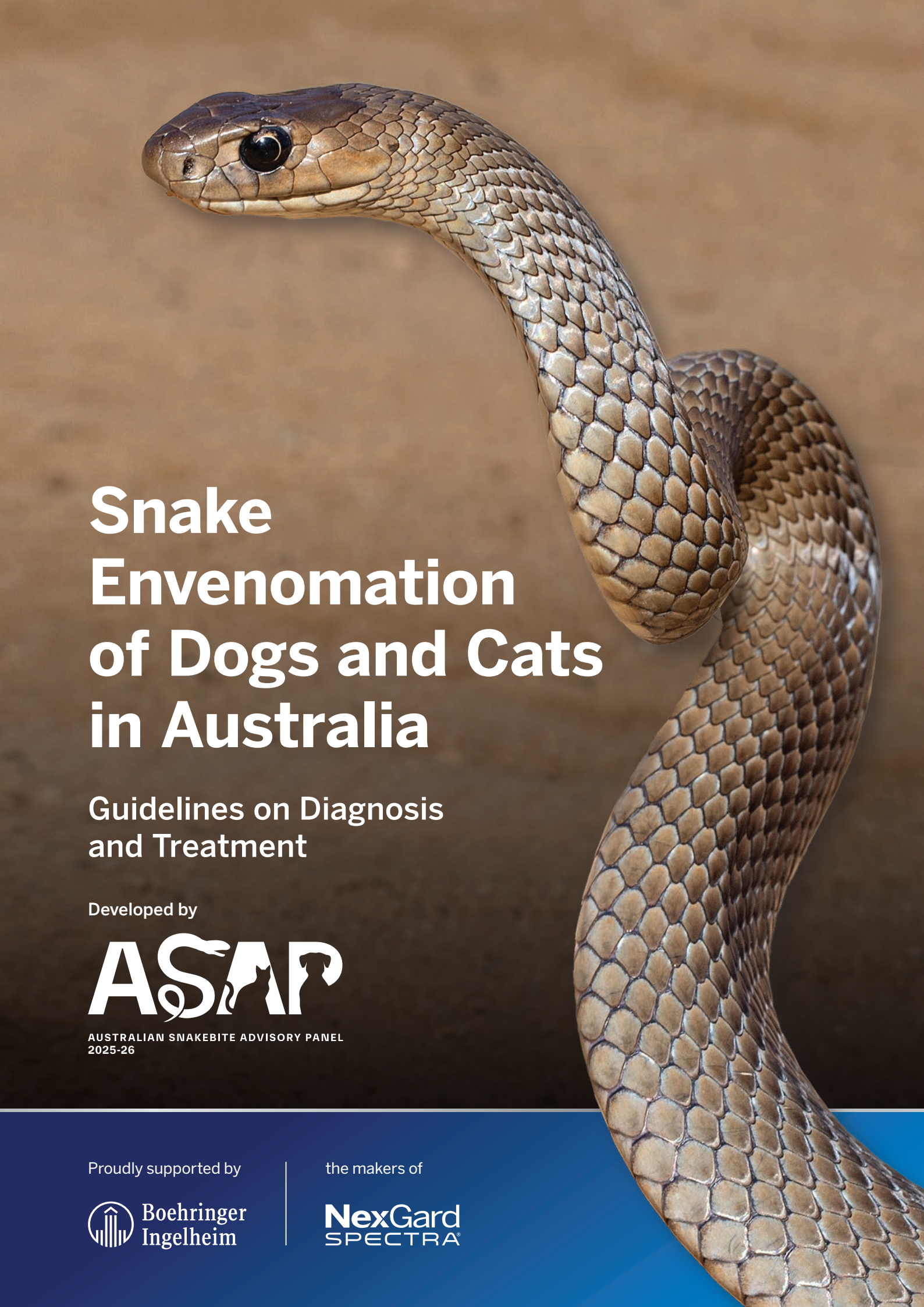


Snake Envenomation of Dogs and Cats in Australia

Guidelines on Diagnosis and Treatment

Developed by

ASAP

AUSTRALIAN SNAKEBITE ADVISORY PANEL
2025-26

Contents

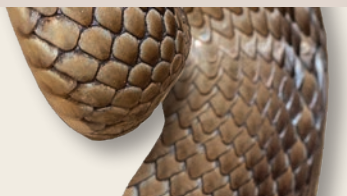
Tiger snake

Abbreviations

ABG	Arterial Blood Gas
AChR	Acetylcholine Receptor
ACT	Activated Clotting Time
AKI	Acute Kidney Injury
aPTT	Activated Partial Thromboplastin Time
AST	Aspartate Aminotransferase
AV	Antivenom
CBC	Complete Blood Count
CI	Confidence Interval
CK	Creatine Kinase
cmH_2O	Centimetres of Water
CPAP	Continuous Positive Airway Pressure
CPR	Cardiopulmonary Resuscitation
CRI	Constant Rate Infusion
CRT	Capillary Refill Time
CT	Clot Time
DDx	Differential Diagnoses
DEA	Dog Erythrocyte Antigen
ECG	Electrocardiogram
$ETCO_2$	End-Tidal Carbon Dioxide
ETT	Endotracheal Tube
FiO_2	Fraction of Inspired Oxygen
FP	Frozen Plasma
FFP	Fresh Frozen Plasma
HME	Heat and Moisture Exchanger
IMHA	Immune-Mediated Haemolytic Anaemia
IPPV	Intermittent Positive Pressure Ventilation
LMN	Lower Motor Neuron
LRS	Lactated Ringer's Solution
MAP	Mean Arterial Pressure
MG	Myasthenia Gravis
MM	Mucous Membranes
MSTS	Minimum Standards Tick Search
MV	Mechanical Ventilation
$PaCO_2$	Arterial Partial Pressure of Carbon Dioxide
PaO_2	Arterial Partial Pressure of Oxygen
PCO_2	Partial Pressure of Carbon Dioxide
PCV	Packed Cell Volume
PEEP	Positive End-Expiratory Pressure
PLA_2	Phospholipase A_2
PLR	Pupillary Light Reflex
POCUS	Point-of-Care Ultrasound
PPV	Positive Pressure Ventilation
pRBCs	Packed Red Blood Cells
PT	Prothrombin Time
$PvCO_2$	Venous Partial Pressure of Carbon Dioxide
RBC	Red Blood Cell
RBBS	Red-Bellied Black Snake
ROTEM	Rotational Thromboelastometry
SBT	Spontaneous Breathing Trial
SpO_2	Peripheral Oxygen Saturation
SWB	Stored Whole Blood
SVDK	Snake Venom Detection Kit
TAS	Tick Antiserum
TEG	Thromboelastography
TP	Total Protein
TIVA	Total Intravenous Anaesthesia
VBG	Venous Blood Gas
VCM	Viscoelastic Coagulation Monitor
VICC	Venom-Induced Consumptive Coagulopathy
WB	Whole Blood
WHO	World Health Organization
WOB	Work of Breathing

Disclaimer: This document aims to provide guidance to veterinarians managing cases of snake envenomation in dogs and cats and should not replace the attending veterinarian's clinical judgement of individual cases. Drugs and dosages represent the opinions of the panel members and are correct at the time of publication.

Boehringer Ingelheim does not promote the off-label use of registered products. Veterinarians should consult relevant legislation, regulations and prescribing guidelines in relation to the off-label use of registered products.



Eastern brown snake

Introduction

There are currently no evidence-based consensus guidelines on the management of snakebite in dogs and cats in Australia. To address this gap, Boehringer Ingelheim Animal Health Australia convened the Australian Snakebite Advisory Panel (ASAP) in 2025-26 with the goal of producing up-to-date and locally relevant guidelines on the diagnosis and treatment of snake envenomation of dogs and cats. The guidelines contain information sourced from peer-reviewed publications, reported experiences, and expert opinions. The overriding goal of this initiative is to provide veterinarians with a comprehensive resource and a strong foundation for consistent and evidence-based management of snake envenomation in dogs and cats to optimise outcomes for affected pets and their owners.

The aims of these guidelines are to:

1. Assist veterinarians in identifying cases of possible snakebite envenomation and guide the early administration of antivenom
2. Improve understanding of the spectrum of, and regional differences in, envenomation syndromes across Australia
3. Establish 'best practice' for the treatment of snake envenomation in dogs and cats
4. Introduce a spectrum of care when managing snakebite cases, including provision of treatment options for resource-limited settings and/or when financial limitations are present

Australian Snakebite Advisory Panel members

Dr Tim Hill

BVSc, MANZCVSc (Small Animal Surgery)

Dr Tim Hill is a veterinarian and owner of Vet Cross, a multi-site veterinary practice serving regional Queensland. He graduated from The University of Queensland in 1993 and has practiced in mixed and small animal veterinary clinics in Australia and the United Kingdom. He holds Membership of the Australian and New Zealand College of Veterinary Scientists in Small Animal Surgery and has additional training in ophthalmology and hip dysplasia assessment. As a practice owner providing emergency and after-hours care in a regional setting, Dr Hill has extensive practical experience managing acute snake envenomation and advising other veterinarians on the topic.

Dr Ellie Leister

BVSc (Hons), FANZCVS (Small Animal Emergency and Critical Care)

Dr Ellie Leister is a veterinary specialist in Emergency and Critical Care at Pet ICU in Brisbane. She began her career in mixed practice in rural New South Wales before moving to the United Kingdom, where she transitioned to small animal and emergency practice. Later she completed a residency program in veterinary emergency and critical care between 2015 and 2017 becoming a Fellow of the Australian and New Zealand College of Veterinary Scientists in 2019. Dr Leister is a long-standing member of the Australian Paralysis Tick Advisory Panel and has contributed extensively to the development of national tick paralysis management guidelines. Her clinical and research interests include tick paralysis and snake envenomation, authoring more than a dozen publications on these topics.

Dr Andrew M. Padula

BVSc (Hons), PhD, MACVSc, Dipl. ECBHM

Dr Andrew Padula is a veterinarian, researcher, and Managing Director of an Australian biotechnology company specialising in the production of hyperimmune sera for veterinary use, including tick antiserum and snake antivenom research. He graduated with Honours in Veterinary Science in 1993 from the University of Melbourne and later completed a PhD, focusing on reproductive physiology and immunology. Dr Padula is internationally recognised for his contributions to the understanding of tick paralysis and snake envenomation in animals and is a contributing expert to the Australian Paralysis Tick Advisory Panel and associated guidelines. He is an Honorary Research Fellow in the Australian Venom Research Unit, Department of Pharmacology & Therapeutics, University of Melbourne and has published multiple papers on venom and antivenom topics in animals. His work bridges clinical practice, antiserum development, and applied toxinology.

Dr Claire R. Sharp

BVSc (Hons), MS, DACVECC (ASAP Chairperson)

Dr Claire R. Sharp is a registered specialist and Associate Professor of Veterinary Emergency and Critical Care at Murdoch University and Section Head of Emergency and Critical Care at The Animal Hospital at Murdoch University in Western Australia. Claire did her specialty training in the United States, becoming a Diplomate of the American College of Veterinary Emergency and Critical Care in 2010. She held a faculty position at the Cummings School of Veterinary Medicine at Tufts University from 2009 to 2015 before returning to Australia to take up her academic role at Murdoch University. Claire's clinical and research interests include transfusion medicine, coagulation, sepsis, anaphylaxis, critical illness biomarkers, and snake envenomation. Claire is involved in the veterinary SnakeMap initiative, and does research on snake envenomation.

Dr Lisa Smart

BVSc (Hons), DipACVECC, PhD

Dr Smart completed her undergraduate degree at the University of Queensland (1999-2003), a rotating internship at Queensland Veterinary Specialists and PetER (2004) and a Small Animal Emergency and Critical Care residency at University of California, Davis. She is board-certified with the American College of Veterinary Emergency and Critical Care (ACVECC) and a registered specialist in Australia. She completed a PhD in Emergency Medicine with Harry Perkins Institute of Medical Research and the University of Western Australia. Dr Smart spent 13 years as a clinical academic at Murdoch University, being productive in laboratory and clinical research. She is also the founder of Track Changers, a consultancy service for veterinary clinical research. She continues her clinical work at the Small Animal Specialist Hospital on the Central Coast, NSW. She has over 20 years clinical experience in treating snakebite in Queensland, New South Wales and Western Australia (and California!). Lisa is also involved in the veterinary SnakeMap initiative.

Dr Rob Webster

BVSc (Hons), FANZCVS (Emergency & Critical Care)

Dr Rob Webster is a registered veterinary specialist in Emergency and Critical Care and one of the founders and Directors of Animal Emergency Service (AES), which operates multiple emergency and referral hospitals across Australia. He graduated from The University of Queensland in 2000 and has worked exclusively in emergency practice since that time.
Dr Webster is a long-standing member of the Australian Paralysis Tick Advisory Panel and has contributed extensively to the development of national tick paralysis management guidelines. His clinical interests include mechanical ventilation and the intensive care of patients with severe neurotoxic envenomation, including paralysis tick and snake envenomation.

Setting the scene

Distribution of venomous snakes of veterinary significance in Australia

Snakes of veterinary relevance in Australia belong to the family Elapidae, and thus are commonly referred to as elapids. In the Australia-wide SnakeMap project, immunotype of envenoming species in dogs and cats that had a positive snake venom detection kit (SVDK) test was 55% brown snake (73/132), 36% tiger snake (47/132), and 7% black snake (9/132), though cases included in the database were predominantly from Victoria and Western Australia (78% of cases).[1]

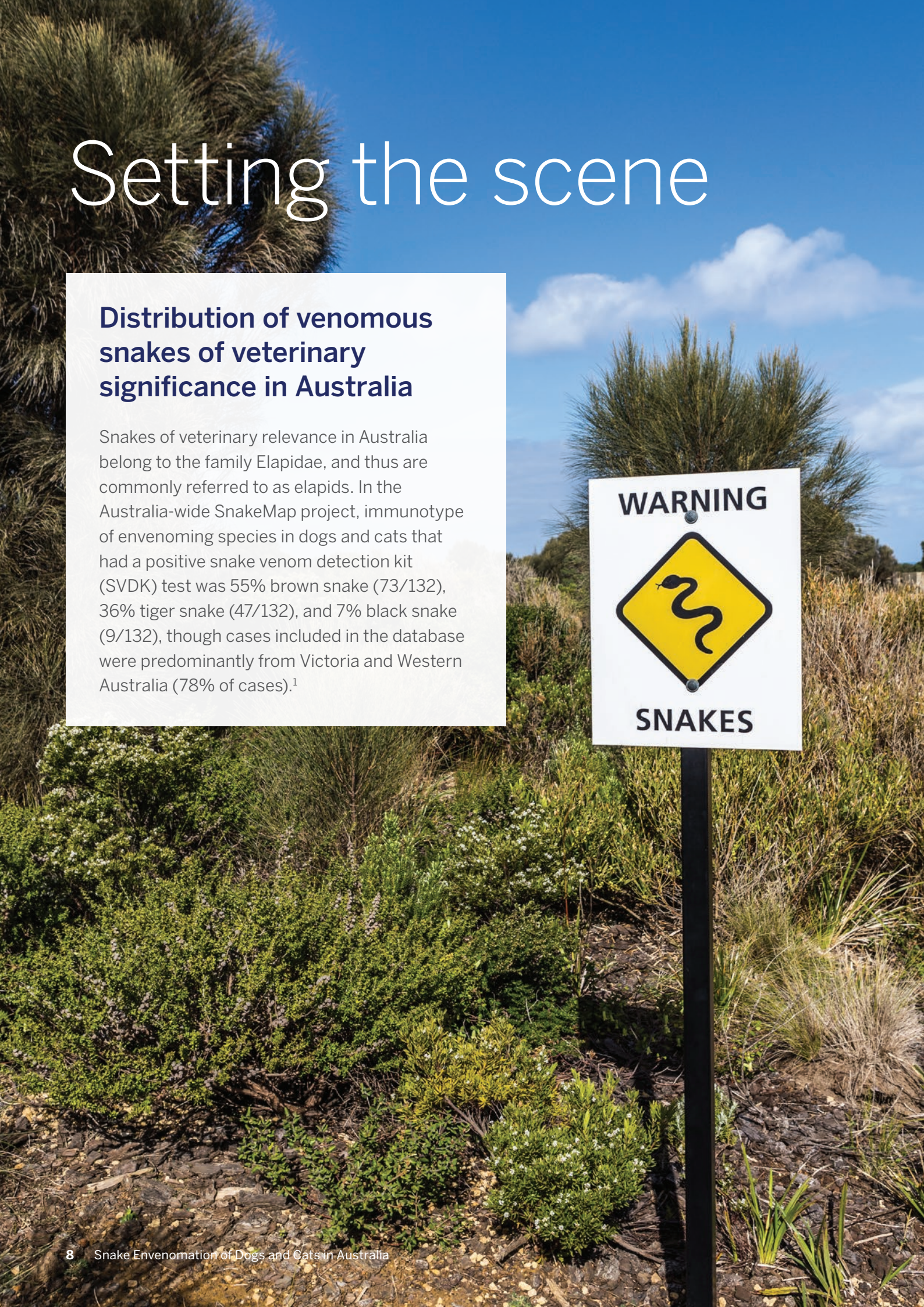


Figure 1. Description of Australian snakes of veterinary significance and their approximate distribution.
These maps have been created from the maps by the University of Melbourne, Australian Venom Research Unit and printed in the book Reptiles & Amphibians of Australia.[2] These are excellent references for snake identification.

Brown Snakes, *Pseudonaja* spp.

There are nine species of brown snakes found in Australia. The eastern brown snake is the most clinically relevant for the eastern regions of Australia. The dugite, western brown snake / gwardar, and ringed brown snake are the most relevant envenoming species for dogs and cats in the west and central areas of Australia.

Eastern brown snake,
Pseudonaja textilis

Dugite,
Pseudonaja affinis

Western brown snake (or Gwardar),
Pseudonaja mengdeni
and
Northern Brown Snake,
Pseudonaja nuchalis

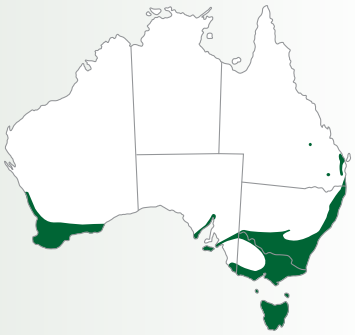
Ringed brown snake,
Pseudonaja modesta

Tiger Snake, *Notechis scutatus*

Tiger snakes are now considered a single species (*N. scutatus*) but historically subspecies names were given to geographically distinct populations.

