

A Clinician's Guide to Australian Venomous Bites and Stings

Incorporating the updated CSL Antivenom Handbook



Principal author
Associate Professor Julian White

The opinions expressed are not necessarily those of bioCSL Pty Ltd. Before administering or prescribing any prescription product mentioned in this publication please refer to the relevant Product Information. Product Information leaflets for bioCSL's antivenoms are available at www.biocsl.com.au/PI.

This handbook is not for sale.

Date of preparation: February 2013.

National Library of Australia Cataloguing-in-Publication entry.

Author: White, Julian.
Title: A clinician's guide to Australian venomous bites and stings: incorporating the updated CSL antivenom handbook / Professor Julian White.
ISBN: 9780646579986 (pbk.)
Subjects: Bites and stings – Australia.
Antivenins.
Bites and stings – Treatment – Australia.

Other Authors/
Contributors: CSL Limited (Australia).
Dewey Number: 615.942

Medical writing and project management services for this handbook provided by Dr Nalini Swaminathan, Nalini Ink Pty Ltd; +61 416 560 258; nalini@iinet.net.au. Design by Spaghetti Arts; +61 410 357 140; spaghettiararts@gmail.com.

© Copyright 2013 bioCSL Pty Ltd, ABN 26 160 735 035; 63 Poplar Road, Parkville, Victoria 3052 Australia. bioCSL is a trademark of CSL Limited. bioCSL understands that clinicians may need to reproduce forms and flowcharts in this handbook for the clinical management of cases of envenoming and permits such reproduction for these purposes. However, except for the purpose of clinical management of cases of envenoming from bites/stings from Australian venomous fauna, no part of this publication may be reproduced by any process in any language without the prior written consent of bioCSL Pty Ltd.

Where applicable, copyright of photos, illustrations and flowcharts included in this handbook that have been reproduced with permission, is attributed to the appropriate person/authority. Maps regarding the distribution of various venomous fauna in Australia obtained courtesy A/Prof Julian White. Cover photos: Box jellyfish photo copyright Dr Lisa-ann Gershwin, Australian Marine Stinger Advisory Services; all other cover photos copyright bioCSL Pty Ltd.

Editorial content: Although great care has been taken in compiling and checking the information provided in this publication to ensure that it is accurate, bioCSL Pty Ltd shall not be responsible for the continued currency of the information, or for any errors, omissions or inaccuracies in this publication.

Before using this handbook

This handbook is sponsored as a service to the healthcare profession by bioCSL Pty Ltd (bioCSL), and is provided for educational purposes only.

Associate Professor Julian White is the principal author of this handbook. The contents represent a compilation of A/Prof White's clinical experience and that of his peers alongside research evidence (published and unpublished, including discussions with colleagues at scientific meetings). Any clinical information and recommendations in this handbook are relevant to Australian venomous fauna only and **should not** be applied to envenoming by non-Australian organisms. Specifically, although the venomous fauna in Papua New Guinea are similar to that of Australia, there are important differences, particularly with regards to snake fauna.

Please note: The purpose of this handbook is to educate and assist clinicians in the emergency management of cases of envenoming and in the appropriate use of antivenom therapy when clinically indicated. The primary intention is to guide the clinician towards current understanding of clinical best practice. The handbook does not purport to be an exhaustive review of the literature. Unreferenced material can be attributed to A/Prof Julian White personal communication.

Envenoming from venomous bites/stings can be life threatening and represents a potential medical emergency. When managing patients with significant systemic envenoming, always seek expert advice. Access to expert advice is available from relevant contacts listed on page 3 of this handbook.

Healthcare professionals should be made aware that the contents of this handbook are based on published evidence, as well as clinical and research experience of the authors. Sometimes the instructions from the authors for use are different from the instructions described in the current respective Antivenom Product Information (PI). bioCSL does not support and is not responsible for the use of its antivenom products outside of that specified in the PI.

Contact Information

| | |
|---|-----------------------|
| Emergency (Police/Ambulance/Fire Services) | 000 |
| Women and Children's Hospital, Adelaide (ask to speak to the Duty Clinical Toxinologist) | (08) 8161 7000 |
| bioCSL Medical Information Department | 1 800 642 865 |
| National Poisons Information Centre | 13 11 26 |
| Australian Venom Research Unit | 1 300 760 451 |

Contents

| | |
|---|----|
| How to use this handbook in an emergency..... | 6 |
| Acknowledgments..... | 8 |
| Foreword..... | 11 |

Section 1

Management of venomous bites and stings: General information

| | |
|---|----|
| Management of venomous bites and stings..... | 15 |
| Envenoming: | |
| Envenoming overview..... | 20 |
| Diagnosis of systemic envenoming..... | 24 |
| Management of envenoming with antivenoms..... | 26 |

Section 2

First aid

| | |
|---|----|
| Basic (critical care) first aid..... | 31 |
| Basic first aid: DRS ABCD..... | 32 |
| Specific first aid for venomous bites and stings: | |
| Appropriate first aid for bites/stings..... | 37 |
| Pressure Bandaging & Immobilisation (PBI)..... | 38 |
| Vinegar..... | 44 |
| Hot water..... | 47 |

Section 3

Snakes

| | |
|-----------------------------------|-----|
| Snakebite: | |
| Clinical presentation..... | 51 |
| First aid..... | 56 |
| Diagnosis..... | 58 |
| Treating snakebite: | |
| Fundamentals..... | 97 |
| Urgent treatment..... | 99 |
| Snakebite management chart 1..... | 102 |
| Snakebite management chart 2..... | 104 |
| Snakebite management chart 3..... | 106 |

| | |
|-----------------------------------|-----|
| Snakebite: General treatment..... | 107 |
|-----------------------------------|-----|

Specific treatment of snakebite:

| | |
|---|-----|
| Antivenom | |
| Antivenom therapy for snakebite..... | 111 |
| Indications, timing, choice of antivenom, dosing, complications, etc..... | 112 |

| | |
|---|-----|
| Administering I.V. antivenom (flowchart)..... | 134 |
|---|-----|

| | |
|--|-----|
| Treating snakebite: Managing venom-induced toxicity..... | 135 |
|--|-----|

Australian venomous snakes

| | |
|------------------------|-----|
| overview: | |
| Brown snake group..... | 143 |
| Tiger snake group..... | 149 |
| Black snake group..... | 156 |
| Death adder group..... | 160 |
| Taipans..... | 163 |
| Sea snakes..... | 166 |

| | |
|---------------------------|-----|
| Snake identification..... | 168 |
|---------------------------|-----|

Section 4

Spiders

| | |
|----------------------------|-----|
| Spiderbite: | |
| Background..... | 177 |
| Clinical presentation..... | 178 |
| First aid..... | 184 |
| Diagnosis..... | 186 |

| | |
|--|-----|
| Spiderbite decision tree based on clinical presentation (flowchart)..... | 192 |
|--|-----|

| | |
|----------------------------|-----|
| Spider identification..... | 193 |
|----------------------------|-----|

| | |
|---|-----|
| Management of envenoming due to spiderbite: | |
| Basic principles..... | 195 |
| Urgent treatment..... | 196 |

| | |
|--|-----|
| Management of suspected or confirmed funnel web spider bite (flowchart)..... | 197 |
|--|-----|

| | |
|---|-----|
| Management of suspected or confirmed red back spider bite (flowchart) | 198 |
| Specific treatment for funnel web spider bite: Antivenom | 199 |
| Specific treatment for red back spider bite: Antivenom | 203 |
| Spider antivenoms: Preparation, administration and complications | 207 |
| Necrotic arachnidism | 217 |
| Necrotic skin lesions: Approach to investigations (flowchart) | 220 |
| Australian spiders overview: | |
| Funnel web spider group | 221 |
| Red back spiders | 223 |
| Mouse spiders | 226 |
| Other spiders | 227 |

Section 5

Other arthropods

| | |
|---|-----|
| Insects, centipedes and scorpions | 233 |
| Paralysis ticks | 235 |

Section 6

Jellyfish

| | |
|--|-----|
| Jellyfish stings: Overview | 243 |
| Jellyfish triage algorithm (flowchart) | 244 |

| | |
|------------------------------------|-----|
| Box jellyfish stings: | |
| Overview & clinical issues | 245 |
| Clinical presentation | 246 |
| First aid & diagnosis | 248 |
| Urgent treatment | 250 |
| Management chart (flowchart) | 253 |
| Antivenom therapy | 254 |

| | |
|--|-----|
| Irukandji jellyfish and Irukandji syndrome | 264 |
|--|-----|

| | |
|-----------------------------|-----|
| Irukandji stings: | |
| Clinical presentation | 266 |
| First aid & diagnosis | 267 |

| | |
|---|-----|
| Management of Irukandji syndrome: | |
| Urgent management | 268 |
| General management: analgesia; hypertension; & other issues | 269 |

| | |
|--|-----|
| Irukandji syndrome management guidelines (flowchart) | 273 |
|--|-----|

| | |
|------------------------------|-----|
| Other jellyfish stings | 274 |
|------------------------------|-----|

Section 7

Stinging fish

| | |
|--|-----|
| Stinging fish: | |
| Background & clinical presentation | 281 |
| First aid & diagnosis | 282 |

| | |
|--|-----|
| Fish stings: General treatment | 283 |
| Specific treatment for stonefish stings: Stonefish antivenom | 284 |
| Stonefish: Overview | 295 |

Section 8

Other marine animals

| | |
|---|-----|
| Stingray stings | 299 |
| Blue ringed octopus, <i>Hapalochlaena</i> spp | 301 |
| Cone snails, <i>Conus</i> spp | 302 |

Section 9

Antivenoms: Practical information

| | |
|--|-----|
| bioCSL's antivenoms: | |
| Specificity | 305 |
| Vial presentations | 306 |
| bioCSL's antivenoms: Initial dose | |
| Snake antivenoms | 307 |
| Spider antivenoms | 312 |
| Marine antivenoms | 313 |
| Stocking antivenoms in hospitals | 314 |

References

| | |
|------------------|-----|
| References | 318 |
|------------------|-----|

How to use this handbook in an emergency

Patient has symptoms/signs of envenoming of unknown aetiology

| | |
|---|----|
| If injury (bite/sting) occurred: | |
| On land | 16 |
| In a marine environment..... | 17 |
| In a freshwater environment..... | 18 |
| Broad/indicative diagnosis based on evenoming signs/symptoms..... | 22 |

Patient requires urgent first aid

| | |
|---|----|
| DRS ABCD | 32 |
| CPR summary | 36 |
| Appropriate first aid for venomous bites/stings | 37 |
| Pressure Bandaging & Immobilisation first aid: | |
| PBI first aid for bites to the lower limb | 39 |
| PBI first aid for bites to the upper limb..... | 41 |
| Vinegar: | |
| When to use..... | 44 |
| How to apply vinegar | 46 |
| Hot water: | |
| When to use..... | 47 |
| How to apply hot water..... | 48 |

Patient presents with suspected or confirmed snakebite

| | |
|---|-----|
| First steps: | |
| Go to Snakebite management chart 1 | 102 |
| Urgent treatment | 99 |
| Clinical presentation | 51 |
| First aid..... | 56 |
| Key early signs of envenoming relating to the type of snake | 64 |
| Is there evidence of envenoming? | |
| History and examination..... | 60 |
| Laboratory investigations..... | 67 |
| Interpreting laboratory test results..... | 72 |

| | |
|--|-----|
| Diagnosis if there is evidence of envenoming: | |
| Determining the most likely type of snake involved based on clinical findings: | |
| Local effects of bite (flowchart) | 94 |
| Systemic effects of bite (flowchart) | 95 |
| bioCSL's Snake Venom Detection Kit | 76 |
| Snake distribution/geography..... | 143 |

| | |
|--|-----|
| Management: If the patient has evidence of systemic envenoming | |
| Go to Snakebite management chart 2 | 104 |
| General treatment of snakebite..... | 107 |
| Antivenom therapy for snakebite..... | 111 |
| If antivenom is not immediately available | 115 |
| What to do if there is an adverse reaction to antivenom..... | 130 |
| Administering I.V. antivenom (flowchart)..... | 134 |
| Managing venom-induced toxicity..... | 135 |

| | |
|--|-----|
| Management: If the patient does not have evidence of systemic envenoming | |
| Go to Snakebite management chart 3 | 106 |
| Laboratory investigations..... | 67 |
| General treatment..... | 107 |

