¹:
 - Geographical location at time of bite
 - Time of bite
 - Visualisation of snake
 - Location of bite
 - First aid
 - Early symptoms particularly collapse (most common with brown snake envenoming and suggestive of severe envenomation), nausea, vomiting, headache, abdominal pain, diarrhoea and sweating.
- Past medical and surgical history

Examination:

Examination should focus on assessing for local and systemic effects of envenomation. Key areas of assessment include:

- Bite site – **This must be examined with PBI in place and can be achieved by cutting a window in the PBI at the bite site:**
 - Fang marks, bruising, swelling or local necrosis
 - Tender draining lymph nodes may support envenomation
- Neurological:
 - Cranial nerves especially the ocular and periorcular muscles assessing for ptosis, ophthalmoplegia and bulbar weakness
 - Limb weakness
 - Respiratory muscle weakness
- Haematological:
 - Evidence of abnormal bleeding including from IV sites, bite site, oral cavity or occult sites including gastrointestinal, urinary or intracranial ¹

The following tables summarise the clinical syndromes associated with Australian snakebites and the effects of clinically important venomous Australian snakes.

Table 1 Characteristics of clinical syndromes from snakebites in Australia¹**Characteristics of clinical syndromes from snakebites in Australia¹****Sudden collapse**

Collapse or syncope occurring within 90 minutes of the bite

- Collapse is associated with hypotension and loss of consciousness
- Spontaneous recovery usually occurs within minutes
- A minority of patients (about 5%) have a cardiac arrest or seizure

Venom-induced consumption coagulopathy (VICC)

Activation of the clotting pathway by prothrombin activator toxins and consumption of clotting factors (fibrinogen, factor V and factor VIII) leads to a consumptive coagulopathy

- INR is high or unrecordable and APTT is prolonged
- Fibrinogen level is low or undetectable and D-dimer level is very high

Complete or severe VICC is defined as:

- Undetectable fibrinogen level
- INR > 3.0 (most often unrecordable)
- Abnormal APTT (outside the laboratory's reference interval)
- Very high D-dimer level (100–1000 × assay cut-off)

Partial VICC (less severe changes) is defined as:

- Low but detectable fibrinogen level (< 1.5 g/L) and INR < 3.0

Neurotoxicity

A descending flaccid paralysis that 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

- CK level is usually normal (within the laboratory's reference interval) on admission and rapidly rises over 6 – 48 hours (peak ranges from 1000 U/L in mild cases to > 100 000 U/L in severe cases)

Anticoagulant coagulopathy

Provides a marker of envenoming by black snakes, including mulga snakes, but is not clinically important

- APTT is moderately abnormal (1.5–2.5 × laboratory's reference interval), with or without mild elevation of INR (> 1.3)
- D-dimer and fibrinogen levels are generally normal (D-dimer < 1.0 mg/L and fibrinogen > 1.5 g/L [or > 2.0 g/L in some laboratories])

Thrombotic microangiopathy

Presence of fragmented red blood cells on blood film (microangiopathic haemolytic anaemia), thrombocytopenia and a rising creatinine level (> 120 mmol/L), which may lead to acute renal failure requiring dialysis

Systemic symptoms

Non-specific systemic symptoms include nausea, vomiting, abdominal pain, diarrhoea, diaphoresis and headache

INR = international normalised ratio. APTT = activated partial thromboplastin time. CK = creatine kinase.

Table 2: Summary of effects of clinically important venomous Australian snakes¹

	Coagulopathy	Neurotoxicity	Myotoxicity	Systemic Symptoms	Thrombotic Microangiopathy	Cardiovascular effects
Brown Snake	VICC	Rare and mild	-	<50%	10%	Collapse (33%), Cardiac arrest (5%)
Tiger snake group:						
Tiger snake	VICC	Uncommon	Uncommon	Common	5%	Rare
Rough-scaled snake	VICC	Uncommon	Uncommon	Common	<5%	Rare
<i>Hoplocephalus spp.</i>	VICC	-	-	<50%	<5%	Rare
Black snake group:						
Mulga snake	Anticoagulant	-	Common	Common	-	-
Red-bellied black snake	Anticoagulant	-	Common	Common	-	-
Death Adder	-	Common	-	Common	-	-
Taipan	VICC	Common	Rare	Common	5%	Uncommon
Sea Snake		Uncommon	Common	Common		