In Tasmania, veterinarians may refer to the tiger snake as a 'black' snake due to its generally black colour.

Tiger snake, *Notechis scutatus*



Black Snakes, *Pseudechis* spp.

There are seven black snake species found on mainland Australia. The species of greatest veterinary significance are the red-bellied black snake and less commonly the king brown.

Red-bellied black snake (RBBS), *Pseudechis porphyriacus*

King brown snake (or Mulga), *Pseudechis australis*

Despite the common name, the king brown (*P. australis*) is taxonomically a *Pseudechis* – a black snake, not a brown.

Copperheads, *Austrelaps* spp.

There are three species of copperhead found in the eastern regions of Australia, but the lowland and highland copperhead are most significant for veterinarians. Copperheads tolerate cool climates and are found on Tasmania and its offshore islands, and elevated mountain regions on mainland Australia.

Copperhead envenomation of cats and dogs is much less common than for tiger, brown and black snakes.

Lowland copperhead,
Austrelaps superbus

Highland copperhead,
Austrelaps ramsayi

Pygmy copperhead,
Austrelaps labialis

Death adders, *Acanthophis* spp.

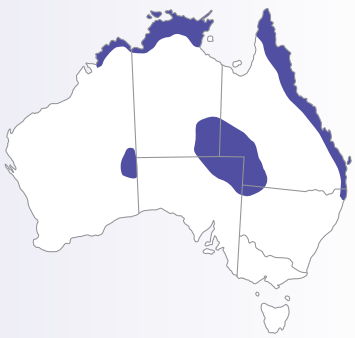
There are eight species of death adders found in Australia. They are a very infrequent cause of envenomation in pet animals in regions where they occur.

Death adders,
***Acanthophis* spp.**

Taipans, *Oxyuranus* spp.

There are three species of taipans found in Australia with the coastal taipan the most relevant. Envenomation of pet animals by coastal taipan is rare but cases occasionally occur in the north of Australia.

Taipans,
***Oxyuranus* spp.**



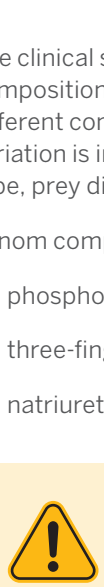
Red-bellied black snake

Snake species and envenomation syndromes

The clinical signs that an animal develops after envenomation depend on the snake venom components and dose. Venom composition is extremely complicated with the venom of a single snake species possibly containing more than a hundred different compounds, including toxic proteins and peptides, as well as non-toxic carbohydrates, lipids, and amines.[3] Venom variation is influenced by factors including snake species, ontogenetic factors (snake age, sex), ecological drivers (prey type, prey diversity, geography, climate), as well as genetic drivers within the snake.[4,5]

Venom components of significance in elapid snake envenomation syndromes include:

- phospholipase A_2 (PLA_2), hyaluronidase, nucleases, metalloproteases, serine proteases
- three-finger toxins, Kunitz-type serine protease inhibitors
- natriuretic peptides, bradykinin-potentiating peptides, and nerve growth factors.[3,4,6]



Common things occur commonly with regard to certain snake species causing particular envenomation syndromes, as highlighted in Table 1 on page 14-15. Nonetheless, any venomous Australian snake can potentially cause any combination of the key envenomation syndromes including pre-paralytic signs, neurotoxicity, coagulopathy, myolysis, haemolysis, and local cytotoxicity at the bite site.

Lowland copperhead

Table 1. Comparative frequency of envenomation syndromes, including commonly observed clinical signs, physical examination findings, and clinicopathologic abnormalities associated with snake envenomation in dogs and cats in Australia. Expected SVDK results and recommended antivenom are also listed.

Envenomation syndrome	Clinical signs, physical examination abnormalities	Clinicopathologic abnormalities	Type of snake (Grouped by common name, Genus)					
			Brown (*Pseudonaja* spp).	Tiger (*Notechis scutatus*)	Black (*Pseudechis* spp.)	Copperhead (*Austrelaps* spp.)	Death Adder (*Acanthophis* spp.)	Taipan (*Oxyuranus* spp.)
Pre-paralytic signs	**Collapse & recovery:** • Sudden collapse soon after bite, temporary recovery then relapse • Vomiting, salivation, excitement, trembling, defecation, and muscle spasm.	N/A	+++	+++	+	++	+	+++
			+++	++++	+	+++	+++	+++
Neurotoxicity	**Acute LMN signs:** • Weakness, ataxia, flaccid paralysis • Abnormal breathing that can progress to respiratory arrest • Reduced to absent spinal reflexes • Reduced to absent withdrawal reflexes Cranial nerve reflex abnormalities • Pupils dilated, pupillary light reflex slow or absent, gag weak or absent	N/A	++++	++++	++	+++	++++	++++
Coagulopathy	**Bleeding** (e.g., from wounds, other locations) • Haemorrhagic shock if large volume haemorrhage • Specific signs due to location of haemorrhage	• Prolonged clotting times (PT, aPTT, ACT) • Low / undetectable fibrinogen • **Discoloured urine - haematuria** (if bleeding into urinary tract)	++++	++++	+ to ++	+/-	-	- to ++++
Myotoxicity	**Myopathy:** • Muscle pain • Reluctance to stand or walk • Stiff gait, weakness • Exercise intolerance • Muscle tremors/fasciculations	• Increased serum muscle enzymes (CK and AST) • Discoloured urine due to pigmenturia (myoglobinuria)	+/-	++++	+++	++	+/-	++ to ++++
Haemotoxicity / haemolysis		• **Discoloured urine (Pigmenturia -** haemoglobinuria) • Spherocytes on blood smear • Progressive anaemia (decreased PCV)	+/-	+/-	+++	+/-	+/-	++
Local cytoxocity	**Swelling, pain, bruising** +/- bleeding at bite site	N/A	-	+	+++	-	+	+
SVDK strongest well			BROWN	TIGER	BLACK	TIGER	DEATH ADDER	TAIPAN
Recommended antivenom type / product			Use Tiger-Brown Snake Antivenom*				Use Specific Antivenom	

Envenomation effects are semi-quantitatively graded based on relative clinical prominence in the literature:
- = absent; + = mild/rare; ++ = moderate; +++ = severe/common; ++++ = predominant feature.
Clinical signs may vary due to time elapsed from bite to presentation, patient body weight, snake subspecies, and regional differences in venom composition.

*Monovalent Brown Snake Antivenom may be used when there is certainty of brown snake envenomation.
For death adder and taipan envenomation, administration of a vial of Tiger-Brown Snake Antivenom while sourcing type-specific antivenom may be beneficial due to potential cross reactivity.
Tiger-Brown Snake Antivenom: one vial = ≥8,000 units (≥4,000 tiger + ≥4,000 brown). Refer to Antivenom choice on p.38.

Small-eyed snake (*Cryptophis nigrescens*) envenomation may be underdiagnosed as SVDK results are negative. Myopathy predominates and can be marked; severe cases develop myoglobinuria and muscle weakness. Tiger-Brown Snake Antivenom has some cross-reactivity and administration is recommended.

Pre-paralytic signs

The term 'pre-paralytic signs' is often used to refer to the syndrome of collapse, gastrointestinal signs and possible recovery that often precedes neuromuscular paralysis. They are early, characteristic clinical signs of elapid envenomation in Australia, particularly in dogs, and occur within minutes to hours of a bite. Importantly, pre-paralytic signs are more likely at higher venom doses,[7] and thus early, aggressive treatment is indicated.

Clinical signs:

- Acute collapse (with or without recovery) and vomiting
- Profuse salivation and diarrhoea (or prompt defaecation)
- Cardiovascular instability (with or without recovery), including severe tachycardia or bradycardia, vasodilatory shock, and/or hypovolaemic shock resulting in abnormal perfusion parameters. These signs may persist to presentation to a veterinary clinic

Pathophysiology:

These signs likely result from the rapid systemic effects of multiple venom toxins, although our understanding is somewhat incomplete. Early cardiovascular collapse appears driven by release of depletable endogenous mediators (histamine, bradykinin, 5-HT) causing vasodilation and hypotension, somewhat analogous to anaphylaxis; rather than direct venom-induced peripheral vasodilation or clotting-factor consumption (mediators replenish within ~1 h).[8,9] There is also evidence that prothrombin activator-like toxin contributes to the rapid cardiovascular collapse induced by *P. textilis* venom.[10] Hypovolaemic shock can also result if envenomed animals experience early bleeding or rapid third space losses into the gastrointestinal tract.

Shock can also mask early neuromuscular paralysis, as it can be hard to distinguish the effects of circulatory shock and progressive paresis.

Neurotoxicity

Neurotoxicity is a feature of most Australian elapid envenomations.

Clinical signs:

- Acute lower motor neuron (LMN) paresis and paralysis that can progress to respiratory muscle paralysis resulting in respiratory failure
- Cranial nerve abnormalities also occur due to inhibition at the neuromuscular junction (Table 1)

Pathophysiology:

Venom neurotoxins present in Australian snake venoms classically cause acute LMN signs via action at the neuromuscular junction, although different neurotoxins have different mechanisms of action.[3] The following is a simplified summary of what we currently understand.

- Presynaptic neurotoxins (also known as ß-neurotoxins) prevent the release of acetylcholine from the pre-synaptic membrane. These are classically considered to have **irreversible** binding and hence their effects are less reversible with antivenom and regeneration of the nerve terminal may be required for resolution of paresis. Presynaptic neurotoxins include PLA_2 enzymes such as textilotoxin (from *P. textilis*), notexin (from *N.scutatus*), and taipotoxin (from *O.scutellatus*)[3]
- Postsynaptic neurotoxins (α-neurotoxins, also called three-finger toxins due to their structure) result in **reversible** antagonism of post-synaptic nicotinic acetylcholine receptors,[11] an effect thought to be rapidly reversible with appropriate antivenom administration[12]
- Venom from Australian elapid snakes, such as tiger snakes, can contain both α-neurotoxins and ß-neurotoxins

Coagulopathy (VICC)

Coagulopathy, commonly referred to as venom induced consumptive coagulopathy (VICC) in elapid snake envenomation is predominantly associated with procoagulant venom components.

Clinical signs:

- May be minimally symptomatic, cause local bleeding, or result in life-threatening systemic bleeding such as pulmonary haemorrhage[13]
- Sites of haemorrhage include:
  - bite site
  - oral cavity (gingival, lingual)
  - venepuncture/IV catheter sites
  - lower genitourinary tract (haematuria, scrotal, preputial, vulval),

  - gastrointestinal (melaena, rectal)
  - upper respiratory tract (nasal)
  - lower respiratory tract (pulmonary haemorrhage)[14]
  - Other less common sites include pleural space, intra-abdominal, pericardial, intramyocardial[15], spinal[16], ocular (hyphaema, scleral)[15], and intracranial

Clinicopathologic abnormalities:

- Coagulopathy is identified by prolonged clotting times and hypofibrinogenaemia
- In unpublished data from the veterinary SnakeMap registry:
  - Prothrombin time (PT) was out of range high in 52.9% of cases in which it was measured (146/276)
  - Activated partial thromboplastin time (aPTT) was out of range high in 62.9% (239/380)
- Coagulopathy may be even more frequent in dogs and cats with eastern brown snake envenomation in south-east QLD based on a recent publication[13]
  - PT out of range high in 151/220 = 68.6% dogs, and 42/86 = 48.8% cats
  - aPTT out of range high in 158/207 = 76.3% dogs, and 52/80 = 65% cats
  - Activated clotting time (ACT) – out of range high in 100% of animals (6/6 dogs, 2/2 cats)

Pathophysiology:

- Multiple procoagulant venom components are recognised including activators of prothrombin (coagulation factor II), factor (F) X, FV, and thrombin-like (FIIa) activators[3,17]
- For example, the notecarin D toxin found in tiger snake venom is structurally similar to FXa, which complexes with endogenous FV, phospholipid, and calcium, producing the prothrombinase complex that activates prothrombin to thrombin, and in turn fibrinogen to fibrin[18]
- The result is rapid consumption of coagulation factors detected by prolonged clotting times and low or undetectable fibrinogen concentrations[19,20]

Thrombosis has also been documented in envenomed animals in organs such as the heart, lungs, and kidneys.[21]

Myotoxicity

Clinical signs:

- Generalised muscle pain
- Myoglobinuria, which is observed as pigmenturia with red to brown to black urine discolouration

Clinicopathologic abnormalities:

- Muscle damage is reflected by a rise in circulating creatine kinase (CK >1,000 U/L), though peak effects are not usually seen until 24 to 72 h after the time of envenomation[22]

Pathophysiology:

Myotoxicity distant to the bite site is thought to be due to several myotoxins in the venom of Australian snakes, most notably PLA_2 toxins. Venom myotoxic enzymes hydrolyse phospholipids in muscle cell membranes causing muscle fibre necrosis and release of myocyte contents such as myoglobin, potassium, phosphate, and CK.

- Clinically-relevant systemic myotoxicity in humans is most likely secondary to envenomation by tiger snakes, RBBS, and the king brown[22,23]
- In dogs and cats, myotoxicity after tiger snake and RBBS envenomation has been well documented,[24,25] though little has been published on king brown envenomation. Other Australian snake species, such as the taipan[26] and brown snake[15,27], have also been shown to cause myotoxicity, albeit milder

Haemotoxicity (Haemolysis)

Haemotoxicity will be used to refer to erythrocyte injury for the purposes of these guidelines, but is sometimes used to refer to abnormalities of coagulation associated with snake envenomation in the human literature.[3,28] We used the terms coagulopathy or VICC to refer to coagulation abnormalities.

Clinical signs and clinicopathologic abnormalities:

- Although signs of immediate red blood cell (RBC) damage and haemolysis can be seen, such as pigmented serum/plasma and spherocytes on blood smear evaluation, clinically relevant haemolytic anaemia often does not occur until 24 to 72 h later
- Haemolysed serum/plasma and pigmenturia have also been reported in envenomed animals without anaemia or spherocytes
- Clinically significant haemolytic anaemia may result in pale mucous membranes, tachycardia, weakness, and pigmenturia

Pathophysiology:

- Haemotoxins, such as PLA_2 enzymes, cause damage to, or destruction of, the RBC membrane that can result in intravascular haemolysis leading to haemoglobinaemia and haemoglobinuria,[30,31] and/or extravascular haemolysis of damaged cells leading to bilirubinaemia and bilirubinuria[30]
- Spherocytes occur early after envenomation as a result of direct RBC membrane damage without immune involvement,[29] or later secondary to splenic macrophages partially phagocytosing damaged RBCs. The latter phenomena is considered appropriate removal of damaged cells and should not be treated with immunosuppression
- Black snake envenomation, particularly RBBS, most commonly causes significant haemolytic anaemia in dogs and cats,[24,29,30,32,33] but milder haemolysis has also been associated with tiger snake envenomation[34]
- Haemolysis is seen clinically with eastern brown snake envenomation but is not comprehensively documented in the veterinary literature, with a single published case.[27] It is likely microangiopathic, secondary to VICC, as documented in humans[94,95]

Venom-induced haemolysis – not immune-mediated haemolytic anaemia (IMHA): Presumed secondary IMHA was reported in 4 tiger-snake envenomed dogs, that were treated with corticosteroids ± azathioprine.[34] Others have now extensively documented haemolytic anaemia in snake envenomed dogs and cats, suspected to be due to direct membrane damage from venom components leading to intravascular haemolysis and/or appropriate extravascular removal of damaged cells by the immune system, that resolves without immunosuppression.[29,30] **As such the panel advises that corticosteroids are not indicated for treatment of haemolytic anaemia secondary to snake envenomation.**

Eastern small eyed snake

Local cytotoxicity

Clinical signs:

- Locally acting **cytotoxins** cause tissue destruction, bruising, oedema, and pain after envenomation
- Local bite site reactions from Australian elapid snakes are uncommon, except for the black snakes. Indeed, more than 50% of dogs envenomed by RBBS show some sort of bite-site reaction[24,30,32]

Pathophysiology:

- Local cytotoxicity results from PLA_2 enzymes and other cytotoxic chemicals that cause muscle and endothelial cell necrosis, and hyaluronidases (also called spreading factors) that break down the extracellular matrix and increase interstitial permeability
- Venom anticoagulants can also contribute to bruising and bleeding at the bite-site

Nephrotoxicity

Indirect nephrotoxicity has been reported in envenomed animals, although there is less evidence of direct nephrotoxicity.

Clinical signs:

- Despite the presence of profound pigmenturia in many cases, clinically relevant acute kidney injury (AKI) appears to be an uncommon complication of snakebite envenomation in dogs and cats
- In two case series of RBBS envenomation 5.7% (5/88)[24] and 5.8% (1/17)[30] of dogs developed AKI
- Severe acute tubular necrosis due to RBBS and tiger snake envenomation has been demonstrated post-mortem in several case reports[31,33,35]
- Some literature suggests direct nephrotoxicity given acute tubular necrosis was evident as early as 1 h after envenomation, and since nephrotic lesions were not closely related to the occurrence of either haemoglobinuria or myoglobinuria[35]

Pathophysiology:

- Both myotoxicity and haemolysis lead to excretion of haeme iron in the urine, which has been associated with AKI
- Haeme iron can cause direct oxidative damage to renal tubular epithelial cells and obstruction of renal tubules due to casts
- Direct nephrotoxicity (i.e., due to direct effects of venom components on the kidney) may also occur[35]

Dog connected to ambu bag

Eastern brown snake

Telephone triage

Seek veterinary attention immediately

Snake encounters and snake bites in dogs and cats in Australia commonly result in envenomation (as opposed to 'dry bites') – any snake encounter should prompt immediate veterinary attention.

Dogs and cats with an acute onset of collapse, paresis, or other neurological abnormalities after being outside should also be seen immediately, even in the absence of an observed snake encounter.

Recommend immediate veterinary attention if any of the following occur:

1. A witnessed interaction with a snake – regardless of whether the snake was thought to bite the pet, and even if the pet is clinically normal.
2. Acute onset of collapse, paresis, or other neurological abnormalities (e.g., tremors, shaking), regardless of snake exposure.
3. Bruising or bleeding of unknown cause, regardless of snake exposure.
4. Acute onset of vomiting, ptyalism, or diarrhoea, regardless of snake exposure.