Patient presents with suspected or confirmed spiderbite

| | |
|--|-----|
| Clinical presentation: funnel web spider bite..... | 182 |
| Clinical presentation: red back spider bite..... | 183 |
| History | 188 |
| Examination..... | 189 |

| | |
|--|-----|
| Differential Diagnosis: | |
| Key symptoms/signs of envenoming based on spider species | 190 |
| Spiderbite decision tree based on clinical presentation (flowchart)..... | 192 |
| If the patient is bitten by a funnel web spider/big black spider: | |
| Urgent treatment | 196 |
| Management of suspected/confirmed funnel web spider/big black spider bite (flowchart)..... | 197 |
| If the patient is bitten by a red back spider: | |
| Management of suspected/confirmed red back spider bite (flowchart)..... | 198 |
| Antivenom therapy: | |
| Antivenom therapy for funnel web spider bite..... | 199 |
| Antivenom therapy for red back spider bite | 203 |
| Spider antivenoms: preparation, administration and complications | 207 |

Patient presents following jellyfish sting

| | |
|---|-----|
| Jellyfish triage algorithm (flowchart)..... | 244 |
|---|-----|

Patient with severe box jellyfish sting

| | |
|---|-----|
| Clinical presentation | 246 |
| First aid (vinegar) if none has been applied..... | 248 |
| Urgent treatment..... | 250 |
| Box jellyfish stings: Management chart (flowchart)..... | 253 |
| Antivenom therapy for box jellyfish stings..... | 254 |

Irukandji syndrome

| | |
|---|-----|
| Clinical presentation | 266 |
| First aid..... | 267 |
| Diagnosis..... | 267 |
| Management: | |
| Urgent management | 268 |
| Irukandji syndrome management guidelines (flowchart)..... | 273 |
| General management of Irukandji syndrome: | |
| Analgesia | 269 |
| Hypertension..... | 271 |
| Other issues | 272 |

Patient presents with fish sting

| | |
|---|-----|
| Clinical presentation | 281 |
| First aid..... | 282 |
| Diagnosis..... | 282 |
| General treatment | 283 |
| Administering bioCSL's Stonefish Antivenom..... | 284 |

Patient stung by stingray, blue ringed octopus or cone snail

| | |
|--------------------------|-----|
| Stingray | 300 |
| Blue ringed octopus..... | 301 |
| Cone snail..... | 302 |

Quick reference: Initial dose of bioCSL's antivenoms

| | |
|-------------------------|-----|
| Snake antivenoms..... | 307 |
| Spider antivenoms | 312 |
| Marine antivenoms | 313 |

Acknowledgments

Handbook contents

The contents of this handbook have been developed by Associate Professor Julian White.

Associate Professor Julian White, Head of Toxinology, Women's and Children's Hospital, Adelaide

Julian White was born in the UK. He undertook medical training at the University of Adelaide and has had a strong association with clinical toxinology and venomous fauna – the latter extending over 40 years. Since treating his first snakebite case in 1976, A/Prof White has been involved in managing thousands of cases of bites/stings.

In 1988, A/Prof White was awarded an MD for his clinical toxinology studies and in 1990 he was appointed inaugural Head of Toxinology at the Adelaide Children's Hospital (now Women's and Children's Hospital) – a position he still holds.

Author of numerous peer-reviewed publications, as well as chapters and books on toxinology, A/Prof White wrote the first CSL Antivenom Handbook soon after his appointment as Clinical Consultant to CSL in 1994. The handbook gained wide usage in the Australian medical community for the emergency management of cases of envenoming and as a training tool for graduate doctors, hospital emergency departments and medical students. This in turn has led to the decision to produce this new and fully updated version, now entitled A Clinician's Guide to Australian Venomous Bites and Stings, which incorporates advances in the field from an additional decade and half of research and the clinical experience of A/Prof White and his colleagues.

A/Prof White is closely involved with the Australian Snakebite Project (ASP), a study of cases of snakebite from around Australia. Additionally, he has co-founded (with Dr John Williamson) the Clinical Toxinology Short Course. Held regularly in Adelaide since 1997, this remains the only complete international-level course in this field.

Currently, A/Prof White is Secretary/Treasurer of the International Society on Toxinology. He is also one of the founding members of the Global Snakebite Initiative. He has been instrumental in developing snakebite guidelines for NSW Health, SA Health and for Australian medical missions to East Timor, the Solomon Islands, Aceh, Nias, and most recently, Pakistan.

bioCSL thanks A/Prof White for all his efforts in helping bring this handbook to fruition through sharing his clinical knowledge and expertise, the exhaustive consideration of reviewers' comments, and for developing the numerous practical flowcharts/algorithms relating to the diagnosis and management of cases of envenoming, which form an integral part of this new handbook.

Review

A panel of experts have reviewed the handbook's contents.

Dr Mark Little, Staff Specialist, Emergency Department, Cairns Base Hospital

Dr Little is an Emergency Medicine physician with extensive experience in the management of cases of envenoming from snakebite and marine stings. He currently holds the position of Staff Specialist and Clinical Toxicologist at Cairns Base Hospital. Dr Little is the author of numerous peer-reviewed publications in the field of toxicology. He regularly teaches at the Clinical Toxinology Short Course held in Adelaide, and most recently co-authored the first Australian Toxicology Handbook.

bioCSL extends a special thanks to Dr Little for his invaluable contribution during all stages of development of this handbook – especially for providing the much-needed ED physician's perspective and for the numerous insightful and practical suggestions to help improve the content and management algorithms, which have greatly enhanced the handbook's useability.

Associate Professor Jamie Seymour, Director, Tropical Australian Stinger Research Unit, James Cook University, Cairns campus

Associate Professor Jamie Seymour has been conducting research on venoms and venomous creatures for over 20 years. Based in Cairns, he is well placed to study the ecology and biology of Australia's venomous fauna. As Director of the Tropical Australian Stinger Research Unit (TASRU), which he established, he leads a world-renowned research group studying the ecology and biology of box jellyfish and medical treatment of box jellyfish envenoming. His research on jellyfish venom and envenoming has had a direct impact on and led to refinements in the treatment protocol for Australian jellyfish stings.

.....acknowledgments continued overleaf

Acknowledgments

A/Prof Seymour has been involved in programs in many parts of the world to help reduce jellyfish envenoming of human beings – including Australia, East Timor, Thailand and Hawaii.

bioCSL thanks A/Prof Seymour foremost for his review of and assistance with sections of this handbook relating to marine and freshwater venomous creatures, and for his valued feedback on other sections including snakebite and spiderbite. Thanks also to A/Prof Seymour for the provision of a number of photos used in this handbook.

Dr Ken Winkel, Director, Australian Venom Research Unit

Dr Winkel is a Past President of The Australasian College of Tropical Medicine. His unit is involved in envenoming research in the Asia-Pacific. In July 2007 he was awarded the Medal for Outstanding Contribution to Tropical Medicine from the Australasian College of Tropical Medicine.

bioCSL thanks Dr Winkel for providing his tropical medicine viewpoint, his kind efforts and assistance with sourcing photos, the assiduous and thorough review of the contents and for providing detailed feedback regarding all aspects of this handbook.

Dr Peter Rischbieth, GP, Murray Bridge, SA

Dr Rischbieth is a GP Partner at Bridge Clinic, Murray Bridge, SA, and Past President (2007-2008) of the Rural Doctors Association of Australia. Currently, he is Vice President – Industrial at the Rural Doctors' Association of South Australia and Principal Medical Officer at Murray Bridge Soldiers Memorial Hospital.

bioCSL is grateful to Dr Rischbieth for his review of this handbook from the valued perspective of the rural/regional GP.

Foreword



I am pleased to present to you A Clinician's Guide to Australian Venomous Bites and Stings, incorporating the updated CSL Antivenom Handbook.

Australia is home to some of the world's most unique and deadliest fauna and bioCSL is proud of its 90 year history of serving the Australian community and heritage of supplying important life-saving products for the treatment of envenoming from snakes, spiders and marine creatures.

Our involvement in the management of venomous bites and stings dates back to 1930 when we produced the first Australian antivenom. Through a long-standing partnership with the Australian Government, we continue to supply these unique and complex medicines in the national interest and as part of our responsibility to the community.

As part of our commitment to improving the clinical management of snakebite, bioCSL actively supports clinical toxicology training and first aid education through a number of initiatives, both globally and regionally, including publication of the original CSL Antivenom Handbook in 1994.

bioCSL is proud to further the education of clinicians through this updated, comprehensive and practical handbook re-authored by Associate Professor Julian White and reviewed by a panel of expert peers. This guide presents current best practice in the management of envenoming, in what is a continually evolving and clinically challenging field. I trust you will find it useful in your endeavours to provide the best possible treatment to Australian patients.

Dr John Anderson

Senior Vice President and General Manager
bioCSL Pty Ltd

Abbreviations

| | |
|------------------------|--|
| ABC | Airway, Breathing, Circulation |
| aPTT | activated Partial Thromboplastin Time |
| AV | Antivenom |
| CK | Creatine kinase (= creatine phosphokinase) |
| CPR | Cardiopulmonary Resuscitation |
| DRS ABCD | Danger, Response, Send for help, Airway, Breathing, Compressions/Circulation, Defibrillation |
| FBC | Full Blood Count |
| FDP | Fibrin(ogen) Degradation Products |
| FFP | Fresh Frozen Plasma |
| GCS | Glasgow Coma Scale |
| GTN | Glyceryl trinitrate |
| HUS | Haemolytic-uremic syndrome |
| IM | Intramuscular |
| INR | International Normalised Ratio (measure of prothrombin time) |
| IPPV | Intermittent Positive Pressure Ventilation |
| IV | Intravenous |
| MAHA | Microangiopathic Haemolytic Anaemia |
| NMJ | Neuromuscular Junction |
| PBI | Pressure Bandaging/Bandage & Immobilisation |
| PIC | Poisons Information Centre |
| PEEP | Positive End-Expiratory Pressure |
| PNG | Papua New Guinea |
| SpO₂ | Oxygen saturation measured by pulse oximetry |
| SVDK | Snake Venom Detection Kit |
| TTP | Thrombotic thrombocytopenic purpura |
| UEC | Urea, electrolytes, creatinine |
| VICC | Venom Induced Consumption Coagulopathy |
| WBCT | Whole Blood Clotting Time |

Section 1

Management of venomous bites and stings: General information

In this section

Management of venomous bites and stings

| | |
|---|----|
| Bites and stings in Australia..... | 15 |
| Injury (bite/sting) occurring on land | 16 |
| Injury (bite/sting) occurring in a marine environment..... | 17 |
| Injury (bite/sting) occurring in a freshwater environment | 18 |
| Managing bites or stings: Basic principles..... | 19 |

Envenoming overview

| | |
|---|----|
| What is envenoming? | 20 |
| Diagnosis | 20 |
| Signs and symptoms of significant local and systemic envenoming | 21 |
| Tetanus prophylaxis..... | 21 |
| Venom actions and associated clinical features | 22 |

Diagnosis of systemic envenoming

| | |
|---------------------------------|----|
| History | 24 |
| Examination..... | 24 |
| Laboratory investigations | 25 |
| Snake venom detection..... | 25 |

Management of envenoming with antivenoms

| | |
|---|----|
| Antivenoms: General principles..... | 26 |
| Antivenom specificity | 26 |
| When to use antivenom..... | 27 |
| Dosing | 27 |
| Adverse reactions to antivenom | 28 |
| Common mistakes during the diagnosis and management of envenoming | 28 |

Management of venomous bites and stings

Bites and stings in Australia¹

Bites and stings are a relatively common problem in Australia – the consequences of which can range from being minor, to death from systemic envenoming or anaphylaxis.

Identification of the culprit animal may be straightforward, but often it is quite obscure and challenging for the clinician. Determining the nature of the patient's problems and the likely cause are vital steps in ensuring an optimal outcome.