Initial investigations

Coagulation studies

International normalised ratio (INR) and APTT should always be performed. Fibrinogen and D-dimer help differentiate between VICC and anticoagulant coagulopathy. D Dimer levels are usually elevated 100 – 1000 times the assay cut off in VICC and modest increases in the absence of other features of envenoming should be interpreted with caution. Point of care (POC) devices has been shown to have a high false negative rate and should not be used.

Full Blood Count (FBC)

Thrombocytopenia and red cell fragmentation indicate a diagnosis of thrombotic microangiopathy. Non-specific lymphopenia and leucocytosis can occur with systemic envenomation.

Biochemistry

Creatinine Kinase (CK) – rises in myotoxicity 6-24 hours post bite. An elevated CK on arrival bloods does not reflect envenomation but provides a baseline value for serial CK levels.

LDH – elevation can occur in thrombotic microangiopathy

Electrolytes, urea and creatinine – serial measurements may assist in assessing renal function

Snake Venom Detection Kits are no longer recommended in the routine management of suspected snakebites due to their inaccuracy.

Initial management

- All suspected snakebites should be initially managed in a monitored, high dependency bed or treatment space
- Apply PBI if not already in place within 4 hours of snake bite.
- Notify senior emergency doctor
- Commence documentation on the [Snake Bite Clinical pathway](#)
- Insert IV cannula and obtain bloods for FBC, UEC, CK, Coagulation studies & D dimer
- Ensure antivenom is available (At QCH, antivenoms are stored in the green zone fridge)

Non-envenomed patients

If the patient remains stable and there is no clinical or biochemical evidence of envenomation, PBI can be removed but the patient needs to be monitored in a critical care area. If there is any deterioration, immediately reapply PBI and contact the clinical toxicologist on call (via the Poisons Information Centre on 131126), as antivenom may need to be administered.

If the patient remains stable, a neurological examination and further bloods (coagulation studies and CK) should be completed **1-hour post-bandage removal**. If these results are within normal limits and the patient shows no signs of envenomation, the patient is to be admitted to Short Stay Ward for further bloods and observation. Formal neurological assessment and bloods (coagulation test and CK) should be repeated at **6 hours post bite and 12 hours post bite (not post PBI removal)**.

If pathology is within normal limits and the patient shows no signs of envenomation after the 12 hours bloods are reported, the patient can be discharged home, provided it is during daylight hours (as subtle neurotoxicity can be missed overnight).

Envenomed patients

Treatment of envenomation

- If the patient shows clinical **or** biochemical signs of envenomation, notify senior emergency doctor immediately
- Move patient to resuscitation area, apply full non-invasive monitoring while attending to ABC assessment
- Contact clinical toxicologist on call regarding choice of antivenom. The choice of antivenom is dependent of the clinical presentation as well as geographical knowledge of local snakes

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All cases of snakebite with signs of envenomation must be discussed with a clinical toxicologist. A clinical toxicologist can be contacted by calling the Poisons Information Centre on 13 11 26.

- Administer appropriate antivenom
 - In cases of severe envenomation, time is critical
 - In cases of unidentified snake bite with VICC in South East Queensland, administration of one vial of monovalent tiger snake antivenom **and** one vial of monovalent brown snake antivenom is recommended²
- Release PBI only after antivenom has been commenced
- All patients who have received antivenom need admission for further observation and repeat bloods (FBC, UEC, Coagulation studies, CK, D Dimer) at **6, 12, 18 and 24 hours post bite** to check for recovery of coagulopathy and development of complications such as thrombotic microangiopathy. Patients who require respiratory support and/or invasive support are admitted to paediatric intensive care unit (PICU). All other patients can be admitted to ESSU, following discussion with a clinical toxicologist. These patients should only be discharged once their clinical envenomation syndrome has resolved.
- All patients who have received antivenom should be counselled on discharge regarding the risk of serum sickness 10 - 14days post antivenom administration. This typically presents with a rash and flu-like symptoms. This is treated with a short course of oral prednisolone 1 mg/kg for 5 days. Steroid prophylaxis is no longer recommended.