Phone advice:

- **Keep calm** – The owner staying calm and keeping their pet calm is important. Panicking is likely to cause stress and an increased heart rate that will increase the rate of venom spread
- **Reduce movement and activity** – This will help to reduce the spread of venom. If possible, carry the pet to the car
- **No first aid** – Do not apply a bandage or tourniquet. Do not attempt to suck venom from the wound or bite site – this is not effective and places the owner at risk
- **Do not search for a bite site** – Bite sites are not identifiable in most cases of snake envenomation in Australia
- **Do not touch the snake, even if it appears dead** – Snake identification is rarely necessary as polyvalent antivenom is given regardless. If a photograph can be taken safely, this may be helpful, but should not delay veterinary attention
- **Prioritise immediate transport** – Get the pet to the nearest veterinarian as quickly as possible

Clinical presentation, initial assessment and diagnostic approach

History

Obtaining a thorough history is very important, including the following:

- Evidence/observation of snake encounter?
- Previous history of snake encounter and treatment (e.g., prior antivenom)
- Environment, such as proximity to waterways or bush areas
- Geographic location
- Season

Treat without delay

Dogs and cats with a known snake encounter that have any clinical signs consistent with envenomation should be treated with antivenom as soon as possible, and regardless of the results of any diagnostic tests.

Pets are more likely to be envenomed by snakes in or near to bushland and waterways. Many cases of envenomation occur in the owner's yard; with SnakeMap documenting envenomation in the owner's yard in 85% of dogs and 26% of cats.[1] In temperate parts of Australia most cases of envenomation occur in spring, summer, and early autumn,[13] and so season can be factored into decision making when considering the likelihood of snake envenomation.

Clinical signs

Table 1 (page 14-15) displays the comparative frequency of commonly observed clinical signs of snake envenomation in dogs and cats, as well as physical examination and clinicopathologic abnormalities.

Physical examination

The initial focus of the physical examination should be on the life-sustaining organ systems (respiratory, cardiovascular, neurologic), similar to the primary survey performed in a trauma patient.

Intubate and ventilate patients with respiratory muscle paralysis

If the patient is paralysed with respiratory arrest or has very weak respiratory excursions, an airway should be secured by intubation and assisted ventilation provided immediately. Small IV doses of induction medications (e.g., propofol, alfaxan) may be required to facilitate intubation.

Antivenom before full assessment

Antivenom administration should commence before a full physical examination is performed in a patient with an imminently life-threatening envenomation syndrome and known snake exposure. Refer to flowchart on pages 34-35.

Have a high index of suspicion for pulmonary haemorrhage

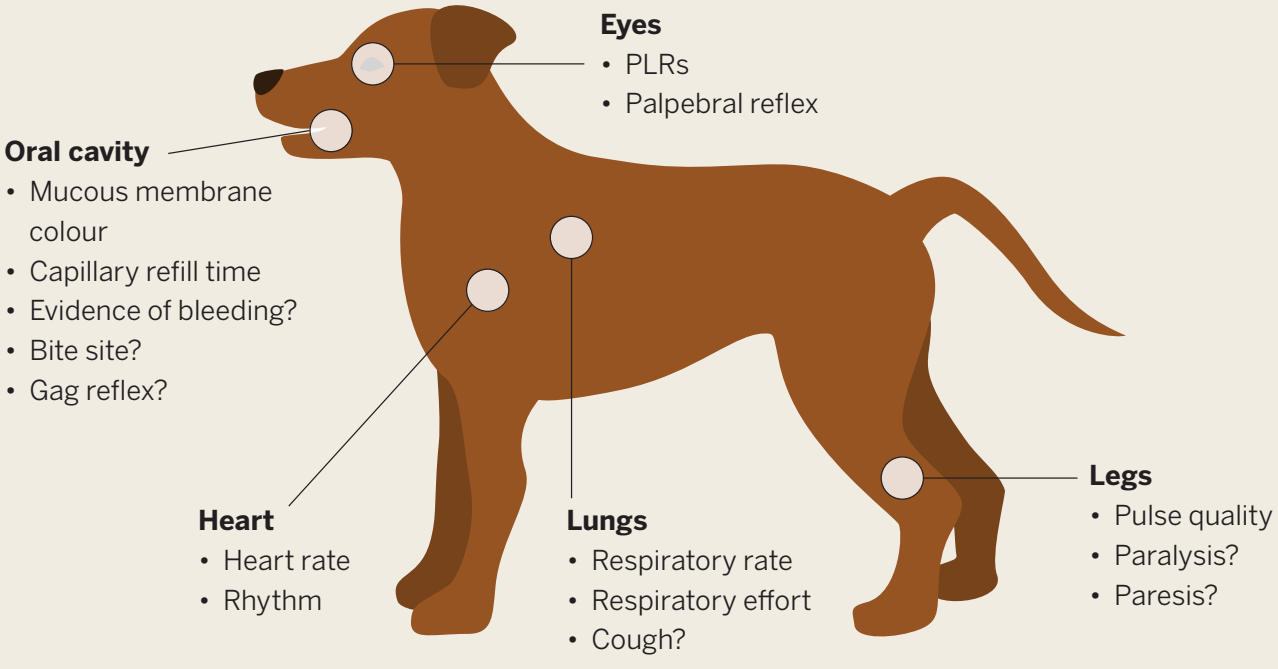
Auscultating or observing a soft cough should prompt concern for pulmonary haemorrhage as progression to haemoptysis has been reported in cases of eastern brown snake envenomation,[14] and described anecdotally in other species.

Shock is uncommon in snake envenomation; when present it is most commonly haemorrhagic (VICC-associated coagulopathy), anaemic (haemolysis), or vasodilatory (venom-mediated inflammation), but other causes should be considered – refer to the blood transfusion and fluid therapy sections on pages 48 and 56.

Ultimately a full physical examination should be performed including documentation of vital signs (heart rate and rhythm, respiratory rate, rectal temperature), thoracic auscultation, perfusion and hydration parameters (refer to fluid therapy section on page 56), and neurologic examination. A bite site or evidence of bleeding may also be evident on examination.

In animals with a known snake encounter but no clinical signs, laboratory evaluation can aid in the decision to give antivenom. Specifically, measurement of clotting times is the single most helpful diagnostic test to perform, and if prolonged in this circumstance should prompt antivenom administration.

In animals with no known snake encounter but clinical signs that could be due to snake envenomation (acute LMN signs, discoloured urine, or collapse followed by recovery), the approach depends on the clinical signs.



Acute LMN signs

The top two differential diagnoses for an acute onset of LMN signs in dogs and cats in Australia are snake envenomation and tick paralysis. While snake envenomation is a problem Australia-wide, tick paralysis is confined to areas where *Ixodes holocyclus* occurs along the eastern coastal strip. In tick endemic areas, differentiation between snake envenomation and paralysis tick toxicosis is essential but can be difficult.

Rapidly progressive LMN signs: treat empirically, don't wait for tests

For dogs and cats with acute LMN signs showing rapid deterioration (e.g., over 20 minutes), prioritise empirical treatment over diagnostic tests. If snake envenomation is more likely than tick paralysis, give antivenom immediately. If tick paralysis is more likely, give tick antiserum per the Australian Tick Paralysis Guidelines. For help differentiating refer to the the tick vs. snake section on page 29.

If LMN signs are not imminently life-threatening nor rapidly progressive, then initial diagnostics should include measurement of clotting times, and a thorough tick search.

- If a tick is found, tick antiserum should be administered
- If clotting times are prolonged, in the absence of an engorged tick, then antivenom should be administered
- If clotting times are normal and no tick is found, the veterinarian must consider the benefit vs. risk of empirical treatment
  - At this time it may be appropriate to perform other diagnostic tests (e.g., measurement of CK, assessing for pigmenturia, SVDK) to aid in clarifying the likelihood of snake envenomation
- Other differential diagnoses for acute LMN signs including botulism, idiopathic polyradiculoneuritis, and generalised myasthenia gravis should also be considered (see Table 3 on page 32)

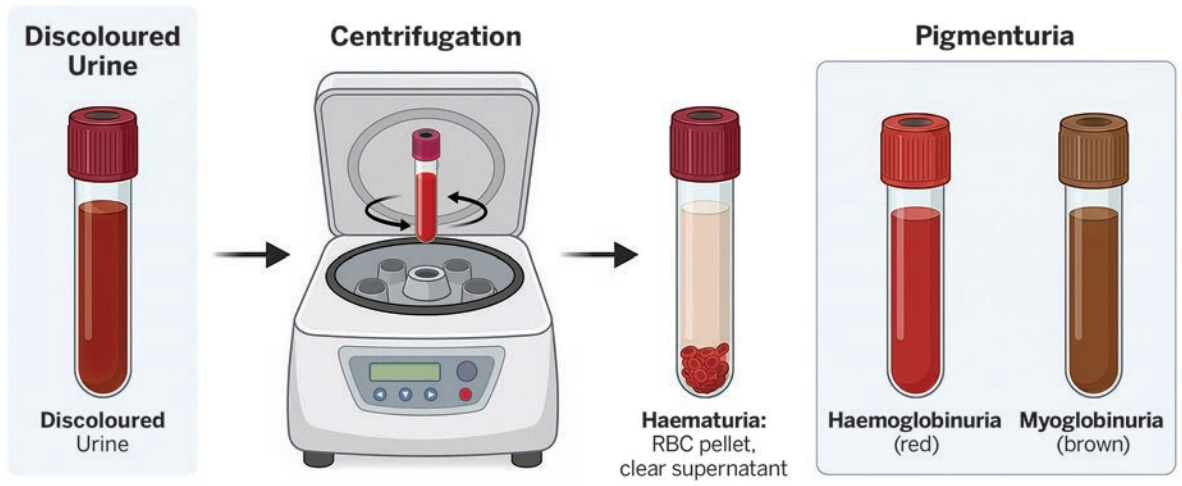
Discoloured urine

Discoloured urine may be evident after spontaneous urination (at home or in a veterinary facility), or upon urine collection.

TIP: If collecting urine, avoid cystocentesis.

Instead use a catheter or gentle expression, as cystocentesis can cause severe bleeding in a coagulopathic patient.

- **Centrifugation** can assist in differentiating pigmenturia from haematuria by separating intact RBCs (seen in haematuria) from dissolved pigments in the urine (pigmenturia). Intact RBCs will sediment into a pellet at the bottom of the tube if haematuria is present, allowing assessment of the supernatant colour. Red or brown discolouration of the supernatant is consistent with haemoglobinuria and myoglobinuria, respectively
- **Causes of pigmenturia:** Pigmenturia can occur in dogs and cats with snake envenomation due to haemolysis (i.e., haemoglobinuria), particularly with black snake envenomation, or rhabdomyolysis (i.e., myoglobinuria) with black, tiger, and, to a lesser extent, brown snake envenomation



Collapse and recovery

Collapse and apparent recovery occurs prior to veterinary presentation in some snake envenomed dogs and cats as part of the pre-paralytic signs.

Give antivenom as soon as possible

Given the association between pre-paralytic signs and a high venom dose, antivenom should be administered as soon as possible in cases where collapse and recovery have occurred, and there is other evidence to suggest a high likelihood of envenomation (e.g., high-risk location such as bushland, appropriate season, previous envenomation).

- The rationale for this recommendation is the high likelihood of progression to a fatal envenomation syndrome if left untreated, and the potential to mitigate this with rapid antivenom administration
- Low-likelihood presentations: In contrast, if collapse and recovery occur in an animal with a low likelihood of envenomation based on history, location, and season, then measurement of clotting times provides adjunctive data to assist diagnosis of envenomation. Abnormal clotting times provide increased evidence for antivenom administration, but other differential diagnoses such as haemoperitoneum due to a bleeding mass, anaphylaxis, or anticoagulant rodenticide toxicity remain. Therefore, a complete physical examination and other point-of-care diagnostic tests should be utilised to narrow down the differential diagnoses

Laboratory investigation

(in order of priority, i.e., most value if cost constraints)

Sample collection tips

Blood collection: Collect a blood sample at the time of IV catheter placement into as many tubes as you think you might need for laboratory investigation. Avoid jugular venipuncture due to the risk of ongoing bleeding due to coagulopathy. If unable to sample from the IV catheter, use a peripheral vein and apply a compression bandage afterwards.

Urine collection: Avoid cystocentesis, instead use a catheter or gentle expression

Clotting times

A variety of coagulation tests can be performed to aid in the diagnosis of snake envenomation. The most commonly measured clotting times are PT, aPTT and activated clotting time (ACT).

- Prolongation of PT suggests reduced activity of factor VII (extrinsic pathway) or factors of the final common pathway of coagulation (Factor X, II [prothrombin], I [fibrinogen])
- Prolongation of aPTT or ACT suggests reduced activity of intrinsic (FXII, XI, IX, VIII) or final common pathway factors
- Prolongation of clotting times provides evidence of depletion of coagulation factors that could be consistent with VICC, although other differential diagnoses should be considered. Other conditions that could cause an acute onset of illness and similarly profound coagulopathy, include anaphylaxis and anticoagulant rodenticide toxicity
- **Prevalence:** Prolonged clotting times are evident in the majority of dogs and cats envenomed by brown and tiger snakes, based on published studies.[13,20,36] In many cases clotting factor depletion is so significant that no clot is formed in the assays at all with the assay result reports as being greater than the upper limit of detection[36]
- Prolonged clotting times are likely **venom-dose dependent**. In experimental tiger snake envenomation, coagulopathy was only seen at higher venom doses (10 lethal doses or greater). Clotting times were normal in dogs receiving lower doses,[7] and as such normal clotting times do not rule out envenomation. Anecdotally some envenomed dogs and cats have had normal clotting times at presentation, but they have become prolonged when rechecked (2–6 h later), so rechecking clotting times should be considered when clinical suspicion for snake envenomation remains
- **Fibrinogen:** While less commonly measured, fibrinogen concentrations are severely depleted in VICC and often below the lower limit of detection of the assays[20,36]
- **Viscoelastic testing:** Results of viscoelastic tests (rotational thromboelastometry [ROTEM], thromboelastography [TEG]) have not been thoroughly documented in snake envenomed dogs and cats, however, VICC is expected to manifest as either prolonged clot formation kinetics (e.g., prolonged ROTEM/VCM clot time [CT], prolonged R in TEG) and a weak clot (reduced maximum clot firmness in ROTEM, or maximum amplitude in TEG), or a flat line trace
- **Resolution of VICC** requires synthesis of new clotting factors. Recovery of clotting times begins approximately 12 h after presentation and antivenom treatment, with normalisation by around 24 h[13,20,36]

Muscle enzymes

- **CK rise and timing:** Muscle damage caused by venom myolysins will result in a rise in CK in affected animals, however the magnitude of the increase is varied, and peak can be delayed for 24–72 h post-envenomation despite appropriate treatment with antivenom[22,29]
- **Early presentation:** If the snake encounter happened shortly before presentation it may take 6–12 h for muscle enzymes to be increased above the reference interval

- **How to measure CK:** CK is part of a full biochemistry analysis performed by commercial veterinary laboratories and available as an individual test on some inhouse analysers (e.g., Idexx catalyst, Heska/Antech Element DCX). CK is not included in the Comprehensive diagnostic profile run on a VetScan VS2 (Zoetis Diagnostic Laboratories) but is included on a large animal profile for this machine
- **Interpret with caution:** While high muscle enzymes are supportive of snake envenomation, there are many differential diagnoses for CK elevation, especially for mild elevations in CK (<1,000 U/L). Pre-analytic factors can artifactually increase CK, including sample haemolysis or inadvertent passage of the venepuncture needle through muscle
- **AST as an adjunct:** Aspartate aminotransferase (AST) may be used alongside CK as a marker of muscle damage, though as a hepatocellular leakage enzyme it should be interpreted in the context of other liver enzymes

Packed cell volume (PCV), total protein (TP), and venous blood gas (VBG)/ acid-base analysis

- **Not required for antivenom decision making in the patient with acute LMN signs:** Performing a PCV, TP, and VBG is not necessary or even recommended for decision making regarding giving antivenom for neurotoxic snake envenomation, but forms part of a complete patient assessment and stabilisation where resources allow

PCV/TP a key antivenom trigger in late black snake presentation

Measuring PCV/TP is **very helpful for delayed black snake presentations** as haemolytic anaemia may be present and should prompt antivenom administration even days post-envenomation. Not only will the PCV be low in these cases but visualising the haemolysis in spun PCV tube is characteristic.