Some general principles must be applied to evaluate a patient for a possible bite or sting and for determining whether significant envenoming has occurred. The flowcharts on pages 16-18 relate to symptomatic patients and broadly assist in reaching a diagnosis, using history, examination, and laboratory tests (where appropriate).¹ If the patient is asymptomatic, a history, examination, and probably laboratory investigations may still be required to establish the identity of the likely biting/stinging organism.¹

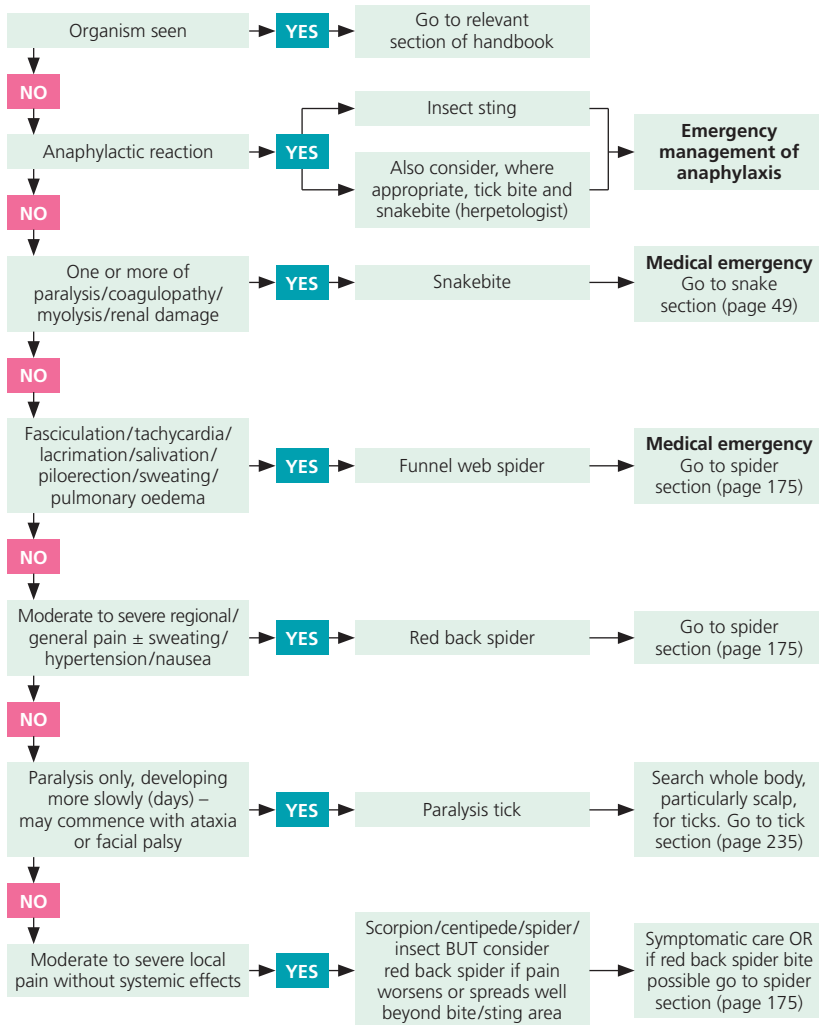
Only a small number of cases of significant envenoming occur each year, as the majority of patients presenting to hospital emergency departments with suspected bites/stings, do not develop envenoming.²⁻⁶ Hence, few clinicians have the opportunity to develop expertise in the management of envenomed patients.

Clinicians are strongly advised to seek expert advice when managing a patient with envenoming.

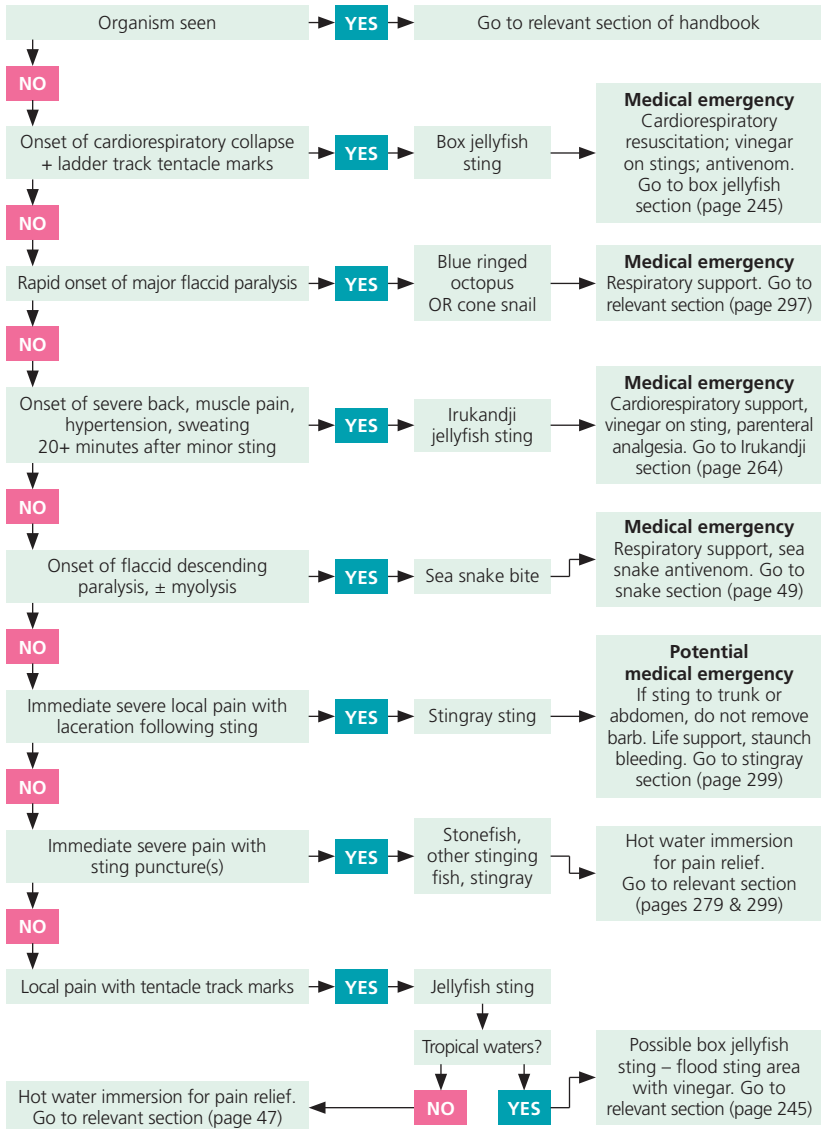
Determining the nature of the patient's problems and the likely cause are vital steps in ensuring an optimal outcome

Management of venomous bites and stings

Injury (bite/sting) occurring on land

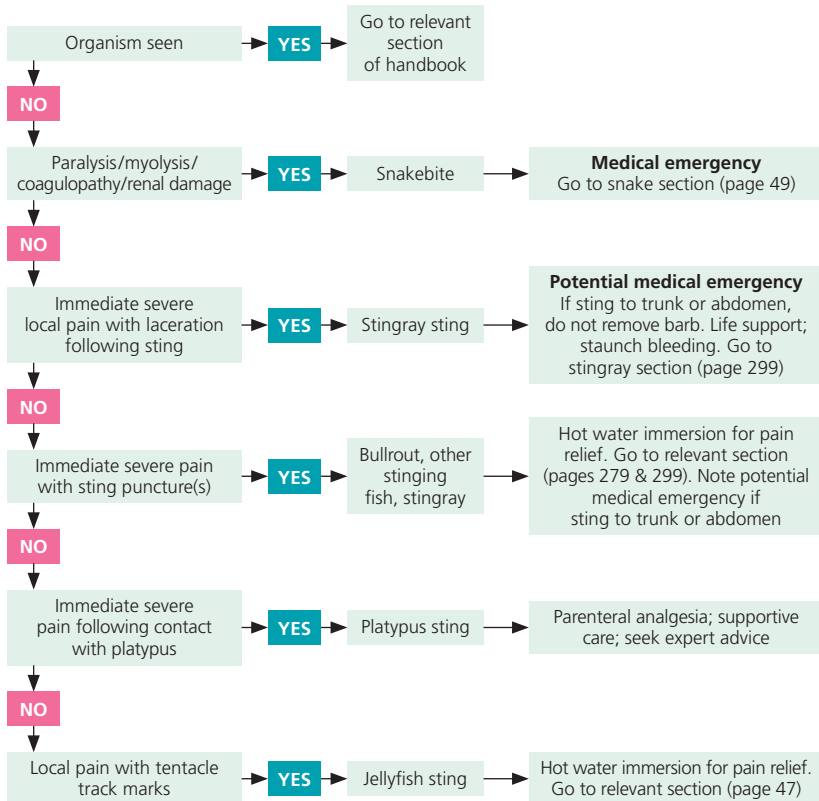


Injury (bite/sting) occurring in a marine environment



Management of venomous bites and stings

Injury (bite/sting) occurring in a freshwater environment



Managing bites or stings: Basic principles¹

- Rapidly triage any patient presenting with suspected bite/sting from a potentially venomous animal.

Address immediate life threats.

Assess airway, breathing and circulation and support as indicated.

Significant impairment of cardiac or respiratory function require priority management.

- If a patient with confirmed or suspected snakebite presents with no first aid in place, immediately apply PBI first aid (i.e. pressure bandage and immobilisation of the bitten limb).
- For potentially life-threatening bites/stings (snakebite; funnel web spider bite; box jellyfish stings; blue ringed octopus), it is important to obtain good venous access early. This is a good opportunity to obtain initial blood samples for laboratory investigations* if indicated.

For potentially life-threatening envenoming from snakebite, funnel web spider bite, or blue ringed octopus bite, PBI first aid should never be removed to inspect the wound if treatment facilities and antivenom (where applicable), are not immediately available.

- To obtain a swab of the bite site (snakebite only) – cut a window in the bandage over the bitten area and reapply bandage after taking the swab.⁷

- Do not remove PBI first aid until the patient is fully assessed including history and examination, blood test results*, and/or snake venom detection results (snake venom detection is performed if the patient has clinical signs or symptoms, or laboratory indicators of envenoming)* – at which point an assessment can be made about the need for antivenom.^{1,7}

If the patient has objective evidence of significant systemic envenoming, antivenom should be commenced and then PBI first aid may be removed.⁷

If there is no clinical or laboratory evidence of significant systemic envenoming, cautiously remove PBI first aid and reassess the patient with further examination and blood tests starting about 1 hour later (or sooner if indicated).⁶

*Blood tests and snake venom detection kit relevant only if snakebite is a possibility.

Envenoming overview

What is envenoming?

Envenoming (also known as envenomation) is the process where venom is introduced into a human being or an animal, and generally refers to the situation where enough venom is introduced to cause clinical effects.

Antivenom is a specific antidote against venom and is usually the most effective treatment for significant envenoming, but the antivenom must be specific for the immunotype of venom involved. Consequently, for cases requiring antivenom therapy, it is important to determine either the venom immunotype (for snakebite) and/or identify the animal that caused the bite or sting.

Diagnosis¹

Identification of the culprit animal may be straightforward, particularly if it is presented with the patient. However, more often than not, it is much less clear.

Patients may present with marks and scrapes that may or may not be related to a bite/sting by an unknown organism. The issue becomes clearer if patients present with symptoms, signs, or laboratory test results indicative of envenoming. However, sometimes the indicators may be non-specific and may point to a variety of etiologies of which envenoming is just one.

With regards to diagnosis, most worrying is the patient who presents with no history of bite or sting and in whom the constellation of symptoms, signs and laboratory tests may not immediately suggest envenoming. A high index of suspicion is key, and in such cases, it is essential to conduct the appropriate tests to exclude or confirm envenoming.

Patients may present with marks and scrapes that may or may not be related to a bite/sting by an unknown organism

Index of suspicion¹

Consider envenoming in unexplained cases of paralysis, myolysis, coagulopathy, renal damage, collapse, convulsions, neuroexcitatory signs, pulmonary oedema, or ataxia.

Signs and symptoms of significant local and systemic envenoming

Never assume that all medical and nursing staff know what to look for when observing a patient with envenoming. Instruct staff to look for specific signs (Table 1).

Table 2 on the following pages provides further information on symptoms/signs of envenoming and the animal(s) likely associated with the clinical features.

Table 1. Envenoming signs and symptoms^{1,8-14}

| |
|---|
| Ptosis (usually earliest sign of developing paralysis) ⁸ |
| Cranial nerve paresis (dysarthria; dysphagia; partial or complete ophthalmoplegia; drooling; pupillary dilatation); general muscle weakness; respiratory paralysis (i.e. paralysis in snakebite, blue ringed octopus bite or tick bite) ⁸⁻¹⁰ |
| Persistent bleeding from bite site, venepuncture sites or gums (indicative of coagulopathy in snakebite) ⁸ |
| Dark urine due to myoglobin (myolysis) or haematuria in snakebite ⁸ |
| Profuse sweating and pain (red back spider bite; possibly Irukandji syndrome) ¹¹⁻¹³ |
| Profound fatigue and mental confusion or disorientation ¹ |
| Profuse salivation, lacrimation, sweating, and piloerection with muscle twitching, pulmonary oedema (funnel web spider bite; possibly Irukandji syndrome) ¹²⁻¹⁴ |

Tetanus prophylaxis¹

Any bite is at risk of secondary infection, which may include tetanus. Ensure tetanus prophylaxis and watch for developing cellulitis.