Antivenom administration

- Antivenom is required in about 5% of cases of Australian snake bite
- Monovalent antivenoms are preferred due to a lower risk of hypersensitivity reactions²
- In cases of unidentified snake bite with VICC in South East Queensland, administration of one vial of monovalent tiger snake antivenom and one vial of monovalent brown snake antivenom is recommended².
- All antivenoms are administered intravenously
- The dose of antivenom is **NOT** weight dependent. One vial of each appropriate antivenom is sufficient to treat envenomed patients²
- Antivenom is to be diluted in a ratio of 1:10 with sodium chloride 0.9% and given over 15 to 30 mins. For larger volumes or in small children, dilution can be 1:5
- There is no role for redosing of antivenom
- There is no role for antivenom >12hrs post bite, as the risk of adverse reactions outweighs the minimal benefit of treatment at this time²



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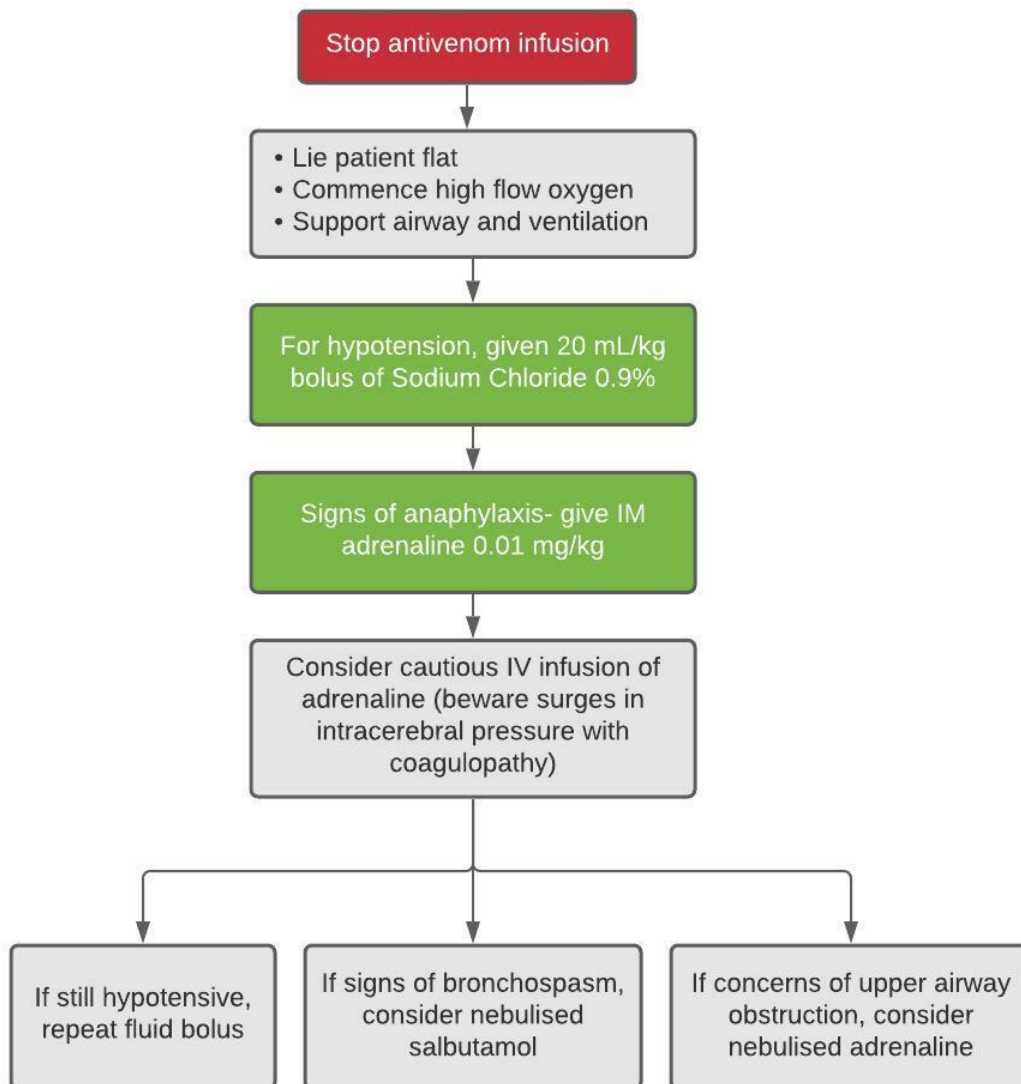
The use of antivenom is associated with risk of anaphylaxis