- **Rapid results:** PCV/TP and VBGs have the advantage of rapidly available results, often more so than a complete blood count and biochemistry, and can help identify life-threatening abnormalities, such as hypercapnia associated with hypoventilation due to respiratory paralysis. The more unwell an envenomed animal is, the more likely these diagnostic tests are to provide useful information
- **Serial monitoring:** Serial assessment of PCV/TP and VBG aids in the observation of trends over time during hospitalisation. For example, a high PCV/TP is likely to be associated with haemoconcentration secondary to dehydration and assists in formulating a fluid therapy plan. Conversely, a declining PCV/TP may suggest blood loss due to coagulopathy, while a drop in PCV with stable TP is consistent with haemolysis in black snake envenomation
- **Assessment of electrolytes** as part of a VBG can aid in tailoring intravenous fluid therapy to the needs of the individual patient

Complete blood count (CBC) and blood smear evaluation

- **When?** Performing a CBC is not necessary for decision making regarding giving antivenom or the diagnosis of snake envenomation, but forms part of a complete patient assessment where resources allow and should be performed in critically ill animals
- **CBC findings:** CBC abnormalities are not well described in snake envenomed animals, with the exception of black snake cases that can develop haemolytic anaemia. Abnormalities of red cell parameters in a CBC can be interpreted in a similar way to PCV as noted above
- **Spherocytosis:** Blood smear evaluation can be helpful to detect spherocytosis,[29] which may be present before anaemia develops
- **Platelets:** Blood smear evaluation also allows manual verification of platelet count, which may be decreased in dogs with VICC[37]

Biochemistry profile

- **When?** Performing a biochemistry profile is not necessary for decision making regarding giving antivenom or the diagnosis of snake envenomation, but forms part of a complete patient assessment where resources allow and should be performed in critically ill animals
- **Biochemistry findings:** A baseline biochemistry profile can help assess for pre-existing abnormalities and changes in muscle enzymes (as above), but other abnormalities are generally non-specific. Obtaining baseline values for assessment of organ function (e.g.,

creatinine as an indicator of kidney function) may prove helpful if there is concern for the development of complications such as AKI during hospitalisation

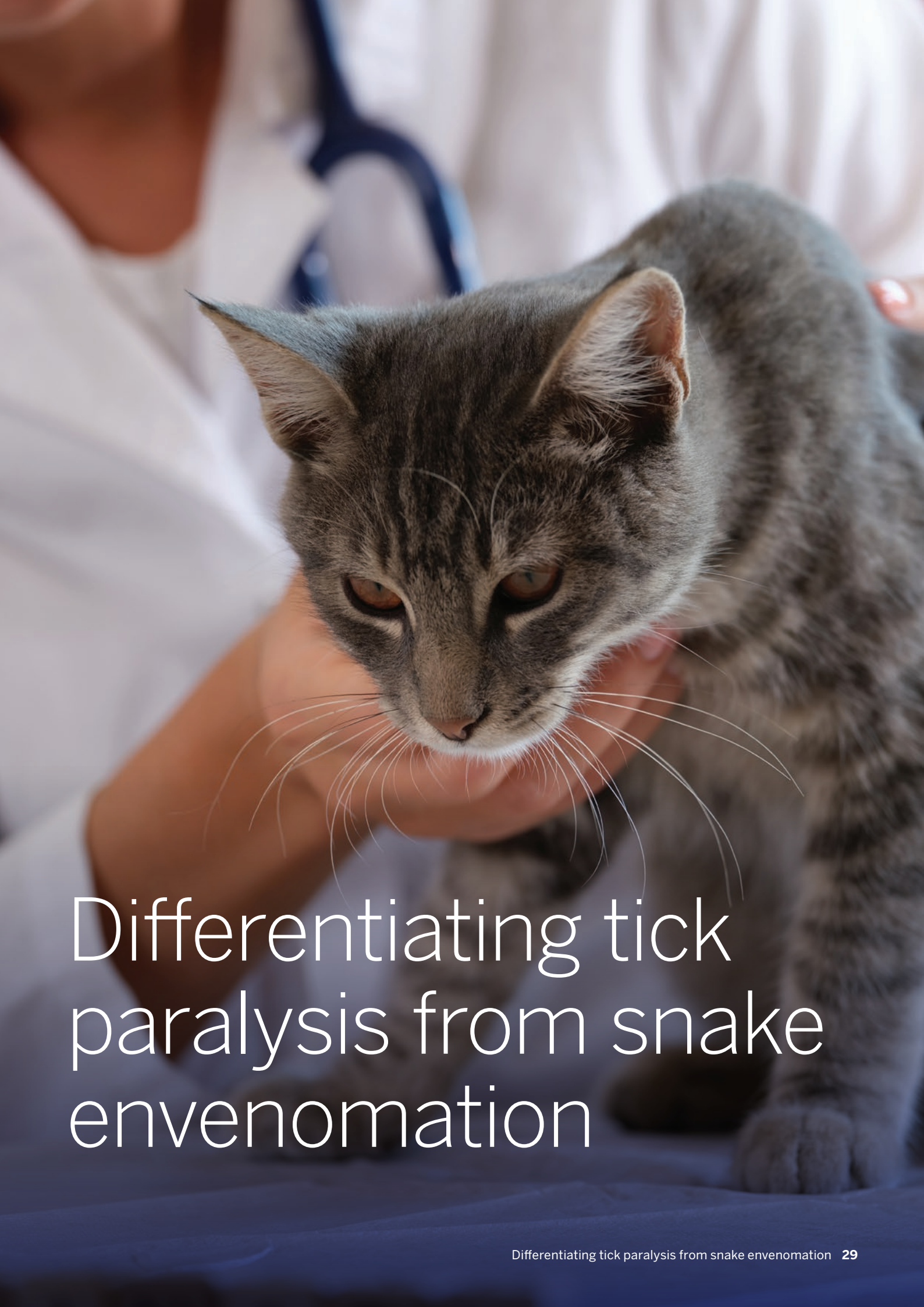
Snake venom detection kit (SVDK)

- **When?** Using an SVDK is not necessary for decision making regarding antivenom administration or the diagnosis of snake envenomation. However, it can be helpful where snakebite envenomation is included as a differential diagnosis for vague clinical signs in order to streamline the diagnostic process. Its value lies in a positive result, which allows the clinician to focus on the diagnosis of snakebite envenomation rather than performing a suite of diagnostic tests for other possible diseases
- **What sample?** Urine is the preferred sample, although blood can be used if unable to obtain urine
- **Interpreting results:** A positive result should be considered diagnostic for snake envenomation since false positives are extremely unlikely if the test is performed correctly. A negative result however does not rule out snakebite envenomation
- **Cost benefit conversation:** A conversation with the owner should include informing them of the costs and benefits of performing an SVDK. In cases of high clinical suspicion of envenomation the client may prefer to invest funds in antivenom administration, rather than an SVDK. However, if clinical signs are vague and the client is not overly cost constrained, then SVDK may prove useful

SVDK results can also guide appropriate antivenom administration where the envenoming snake is uncertain.

- Tiger-brown snake antivenom should be used for patients that are positive in the tiger, brown, or black snake wells of the SVDK
- Death adder antivenom should be used for patients positive in the death adder well, and taipan specific antivenom should be used for patients positive in the taipan well

Eastern brown snake in defensive stance



Differentiating tick paralysis from snake envenomation

Where are you? That is your starting point.

Before examining the patient, your location may already tell you the most likely diagnosis. In tick-endemic coastal eastern Australia, tick paralysis and snake envenomation are both common and should be considered in parallel. Inland and in WA, SA and NT, an acute LMN case should be considered snake envenomation until proven otherwise. Use geography, season and local species prevalence to guide initial probability, then refine with history, physical examination, and diagnostics.[38-40]

Tick paralysis and elapid snake envenomation can both cause acute LMN paralysis in dogs and cats. Treatment differs entirely – early differentiation prevents delays in administration of antivenom or tick antiserum.[38,41]

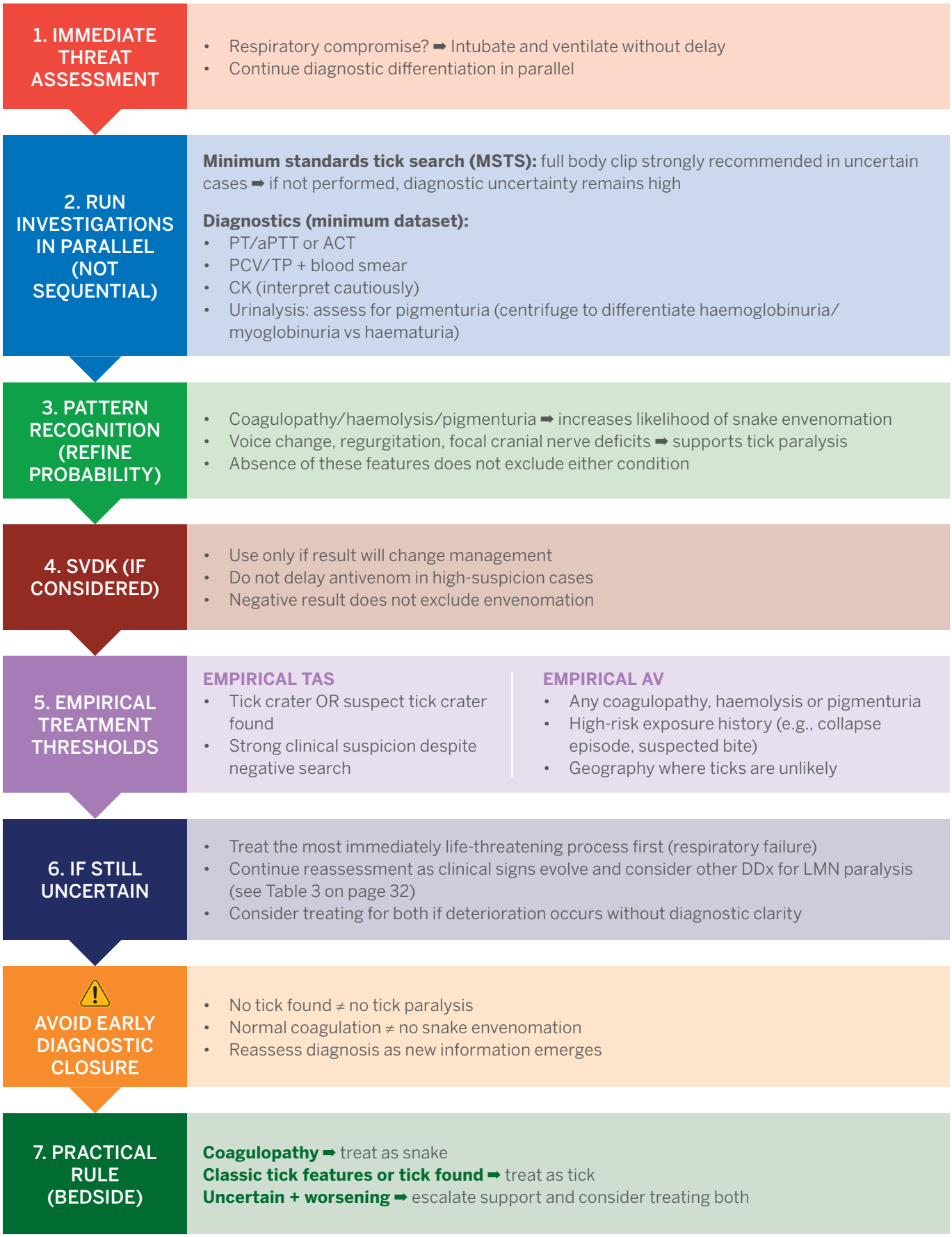
- Early presentations of both tick paralysis and snake envenomation may lack classic LMN paralysis and be difficult to differentiate – diagnostic clarity typically emerges as signs progress[38,41]
- Both may produce ascending or generalised flaccid LMN paralysis; vomiting and mydriasis may be reported in both[13,38,41]
- Onset of paralysis may not differentiate – both can cause rapidly progressive LMN signs. There are no clinical signs while a tick engorges; salivary glands mature around 72 h after attachment and toxin release begins, with signs then able to progress rapidly[42]
- Normal clotting times do not exclude snake envenomation – VICC may be absent early or species-dependent[36,43,44]
- CK is inconsistent and time dependent. It may be increased in some cases of snake envenomation but is often normal at presentation and should not be used exclusively to confirm or exclude the diagnosis[24,44-46]

Table 2 includes the features that reliably point towards one diagnosis over the other. A practical decision pathway is also provided for uncertain cases (Figure 2).

Table 2. *Key differentiating features between snake envenomation and tick paralysis in dogs and cats in Australia.*

Points to SNAKE envenomation	Points to TICK paralysis
Witnessed or suspected snake encounter	**Tick found.** Perform a whole body search; pay particular attention to head and neck, skin folds (axilla, groin, vulva) and interdigital spaces[38]
Coagulopathy/haemolysis/pigmenturia prolonged PT/aPTT or ACT, haemoglobinaemia, spherocytosis, pigmenturia[13,20,24,25,30,36,47,48]	**Change in bark (dogs) or change in meow (cats)**[38]
Pre-paralytic collapse + recovery	**Regurgitation/retching/expiratory grunt (dogs)**[38]
Bite site swelling some species – e.g., RBBS, whipsnake[24,25,29,30,45,49]	**Anisocoria, absent palpebral reflex or unilateral facial paralysis**[38,50]
Elevated CK may support snake; normal CK does not exclude[24,44,46]	

Figure 2. Practical decision pathway where snake envenomation and tick paralysis are both possible causes of acute LMN signs in a dog or cat.



Concurrent tick paralysis and snake envenomation

Rare but documented. A confirmed diagnosis of one does not exclude the other – search for a tick in any snake-envenomed patient, especially in tick season, and keep snake on the differential in any tick paralysis case with atypical features.

Other differential diagnoses for acute LMN signs should also be considered, particularly when a tick search is negative, clotting times are normal, haemolysis and pigmenturia are absent, and SVDK is negative.[51]

Table 3. Key differential diagnoses for acute LMN signs in dogs and cats: distinguishing features from snake envenomation and tick paralysis.

Condition	How it differs from tick paralysis and snake envenomation	Key diagnostic test
Idiopathic polyradiculoneuritis	Slow onset over days (not hours). Tail wag and perineal reflexes preserved. Alert and bright. No megaoesophagus. Can usually urinate and defecate.	Diagnosis of exclusion[41] Electromyography helpful.[51] Consider AChR titre to exclude myasthenia gravis
Myasthenia gravis (generalised)	Slow onset with weakness progressing over days to weeks (unless fulminant). Exercise intolerance with recovery at rest. Megaoesophagus common (regurgitation). Dysphonia. Can mimic tick paralysis. Spinal reflexes typically preserved.[41]	Positive neostigmine response, AChR antibody titre
Botulism	History of carrion or spoiled food ingestion. LMN signs may be accompanied or preceded by GI signs. Usually clinically unwell. Tail wag and perineal reflexes often preserved (as in polyradiculoneuritis). Rare; dogs > cats.[41,51] Cranial nerve and autonomic signs are more common than in other acute LMN diseases, e.g., facial paresis/paralysis, decreased gag tone, mydriasis with decreased pupillary light responses, decreased tear production, alterations in heart rate.[51]	Clinical signs (GI, systemically unwell) + history of rotten food
Tetrodotoxicosis (secondary to puffer fish ingestion)	Beach or marina access; ingestion of discarded fish. Very rapid onset. Vomiting, ataxia, profound LMN tetraparesis, possible cardiac arrhythmias.[41]	History; no specific confirmatory test
Organophosphate toxicity	SLUDGE signs (salivation, lacrimation, urination, defaecation, GI signs, emesis). Miosis. Bradycardia. History of insecticide or chemical exposure.[52]	History; erythrocyte cholinesterase activity
Protozoal myopathy (*Toxoplasma/ Neospora*)	Slower onset over days to weeks. Marked CK elevation. Muscle pain or stiffness. May cause hindlimb rigidity in extension (*Neospora*, especially young dogs). May have fever or systemic signs.[53]	*Toxoplasma/Neospora* serology; CK


SurgiVet
101
PSIMV+
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Modes
Monitoring
Graphics
Tools
Events
System
Controls
Alarms
HAMILTON-G5
INTELLIVENT-ASV
HAMILTON MEDICAL
ICU
WEAR GLOVES
CPR
Clinical Study
14

SUSPECTED SNAKEBITE TREATMENT DECISION TREE

Developed by the ASAP AUSTRALIAN SNAKEBITE ADVISORY PANEL

Known Snake Encounter

ANY clinical signs consistent with envenomation

- No → Measure clotting time(s)
  - Normal → Vet determines benefit > risk for antivenom treatment*
    - No → Wait/watch/ consider other differentials
    - Yes → TREAT WITH ANTIVENOM
  - Prolonged → TREAT WITH ANTIVENOM
- YES → TREAT WITH ANTIVENOM

No Known Snake Encounter

ACUTE LMN SIGNS

Rapidly progressive (e.g., visibly deteriorating within 20 min)

- Yes → Vet determines likelihood of snake > paralysis tick
  - Yes → TREAT WITH ANTIVENOM
  - No → Give tick antiserum, see Tick Guidelines
- No → Clotting time(s) and tick search (if in paralysis tick endemic area)
  - Tick found → Give tick antiserum, see Tick Guidelines
  - No tick, prolonged clotting time(s) → TREAT WITH ANTIVENOM
  - No tick, normal clotting time(s) → Vet determines benefit > risk for antivenom treatment*
    - Yes → TREAT WITH ANTIVENOM
    - No → Wait/watch/ consider other differentials

DISCOLOURED URINE

Centrifuge urine to differentiate pigmenturia from haematuria

- Haematuria → Consider other differentials if no other signs
- Pigmenturia → Measure clotting time(s) and CK
  - Normal clotting time(s) and CK → Vet determines benefit > risk for antivenom treatment*
    - No → Wait/watch/ consider other differentials
    - Yes → TREAT WITH ANTIVENOM
  - Prolonged clotting time(s) and/or increased CK → TREAT WITH ANTIVENOM

COLLAPSE AND RECOVER

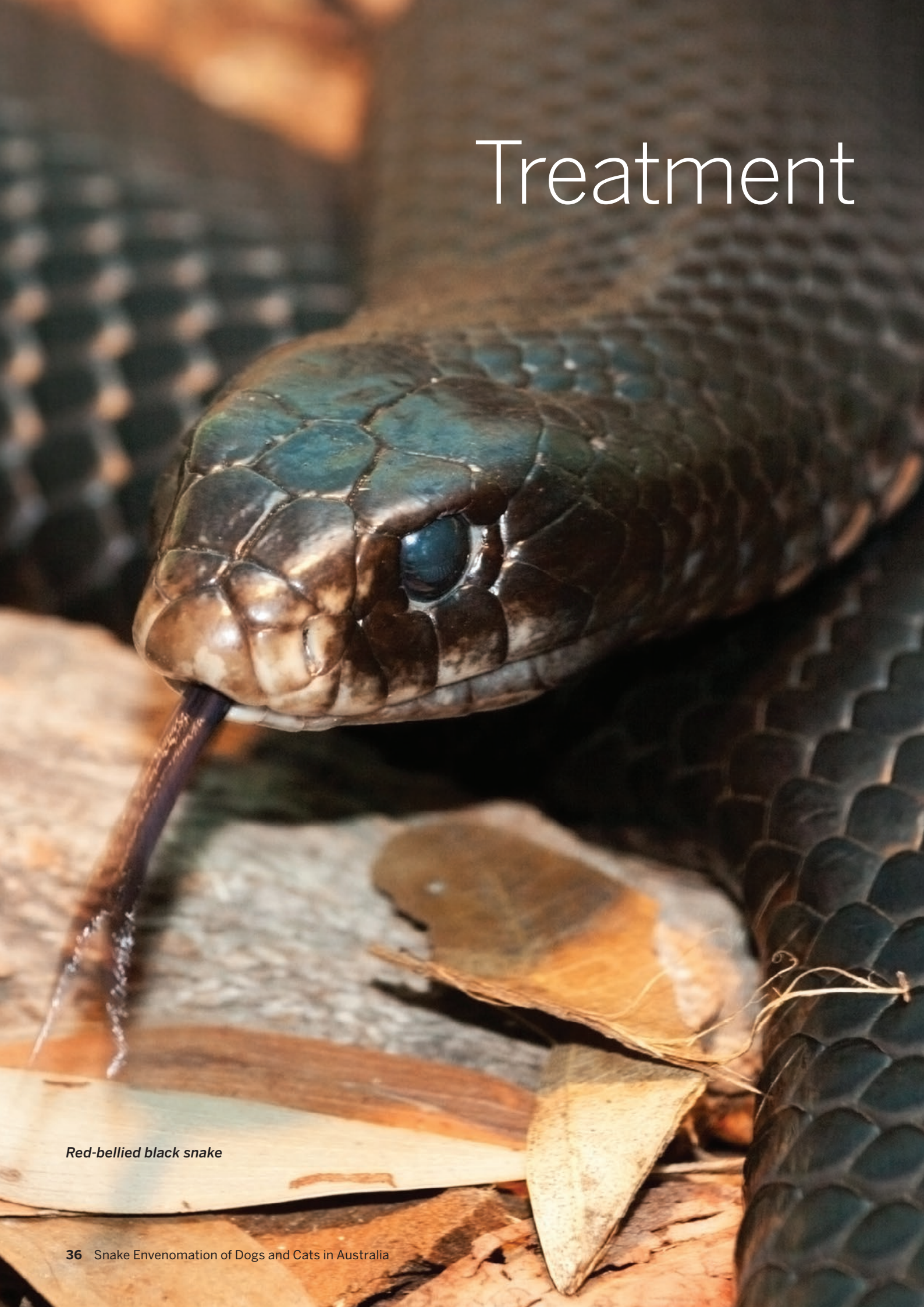
Measure clotting time(s)

- Prolonged → High likelihood of snake envenomation
  - Yes → TREAT WITH ANTIVENOM
  - No → Vet determines benefit > risk for antivenom treatment*
    - Yes → TREAT WITH ANTIVENOM
    - No → Wait/watch/ consider other differentials
- Normal → TREAT WITH ANTIVENOM

***Supportive evidence for envenomation**

- Location, geography, season, previous history of envenomation
- Positive SVDK (snake venom detection kit)
- Increased CK (creatine kinase)
- Pigmenturia
- Haemolysis

Treatment



Red-bellied black snake

Antivenom

Antivenom indications

Give antivenom as soon as possible

Antivenom limits neuromuscular paralysis, haemolysis, and myolysis, reducing the need for mechanical ventilation and blood products, and lowering AKI risk.[24,54,55] It should be administered as soon as possible.