Note: To avoid muscle haematoma, patients with coagulopathy should not receive intramuscular injections until the coagulopathy has resolved.

Envenoming overview

Venom actions and associated clinical features

Table 2. Broad/indicative diagnosis based on envenoming signs/symptoms^{1,8-17}

| Type of venom action | Symptoms and signs | Consider |
|---|---|---|
| Paralytic neurotoxins [Cause progressive flaccid paralysis] | Partial or complete cranial nerve paresis – [ptosis; ophthalmoplegia; dysarthria; dysphagia; drooling; fixed dilated pupils; limb weakness; decreased or absent deep tendon reflexes; respiratory compromise/paralysis] Progressive ataxia | Snake Blue ringed octopus Paralysis tick Cone snail Paralysis tick Blue ringed octopus (often quick) |
| Excitatory neurotoxins [Cause general or specific excitation of the nervous system] | Autonomic (sympathetic and/or parasympathetic) nervous system stimulation – [increased lacrimation; salivation; sweating; piloerection; muscle fasciculation; muscle spasm/pain; alteration in HR or BP; pulmonary oedema] | Funnel web spider Red back spider Irukandji syndrome |
| Myotoxins [Systemic rhabdomyolysis] | Muscle pain, tenderness or weakness; myoglobinuria; major elevation of plasma CK | Selected snake species |
| Procoagulants [Cause activation of coagulation pathways, resulting in consumption of fibrinogen and other factors – i.e. defibrination coagulopathy] | Persistent oozing of blood from bite site, venepunctures or gums; bruising; haematuria; haematemesis | Selected snake species |

The above table suggests the probable organism associated with particular venom actions and resultant clinical features. Refer to the relevant section (i.e. 'Snakes'; 'Spiders'; etc) for additional information (including detailed diagnostic algorithms).

Note also that some snake venoms may have atypical effects. This should be considered when managing cases of suspected or confirmed snakebite.

....table continues

Table 2. Broad/indicative diagnosis based on envenoming signs/symptoms^{1,8-17} ... cont'd

| Type of venom action | Symptoms and signs | Consider |
|--|--|---|
| Anticoagulants [Cause inhibition of coagulation pathways, resulting in increased bleeding tendency] | Persistent oozing of blood from bite site, venepunctures or gums; bruising; haematuria; haematemesis | Selected snake species |
| Renal damage [Usually secondary] | Elevation of blood creatinine/urea; polyuria; oliguria; anuria | Snakes Potentially (but rarely), other venomous animals |
| Pain producing toxins | Local severe pain – Immediate severe local pain | Box jellyfish; stinging fish; stingrays; scorpions; centipede; insects (bees, wasps, some ants) |
| | – Delayed severe local pain | Red back spider and selected snake species |
| | Generalised severe pain | Red back spiders and Irukandji jellyfish |
| Cardiotoxins | Cardiac failure/decompensation; cardiac arrhythmia; cardiac arrest | Box jellyfish |

The above table suggests the probable organism associated with particular venom actions and resultant clinical features. Refer to the relevant section (i.e. 'Snakes'; 'Spiders'; etc) for additional information (including detailed diagnostic algorithms).

Note also that some snake venoms may have atypical effects. This should be considered when managing cases of suspected or confirmed snakebite.

Diagnosis of systemic envenoming

History^{1,18}

A thorough history is fundamental to aiding diagnosis and decisions regarding treatment. Consider the following:

- Circumstances of the bite or sting (e.g. time; activity; geographic location).
- Details of organism involved (if seen).
- Symptoms (including time of onset).
- Details of first aid (e.g. type; delay in application; activity before application).
- Medical history and medications, including prior bites and antivenom treatment.

Examination

A detailed examination of the patient is key. Look for local (bite/sting site), general and systemic signs of envenoming (Table 3).

Table 3. Examining a patient with bite or sting: Key parameters^{1,11,14,18}

| Examination | Look for |
|---------------------------------------|--|
| General signs of envenoming | – Alterations in HR, BP, respiratory rate, body temperature, SpO ₂ , or consciousness level |
| Bite/sting site | – Swelling – Increased sweating – Piloerection – Local trauma (stingray) – Tentacle marks (jellyfish) – Persistent bleeding – Bruising – Multiple bites/stings – Blistering – Necrosis |
| Draining lymph nodes | – Tenderness or swelling |
| Specific signs of systemic envenoming | – Neurotoxic paralysis (ptosis and other cranial nerve effects; limb weakness; respiratory paralysis) – Rhabdomyolysis (muscle pain, tenderness or weakness) – Coagulopathy (increased bleeding or bruising) – Neuroexcitatory envenoming (increased salivation, lacrimation, sweating) |

Laboratory investigations¹

Laboratory tests are only of value in certain types of bites/stings. In particular, tests are vital in the diagnosis of envenoming in snakebite cases (Table 4), but are of no value in assessing spiderbite and may play a limited role in the assessment of cases of marine envenoming (e.g. Irukandji syndrome).

Table 4. Key laboratory investigations for snakebite^{1,6}

| |
|---|
| Coagulation studies [INR as minimum, but preferably INR + aPTT + fibrin(ogen) degradation products (FDP, d-dimer), and where available, direct fibrinogen] |
| Full Blood Count including blood film. [Blood film to be checked for schistocytes if there is evidence of systemic envenoming or evidence/suspicion of haemolysis – e.g. falling haemoglobin or low platelet count] |
| Biochemistry (creatinine, urea, electrolytes, CK, bilirubin) |
| Snake venom detection kit (perform in selected cases only – see Section 3) |

Snake venom detection

- A snake venom detection test is available for Australian and Papua New Guinea snake venoms. It is performed using the bioCSL's Snake Venom Detection Kit (SVDK).
- The primary purpose of the SVDK is to determine the appropriate neutralising monovalent antivenom to use if a patient is envenomed and antivenom therapy is clinically indicated.¹⁹
- SVDK should not be used to confirm or exclude snakebite or to determine whether a patient is envenomed.¹⁹⁻²¹
- From a practical perspective, the best sample for venom detection is a bite site swab, then urine.¹⁹

Laboratory investigations are vital in assessing snakebite... but are of no value in assessing spiderbite

Management of envenoming with antivenoms

Antivenoms: General principles¹

Always seek expert advice when managing a patient who may require antivenom therapy.

Antivenom specificity

- Antivenom is a specific antidote against venom toxins and therefore it is vital to match antivenom against the venom involved.²²⁻³²
- Where possible, monovalent antivenom is the preferred option.
- For venomous snakebite, bioCSL manufactures monovalent antivenoms against the 5 main snake venom immunotypes found in land snakes in Australia and Papua New Guinea (PNG).²²⁻²⁶

Note: Monovalent snake antivenoms may contain varied levels of the other snake antivenoms due to the nature of manufacturing processes.³³

Importantly however, the stated minimum neutralising potency applies only to the 'monovalent' snake venom immunotype listed on the product.²²⁻²⁶ No assumptions should be made about non-listed neutralising potency.

- Additionally, bioCSL manufactures Polyvalent Snake Antivenom, which is prepared with neutralising potency covering all five snake venom immunotypes predominating in land snakes in Australia and PNG.²⁷
- bioCSL also manufactures Sea Snake Antivenom, which covers bites by most medically important sea snake species.²⁸
- For all other antivenoms it is imperative to use only the antivenom specific for that group of organisms – e.g. bioCSL's Red Back Spider Antivenom is indicated for the treatment of envenoming from red back spider bite only³⁰ and **should not** be used to treat envenoming from funnel web spider bites.

When to use antivenom

- **Antivenom should be used only when there are clear clinical and/or laboratory indicators of systemic envenoming or major local/regional envenoming (e.g. marine stings).**²²⁻³²
- Indications for antivenom vary depending on the type of animal and the type of antivenom.²²⁻³²

The dose of antivenom given is the same for children and adults

Dosing

- Antivenom needs to achieve high blood levels rapidly to counteract high levels of circulating venom. Therefore, for most antivenoms, the I.V. route is required.^{22-29,31}
- The dose of antivenom depends on the particular antivenom involved and how much venom was injected, but is independent of the size or age of the patient. Snakes inject the same dose of venom into children and adults. Thus, the dose of antivenom given is the same for children and adults.²²⁻³²
- Repeat doses of antivenom may sometimes be required.²²⁻³²
- In most cases, it is preferable to dilute antivenom up to 1 in 10 in a standard I.V. fluid (e.g. normal saline, Hartmann's solution*).^{22-28,30-32}
- However, where volumes are large and the patient small (i.e. children) or there is a risk of cardiac decompensation, high dilutions may not be possible. In children, calculate the amount of crystalloid to be used based on the total volume to be delivered **not** exceeding 10 mL/kg.^{34,35}

*Product Information leaflets for a number of bioCSL's antivenoms recommend dilution using Hartmann's solution. The use of other isotonic crystalloid such as normal saline to dilute these antivenoms is based on expert clinical experience and is accepted current clinical practice [1,22-27,30,36].

Management of envenoming with antivenoms

Adverse reactions to antivenom

- All antivenoms consist of animal immunoglobulin and have the potential to cause adverse reactions, which may be early or delayed. Therefore antivenoms should only be administered when there is a clear clinical indication.²²⁻³²
- **Whenever antivenom is given, adrenaline and resuscitation equipment must be immediately available in case the patient develops severe anaphylaxis.**²²⁻³²

Common mistakes during the diagnosis and management of envenoming¹

- Failure to obtain a detailed enough history to guide accurate diagnosis.
- Assuming an initially well patient will remain well.
- Failure to look for or detect early evidence of paralysis.
- Failure to repeat tests/examination frequently enough to pick up change in clinical circumstance.
- Failure to correctly use bioCSL's SVDK (Snake Venom Detection Kit) and/or interpret its results.
- Patient discharged too early.
- Giving I.M. injection (such as tetanus vaccine) prior to resolution of venom-induced coagulopathy.

Bites/stings observation foldout chart

A template chart for recording serial observations in cases of snakebite, spiderbite or marine stings and details regarding antivenom therapy (if administered), is provided at the back of this handbook.

Note: The chart should be used alongside information within this handbook. Other Investigations/observations (in addition to those mentioned in the chart) may be required for bites/stings by various venomous creatures. Consult the relevant section of this handbook for further information.

Section 2

First aid

- Basic first aid: DRS ABCD
- Pressure Bandaging
& Immobilisation (PBI)
- Vinegar
- Hot water

In this section

Basic (critical care) first aid

First aid for envenoming: Key principles 31

Basic first aid: DRS ABCD

| | |
|----------------------|----|
| Danger | 32 |
| Response | 32 |
| Send for help | 33 |
| Airway | 33 |
| Breathing | 34 |
| Compression | 35 |
| Defibrillation | 35 |
| CPR Summary | 36 |

Specific first aid for venomous

bites and stings 37

Pressure Bandaging & Immobilisation (PBI)

| | |
|--|----|
| Background | 38 |
| Purpose of the PBI technique | 38 |
| When to use the PBI technique | 39 |
| How to apply PBI first aid: Bites to the lower limb | 39 |
| How to apply PBI first aid: Bites to the upper limb | 41 |
| PBI technique: Tips | 42 |
| Additional information | 42 |
| Timing of PBI first aid removal | 43 |
| PBI technique: Common mistakes | 43 |

Vinegar

| | |
|---|----|
| When to use vinegar as first aid | 44 |
| Tropical jellyfish stings | 44 |
| Rationale for using vinegar | 44 |
| How to apply first aid with vinegar | 46 |

Hot water

| | |
|---|----|
| When to use hot water as first aid | 47 |
| Rationale | 47 |
| How to apply hot water as first aid | 48 |

Basic (critical care) first aid

First aid for envenoming: Key principles¹

In persons bitten by a venomous creature, the application of appropriate first aid potentially can be critically important in ensuring patient survival. This includes not just first aid directed against venom (such as PBI – i.e. Pressure Bandaging & Immobilisation technique) but also critical care first aid such as DRS ABCD.

For venom-specific first aid, the key principles are:

- (a) Do no harm.
- (b) Where applicable, minimise venom effects through measures such as immobilising venom around the bite site to delay entry into the general circulation. (Note: For some venoms this type of local immobilisation is either ineffective or potentially dangerous – see Table 6 on page 37).