Treatment of anaphylaxis and allergic reactions to antivenom

The use of antivenom is associated with high rates of acute systemic hypersensitivity, with allergic reactions occurring in approximately 24% of patients and severe anaphylaxis in 6%³. There is no role for premedication with steroids, antihistamines or adrenaline when administering antivenom².

In cases of anaphylaxis, modification of standard anaphylaxis guidelines is recommended due to the risk of coagulopathy, as shown below¹.

Management of Antivenom Anaphylaxis 1



Expected response to antivenom

- Following the administration of antivenom, good supportive care is the mainstay of management of the envenomed patient.
- Clinical and biochemical response to antivenom in cases of severe envenomation can often be difficult to assess².
- Non-specific symptoms of systemic envenomation are potentially reversible with the administration of antivenom, as is anticoagulant coagulopathy².
- Once established, VICC, neurotoxicity and myotoxicity are irreversible. Coagulation studies in patients with VICC will normalise over days, as the liver resynthesises clotting factors. Markers of myotoxicity and renal impairment (e.g. CK and creatinine) may rise or remain high after antivenom administration².

Consultation

Key stakeholders who reviewed this version:

- Paediatric Emergency Specialist
- Clinical Toxicologist, PA Hospital
- Consultant Pharmacist, Queensland Poisons Information Centre
- Paediatric Emergency and Guideline Specialist

Definition of terms

Term	Definition
PBI	Pressure bandage with immobilisation
CK	Creatine kinase
SVDK	Snake Venom Detection Kit
INR	International normalised ratio
APTT	Activated partial thromboplastin time
ESSU	Emergency short stay unit
VICC	Venom induced consumptive coagulopathy
IV	Intravenous
FBC	Full Blood Count
UEC	Urea, electrolytes, Creatinine
POC	Point of care