Where the probability of envenomation is high, antivenom should be administered without delay. This includes any of the following scenarios as outlined in the decision tree:

- **Known snake encounter + any clinical sign:** this could be as mild as muscle tremors or slight weakness. Collapse is recognised as a clinical sign even if it is followed by recovery
- **Acute LMN signs:** rapidly progressive deterioration when snake envenomation is more likely than tick paralysis
- **Pigmenturia:** confirmed by centrifugation; supports envenomation when accompanied by clinical findings or other laboratory abnormalities (e.g., prolonged clotting times, increased CK)
- **Collapse and recover + prolonged clotting times**

Early clinical signs vary and may be non-specific; a high index of suspicion should be maintained in at-risk patients, particularly in endemic regions or when exposure is possible. Refer to the "Supportive evidence for envenomation" box on the decision tree, page 34.

When the case does not fit the decision tree

Clinical presentation varies between species and geographically within a species. Rely on local knowledge and clinical judgement. If the presentation does not clearly fit, weigh benefit versus risk across the full clinical picture:

- Signalment and history
- Geographic location and season
- Previous envenomation
- Supporting evidence: increased CK, pigmenturia, haemolysis ± spherocytes, positive SVDK, absence of a paralysis tick in tick-endemic areas

Antivenom considerations

- **Do not delay antivenom for test results:** if clinical signs are present, give antivenom immediately and run diagnostics concurrently
- **Delayed presentation still warrants antivenom:** treat if clinical signs are present, regardless of time since bite. How long free venom circulates is unknown. Antivenom acts primarily on free venom, so efficacy falls as venom becomes tissue-bound. Nonetheless, anecdotally many cases of dog and cat envenomation experience clinical benefit despite delayed antivenom
- **Prior antivenom is not a reason to withhold treatment:** any increased reaction risk is uncertain and generally outweighed by the risk of untreated envenomation. All of the panelists have successfully re-treated dogs and cats with antivenom across separate envenomation events
- **Antivenom is not indicated without clinical signs of envenomation;** monitor a suspected bite closely and treat if any signs develop

Antivenom choice

In Australia, approximately 90% of veterinary snakebite cases involve species neutralised by polyvalent tiger-brown snake antivenom. Often a definitive snake identification is not possible at presentation, so start with tiger-brown snake antivenom.[55]

- **Polyvalent tiger-brown snake antivenom, first-line:** directly covers tiger and brown snake, and cross-reacts with most black snake and copperhead. Use when the species is unknown.[55] One vial contains ≥8,000 units (≥4,000 units tiger + ≥4,000 units brown snake antivenom)
- **Monovalent brown snake antivenom:** is an option where only brown snakes occur, although the larger neutralising dose of tiger-brown snake antivenom is often preferred and more cost effective
- **Species-specific antivenom (death adder, taipan):** the venom of the death adder and taipan is not neutralised by tiger-brown snake antivenom, and thus antivenom specifically developed to neutralise the venom of these species is recommended.[12,26,56] Only human-registered products exist, which are often expensive and hard to obtain quickly. Where the snake isn't identified, a positive death adder or taipan SVDK result confirms the species; refer to distribution maps on pages 9-11. If unavailable, consider giving tiger-brown snake antivenom while sourcing or referring
- **Lesser-known species (e.g., small-eyed snake):** give tiger-brown snake antivenom early. Multiple vials may be required since there is low cross-reactivity with small-eyed snake venom components[57]
- **Product purity:** High-purity products are recommended, such as the currently available tiger-brown snake antivenom products, to minimise the risk of adverse reactions. The reported rate of hypersensitivity reactions in dogs and cats is very low (<5%) with these products.[1] Unrefined serum carries a greater risk of adverse reactions from residual host proteins.[58] Product colour is a subjective indicator of purity; the more refined a product the more likely it is to be colourless, rather than the yellow or amber colour of horse plasma

Initial antivenom administration

Initial antivenom administration: practical steps

- **Dose:** at least one vial IV. One vial is designed to neutralise the average venom yield per species, although venom dose varies widely[55]
  - The panellists commonly give 2 vials of tiger-brown snake antivenom for those animals that require emergent intubation and ventilation, have life-threatening haemorrhage, or pre-paralytic signs
- **Route:** IV infusion only; SC and IM are absorbed too slowly to be effective[54,59]
- **Rate:** IV administration can be either by an IV "push" or by IV infusion,[59] the rate of which should be determined by the severity of the envenomation syndrome
  - Rapid administration (over 1–5 minutes) is recommended for patients in an imminently life-threatening condition or if animals had pre-paralytic signs. The World Health Organisation recommends a maximum rate of 2 mL/min (i.e., 10 mL vial in 5 min) for the IV "push" although panel members describe administering antivenom more rapidly than this in some cases[59]
  - IV infusion should be completed in no more than 60 minutes. Slower infusion is appropriate for stable patients or delayed presentations. Slower infusion potentially aids in the detection of a hypersensitivity reaction, although leads to slower time to venom neutralisation
- **Dilution:** sterile saline dilution may be used where patient weight allows a larger volume, but dilution is not required
- **Monitoring:** continuous monitoring of patient vital signs during infusion and for at least 1–2 h afterwards is recommended
- Follow the Manufacturer's instructions for administration
- **Expiry dates:** based on research performed, the World Health Organisation guidelines for the management of snakebites state that "Ideally, antivenoms should be used before stated expiry dates, antivenoms retain useful activity for months or even years after these dates. In patients with severe envenoming, recently expired antivenoms may be considered if there is no alternative"[59]

Routine premedication (antihistamines/corticosteroids) is NOT recommended

There is no evidence that premedication with antihistamines or corticosteroids prevents anaphylaxis.[58,60,61]

More than one vial? The evidence to support or reject administration of additional vials of antivenom is limited. Extra vials may be justified by clinical severity, regional venom differences, local experience, or practice protocols; use judgement and weigh cost. In the largest case series of eastern brown snake envenomation, antivenom improved survival versus none, but there was no added benefit of two vials over one.[13] Animals given more antivenom trended towards a worse outcome, but this was most likely because clinicians gave more to the sicker cases that had the highest probability of non-survival.[13] The retrospective nature of this study, and confounding by case severity, means that it cannot be used to determine the optimal number of vials of antivenom. Rather the decision remains case-specific and clinician-dependent.

Continued treatment after antivenom

Antivenom is not the only treatment required for snake envenomation. Mechanical ventilation and blood product transfusion is often life-saving; intravenous fluids, analgesia and nursing care further support recovery.

- **Mechanical ventilation:** see Mechanical Ventilation section on page 43
- **Fluid therapy:** see Intravenous Fluid Therapy section on page 56
- **Blood products:** see Blood Product Transfusions section on page 48
- **Analgesia and anxiolytics:** see Analgesia section on page 60
- **Nursing care:** see Recumbent Patient Care section on page 63

Repeat antivenom dosing

Most cases are well managed by the initial antivenom dose. If the patient is deteriorating, before repeating antivenom, reassess ventilation, perfusion, and the need for adjunctive therapies (e.g., mechanical ventilation, blood products), as these are often the determinants of outcome.

When to repeat antivenom

Repeat antivenom IS indicated for:

- **Severe, ongoing haemolysis (red-bellied black snake), especially if transfusion is required:** a further vial may be considered based on panel experience and two publications.[29,30] See Haemolysis section on page 50
- **Persisting respiratory paralysis or severe tetraparesis after brown snake envenomation in Western Australia:** anecdotally, additional vials have been given with continued neurological improvement after each vial;[15] proposed to reflect a reversible postsynaptic neurotoxin component although the exact pathophysiology has not been studied

Repeat antivenom IS NOT indicated for:

- **Coagulopathy:** repeat antivenom does not hasten recovery from VICC; give plasma for clinically significant bleeding, not more antivenom[19,20]
- **A persistently positive SVDK:** urine SVDK results may stay positive for hours after effective antivenom administration as venom antigens are filtered freely by the kidney into the urine while antivenom is excreted far more slowly[27,36,45]
- **Established LMN signs (paralysis) due to presynaptic neurotoxins:** for example, a paralysed eastern brown snake envenomation that has already had 1 to 2 vials of antivenom. Paralysis will not reverse with more antivenom, so prioritise ventilatory support[13,36,62-64]

Risks of additional vials: the evidence to support or reject administration of additional vials of antivenom is limited. Additional vials might slightly increase the risk of acute reactions (mild angioedema to serious anaphylaxis), delayed reactions (serum sickness), or sensitise the patient to future reactions if antivenom is administered again. Highly purified antivenoms are preferred and carry a lower reaction risk[58]

Adverse reactions to antivenom

The reported rate of hypersensitivity reactions to modern, high-purity, antivenom in dogs and cats in Australia is very low based on recent data.[1,13] The SnakeMap publication reported hypersensitivity reactions in 4% of dogs (14/358), 95% confidence interval [CI] 2–7%) and 0.6% of cats (1/165, 95% CI 0.1–3%), but the severity of these reactions was not specifically described.[1] The largest publication of eastern brown snake envenomation reported potential reactions to antivenom in 6% of dogs (12/199) and no cats (0/66).[13] The nature of the reactions reported in the latter publication included mild to moderate facial angioedema (9 dogs), brief self-resolving bradycardia (1), vomiting (1), and mild hypotension (1).[13]

Reactions to antivenom form a spectrum from a mild type 1 hypersensitivity reaction with dermatologic signs only, to life-threatening anaphylaxis, including cardiovascular collapse and/or respiratory compromise. The historical distinction between "anaphylactic" (IgE-mediated) and "anaphylactoid" (non-IgE-mediated) reactions is no longer recommended, as the two are clinically indistinguishable and management is guided by severity rather than mechanism.[60,61]

Acute reactions

Severity guides management

Most reactions are mild and cutaneous (e.g., angioedema, urticaria). Gastrointestinal, respiratory or cardiovascular signs indicate more severe anaphylaxis. When severe, dogs predominantly show cardiovascular and gastrointestinal signs, while cats show respiratory signs, the lung being their primary shock organ[60,61,65-67]

- Mild signs are usually self-limiting and resolve without treatment; an antihistamine can speed recovery and improve patient comfort
- Adrenaline is indicated for moderate to severe reactions and should always be easily accessible

Table 4. Recognition and recommended treatment of acute adverse reactions to antivenom in dogs and cats based on severity.

Reaction Severity			
Clinical Signs	**Mild**	**Moderate**	**Severe**
Both species	**Urticaria, erythema, pruritus; facial angioedema**	**Shock – Abnormal perfusion parameters (pale mucous membranes, prolonged CRT, poor pulse quality, hypothermia)** **Tachycardia or bradycardia** **Hypotension**	**Cardiovascular collapse, profound hypotension, arrhythmias, obtundation**
Dogs		**Vomiting, diarrhoea**	**Haemorrhagic diarrhoea, coagulopathy**
Cats		**Dyspnoea, tachypnoea, hypoxaemia (± bronchoconstriction), piloerection, vomiting, diarrhoea** *Cats can deteriorate very rapidly.*	**Respiratory collapse, severe dyspnoea, non-cardiogenic pulmonary oedema**
Recommended treatment	• Slow antivenom administration • Antihistamine • Adrenaline not needed; escalate if signs progress	• Stop antivenom administration • **Adrenaline IM (IV if known coagulopathy) ± CRI if signs persist (see Dosing)** • IV bolus of isotonic crystalloid fluid 10 mL/kg (dogs), 5–10 mL/kg (cats), repeat up to 40 mL/kg (dogs), 20 mL/kg (cats) • O_2; intubate and PPV if indicated	• Stop antivenom administration • **Adrenaline CRI ± IV bolus to start if indicated (see dosing)** • IV bolus of isotonic crystalloid fluid 10 mL/kg (dogs), 5–10 mL/kg (cats), repeat up to 40 mL/kg (dogs), 20 mL/kg (cats) • O_2; intubate and PPV if indicated • If hypotension refractory: increase adrenaline CRI ± second-line vasopressor (dopamine or vasopressin)

Restarting antivenom after a reaction

Restarting antivenom is an individual decision, balancing the ongoing need for venom neutralisation against the risk of ongoing or worsening adverse reaction. Consider whether envenomation is confirmed or only possible, the severity of the envenomation syndrome, the severity of the adverse reaction, and the patient's stability after initial treatment. If the benefit of the antivenom still outweighs the risk, restart antivenom at a lower rate, once the clinical signs of the adverse reaction are controlled. This may mean running the antivenom infusion over multiple hours.

Table 5: Adrenaline dosing for treatment of anaphylaxis.

Adrenaline Dosing			
Bolus (IM or IV)	0.01 mg/kg, repeat q5–15 min as needed. IM first-line; IV bolus before a CRI or if the IM route is contraindicated (i.e., due to coagulopathy/VICC)		
CRI	0.05 µg/kg/min IV (range 0.005–0.5), titrate to effect; wean gradually, do not stop abruptly		
Patient Weight (kg)	**Bolus IM or IV (mL)** neat 1:1,000	**Bolus IM or IV (mL)** 1:10,000 dilution 1 mL adrenaline + 9 mL crystalloid fluid (e.g., 0.9% NaCl)	**CRI (mL/h)** 1 µg/mL 1 mL adrenaline in 1,000 mL crystalloid fluid (e.g., 0.9% NaCl)
2.5	0.025	0.25	7.5
5	0.05	0.5	15
10	0.1	1	30
20	0.2	2	60
40	0.4	4	120
60	0.6	6	180

The IV bolus may be lowered to ¼ or ½ the dose (2.5–5 µg/kg) where a more cautious dose is preferred.

In coagulopathy (VICC), an IM injection can cause bleeding/haematoma at the site and reduce absorption; give adrenaline IV.

Delayed reactions

Delayed reactions (serum sickness, 5–14 days post-AV)

A Type III immune-complex reaction. Host antibodies against the foreign equine protein form complexes that deposit in the skin, joints and kidneys, causing skin eruptions, joint pain/swelling and lethargy. A short tapering course of corticosteroids usually relieves signs until they resolve[55,58]

Mechanical ventilation (MV)

Respiratory paralysis is the most immediately life-threatening complication of elapid envenomation.[13,36,45,48,68,69] MV for snake envenomation is typically shorter than for tick paralysis.[36,48,70] Postsynaptic neurotoxins are reversed by antivenom,[11] while presynaptic neurotoxins require nerve-terminal regeneration over days.[3] Most snake-envenomed patients have normal lungs at intubation and rarely aspirate, thus overall MV prognosis is better than for tick paralysis.[36,48,69,70] Pulmonary parenchymal disease at the time of MV (i.e., aspiration pneumonia or pulmonary haemorrhage) is uncommon but significantly worsens outcome.[14,85]

Know your local snake species

The need for, and duration of, MV vary by envenoming species and geographic region, ranging from hours to days;[13,15,48,69] local knowledge of prevalent species and presenting syndromes is essential.

Early recognition of the need for MV – before respiratory fatigue or arrest – is the critical clinical skill in these cases. Most require intubation at presentation or as progressive fatigue makes ventilation untenable.

Do not wait for respiratory arrest

Early intubation improves prognosis.[68,69] Anticipate MV in any patient with progressive LMN paralysis.

Indications for mechanical ventilation

Indications for MV include:[68,69,71,73]

- **Severe hypoxaemia** – SpO_2 <90% (or PaO_2 <60 mmHg) on supplemental O_2
- **Severe hypercapnia** – $PaCO_2$ / $PvCO_2$ / $ETCO_2$ >60 mmHg
- **Unsustainable work of breathing (WOB)** – clinical judgement. Minimal chest excursions in LMN paralysis represent unsustainable effort. Note that oxygenation and ventilation parameters may be normal until exhaustion
- **Respiratory arrest**

Recognising unsustainable WOB in snake envenomation: a clinical judgement

Unsustainable WOB in snake envenomation may be more subtle than from other causes because of the paralysis. Snake-paralysed patients with tetraparesis and respiratory paralysis will have minimal chest excursion and flaccid paradoxical abdominal movement. Respiratory effort may appear mild to moderate, and the respiratory rate may be very slow or fast.

Managing respiratory failure without a mechanical ventilator

In the absence of a ventilator, intubate and provide manual positive pressure ventilation (PPV) via an ambu bag under general anaesthesia. Some snake envenomation cases require only hours of respiratory support before neurotoxin effects begin to resolve – manual PPV may be sufficient. Where prolonged support is needed, maintain manual PPV during transfer to a referral centre. In remote areas where transfer is not immediately possible, manual PPV should be continued with periodic attempts to wean.