The following pages 32-36 provide a quick reference to basic (critical care) first aid (i.e. DRS ABCD). For more comprehensive information – refer to the appropriate guidelines on the Australian Resuscitation Council website www.resus.org.au.

In persons bitten by a venomous creature, the application of appropriate first aid potentially can be critically important in ensuring patient survival. This includes not just first aid directed against venom (such as PBI) but also critical care first aid such as DRS ABCD

The information on pages 32-48 has largely been adapted from various sources including the Australian Resuscitation Council guidelines (www.resus.org.au) with additions by A/Prof Julian White and the handbook's reviewers. Please refer to the Australian Resuscitation Council website www.resus.org.au for official first aid guidelines and further information.

Basic first aid: DRS ABCD

D.... Danger^{1,2}

Assess the scene for danger. Ensure you, others and the person bitten or stung, are safe.

Avoid the culprit animal and DO NOT try to kill or catch it. If it is already dead, consider carefully collecting it and transporting it with the person, if this can be done safely without risk. Attending to the person takes priority.

R.... Response^{2,3}

Check for a response (ask the person to open their eyes and/or squeeze your hand) or see if they respond to touch (squeeze the person's shoulders firmly). A person who fails to respond or shows only a minor response (e.g. groaning without opening the eyes) or is not breathing normally, should be managed as if the person is unresponsive.³

| | |
|--|--|
| If the person is responsive | Apply specific first aid (see Table 6 on page 37) and seek medical assistance |
| If the person is unresponsive | Send for help – then open the airway and check for breathing (see pages 33-34) ² |
| If the person is breathing normally ³ | Care of the airway takes priority over any injury (including the possibility of spinal injury). ³ Carefully place the person on their side to establish and maintain a clear airway to facilitate drainage and reduce the risk of inhaling foreign material. ² Apply specific first aid if applicable (Table 6 on page 37) |
| If the person is not breathing normally ² | Immediately begin CPR. Important note: If snakebite coagulopathy is an issue, chest compressions may be problematic. ¹ Please see additional information on page 34 in the section entitled 'B.... Breathing'. To administer CPR, give 30 chest compressions followed by 2 rescue breaths. Continue CPR until the person's breathing is normal and stable, or until medical assistance arrives. ² Once breathing is normal and stable, apply specific first aid (Table 6 on page 37). |

S.... Send for Help²

Immediately send for help.

A.... Airway^{2,4}

Open the mouth, check the airway, and clear any potential obstruction from the mouth to ensure the person can breathe. Turn the mouth slightly downwards to allow any obvious foreign material (e.g. food, vomit, blood and secretions) to drain using gravity. Visible material can be removed by the rescuer's fingers.



B.... Breathing

See instructions on page 34.



....continued overleaf

Basic first aid: DRS ABCD

B... Breathing^{2,5}

– Check for breathing with your cheek close to the person's mouth.

Look – for movement of the upper abdomen or lower chest

Listen – for the escape of air from the nose and mouth

Feel – for movement of the chest and upper abdomen

– Take 5 to 10 seconds to check for normal breathing if the person is unresponsive.

– If the person is not breathing normally and there is no perceptible pulse, immediately begin **Chest Compression**. Be aware that in cases of envenoming with paralysis, initially, breathing may be absent, but cardiac function may be unaffected. If there is a venom-induced coagulopathy, there is a risk of bleeding, and vigorous chest compression may potentially cause trauma and internal bleeding. Therefore, in snakebite cases it is important to avoid chest compression if cardiac function is adequate, as evidenced by an adequate pulse. In this sense, DRS ABCD first aid in snakebite may differ from general guidelines on DRS ABCD. However, do not delay or avoid chest compression in suspected snakebite if an adequate pulse is not quickly detected. The dangers of prolonged, untreated cardiac arrest outweigh the dangers of chest compression in the presence of coagulopathy.¹

– **Then** perform **Rescue Breathing**:

Tilt – place the person on their back, tilt the head back and lift the jaw to open the airway.

Blow – pinch/close the person's nose and give them 2 normal breaths (not deep breaths). Breathe air into their mouth to inflate the chest – do this twice at 1 second per breath (this is referred to as 2 rescue breaths).

Look – to ensure the chest rises with each breath.

Listen and Feel – for air being exhaled.



C.... Compression^{2,6}

- Different methods are used depending on the age of the person – refer to CPR summary table on page 36.
- Placing the heel of one hand on the centre of the chest, and covering this hand with the other, push directly downwards, giving equal time for compression and relaxation. Avoid rocking backwards and forwards, or using thumps or quick jabs.
- Apply compressions at a rate of 100 per minute (i.e. almost 2 compressions per second). Perform 30 chest compressions followed by 2 breaths – then repeat.
- If the rescuer is unable or unwilling to provide breathing assistance, **it is vital to perform the chest compression component of CPR (at the rate of 100 compressions per minute).**²
- Apply specific first aid as soon as possible while maintaining CPR.
- Continue CPR until the person's breathing is normal and stable, or until medical assistance arrives.²
- See CPR summary table on page 36.






D.... Defibrillation²

If available, attach an AED (Automatic External Defibrillator) and follow the prompts.

Basic first aid: DRS ABCD

Table 5. CPR Summary³⁻⁶

| | | | |
|--|--|---|---|
| Response | Check if the person is responsive | | |
| Airway | Open airway – tilt head – lift chin | | |
| Breathing | After performing 30 chest compressions (see below), provide 2 rescue breaths at 1 second per breath | | |
| Compression | | | |
| | Infant (< 1 year) | Child (1 to 8 years) | Adult (> 8 years) |
| Hand position | Just below nipple line | | In the centre of the chest between the nipples |
| Compression method Push hard and fast Allow complete recoil | 2 fingers  | 1 hand: Heel of one hand or 2 hands: Heel of one hand with second hand on top  | 2 hands: Heel of one hand with second hand on top  |
| Compression depth | About 1/3 the depth of the chest | | |
| | About 4 cm | About 5 cm | Greater than 5 cm |
| Compression rate | 100 per minute | | |
| Breathing-Compression cycle | Give 30 chest compressions (at almost 2 compressions per second) followed by 2 breaths – then repeat | | |

Specific first aid

- Manage wounds, bleeding and shock if present.
- Once the person is stable, proceed to apply first aid specific for the venomous creature (see Table 6 on page 37).

Specific first aid for venomous bites and stings

Table 6. Appropriate first aid for bites/stings^{1,7-16}

| Organism | First aid method | Comments |
|--|--|---|
| Venomous snakes Funnel web spider Blue ringed octopus Cone snail | Pressure Bandaging & Immobilisation (PBI) technique ⁷ Note: The PBI technique applies to all the creatures mentioned in the left hand column. See page 38 for detailed information | Dos and Don'ts relating to PBI technique are shown on page 42 Venom from snakes, blue ringed octopus and cone snails may cause the person to stop breathing. Extended breathing assistance (expired air resuscitation) may be required until medical assistance is reached ¹ |
| Box jellyfish and related chirodroid jellyfish | Vinegar for at least 30 seconds to stung area ⁸ See page 44 for detailed information | PBI first aid is contraindicated for box jellyfish stings as it may increase the extent of envenoming ⁷⁻⁹ |
| Irukandji jellyfish | Vinegar over the sting site/remaining tentacles ⁸ if the person is aware of having been stung Do not use PBI for Irukandji jellyfish stings ^{7,8} | Typically stings cause minimal discomfort and symptoms generally develop about 20-40 minutes later (i.e. generalised muscular pain, especially back pain). ^{8,10-12} At this point the person is already systemically envenomed and requires urgent medical care |
| Stinging fish (e.g. Stonefish and other species) Stingray Bluebottle jellyfish Non-tropical jellyfish (i.e. bluebottles and other jellyfish found in non-tropical waters) | Hot (but not scalding) water that is no hotter than 45°C may be used for all the organisms mentioned in the left hand column ^{8,13-15} See page 47 for further information Do not use the PBI technique for fish stings ^{7,13} | After administering first aid with hot water, thoroughly clean marine puncture wounds (from stinging fish) with clean fresh water and apply antiseptic to prevent infection ¹ Note that chest or abdominal stingray injuries are a medical emergency. The barb should not be removed pre-hospital in such cases. Any stingray injury can cause a significant wound trauma with bleeding. Application of local pressure to stem the bleeding is appropriate ¹ Ensure tetanus immunisation is up to date ¹ |
| Red back spider | Ice pack or cold compress. PBI is contraindicated (may worsen pain) ¹⁶ | |

Note: In cases of suspected funnel web spider bite, if the spider suspected of biting the person is captured or killed (even if it has been squashed) – it may be preserved in methylated spirits or similar for identification, although this is not vital.

Pressure Bandaging & Immobilisation (PBI)

Background

This is an important first aid method for snakebite and a variety of other bites and stings. Developed by Dr Struan Sutherland and colleagues at CSL – the technique is based on an understanding of the movement of important snake and spider toxins.

For certain types of venomous bites and stings, timely and correct application of Pressure Bandage & Immobilisation (PBI) first aid is crucial for facilitating optimal clinical outcomes.

Purpose of the PBI technique

Many Australian animal venoms are composed of high molecular weight proteins, which are transported around the body by the lower pressure lymphatic system. Clinically, enlargement or tenderness of nodes draining the bite site is often an early sign of absorption of venom.

The aim of the PBI technique is to retard venom transport within the lymphatic system without impeding blood circulation.^{17,18} This is achieved by:

1. **Pressure:** compressing lymphatic vessels at the bite site by bandaging, extending upwards along as much of the bitten limb as possible
and
2. **Immobilisation:** slowing or stopping the movement of lymph in the vessels by splinting the limb and keeping the person still. Splinting restricts the pumping effect of muscle movement.

For the PBI technique to be effective, both steps are crucial, i.e. the application of pressure (bandaging) and immobilisation of the bitten limb.

For the PBI technique to be effective, both steps are crucial, i.e. the application of pressure (bandaging) and immobilisation of the bitten limb

When to use the PBI technique^{7,9}

| Use for | Do NOT use for |
|---|---|
| Bites by: <ul style="list-style-type: none">– Snakes – including sea snakes– Funnel web spider– Blue ringed octopus– Cone snails | Bites and stings by: <ul style="list-style-type: none">– Jellyfish (including Irukandji stings and box jellyfish stings)*– Other spiders (including red back spider)– Scorpions, centipedes or beetles– Fish (including stonefish) <p>*Note: In the past, the PBI technique was recommended first aid for Box jellyfish stings. However, further research has indicated that application of pressure may actually increase the extent of envenoming following box jellyfish sting and therefore PBI first aid is not only discarded as a recommendation, but considered a contraindicated technique by experts⁹</p> |

How to apply PBI first aid: Bites to the lower limb^{1,7,19,20}

1. Maintain basic life support if required. Refer to 'Basic (critical care) first aid' on pages 32-36.
2. Do not wash the wound. As soon as possible, apply a broad pressure bandage starting below the bite site continuing upward along the bitten limb. Starting at the toes, bandage upwards as far as possible. Leave the tips of the toes unbandaged to allow for circulation to be checked.

Use elastic bandages if available, or crepe bandages (but any flexible material that can be cut into 10-15 cm wide strips may be used). Emerging evidence supports the use of elastic bandages (over crepe bandages).^{7,19} However, extra care is required to avoid tourniquet action with an elastic bandage.

Do not remove pants or trousers – simply bandage over the top of the clothing.

Take note of the location of the bite – this becomes important later for marking the bandage to indicate the bite area.¹

.....continued overleaf

Pressure Bandaging & Immobilisation (PBI)

How to apply PBI first aid: Bites to the lower limb^{1,7,19,20} ... cont'd

3. Bandage as firmly as for sprained ankle, but not so tight that circulation is prevented. Continue to bandage upwards from the lower portion of the bitten limb.
4. Apply the bandage as far as possible up the limb to compress lymphatic vessels.
5. It is vital to now **apply a splint**. Bind a stick or suitable rigid item over the initial bandage to splint the limb. Secure the splint to the bandaged limb by using another bandage (if another bandage is not available, use clothing strips or similar). It is very important to keep the bitten limb still.
6. Bind the splint firmly to as much of the bitten limb as possible (including the joints on either side of the limb), to prevent muscle, limb and joint movement. This will restrict venom movement.
7. On the overlying bandage, mark the location of the bite with a pen, tape or any other material.¹
8. It is crucial to keep the bitten limb (and the person) still.
9. Seek urgent medical assistance once first aid has been applied. Bring transport to the person, if possible.