References and suggested reading

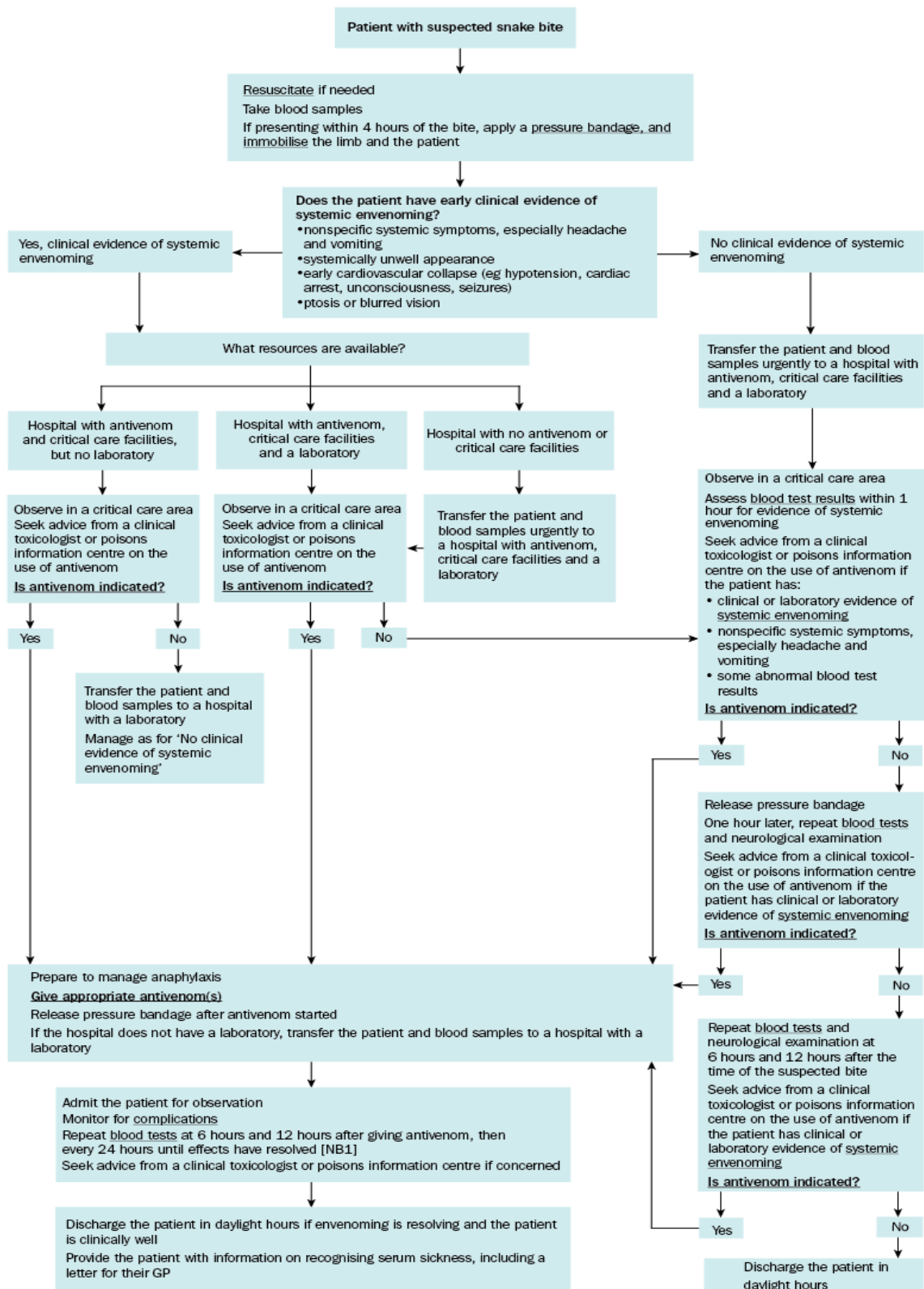
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2. Snake bite [published August 2020]. In: *eTG complete* [digital]. Melbourne: Therapeutic Guidelines Limited; 2021 March https://tgldcdp.tg.org.au/viewTopic?topicfile=toxology-snake-bite&guidelineName=Toxicology_and_Toxinology#toc_d1e754 (accessed 7th July 2021)
3. Johnston CI, Ryan NM, Page CB, Buckley NA, Brown SG, O'Leary MA, et al. The Australian Snakebite Project, 2005–2015 (ASP-20). *Med J Aust* 2017;207(3):119–25

Guideline revision and approval history

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1.0 10/08/2017	Director Paediatric Emergency Department	Divisional Director, Critical Care	Executive Director Hospital Services
2.0 24/01/2022	Senior Medical Officer Emergency Department	Director Emergency Department	Divisional Director, Critical Care
2.1 07/11/2023	Paediatric Emergency Physician	Paediatric Emergency Physician	Paediatric Emergency Physician

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Accreditation references	NSQHS Standards (1-8): 1 Clinical Governance, 3 Preventing and Controlling Healthcare Associated Infections, 6 Communicating for Safety, 7 Blood Management ISO 9001:2015 Quality Management Systems: (4-10)

Appendix 1: Summary Management of Snake Bites



Appendix 1: Summary management of snake bite. Taken from eTG complete [Internet].

NB1: Coagulopathy may not begin to improve until about 12 hours after the snake bite. Persistent coagulopathy is not an indication for additional antivenom. Seek advice if concerned.