Pre-intubation preparation

Don't move the patient to intubate

A patient with unsustainable WOB can deteriorate acutely due to respiratory or cardiopulmonary arrest. Intubate in the cage or on the crash bench, confirm vitals are stable on PPV, then move the patient.

- **IV access:** large-bore catheter; draw bloods at the same time
- **Monitoring:** ECG, SpO_2, non-invasive blood pressure
- **Pre-oxygenate:** 100% O_2 by mask or flow-by until intubated
- **Equipment:** laryngoscope, cuff syringe, tube ties, suction, ambu bag, capnography
- **ET tube:** appropriate size, with one smaller and one larger at hand. Use a sterile tube where time permits; if an emergency intubation requires a non-sterile tube, change it for a sterile one at the earliest convenience
- Crash cart and emergency drugs immediately accessible.

Referral

MV is resource- and staffing-intensive. Where referral to a specialist centre is available, it should be considered early. Manual ventilation may be needed during transport.

Induction and maintenance of total intravenous anaesthesia (TIVA)

Multiagent TIVA is the standard for MV anaesthesia in dogs and cats.

Induction

- Propofol 4–6 mg/kg IV to effect; start at 2 mg/kg and titrate (less is often sufficient in paralysed patients)
- Alfaxalone 2–5 mg/kg IV to effect; preferred in cats

Maintenance CRI options

Combining an anaesthetic agent with an opioid (pure or partial μ-agonist) and sedative minimises the cumulative dose of each drug, supports cardiovascular stability, and may reduce the risk of propofol-associated oxidative injury.[72-74] Titrate each to effect. Suggested starting doses are included in Table 6.

Table 6. Recommended agents, including starting doses, for total intravenous anaesthesia in dogs and cats receiving mechanical ventilation for treatment of snake envenomation.

Drug	Starting CRI dose rate	Notes
Anaesthetic agents		
Propofol (dogs)	0.05–0.6 mg/kg/min	Practical starting rate: 0.5 mL/kg/h of propofol 1% (≈0.08 mg/kg/min), titrate up as needed. If MV >48 h is anticipated, minimise propofol amounts, or transition to alfaxalone – Heinz bodies have been reported at 47–96 h of propofol CRI in a published case series in dogs.[72] Daily blood smears.
Alfaxalone	1–4 mg/kg/h	First-line maintenance in cats (avoid propofol due to oxidative haematotoxicity). Preferred over propofol for prolonged MV in dogs.
Opioids		
Fentanyl	2–10 µg/kg/h	Pure μ-agonist. Analgesia and sedation synergy; give 2–5 µg/kg IV bolus before starting CRI.
Butorphanol	0.1–0.5 mg/kg/h	May provide sedation but minimal analgesia; not suitable if pain present.
Sedatives		
Midazolam	0.1–0.5 mg/kg/h	Sedation and muscle relaxation; effective in combination with opioids and propofol/alfaxalone.
Dexmedetomidine or medetomidine	0.5–3 µg/kg/h	Potent sedation-sparing agent, useful during weaning and post MV. Medetomidine may produce more pronounced cardiovascular depression at equivalent dosing.
Ketamine	0.1–0.5 mg/kg/h	Sympathomimetic cardiovascular support; use as part of multiagent TIVA – not recommended as sole agent.

Ventilator settings

The ventilator settings outlined in Table 7 represent pragmatic veterinary targets adapted from key references.[71,73,75] While lung-protective strategies (lower tidal volume and higher positive end expiratory pressure [PEEP]) are widely used in human critical care medicine, dogs have greater airway dead space and often require higher tidal volumes.[76,77] Ventilator settings should be individualised to gas exchange and respiratory mechanics rather than applied rigidly. Once the patient is stable on the ventilator, settings can then be titrated to the least aggressive parameters required to achieve acceptable oxygenation and ventilation.

Table 7. Recommended ventilator settings for mechanical ventilation of dogs and cats.

Setting	Healthy Lungs	Lung Disease
FiO_2	100% initially; reduce to ≤60% when stable and as soon as possible	100% initially; reduce to ≤60% when stable and as soon as possible
Tidal volume (mL/kg)	10–15	8–12
Respiratory rate (breaths/min)	10–20	15–30
Pressure above PEEP (cmH_2O)	8–10	10–15
PEEP (cmH_2O)	0–5	5–8
Inspiratory flow rate (L/min)	40–60	40–60
Inspiratory time (s)	0.8–1	0.8–1
Rise time (s)	0.1–0.3	0.1–0.3
I:E ratio	>1:2	1:1 to >1:2
Inspiratory trigger	1–2 cmH_2O or 1–2 L/min	1–2 cmH_2O or 1–2 L/min

Monitoring during MV

Patients should be connected to a multiparameter monitor for continuous assessment of parameters including SpO_2, $ETCO_2$, ECG, temperature and blood pressure, during MV. Table 8 includes recommended monitoring of the mechanically ventilated patient, adapted from key references.[71,73,78]

Table 8. Recommended patient monitoring of dogs and cats during mechanical ventilation.

Parameter	Frequency	Notes
SpO_2	Continuous	Target ≥95%
$ETCO_2$	Continuous	Target 35–50 mmHg
ECG	Continuous	Arrhythmia detection
Temperature	Continuous or q1h	Active warming PRN to maintain normothermia
Blood pressure	Continuous or q1h	Target MAP ≥60–65 mmHg
Venous blood gas (VBG, for assessment for $PvCO_2$)	Minimum q6h	Preferred while coagulopathic over arterial blood gases. $PvCO_2$ correlates well with $PaCO_2$ for ventilation assessment
Arterial blood gas (ABG)	Once coagulopathy resolved if concerns for oxygenation	**Do not perform while VICC is active** – arterial catheterisation/ sampling risks haemorrhage. Rely on $PvCO_2$, $ETCO_2$ and SpO_2 until clotting times normalise.
PCV/TP + blood smear	q12–24h	Monitor for anaemia (haemorrhage or haemolysis). Monitor for Heinz bodies if propofol in use[72]
CBC + Biochemistry	q24–48h	Frequency depends on clinical status, e.g., risk of AKI, hepatopathy
Fluid balance	q4–8h	Inputs and outputs; avoid over-hydration
Cytology of ETT sputum	At each tube change	Intubated and ventilated patients are at risk of pneumonia. Performing cytology of purulent exudate on the ETT can aid in early detection

Nursing and airway care of the ventilated patient

Infection Control

Wear gloves and wash hands before every patient contact; wash hands after removing gloves. Hand hygiene is one of the most effective measures for preventing hospital-acquired infections.[78-80]

Diligent nursing care of the ventilated patient maximises patient comfort and reduces the risk of complications and secondary infection. Recommendations are adapted from key references.[73,78,80]

- **Reposition:** q2–4h. During MV the patient's thorax is generally kept in sternal recumbence while the hips are 'flipped' q2–4h to the left and right. Use soft bedding or foam mattress
- **Ocular care:** q1–2h eye lubrication. Daily assessment for corneal ulceration
- **Oral care:** q4h – 0.05% aqueous chlorhexidine rinse then saline; glycerine to the tongue
- **Analgesia:** pain is under-recognised in the ventilated/recumbent patient. Pure μ-opioids ± paracetamol are first-line; avoid NSAIDs. See Analgesia section on page 60

Airway care ensures effective maintenance of an airway and reduces the risk of complications such as airway obstruction or secondary pneumonia.[78,80-82]

- **ET tube ties:** IV giving-set tubing is recommended over material tube ties since the latter become contaminated with oral secretions. Ties should be soft and tied so as to minimise pressure necrosis of the skin. Changing the tie position q4h may aid in reducing pressure injury
- **Cuff pressure:** 20–25 cmH_2O; check q4–8h
- **Suction:** ET tube and oropharynx aseptically, minimum q4h, or sooner for audible/visible secretions, rising peak inspiratory pressure or saw-tooth appearance on flow-volume loop, capnogram waveform changes, or falling SpO_2
- **ET tube change:** not performed routinely. Replace only for persistent high resistance or ET occlusion unresolved by suction, visible tube/cuff damage
- **Humidification:** heated water humidification of the ventilator circuit with a humidifier is ideal, although HME (heat and moisture exchanger) filters can also be used. Saline nebulisation q4–6h if secretions thicken

Weaning and extubation

Weaning from mechanical ventilation can occur when ALL of the following are met:[73,83]

- Spontaneous respiratory effort present and improving
- SpO_2 maintained on FiO_2 ≤0.4 with sustainable work of breathing
- Tolerates a ventilator-supported spontaneous breathing trial [SBT]. Ventilator support may include continuous positive airway pressure (CPAP) and/or pressure support (PS)
- Tolerates an unassisted SBT (intubated, no CPAP/PS) for ≥1 h without fatigue
- $ETCO_2$ cross-checked with $PvCO_2$ at 5 min and again before extubation; target $PvCO_2$ <60 mmHg (50–60 = grey zone, continue based on clinical and financial context)

Fail criteria – return to MV for 12–24 h (snake species-dependent):[73,83]

- Unsustainable RR or effort
- $ETCO_2$ trending >60 mmHg, confirmed on VBG

Post-extubation supplemental oxygen can be administered as required. Close monitoring is required for fatigue/deterioration, particularly in the first few hours after extubation. Patients are generally maintained nil per os for 4–8 h given the risk of aspiration.

Jack Russell Terrier connected to an ambu bag

Blood product transfusions

Blood products are required in only a minority of envenomations, and frequency varies by species: up to 4% of eastern brown cases,[13,64] and up to 17% of RBBS cases.[24,30] Frequency of blood product transfusion for dogs and cats envenomed by tiger snakes, mulga (king brown) or other brown snakes (dugite, western brown) are not separately published. The two indications for transfusion after envenomation are **haemorrhage and haemolysis**.

When to transfuse? Clinical decision-making should be guided by the presence and severity of bleeding or haemolysis, not laboratory abnormalities alone.

Haemorrhage

Prolonged clotting times do not predict bleeding: an animal may have unclottable blood yet not bleed. The severity of active bleeding guides plasma and red cell use.

Venom-induced consumption coagulopathy (VICC), seen with several Australian elapids (most commonly eastern brown, tiger and taipan), consumes clotting factors and carries the highest risk of life-threatening bleeding; it is the coagulopathy most likely to require plasma transfusion. While antivenom neutralises circulating venom and thereby prevents further clotting factor consumption, it does not reverse established VICC; recovery depends on hepatic synthesis of new clotting factor proteins. Clotting factors begin to recover within approximately 12 h, although full normalisation usually takes around 24 h (but may take longer).[13,20,36]

Mild haemorrhage from VICC (e.g., oozing from venepuncture sites) usually stops within 12-24 h. Apply pressure where possible for control of bleeding. More significant haemorrhage or pulmonary haemorrhage requires clotting factor replacement with plasma transfusion +/- packed red blood cells (pRBCs) or whole blood.

Anticoagulant coagulopathy, more typical of black snakes (RBBS, king brown), is reversed by antivenom alone,[84] and is less likely to cause clinical bleeding. However, clinically significant bleeding, including fatal pulmonary haemorrhage, has been reported with RBBS,[24] possibly reflecting the procoagulant, consumptive effect RBBS venom shows at high concentrations in vitro.[84]

Treat the bleeding, not the laboratory defined coagulopathy

Manage clinically significant bleeding the same way regardless of mechanism: antivenom first, then plasma, with or without red cells. Minor oozing or prolonged clotting times alone are not an indication for transfusion.

Table 9. Assessing the severity of clinical bleeding in patients with venom induced consumptive coagulopathy to guide blood product transfusion.

Clinical Bleeding in Patients with VICC			
NO CLINICAL BLEEDING	**MILD**	**SIGNIFICANT**	**PULMONARY HAEMORRHAGE**
No bleeding identified clinically. Clotting tests prolonged.	Mild bleeding present but patient haemodynamically stable. Typically self-resolving as clotting factors regenerate.	Active bleeding with haemodynamic compromise, or bleeding into a critical site.	Distinct, rapidly progressive bleeding into the lungs; can be fatal within hours.
Clinical signs • No external or internal haemorrhage • Monitor for development of clinical bleeding	**Clinical signs** • Peripheral venepuncture haematoma • Bite-site ooze or bleeding • Mild gingival haemorrhage[13,36,64] • Monitor closely; severe bleeding may develop at other sites	**Clinical signs** • Haemoabdomen or haemothorax[13] • Intramyocardial haematoma[15] • Extradural haematoma[16] • Severe or ongoing external haemorrhage • Rapidly falling PCV with active bleeding	**Clinical signs** • Soft cough ➡ hypoxaemia ➡ respiratory distress ➡ haemoptysis[14,85] • POCUS: B-lines and pleural effusion[85] • Thoracic radiographs: interstitial/alveolar pattern (do not delay treatment for imaging)
Plasma not indicated.	**Plasma not required.**	**Give plasma (10-20 mL/kg) over ≤60 min.**	**Give rapid plasma bolus (10-20 mL/kg) over ≤15-30 min.**

Fatal pulmonary haemorrhage

Dogs may present ambulatory with only a soft cough before overt haemoptysis; do not wait for haemoptysis to suspect pulmonary haemorrhage. If suspected, perform thoracic point-of-care ultrasound (POCUS) or thoracic radiographs. Increased B-lines, generalised or patchy interstitial to alveolar pattern, and/or falling PCV/TP are indications to act immediately with plasma ± RBC product transfusion. Mortality is high if unrecognised or treatment is delayed.[14,24,85] Reported with eastern brown,[14,85] and RBBS,[24] and tiger snakes (anecdotal, unpublished).

Red-bellied black snake

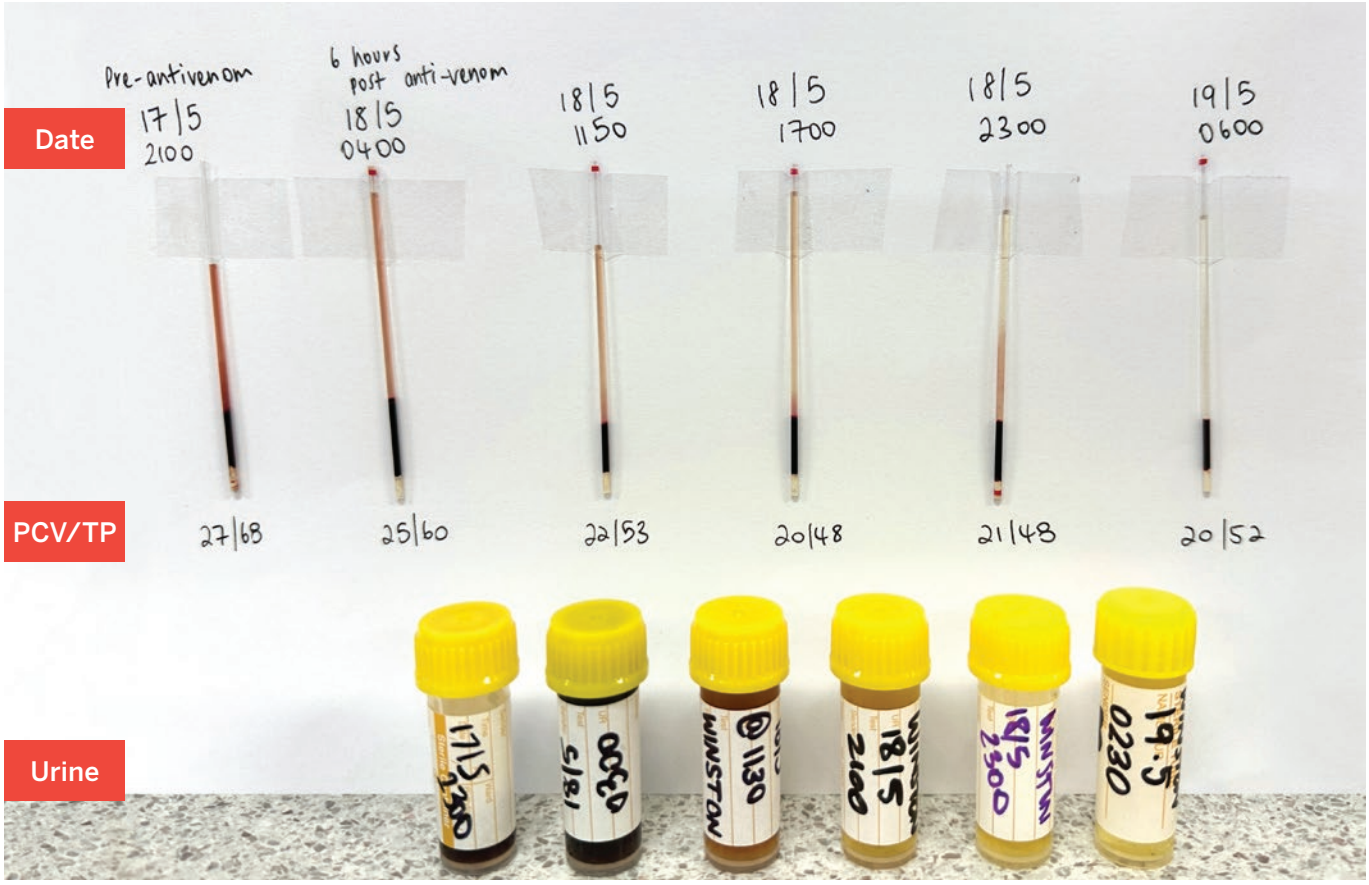
Haemolysis

Red cell transfusion is reserved for those patients with haemolysis that develop anaemia severe enough to impair oxygen delivery, not haemolysis alone. Red cells can be provided in the form of pRBCs or whole blood.

Continued haemolysis

- Haemolysis onset may be delayed: dogs can appear stable at presentation then develop worsening pigmenturia, haemoglobinaemia and a falling PCV around day 3–5, whether still hospitalised or after discharge; this can be fatal if missed[24,29,30,32,33]
- Keep monitoring inpatients, and advise owners to return for darkening urine, pale gums or lethargy
- **Corticosteroids are not indicated:** spherocytosis and a delayed, progressive course can mimic immune-mediated haemolysis, but this is direct venom-mediated red cell membrane damage, not IMHA
- **Additional antivenom:** in cases of severe, ongoing haemolysis, especially if transfusion is required, a further vial of antivenom may be considered. Although the evidence is weak, this recommendation is based on panel experience of benefit and supported by two published cases.[29,30] Both dogs received one vial initially and a second on day 2–4 for ongoing haemolysis (See Repeat antivenom dosing on page 40). In these cases circulating venom was undetectable after the first antivenom dose, but persistent venom activity cannot be excluded based on clinical evidence of benefit after the second vial

Figure 3. Resolution of haemoglobinaemia and pigmenturia in a red-bellied black snake-envenomed dog over ~30 h following a second vial of tiger-brown snake antivenom on day 4 for ongoing intravascular haemolysis; PCV stabilised ~14 h post AV.