Basic (critical care)
first aid DRS ABCD

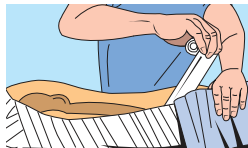
Step 1.



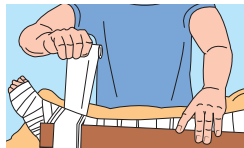
Step 2.



Step 3.



Step 4.



Step 5.



Step 6.

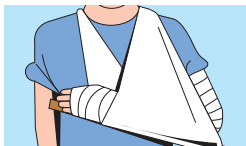
How to apply PBI first aid: Bites to the upper limb^{1,7,19,20}

1. Maintain basic life support if required. Refer to 'Basic (critical care) first aid' on pages 32-36.
2. Quickly remove any rings or other potentially restrictive jewellery from all fingers in the bitten limb
3. Do not wash the wound. As soon as possible, apply a broad pressure bandage from the fingers of the affected arm, bandaging upward as far as possible. Use elastic bandages if available, or crepe bandages (but any flexible material that can be cut into 10-15 cm wide strips may be used). Emerging evidence supports the use of elastic bandages (over crepe bandages).^{7,19} However, extra care is required to avoid tourniquet action with an elastic bandage.

Do not remove any clothing – simply bandage over the top of the clothing.

Take note of the location of the bite – this becomes important later for marking the bandage to indicate the bite area.

4. Bandage the arm with the elbow bent, to ensure the person is comfortable with their arm in a sling. Leave the tips of the fingers unbandaged to allow for circulation to be checked.
5. **Bind a splint along the forearm.**
6. Use a sling to prevent limb movement.
7. On the overlying bandage, mark the location of the bite.¹
8. Keep the bitten limb (and the person) still.
9. Seek urgent medical assistance after first aid has been applied. Bring transport to the person if possible.



Pressure Bandaging & Immobilisation (PBI)

PBI technique: Tips^{1,7,20}

| Do | Do NOT |
|--|---|
| <ul style="list-style-type: none">– Rapidly apply the bandage– Use strips of clothing or similar material to apply pressure if a bandage is unavailable. Additional bandage or clothing strips can be used to bind the splint– Extend the bandages as high and as low as possible on the affected limb– If possible, mark the bite site with a pen (on top of the bandage)– Check unbandaged tips of fingers or toes for circulation– Reassure and keep the person at rest and bring transport to the person, if possible | <ul style="list-style-type: none">– Do not cut or suck the bite site or stung area– Do not use a tourniquet, as this can stop blood flow and can be dangerous if left on for too long– Do not wash the bite site– Do not remove the person's clothing as the movement in doing so may force more venom into the bloodstream– Do not allow the bitten person to walk– Do not attempt to catch or kill the snake as it increases the risk of a repeat bite or another person being bitten. The snake is not required for medical treatment |

Additional information^{1,20}

- A correctly applied bandage and splint will be firm but comfortable and may be left on for several hours.
- A bandage applied too tight will act as a tourniquet and will be painful after only a few minutes in place.
- Conversely, a bandage that is too loose will be ineffective as first aid. If venom movement is not impeded, the person is at risk of developing major envenoming.
- Bites to the head or neck require urgent transport and medical assistance. The PBI method cannot be applied to the head or neck.
- Bites to the trunk require firm pressure over the bitten area. Do not restrict chest movement.
- Bandages should not be removed until the person has reached medical care. [See page 43 regarding timing of PBI first aid removal].

Timing of PBI first aid removal

Once the person has been transported to an adequately resourced medical facility, a number of steps need to be undertaken before PBI first aid may be safely removed.

The optimal timing for removing the pressure bandage and splint is discussed further in the relevant sections of this handbook. [See pages 56-57 for 'Snakes'; pages 184-185 for 'Funnel web spiders'; page 301 for 'Blue ringed octopus'; page 302 for 'Cone snails'].

Important notes

- The Pressure Bandage & Immobilisation technique is only first aid.
- It is not definitive treatment for envenoming.
- First aid should be removed only when the person is in a hospital that stocks appropriate antivenom, has a laboratory that is able to perform requisite investigations and a clinician who is able to manage the patient.

PBI technique: Common mistakes^{1,21}

- Bandages applied too loosely – i.e. ineffective retardation of venom movement.
- Limb is not immobilised with a splint.
- Bandages applied too tightly – becomes a form of tourniquet.
- Insufficient bandages used to adequately cover the bitten limb.
- Bandages applied too late and/or only after patient has been active following the bite.
- Person is physically active after PBI is applied (e.g. walking to transport).
- Bite site is washed (impairs later venom detection).
- Bite area is not marked on overlying bandage.

A bandage that is too loose will be ineffective as first aid. If venom movement is not impeded, the patient is at risk of developing major envenoming

Vinegar

When to use vinegar as first aid

Vinegar is appropriate first aid for tropical jellyfish stings,⁸ i.e.:

- Box jellyfish.
- Related chirodropid jellyfish.
- Irukandji jellyfish (see note below).
- For persons stung by an unknown tropical marine animal.

Note: Vinegar may be used for Irukandji jellyfish stings if the person is aware of having been stung. However, from a practical perspective, many individuals may be unaware of the sting until approximately 20-40 minutes later when they develop symptoms of envenoming, i.e. limb pain and/or back pain (not local pain) [8,10-12]. Since the sting site is often not apparent and envenoming has already occurred, at this stage, urgent medical care is required [1].

Tropical jellyfish stings

Large jellyfish such as the box jellyfish have tens of millions of stinging organelles called nematocysts. Unlike most other forms of envenoming, jellyfish nematocysts inject a proportion of their venom directly into capillaries, thus causing rapid and potentially catastrophic systemic envenoming.⁸

Rationale for using vinegar⁸

During the process of stinging, pieces of tentacle or unfired nematocysts may remain on the skin. For life-threatening stings, it is important to inhibit the unfired nematocysts so that subsequent handling does not cause them to fire and inject more venom. Vinegar has no effect on venom already introduced into the patient.

.....continued

Vinegar is primarily recommended in tropical areas where potentially fatal stings may occur

Nematocysts from different species of jellyfish are inhibited or fired by different substances.⁸

Box jellyfish and Irukandji jellyfish cause the most serious cases of marine envenoming and occur most frequently in tropical areas.⁸

For stings by box jellyfish and Irukandji jellyfish, vinegar (4-6% acetic acid in water) is an effective nematocyst inhibitor.^{8,22} Flooding the sting area with vinegar will inactivate any unfired nematocysts – and ensures no further venom is injected in the patient. Once this is done it is safe for bystanders to remove adherent tentacle(s). Copious amounts of vinegar are likely to be required if there is an extensive area of tentacle contact on the skin.

For some other jellyfish stings (e.g. bluebottle), vinegar may cause nematocyst discharge. Therefore, vinegar is primarily recommended in tropical areas where potentially fatal stings may occur.⁸

Vinegar does not relieve the pain already present as a result of venom introduction. Other pain relief measures may be used, but only after vinegar has been applied for box jellyfish, Irukandji jellyfish or unknown tropical marine animal stings.⁸

**For some other jellyfish stings (e.g. bluebottle),
vinegar may cause nematocyst discharge**

Vinegar

How to apply first aid with vinegar^{1,8,22}

1. Remove the person from the water and restrain if necessary.
2. Call for an ambulance (dial Triple Zero – 000) and seek assistance from a lifesaver/lifeguard if available.
3. Assess and commence resuscitation as necessary. Refer to 'Basic (critical care) First Aid' on pages 32-36.
4. Flood the stung area liberally with vinegar for at least 30 seconds, to neutralise unfired nematocysts (stinging organelles). Do not use fresh water, alcohol or methylated spirits.^{1,8,22}
5. Gently pick off any tentacles with tweezers, forceps or gloved fingers.
6. If vinegar is unavailable, pick off any remnants of the tentacles (this is not shown to be harmful to the rescuer) and rinse the stung area well with sea water (do not use fresh water as it may cause unfired nematocysts to discharge).
7. Apply a cold pack or dry ice in a plastic bag for pain relief.
8. If pain relief is required, provide this only after vinegar has been applied.
9. Reassure and keep the person still until medical assistance arrives.
10. Persons who initially appear stable but experience severe symptoms in the following 30 minutes may have developed Irukandji syndrome and require urgent medical care.

Note: In tropical waters, if the person has **clearly** been stung by bluebottle jellyfish and is assessed as having a localised sting, and is stable and does not require an ambulance, vinegar should **not** be applied. Instead, manage as for other non-tropical jellyfish stings (hot water first aid).

PBI first aid is not recommended for jellyfish stings.⁷

Flood the stung area liberally with vinegar for at least 30 seconds, to neutralise unfired nematocysts

Hot water

When to use hot water as first aid^{1,8,13-15,23}

- Fish stings (e.g. Stonefish).
- Stingray.
- Bluebottle jellyfish.
- Jellyfish stings in non-tropical waters (i.e. bluebottles and other jellyfish).

Rationale

The spines of a variety of fish and tentacles of some jellyfish can cause painful local reactions. For some fish (e.g. stonefish), venom glands in spines can deposit venom very deeply into the skin and cause excruciating pain. Very rarely, severe general toxic effects occur.¹³

Studies have shown that for most fish stings and for bluebottle (*Physalia sp*) jellyfish stings, hot water is significantly more effective than cold packs at reducing pain caused by envenoming.^{14,23} Additionally, emerging evidence suggests hot water may be efficacious for other non-tropical jellyfish stings.^{15,23}

Although precise mechanisms require further research, it has been postulated that hot water may deactivate heat-labile venom toxins or modulate pain receptors, thereby reducing pain.²³

For potentially lethal jellyfish stings (e.g. box jellyfish), research has yet to confirm whether hot water is effective – and therefore, hot water is not recommended currently for stings by these animals.¹

For most fish stings and for bluebottle jellyfish stings, hot water is significantly more effective than cold packs at reducing pain caused by envenoming

Hot water

How to apply hot water as first aid^{1,8,13-15,23,24}

1. Keep the person at rest. Reassure and keep under constant observation.
2. Prevent rubbing of the stung area.
3. Place the stung hand or foot in hot water (no hotter than the rescuer can tolerate and no hotter than 45°C).^{14,15,23} If possible, also place the non-stung limb into hot water.^{1,24}
4. For bluebottle or other non-tropical jellyfish stings use a hot shower. Prior to this, if possible, pick off any adherent tentacles and rinse the stung area in sea water to remove invisible stinging cells.^{8,23}
5. Do not immerse the stung hand or foot for more than approximately 20 minutes. [Do not immerse if local anaesthetic has been used].^{1,8,24}
6. Remove briefly before re-immersing.
7. Continue this cycle if pain persists, but for no longer than 2 hours.^{1,24}
8. For fish stings, if pain is not relieved with hot water, a regional local anaesthetic block may help to relieve pain.^{1,24}
9. Transport the person to a medical facility.
10. For bluebottle or other non-tropical jellyfish stings, if pain persists or is generalised, if the sting area is large (half of a limb or more), or involves sensitive areas (e.g. the eye) call an ambulance (dial Triple Zero - 000) and seek assistance from a lifesaver/lifeguard if available.⁸
11. Do not use Pressure Bandaging & Immobilisation technique, because for fish stings, the venom tends to stay in the stung area.¹³
12. Antivenom is available for stonefish envenoming.

Do not immerse the stung hand or foot for more than approximately 20 minutes. Remove briefly before re-immersing

Section 3

Snakes

- Snakebite: Clinical presentation
- Snakebite: First aid
- Snakebite: Diagnosis
- Treating snakebite: Urgent & general treatment & antivenom
- Managing venom-induced toxicity
- Australian venomous snakes overview

In this section

Snakebite: Clinical presentation

| | |
|---------------------------------|----|
| Local signs/symptoms | 51 |
| General systemic effects..... | 51 |
| Specific systemic effects | 51 |
| Snakebite in children..... | 53 |
| Problem presentations..... | 54 |

Snakebite: First aid

| | |
|---|----|
| First aid for snakebite | 56 |
| Snakebite first aid – what not to do | 56 |
| Timing of removal of PBI first aid for snakebite..... | 56 |

Snakebite: Diagnosis

| | |
|--|----|
| Fundamentals of diagnosis..... | 58 |
| History | 60 |
| Examination..... | 61 |
| Key early signs of envenoming relating to the type of snake..... | 64 |
| Laboratory investigations | 67 |
| Key laboratory investigations for snakebite..... | 68 |
| Laboratory investigations request form | 68 |
| Clinical pathway for suspected & confirmed snake bite | 69 |
| Tests in rural hospitals with limited laboratory facilities..... | 70 |
| Interpreting laboratory test results..... | 72 |
| Determining the type snake/venom involved: General principles..... | 74 |
| Snake venom detection: Fundamentals | 76 |
| bioCSL's Snake Venom Detection Kit (SVDK)... .. | 77 |
| Diagnostic algorithms: Determining the most likely type of snake involved..... | 93 |
| Diagnostic algorithm: Local effects of the bite (flowchart)..... | 94 |
| Diagnostic algorithm: Systemic effects of the bite (flowchart)..... | 95 |
| Identifying a snake specimen | 96 |

Treating snakebite: Fundamentals

| | |
|--------------------------------------|----|
| Basic principles of management | 97 |
|--------------------------------------|----|

Treating snakebite: Urgent treatment

Snakebite management

| | |
|------------------------------------|-----|
| charts/algorithms | 101 |
| Snakebite management chart 1 | 102 |
| Snakebite management chart 2 | 104 |
| Snakebite management chart 3 | 106 |

Snakebite: General treatment.....

Specific treatment of snakebite: Antivenom

| | |
|--|-----|
| Antivenom therapy for snakebite | 111 |
| Indications for antivenom..... | 112 |
| Timing of antivenom therapy | 112 |
| Choice of antivenom..... | 113 |
| When to use bioCSL's Polyvalent Snake Antivenom..... | 113 |
| If antivenom is not immediately available | 115 |
| Snake antivenoms: Initial dose | 117 |
| Evaluating patients after initial antivenom therapy..... | 122 |
| Follow-up doses of antivenom | 123 |
| Preparation prior to commencing antivenom therapy..... | 125 |
| How to administer snake antivenoms..... | 126 |
| Observation during antivenom therapy | 127 |
| Premedication prior to administering antivenom | 128 |
| What to do if there is an adverse reaction to antivenom..... | 130 |
| Antivenom therapy: Commonest mistakes... .. | 131 |
| Complications of antivenom therapy..... | 132 |
| Management of serum sickness | 133 |

Administering I.V. antivenom

(flowchart).....

Treating snakebite: Managing venom-induced toxicity

| | |
|--|-----|
| Haematological effects of snake venom | 135 |
| Managing the haematological effects of snake venom | 136 |
| Myotoxicity: Snake venom-induced rhabdomyolysis | 139 |
| Renal/nephrotoxic effects | 140 |
| Cardiovascular effects | 140 |
| Neurotoxic effects – pre-synaptic and post-synaptic neurotoxicity..... | 141 |
| Management of neurotoxic paralysis..... | 141 |
| Local cytotoxicity..... | 142 |

Australian venomous snakes overview

| | |
|-------------------------|-----|
| Brown snake group..... | 143 |
| Tiger snake group | 149 |
| Black snake group..... | 156 |
| Death adders | 160 |
| Taipans | 163 |
| Sea snakes | 166 |

Snake identification

Snakebite: Clinical presentation

Local signs/symptoms^{1,2}

- No symptoms, to swelling, bruising or bleeding (if bleeding is persistent – may indicate coagulopathy).
- Bite may be painful (immediate or delayed pain), but also can be painless and the patient may be unaware of being bitten.
- Bite marks can vary from obvious punctures and scratches to being virtually invisible – see page 62 for photos of bite sites.
- Lymph nodes draining the bite area may sometimes be swollen and/or tender, suggesting venom movement.

General systemic effects^{1,2}

- Nausea/vomiting*
- Headache*
- Abdominal pain*
- Collapse
- Hypertension/Hypotension

*May occur due to envenoming, but also may reflect anxiety.

Specific systemic effects

Envenoming from snakebite can lead to a number of specific systemic effects manifesting as varied presentations (see Table 7 on page 52).

Snakebite: Clinical presentation

Table 7. Specific systemic effects of snake venom: Presenting signs/symptoms¹⁻⁵

| Systemic effect of snakebite | Presentation |
|---|--|
| Neurotoxic paralysis | <ul style="list-style-type: none"> – Ptosis – drooping upper eyelids (see page 63 for photos) – Double vision – may progress to fixed forward gaze – Fixed dilated pupils – Slurred speech – Drooling – pooling of secretions; may have difficulty swallowing – Limb weakness – Loss of deep tendon reflexes – Loss of withdrawal reaction to painful stimuli (may still feel pain) – Respiratory paralysis |
| Systemic myolysis | <ul style="list-style-type: none"> – Muscle pain and tenderness (pain/weakness on contracting muscles against resistance) – Muscle weakness (sometimes mild ptosis) – Evidence of muscle damage – e.g. dark or red urine (myoglobinuria) |
| Coagulopathy | <ul style="list-style-type: none"> – Persistent oozing of blood from bite site, venepunctures or gums; bruising; haematuria; haematemesis – Signs of cerebral irritation (if intracranial haemorrhage) |
| Renal damage | <ul style="list-style-type: none"> – Rising creatinine/urea – Polyuria; oliguria; anuria |
| Microangiopathic haemolytic anaemia | <ul style="list-style-type: none"> – Intravascular haemolysis – Anaemia – Thrombocytopenia – Renal failure |
| Anaphylaxis | Individuals previously exposed to snake venom, either through bites or from working with venom, are at risk of an anaphylactic reaction to subsequent snakebites |
| Varied systemic effects leading to early collapse ± convulsions | <ul style="list-style-type: none"> – Collapse within 15-60 min of snakebite occurs in a minority of cases – In many cases there is rapid spontaneous recovery, but if cardiac arrest is the cause of collapse, recovery is uncertain – Collapse due to cardiac arrest is more likely to occur with brown snake bite – Collapse with convulsions, then recovery, is more common in small children, but can occur in older children and adults |

Snakebite in children^{1,2}

- Snakebite in children, especially young children, can result in more severe and rapidly developing envenoming.
- Obtaining an adequate history may prove difficult.
- Detailed neurological examination such as formal testing for ptosis may not be possible, so look for loss of facial expression/flat facies as evidence of developing early neurotoxic paralysis.
- See 'Problem presentations' on page 54 for further discussions regarding children.

[In children]... look for loss of facial expression/flat facies as evidence of developing early neurotoxic paralysis



Child's face with paralytic features.

Snakebite: Clinical presentation

Problem presentations^{1,2}

There are a number of ways in which a patient (including children) bitten by a snake may present without snakebite being an obvious diagnosis.

Unexplained collapse/convulsions²

- In small children, no history of a bite may be given. The child may run inside crying, collapse and possibly have a *grand mal* convulsion.
- Unless bite marks are obvious and looked for, the parents may not suspect snakebite and the child may be presented to the doctor as a case of convulsions.

Generalised progressive unwellness in children¹

- Again, in children, if development of envenoming is less precipitate than above, but also with no history of a bite, the child may be presented as becoming progressively unwell, lethargic, with vomiting, mild fever, and possibly, apparent neck stiffness and fixed dilated pupils.
- If checked, the urine may be dark and show “haematuria”, with abdominal pain.
- Careful examination in such cases may show progressive paralysis, notably ptosis.
- The “haematuria” usually is myoglobinuria.
- The neck stiffness is due to myolysis and the fixed dilated pupils due to the effect of neurotoxins.

Unless bite marks are obvious and looked for, parents may not suspect snakebite and the child may be presented to the doctor as a case of convulsions

General symptoms/collapse in adults²

Adults also may be unaware of being bitten by a snake, such as when walking through long grass, and may present later with headache, nausea and vomiting, or worse still, collapse and are presented unconscious, with no history of a bite.

Renal failure¹

An unnoticed bite might also cause renal failure, presenting a day or more after the bite.

The above are but a few of the many cryptic presentations for snakebite. Also remember that snakes can, and do, enter houses and unnoticed snakebites can occur inside the patient's house!

An unnoticed bite might also cause renal failure, presenting a day or more after the bite

Snakebite: First aid

First aid for snakebite⁶

First aid for snakebite involves:

1. Assessment of ABC (Airway; Breathing; Circulation) and provision of CPR if required (see pages 32-36).
2. Rapid application of Pressure Bandage & Immobilisation (PBI) first aid after the person has been bitten.
3. Keeping the person and the bitten limb still until medical assistance arrives or they can be transported safely to hospital.

For information on the proper application of PBI first aid for snakebite – see pages 38-43 in section 2.

Snakebite first aid – what **not** to do^{1,6}

- Do not cut (i.e. either incision or excision) at or around the bitten area.
- Do not inject chemicals in or around the bitten area.
- Do not wash the bitten area.
- Do not freeze the bitten area.
- Do not use a tourniquet.
- Do not apply suction to the bite site.
- Do not apply high-voltage shock to the bite site.

Timing of removal of PBI first aid for snakebite^{1,7}

- PBI first aid should not be left on for prolonged periods once the person has reached adequate medical care, i.e. a hospital that stocks appropriate antivenom, is equipped to monitor and treat the patient for envenoming, and has a laboratory that can perform the requisite investigations.
- Importantly however, the removal of first aid may precipitate severe envenoming. Consequently, PBI first aid should be removed only after specific procedures/measures have been implemented (see Table 8 on page 57).
- If a clinically well patient develops signs of envenoming immediately after removing first aid, reapply PBI first aid, perform repeat laboratory investigations and administer antivenom as appropriate.⁷

PBI first aid should be removed only after specific procedures have been implemented

Table 8. Timing and process of PBI first aid removal in snakebite^{1,2,8-10}

| | |
|--|---|
| Key principle | Snakebite should be managed as a medical emergency |
| Removal of PBI first aid in snakebite: General principles | <p>Patient must be stable – if the patient is unwell, or exhibits unstable observations or is obviously envenomed, do not remove the PBI first aid</p> <p>If the patient does not show clinical or laboratory signs of envenoming at presentation, PBI first aid should be removed only if appropriate antivenom is immediately available along with a clinician experienced in resuscitation. Seek expert advice</p> |
| Process prior to PBI first aid removal | <p>If a patient with suspected/confirmed snakebite presents to hospital without effective PBI first aid in place, immediately apply PBI first aid before proceeding further</p> <p>First aid measures for snakebite should remain undisturbed until all of the following steps have been undertaken (in most cases this will take 1-2 or more hours)</p> <ul style="list-style-type: none"> – The patient is admitted to hospital – An I.V. line is in place (this is a good opportunity to take blood samples for testing). In most cases, administer an I.V. fluid load – The patient has been fully assessed including history and examination*, blood test results, and if indicated, snake venom detection results (Note: Snake venom detection is performed if the patient is symptomatic or if blood tests, history or examination indicate that the patient is envenomed), at which point an assessment can be made about the need for antivenom and choice of antivenom – The doctor has assembled the appropriate antivenom and the drugs that may need to be administered when the bandage and splints are removed – Adrenaline and resuscitation equipment are immediately available in case of a severe adverse reaction to antivenom |
| After completing the above steps | <p>If the patient has clinical or laboratory evidence of significant envenoming requiring antivenom therapy, antivenom should be commenced prior to removal of PBI first aid</p> <p>Alternatively, if the patient does not appear to be envenomed, remove first aid and fully reassess starting 1 hour later (or sooner if symptoms/signs of envenoming develop more rapidly)</p> |

*To examine the bite area and swab for venom detection, cut away the dressing over the bite and reapply fresh bandages after taking the swab. It is good practice to take a bite site swab early and store for use in snake venom detection if indicated. [Also keep the old overlying bandage for snake venom detection if required].