When to transfuse – Transfusion triggers

Transfusion triggers: clinical signs that indicate transfusion (not lab values alone)

Anaemia with impaired oxygen delivery

- Dull mentation, lethargy, weakness
- Tachycardia, weak pulses, prolonged CRT
- Pale mucous membranes. Caution: with severe intravascular haemolysis mucous membranes can remain pink (see Figure 4) and CRT is unreliable; assess HR, pulse quality, mentation and lactate instead
- Hyperlactataemia (lactate concentration >2 mmol/L)
- PCV <15% regardless of clinical signs, or <20–25% with clinical signs of impaired oxygen delivery

Active bleeding

- Clinically significant bleeding, internal or external
- Pulmonary haemorrhage (soft cough, haemoptysis, B-lines on POCUS)
- Falling PCV/TP, although it may be normal early in acute haemorrhage and falls only as interstitial fluid shifts into the circulation, and with IV fluids; splenic contraction can also mask the drop in PCV
- Bleeding into a critical site is an indication for transfusion on its own: myocardium (intramyocardial haematoma), pericardium, spinal cord (extradural haematoma) or intracranial, eye (scleral haemorrhage, hyphaema)

Minor oozing, or prolonged clotting times alone, are not transfusion triggers.

Figure 4. Pink mucous membranes in a red-bellied black snake-envenomed dog despite severe haemolytic anaemia (PCV 7%). Haemoglobinaemia keeps the gums pink, so assess anaemia and perfusion by HR, pulse quality, mentation and lactate concentration, not mucous membrane colour.

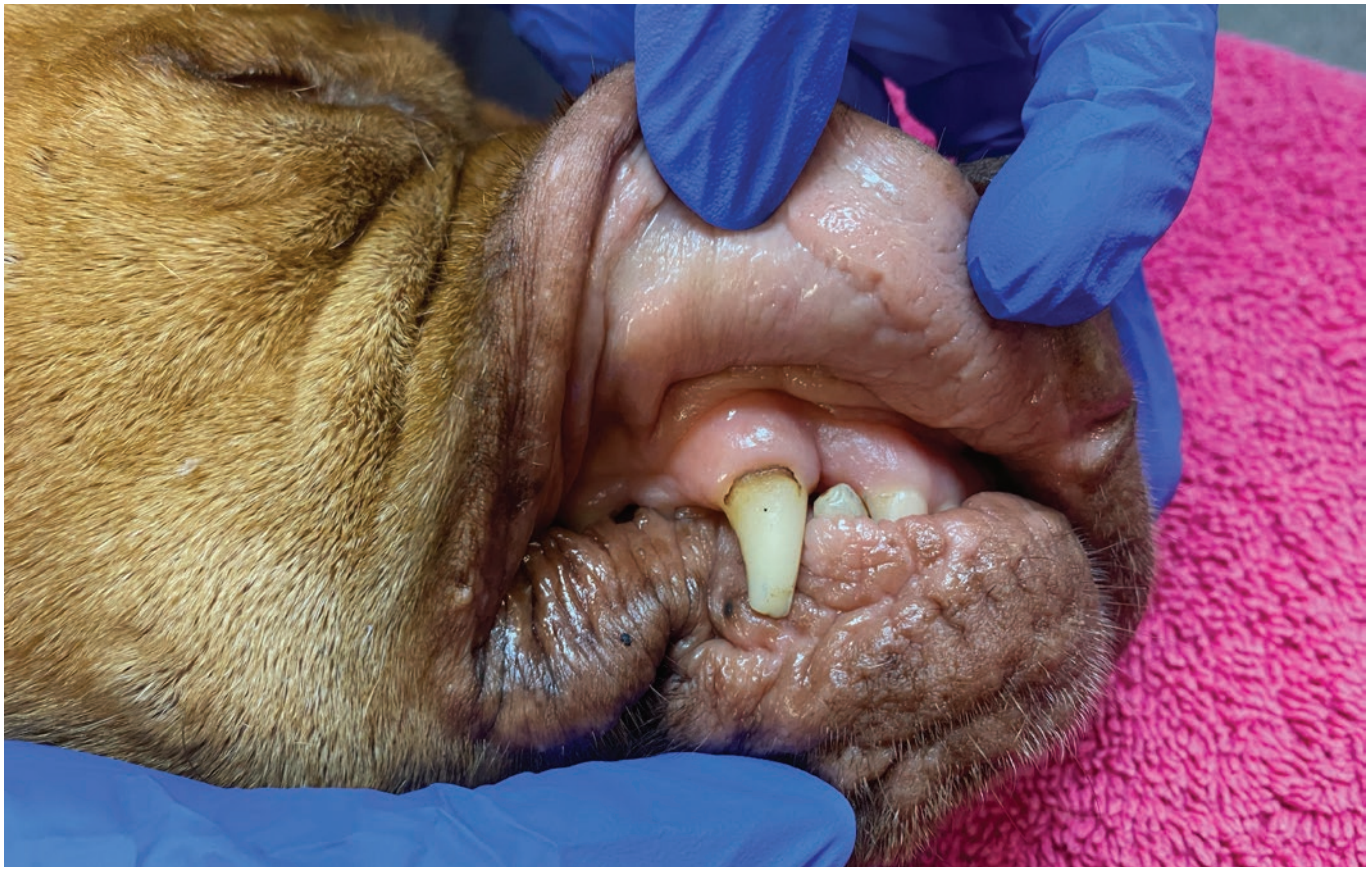
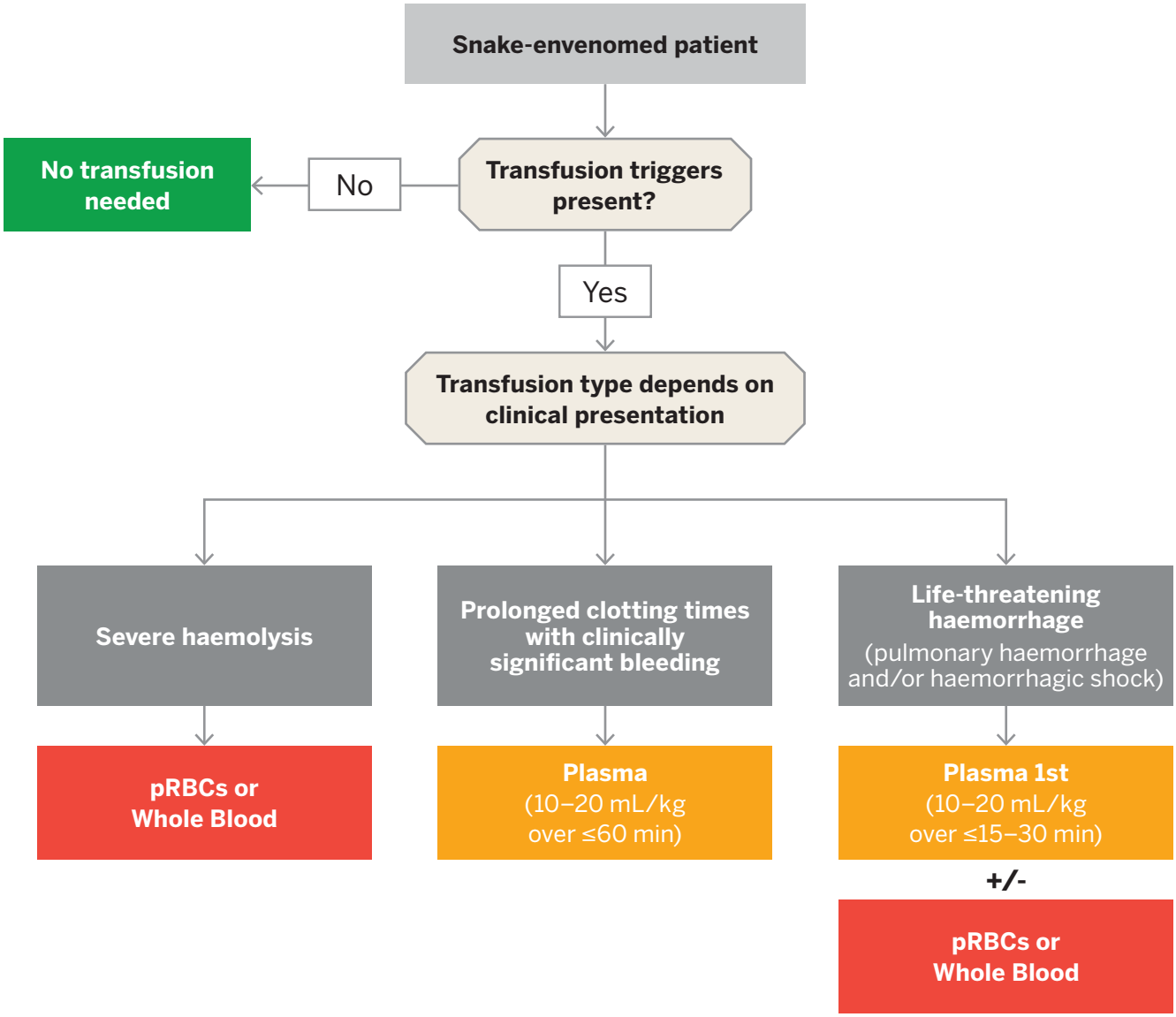


Figure 5. Transfusion decision tree in snake envenomed dogs and cats.




Tiger snake

Administering blood products

Antivenom first, then blood products

Neutralise circulating venom with antivenom before giving plasma or red cells.[86,87]

Plasma transfusion

Plasma is the primary blood product for treatment of clinically significant haemorrhage secondary to VICC, since it replaces consumed clotting factors. Indication and urgency follow the clinical bleeding severity (Table 9, Figure 5), not coagulation times.

Plasma administration: practical steps

Thawed (refrigerated) plasma: this is fresh frozen plasma that has been thawed ahead of time and stored refrigerated so that it is ready to use. Coagulation factor activity remains acceptable for transfusion for 28-35 days of refrigerated storage after thawing.[88,89] Consider keeping a unit of refrigerated plasma ready if you see high VICC caseloads requiring transfusion (especially brown snake species).

Fresh frozen plasma (FFP)/frozen plasma (FP): requires ~35 min thaw in 37°C warm-water bath.[90] Do not microwave.

1. **Dose:** 10–20 mL/kg, rounded up to nearest bag. Minimum one unit unless dog <10 kg
2. Administer through a blood filter
3. Use commercially available plasma or type-matched plasma. No cross-match necessary
4. Give plasma empirically if clinically significant bleeding is evident but coagulation testing is unavailable
5. **Rate:** bolus administration is recommended in patients with pulmonary or life-threatening haemorrhage / haemorrhagic shock (i.e., in ≤15–30 min); otherwise over ≤60 min
6. Monitor for transfusion reactions throughout, especially in the first 15 min
7. Recheck clotting times (ACT or PT/aPTT) after administration
8. Reassess perfusion parameters (HR, RR, MM colour, CRT, pulse quality, mentation) and for evidence of any active bleeding. Repeat plasma if clinically significant bleeding persists or recurs (prolonged coagulation times alone are not an indication)
9. **Endpoints:** cessation of clinical bleeding, perfusion parameters returning towards normal, and improving clotting times

Cats: cat plasma is less readily available; where blood-type-specific (type A or B) cat plasma cannot be sourced, type-matched whole blood is an acceptable emergency alternative. Where cat blood is not available and a cat patient is experiencing life-threatening haemorrhage xenotransfusion of canine plasma or whole blood may be required to prevent exsanguination.

Red blood cell transfusion

Red cell transfusion is indicated for anaemia that impairs oxygen delivery, whether from blood loss, haemolysis, or both. Both can be missed at presentation: haemolytic anaemia may be present early or develop over 24–72 h and progress for up to a week,[24,29,30,32] and clinical bleeding is often occult rather than obvious. Serial monitoring is therefore essential: PCV/TP for falling values, and perfusion parameters (HR, pulse quality, mentation, lactate) for early signs of haemorrhagic shock. Transfuse according to the aforementioned transfusion triggers.

Red cell administration: practical steps

- **Product:** packed red blood cells (pRBC) for haemolytic anaemia; stored whole blood (SWB) or fresh whole blood (FWB) for combined anaemia and coagulopathy, or when components are unavailable
- **Dose:**
  - pRBC: dog 10–20 mL/kg, cat 5–10 mL/kg
  - FWB/SWB: dog 20–25 mL/kg, cat 10–15 mL/kg
- **Compatibility testing – Typing and crossmatching:**
  - Dogs should receive type-matched, or DEA 1-negative, pRBCs or WB if possible. If typing is not possible before a first transfusion, blood can be given. Crossmatching is required before second and subsequent transfusions
  - Cats should receive type-matched pRBCs or WB. Cross-matching is also recommended before all cat RBC containing transfusions to reduce the risk of acute haemolytic transfusion reactions
  - If transfusion is urgent and type-matched/cross-matched blood is unavailable, perform an in-house slide agglutination cross-match first
- **Administration:** through a blood administration filter via a dedicated IV line; do not co-infuse with calcium-containing fluids
- **Rate:** if rapid administration is indicated based on clinical signs of shock, give at bolus rates. Otherwise complete within 4 h
- **Monitor:** HR, RR, temperature and MM every 5 min for the first 30 min, then every 15 min until finished; watch for transfusion reactions (febrile, urticarial, haemolytic, cardiovascular instability).[91] Post-transfusion PCV at 1–2 h; in ongoing RBBS haemolysis recheck PCV q8–24h. Monitor urine output and colour, and renal values
- **Endpoints:** resolution of transfusion triggers with improving perfusion parameters; ideally PCV >20%

Haemorrhagic shock: rapid administration

Rarely, haemorrhagic shock develops, needing rapid plasma and RBC transfusion to restore intravascular volume and oxygen-carrying capacity.

- **Volume:** as per the plasma and RBC doses above; severe haemorrhage may need more
- **Rate:** as rapidly as possible until perfusion parameters improve, then slow to give the remainder over 1–2 h
- **Products:** give both plasma and red cells; refrigerated plasma (no thaw delay) + SWB or pRBCs preferred; FFP/FP + pRBCs, or FWB are also acceptable. Being able to rapidly commence the blood product is more important than selection, with the primary aim to replace clotting factors and RBCs
- **Calcium:** rapid infusion, particularly of plasma products, and volumes >20 mL/kg can lead to hypocalcaemia from citrate in the blood products. Monitor ionised calcium and treat hypocalcaemia with calcium gluconate via a separate line. Do not infuse with blood products because it will cause them to clot on contact[92]
- **Warmth:** warm the patient; warm blood products if possible since severe haemorrhagic shock may result in, and be exacerbated by, hypothermia

Blood products: quick reference

Table 10. Description of different blood product types and their indications in snake envenomation.

Product	Indication in Snake Envenomation
Refrigerated/Thawed Plasma	First choice for haemorrhage; no thaw delay. Haemostatic activity remains acceptable for transfusion for 28–35 days.[88,89]
Fresh Frozen Plasma (FFP)	Second choice; thaw ~35 min at 37°C. Coagulopathy with significant haemorrhage.
Frozen Plasma (FP)	When FFP and refrigerated plasma unavailable. Reduced FV/FVIII activity, but vitamin K-dependent factors retained.
Packed Red Blood Cells (pRBC)	First choice for improving oxygen carrying capacity in haemolytic anaemia. Adjunct in haemorrhagic shock alongside plasma.
Fresh Whole Blood (FWB)	Combined coagulopathy + anaemia; or when component products unavailable. Life-threatening haemorrhage when components not available.
Stored Whole Blood (SWB)	Substitute for FWB when unavailable. Combined anaemia and coagulopathy.

Consider phoning a critical care specialist

For complex or severe envenomation cases, particularly haemorrhagic shock, pulmonary haemorrhage, or continued haemolysis, consider phoning a critical care specialist at a referral hospital for case-specific guidance.

Intravenous fluid therapy

Intravenous fluid therapy is indicated in snake envenomed animals for one or more of the following reasons:

- **Hypovolaemic shock**
- **Rehydration** if dehydrated
- **Maintenance** in animals not eating and drinking
- **Replacement** of abnormal ongoing losses

Initial stabilisation – Assessment of shock

Dogs and cats presenting soon after envenomation show a spectrum of cardiovascular abnormalities and poor perfusion:

- **Assess perfusion immediately on triage:** abnormalities of the six perfusion parameters – mentation, mucous membrane colour, CRT, heart rate, pulse quality and peripheral temperature are used to recognise shock (see Table 11a below and 11b on page 58)
- **Measure blood pressure early:** perfusion parameters can be mixed or appear normal despite severe hypotension
- **Consider neuromuscular paralysis concurrently:** rapid intubation and ventilation may need to occur concurrent with shock treatment. Hypoxaemia (e.g., due to paralysis, pulmonary haemorrhage) causes vasoconstriction, while severe hypercapnia from hypoventilation causes relative vasodilation. Address circulation and respiration together

Table 11a. Changes in the six perfusion parameters that occur with vasoconstrictive and vasodilatory shock.

Parameter	Vasoconstrictive Shock	Vasodilatory Shock
Mentation	Dull to obtunded	Obtunded
Mucous membranes	Pale to white	Injected/red (sometimes pink)
Capillary refill time	Prolonged (>2 s)	Rapid (<1 s) early; prolonged if decompensating
Heart rate	Tachycardia	Tachycardia (relative bradycardia possible)
Pulse quality	Weak, thready	Bounding early, then weak
Peripheral temperature	Cold	Warm (early)

Treatment of shock may form part of initial stabilisation along with early antivenom administration and treatment of significant and life-threatening bleeding.

Figure 6. Initial stabilisation of dogs and cats presenting for snake envenomation.

Snakebite stabilisation: initial approach

Primary survey first; IV access and oxygen early; antivenom without delay.

1

Rapid primary survey (ABC)

- **Airway and breathing:** if respiratory arrest is imminent, intubate and ventilate (IPPV)
- **Circulation:** if the patient is in cardiopulmonary arrest, start CPR

2

Assess for signs of shock

- **Assess the six perfusion parameters:** heart rate, mucous membrane colour, capillary refill time, pulse quality, mentation, peripheral temperature

3

IV access and oxygen

- **Place IV catheter and give flow-by or mask oxygen**
- **Collect baseline bloods from the IV catheter if possible:** PCV/TP, clotting times (± other diagnostics depending on geographical area and preference: CK/biochem, blood gas, blood smear)

4

Give antivenom immediately

- If snakebite is confirmed or highly probable: 1 to 2 vials of tiger-brown snake antivenom
- Type-specific antivenom only if taipan or death adder is confirmed and the specific product is available

5

Assess for clinically significant active bleeding

- **Look for:** soft cough, haemoptysis, obvious external bleeding; abdominal and thoracic bleeding (POCUS)
- **Give blood products if indicated**

6

If perfusion is still poor: IV fluid resuscitation

- **Incremental isotonic crystalloid:** 10 mL/kg bolus of a balanced isotonic crystalloid (e.g., LRS or Hartmann's); reassess perfusion and repeat, up to 40 mL/kg (dogs), 20 mL/kg (cats)
- **Note:** large-volume crystalloid resuscitation is not commonly needed in snake envenomation
- **If more than 20 mL/kg is needed:** reassess for active bleeding and for signs of anaphylaxis

7

Secondary survey

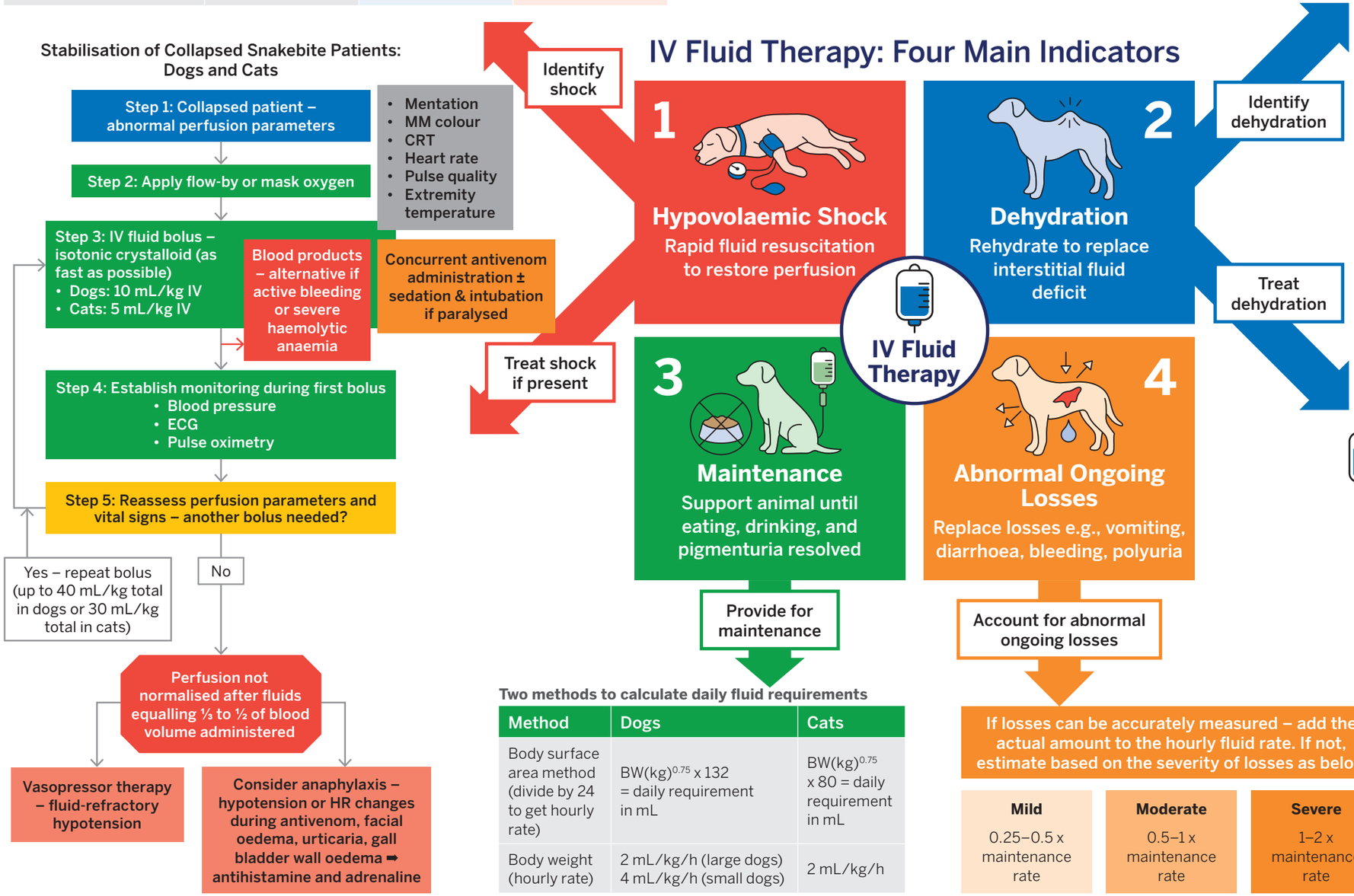
- Perform a thorough, systematic head-to-tail examination
- Reassess perfusion (the six perfusion parameters) and respiration regularly
- Assess for signs of dehydration to inform the ongoing fluid plan

Table 11b. Identifying vasoconstrictive or vasodilatory shock based on physical examination using abnormalities of the six perfusion parameters

Perfusion parameters	Normal	Vasoconstriction	Vasodilation
Mentation	Appropriate	Obtunded	Obtunded
Mucous membrane colour	Pink	Pale-white	Hyperemic
Capillary refill time	1–2 s	>2 s	<1 s
Heart rate (bpm)	60–140 (dog) 120–180 (cat)	>160–180 (dog) >200–240 (cat) <120 (cat)	>160–180 (dog) >200–240 (cat) <120 (cat)
Pulse quality	Good	Weak or thready Not palpable	Bounding or strong
Peripheral temperature	Normal	Cool to cold	Warm

Identifying dehydration, and estimating severity, based on physical examination. (Table taken from Canine Parvovirus Guidelines)

Physical examination Hydration parameters	Estimated severity of dehydration, as % of body weight		
	5-6%	7-9%	10-15%
Skin tent	Subtle loss of skin elasticity	Delayed	Stays tented for >2 seconds
Mucous membranes	Tacky	Tacky to dry	Dry
Tear film	Normal to reduced	Reduced	Dry
Sunken eyes?	No	Possibly	Yes
Abnormal perfusion parameters?	No	Possibly	Yes



Two methods to calculate daily fluid requirements

Method	Dogs	Cats
Body surface area method (divide by 24 to get hourly rate)	$BW(kg)^{0.75}$ x 132 = daily requirement in mL	$BW(kg)^{0.75}$ x 80 = daily requirement in mL
Body weight (hourly rate)	2 mL/kg/h (large dogs) 4 mL/kg/h (small dogs)	2 mL/kg/h

If losses can be accurately measured – add the actual amount to the hourly fluid rate. If not, estimate based on the severity of losses as below

Mild	Moderate	Severe
0.25–0.5 x maintenance rate	0.5–1 x maintenance rate	1–2 x maintenance rate

Factors that can confound physical examination assessment of perfusion and hydration parameters

Parameter	Confounding factors
Mucous membrane moistness	Panting, tachypnoea, dyspnoea, open mouth breathing can result in dry mm despite normal hydration Nausea, ptyalism, vomiting can result in moist mm despite dehydration
Mucous membrane colour and capillary refill time	Anaemia or vasoconstriction caused by pain or anxiety can make the mm appear pale in the absence of shock, and make CRT difficult to assess
Skin tent/turgor	Young or obese animals may have normal skin turgor despite dehydration Geriatric or cachexic animals may have increased skin tent despite normal hydration

% Dehydrated x Body Weight (kg) = Fluid Deficit (L)

e.g., 10 kg dog, 7% dehydrated = 0.7 mL (i.e., 700 mL) deficit

Calculate deficit ➡ Determine time-frame

Replace Quickly (6–12 h)	Replace More Slowly (>12–24 h)
• Rapid onset dehydration • Minimal risk of fluid overload (normal heart and kidney function)	• Risk of fluid overload • Cats

Reassess hydration parameters

Assessment and maintenance of hydration

- **May present late and dehydrated:** tacky to dry mucous membranes, reduced skin turgor; from gastrointestinal losses (vomiting, diarrhoea), reduced drinking, and sometimes active bleeding
- **Rehydrate with isotonic balanced crystalloids** (e.g., LRS / Hartmann's, Plasma-Lyte 148); replace the estimated deficit over 8–12 h (adjust for chronic kidney or cardiac disease); account for ongoing losses
- **Monitor urine output**, especially with pigmenturia from haemolysis or myolysis.

If AKI develops (rising creatinine, oliguria): give fluids cautiously to avoid fluid overload.

Pigmenturia: fluid therapy

- **Give IV fluids until urine is grossly clear** to reduce contact time between haeme-containing proteins and the renal tubules
- **Maintain normal hydration and urine output >2 mL/kg/h** until the patient is eating and drinking. Fluid rates greater than needed to provide for maintenance and abnormal ongoing losses are not necessary
- **CK elevation alone (without pigmenturia)** should not guide fluid therapy duration. Persistently elevated CK should not preclude discharge if the patient is eating, drinking and the urine is grossly non-pigmented

Analgesia

Pain may arise from the bite site, generalised myonecrosis, or visceral (gastrointestinal) effects. Black snakes (e.g., RBBS) cause significant local oedema, bruising and pain.

Tetraparesis and prolonged recumbency can themselves cause muscle and joint pain, independent of myopathy; large-breed and older arthritic dogs are particularly affected.

Treat pain in every symptomatic patient

- **Treat pain empirically** for envenomation syndromes associated with myalgia (tiger and black snakes)
- **Provide analgesia where pain is likely** – pain is hard to detect; err towards treating, for welfare
- **Antivenom treatment** often rapidly improves pain by halting ongoing venom-mediated tissue injury[93]
- **Assess frequently and titrate** analgesia using pain scores
- **Continue analgesia after discharge** if pain persists

Opioids: first line

Prioritise opioids since they provide effective analgesia with minimal cardiovascular effects.

- **Pure μ-agonists (methadone, fentanyl CRI):** first line for analgesia; suit hypotensive and critically ill patients
  - Methadone (e.g., 0.1–0.3 mg/kg IV q4–6h)
  - Fentanyl CRI: rapid onset and easily titrated
- **Buprenorphine (partial μ-agonist):** longer dosing interval, convenient; reassess adequacy frequently
- **Butorphanol (agonist–antagonist):** provides more sedation than analgesia, so may be useful in circumstances such as the anxious patient at risk of upper-airway obstruction

Paracetamol: adjunct (dogs only)

- **Adjunct in dogs**, IV or oral; cost-effective and easy, especially beyond discharge, although has not been evaluated in snake envenomation and is variable in existing canine studies
- **Possible renal benefit** via prostaglandin inhibition in rhabdomyolysis (laboratory studies only)

NEVER give paracetamol to cats: they are exquisitely sensitive to hepatotoxicity, methaemoglobinaemia and Heinz-body haemolysis

Avoid NSAIDs and glucocorticoids

- **Avoid NSAIDs in the acute phase** since they can exacerbate the VICC bleeding risk, the risk of AKI from myoglobinuria, haemoglobinuria, or hypovolaemia, and the risk of gastric ulceration
- **Post-discharge:** continue to avoid NSAIDs, especially in patients that had rhabdomyolysis or delayed haemolysis (risk of AKI); paracetamol (in dogs only) is usually sufficient
- **Avoid glucocorticoids**

Wound complications and management

Most bites show no wound or only a small puncture. Black snakes, and other species with a cytotoxic venom component, can cause marked local reactions: bruising, oedema and pain at the bite site.

- **Most local reactions:** manage with analgesia alone; consider a cold compress if tolerated
- **Face/mouth oedema affecting eating:** analgesia and soft food; feeding tube rarely needed
- **Head/neck oedema risking airway (esp. brachycephalics):** monitor closely; adjunctive sedation may be required
- **Brachycephalics with stress and upper-airway swelling/obstruction:** a one-off dexamethasone injection may be considered

Antibiotics are rarely needed

- **Small skin necrosis** (darkened skin at the puncture) is common after black snake bites and rarely sloughs
- **Reserve antibiotics for compromised skin integrity** (sloughing, purulent discharge): likely gram-positive aerobes; empirical 1st/2nd-gen cephalosporin (cephalexin, cefazolin) or beta-lactam (amoxicillin ± clavulanate), pending culture and susceptibility
- **Large slough or abscess with systemic signs** (firm, warm, painful swelling ± fever): consider surgical debridement

Supportive care for gastrointestinal signs

- Vomiting is a common early sign, so antiemetics are appropriate
- Maropitant first line (registered for acute vomiting in dogs and cats)
- Add ondansetron or metoclopramide only if vomiting or nausea is ongoing; multimodal antiemesis is rarely needed
- Regurgitation, gastroparesis, ileus: infrequent after elapid envenomation; if present, give a prokinetic (metoclopramide, erythromycin)
- If ileus persists: review contributory opioids (e.g., fentanyl) alongside prokinetics

Table 12. Commonly used antiemetics and prokinetics in dogs and cats

Drug	Recommended Dosing
Antiemetics	
Maropitant	1 mg/kg IV (or SC) q24h, or 2 mg/kg PO q24h
Ondansetron	0.5 mg/kg IV q8h
Metoclopramide	0.5 mg/kg IV q6–8h, or 2 mg/kg/day CRI
Prokinetics	
Metoclopramide	0.5 mg/kg IV q6–8h, or 2 mg/kg/day CRI
Erythromycin	1–3 mg/kg IV q8h

Treatments that are not recommended

Not recommended

- **Vitamin C and Vitamin K:** no evidence of benefit in confirmed snakebite
- **Prophylactic antibiotics:** not indicated; case-by-case only for infection at a site of skin necrosis after black snake bite

Recumbent patient care

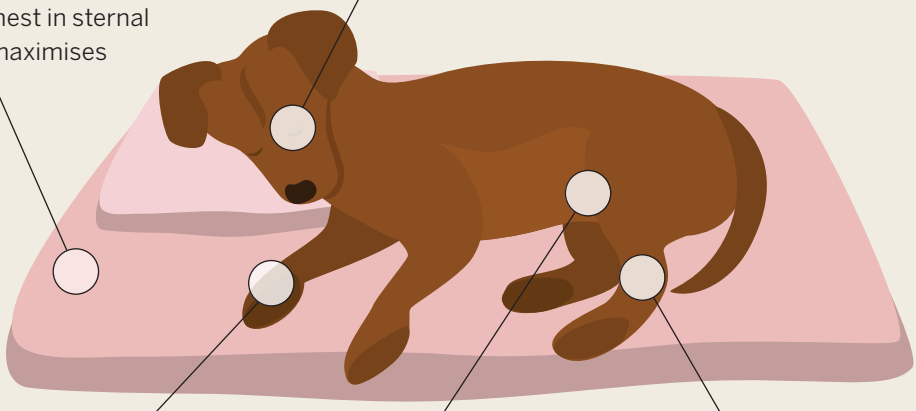
Recumbent patient care is required for paralysed patients until they regain the ability to ambulate, including during mechanical ventilation.[80]

Eye care (if unable to blink)

- Lubrication:
  - Cellulose-containing drops (e.g., Ocunovis®, Hylo-Forte®, Viscotears® or Celluvisc®) every 1-4 h
  - Lubricating eye ointment containing paraffin (e.g., VitA-POS®, Poly Visc®), every 2-4 h
- Corneal examination ± fluorescein staining at least once daily
- Add appropriate antibiotic topically if corneal ulceration

Soft, padded bedding

- Reposition slightly every 4-6 h
- Either sternal recumbence or rotate between lazy lateral and sternal
- Keeping the chest in sternal recumbence maximises oxygenation



Apply physiotherapy principles of recumbent patients

- Passive range of motion
- Gentle massage if not contraindicated

Bladder care

- If extended period of recumbence expected place a Foley urinary catheter with closed collection system
- Gentle bladder expression every 4-6 h if indicated

IV catheter care

- Assess at least once daily
- Monitor for signs of phlebitis, infection

Prognosis

Prognosis for survival to discharge for dogs and cats after snake envenomation is good when treated with antivenom. 92% survival has been reported in dogs in two large studies.[1,13] The same studies reported survival in 95% of cats Australia-wide,[1] and 77% of cats treated for eastern brown snake envenomation in south-east QLD.[13]

Criteria for discharge

Criteria for discharge for an envenomed dog or cat are similar to those for most other hospitalised patients. Ideally, they are ambulatory, eating and drinking, and able to take oral medications if oral medications are required. While not imperative to recheck, particularly if resource constraints are present, a veterinarian may want to see normalisation or improvement in clotting times, and resolution of pigmenturia, prior to discharge if present as part of the envenomation syndrome.

Discharge instructions

Discharge instructions should be provided to guide owners regarding home care. Templates can be used, although individualisation, particularly for recheck requirements is recommended.

Recommendations following discharge include:

- Rest for a minimum of 5–7 days
  - Dogs should be strictly rested with leash walks only – no running, jumping, or exuberant play
  - Indoor/outdoor or outdoor cats would ideally be kept indoors for 7 days, to allow owner observation, and given their increased risk of trauma during recovery
  - Significant myotoxicity: extend strict rest, up to ~4 weeks, as movement may worsen myonecrosis[35]
- Awareness of specific risks in the recovery period:
  - Risk of recurrence of neuromuscular weakness can occur after discharge, especially with overexertion or fatigue
  - Risk of haemorrhage if clotting times have not normalised by discharge (or are not known)
  - Risk of progression of anaemia in haemolytic syndromes
  - Risk of progression/development of AKI if pigmenturia or AKI present during hospitalisation. Veterinarians should ensure that patients with pigmenturia or AKI during hospitalisation continue to drink after discharge and that nephrotoxic drugs are avoided
  - Risk of serum sickness secondary to antivenom – while rare, it is important for owners to be aware of. Serum sickness can manifest as skin rash, pruritus, joint swelling, pain, and fever. The time frame for development of clinical signs is usually 5–14 days after antivenom
- Recheck of blood tests may be recommended if blood tests were abnormal during hospitalisation, especially PCV/RBC parameters in those with haemolytic syndromes, and creatinine/kidney parameters in those with AKI

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