Snakebite: Diagnosis

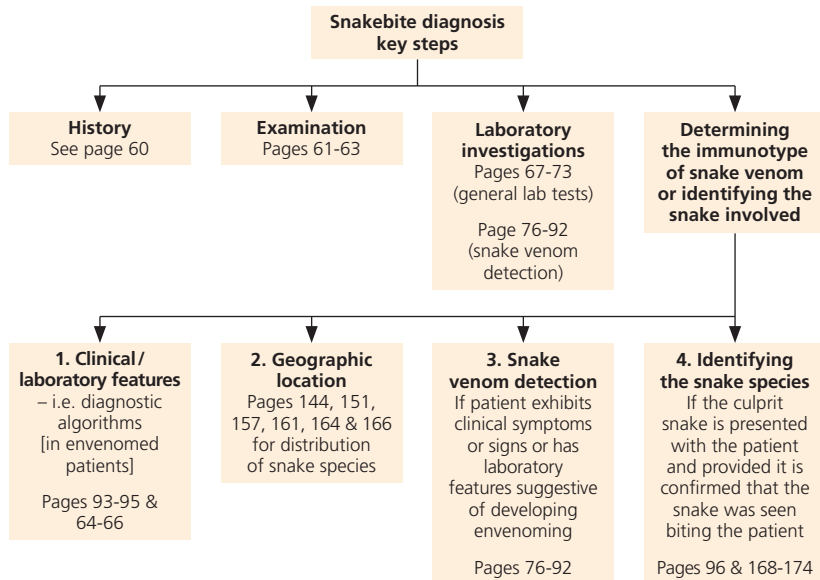
Fundamentals of diagnosis^{1,2,10-12}

- Rapid and accurate diagnosis is crucial to effective management of snakebite.
- Basic diagnosis relies on the combination of a good history, targeted examination and appropriate laboratory investigations.
- In patients presenting with a history of probable/confirmed snakebite, a crucial diagnostic step has already been performed. For other cases, it is important to maintain a high index of suspicion – i.e. in a patient who may have been bitten but is unaware of this and is not able to provide a history (see problem presentations on page 54).
- Quickly ascertain if a snake was seen or the bite was felt and if symptoms of envenoming have developed subsequently.
- While obtaining the history, observe for any ptosis or dysarthria and test for ophthalmoplegia or other muscle weakness.
- There is a general misconception that any bite by a dangerous snake will result in lethal envenoming unless urgent remedial action is taken.

Health professionals managing cases of snakebite must remember that venomous snakes may bite without injecting a dangerous quantity of venom. Thus, a bite by even the most lethal snake species with clear bite marks and venom on the skin surface may not lead to the development of envenoming.

Consequently, the key question when managing snakebite is whether significant systemic envenoming is present, as this is the usual indication for commencing antivenom therapy.
- Some patients may remain symptom free and apparently well with systemic envenoming becoming evident only through laboratory investigations.
- Patients who are symptom free at presentation with promptly-applied effective first aid in place, may develop envenoming rapidly after removal of first aid or it may take several hours before envenoming develops. These patients require frequent reassessment.

.....continued



- In envenomed patients, the pattern of clinical and laboratory features is a vital aid to diagnostic decision making (see diagnostic algorithms – pages 94-95).
- If significant systemic envenoming has developed, the results of a snake venom detection test on an appropriate sample (e.g. bite site swab) may assist in choosing the appropriate monovalent antivenom.
- The key indications for antivenom therapy are listed on page 112.
- Non-specific general symptoms such as headache, nausea/vomiting and abdominal pain can be caused by anxiety, but also may indicate developing systemic envenoming.

These non-specific symptoms generally cannot be used as an indicator of either the degree of envenoming, or the need for antivenom.

Nevertheless, a patient with snakebite who develops significant headache, vomiting or abdominal pain, requires urgent re-evaluation, including repeat laboratory testing to ensure progressive systemic envenoming is not missed.

Snakebite: Diagnosis

History

Obtaining a thorough history is vital to aiding diagnosis and in the management of a patient with snakebite. History taking involves 5 key steps (Table 9).

Table 9. Snakebite cases: Taking a detailed history^{1,2}

| | |
|--|--|
| Circumstances of the bite | <ul style="list-style-type: none">– Time of day– Activity/what the patient was doing at the time of bite– Geographic location to limit the range of snakes to be considered– Number of bites – multiple bites are often more severe– Did anyone actually see the snake bite the patient?– What clothing was the patient wearing in the region of the bite site? |
| Details of the snake if seen | <ul style="list-style-type: none">– Length– Colouration– Distinguishing features |
| Symptoms, including time of onset | <ul style="list-style-type: none">– See pages 64-66 for key early signs based on the type of snake |
| Details of first aid | <ul style="list-style-type: none">– Type of first aid– Any delay in application– Activity before and after application (a patient who was physically active before or after first aid application is less likely to have effective first aid. Hence, if the patient is symptom free several hours later, it is more likely to have been a dry bite) |
| Medical history and medications | <ul style="list-style-type: none">– Past exposure to snakebite or antivenom– Allergy to antivenom– Allergy history– Significant pre-existing medical conditions such as cardiac, renal or respiratory disease– Medications that may interfere with laboratory testing, e.g. warfarin |

Examination

Carefully examine the patient for local, general and specific signs of systemic envenoming (Table 10). Serial observations may be required.

Table 10. Patient with snakebite: Physical examination^{1,2,8,10,12,13}

| | |
|--|--|
| ABC | Check airway, breathing and circulation and assess the presence of any immediate life threats |
| Bite site | <p>Look for swelling, persistent bleeding, bruising, fang marks, multiple bites (see pictures on page 62)</p> <p>If PBI first aid is in place, cut a window in the bandage over the bite site to check the bite site. Also swab the bite site (sterile swab stick moistened in sterile saline). Do not wash or interfere with the wound and keep the removed bite site bandage in case needed for later testing. Rebandage the bite site. Swab is for snake venom detection if indicated now or later (the swab, if stored, should be refrigerated and not frozen)</p> |
| Draining lymph nodes | Tenderness or swelling |
| General signs of envenoming | <p>HR (typically tachycardia)</p> <p>BP (hypotension or hypertension)</p> <p>Respiratory rate (increasing if progressive respiratory failure due to paralysis; may also have shallow respiration or use accessory muscles for respiration)</p> <p>Oxygen saturation</p> <p>Reduced level of consciousness</p> |
| Specific signs of systemic envenoming | <p>Look for evidence of specific systemic effects of snakebite including coagulopathy, neurotoxicity, myolysis, microangiopathic hameolytic anaemia, or renal failure</p> <ul style="list-style-type: none">– Refer to Table 7 on page 52 for the specific systemic effects of snakebite– Also see pages 65-66 for key early signs of envenoming |

Snakebite: Diagnosis

Examination ... cont'd

Bite site examination



Brown snake bite x1 just above ankle. Bite unnoticed, but severe envenoming.



Brown snake bite x1 to thumb, with scratch marks, not punctures; severe envenoming.



Tiger snake bite x2 to thigh; severe envenoming. Note classic bruising around bite, typical of tiger snakes.



Mulga snake bite to base of thumb with classic swelling; severe envenoming.



Death adder bite to finger. Puncture marks only; severe envenoming.



Taipan bite to base of thumb. Major chewing bite with multiple fang and other tooth marks; severe envenoming.

Examining a patient for ptosis¹

- Stand facing the patient. Hold your finger or a pen horizontally at the patient's eye level.
- Instruct the patient to follow the movement of the finger/pen with his/her eyes while keeping the head motionless.
- Slowly move the finger/pen upwards.
- In 'normal individuals', i.e. in the absence of ptosis, the upper eyelids would move upwards along with the patient's gaze.
- However, in patients with ptosis (depending on the degree of paralysis) there is failure of this upward gaze – the upward movement of the upper eyelids is reduced in extent or absent.

Examining a patient for lateral ophthalmoplegia¹

- Hold a finger/pen in the vertical position at the patient's eye level.
- Instruct the patient to remain still while following the movement of the finger/pen with the eyes.
- Move the finger or pen laterally – to the right and the left.
- Observe whether the eyes move together to each side. If yes (i.e. conjugate lateral gaze), the patient does not have lateral ophthalmoplegia.
- Patients with lateral ophthalmoplegia exhibit disconjugate gaze (due to failure of lateral gaze as the eyes cease moving together).
- If disconjugate gaze is apparent, ask whether the patient is experiencing double vision.



Ptosis.



Disconjugate gaze + ptosis.

Snakebite: Diagnosis

Key early signs of envenoming relating to the type of snake¹

- Pages 65-66 provide a list of key early signs based on venom effects, and the snakes likely to be involved.
- However, occasionally a snake from a given species may have an atypical venom, resulting in atypical effects. Any diagnostic decision should be made with this caveat in mind.
- Additionally, many snakes have limited ranges of distribution, and consideration should be given to the species that are likely to be found in the area where the patient was bitten. Where appropriate, the interpretation may involve exclusion of species that are not likely to be found in the area.
- Bear in mind however that a number of snakes, e.g. taipans (and others) are sought by reptile collectors, and a considerable number of these snakes are kept alive in both public and private collections in capital cities and some rural towns. Thus, bites might at times present well outside of the snakes' natural range of distribution.
- Also be aware that bites in reptile collectors may involve exotic snakes and/or misidentified snakes.

Snake classification and colour

- The term 'brown snake', 'black snake', etc does not refer to colour.
- Instead, the category, e.g. brown snake group, typically includes snakes of a particular genus but different species. Snakes belonging to the brown snake group can vary substantially in colour. All snakes included within this group share a common venom immunotype (due to evolutionary relationship).
- Some groups, e.g. the tiger snake group include snakes from more than one genus. Once again, all the snakes within this group share a common venom immunotype.
- Black snakes (red-bellied black snake; blue-bellied/spotted black snake) belong to the black snake group (genus *Pseudechis*).

Neurotoxicity (flaccid paralysis)^{1,2,14,15}

| Key early signs | Late signs | Consider |
|---|---|---|
| Ptosis (see page 63 for further explanation on examination for ptosis) | Limb weakness | – Tiger snakes – Taipans |
| Partial ophthalmoplegia with diplopia (see page 63) | Decreased or absent deep tendon reflexes | – Death adders – Rough scaled snake – Copperheads |
| Dysarthria, dysphagia, drooling | Loss of withdrawal response to painful stimuli (patient still feels pain) | – Rarely, brown snake – Sea snakes |
| Loss of facial expression | Fixed dilated pupils | |
| Ptosis alone may occur with severe myolytic envenoming from mulga snakes and sea snakes | Fixed forward gaze | |
| | Use of accessory muscles for respiration | |
| | Respiratory failure/cyanosis | |

Coagulopathy: Procoagulant (defibrination) effects^{*1,2,14}

| Key early signs | Late signs | Consider |
|--|--------------------|---|
| Persistent ooze from the bite site, gums or venepuncture sites | As for early signs | – Brown snakes – Tiger snakes – Taipans |
| Signs of cerebral irritation (if intracranial haemorrhage) | | – Rough scaled snake – Broad headed snake – Pale headed snake – Stephens' banded snake |

Coagulopathy: Anticoagulant effects^{*1,2,14,16}

| Key early signs | Late signs | Consider |
|--|--------------------|---|
| Persistent ooze from bite site, gums or venepuncture sites | As for early signs | – Mulga snakes – Collett's snake – Black snakes |

*When coagulopathy is evident, it is important to distinguish (through laboratory tests) whether this is a consequence of defibrination (i.e. procoagulant) or anticoagulant effects of venom (as venom from different snake species cause different types of coagulopathy).

....continued overleaf

Snakebite: Diagnosis

Myolysis (systemic rhabdomyolysis)^{1,2,14,15}

| Key early signs | Late signs | Consider |
|--|--------------------|---|
| <p>Pain on contracting muscles against resistance</p> <p>Muscle tenderness</p> <p>Muscle weakness (may mimic paralytic signs)</p> <p>Dark urine which tests positive for blood on dipstick (most likely indicating myoglobin, not haemoglobin in this setting)</p> | As for early signs | <ul style="list-style-type: none"> – Tiger snakes – Mulga snakes – Collett's snake – Black snakes – Taipans – Rough scaled snake – Sea snakes – Copperheads |

Nephrotoxicity^{1,2,14}

| Key early signs | Late signs | Consider |
|--|--------------------|---|
| <p>Polyuria, oliguria or anuria (none of which are common)</p> <p>Increased creatinine and urea only</p> | As for early signs | <p>Any snake, but particularly:</p> <ul style="list-style-type: none"> – Brown snakes – Tiger snakes – Mulga snakes – Taipans – Rough scaled snake |

Local cytotoxicity^{1,2,4,17}

| Key early signs | Late signs | Consider |
|--|---|--|
| <p>Marked local swelling or mild local swelling with ecchymosis and pain</p> <p>Bites from some species may cause little swelling but there may be marked pain</p> | As for early signs. Local swelling may take 1-3 days to reach maximum extent | <ul style="list-style-type: none"> – Mulga snakes – Collett's snake – Black snakes – Tiger snakes – Taipans – Death adders |

Laboratory investigations

- Laboratory investigations are vital in assessing snakebite.
- Initial results may be normal – therefore it is essential to conduct repeat tests to capture envenoming that develops later, which can be symptom free.¹⁰
- **Laboratory investigations should be performed at regular intervals.**
A recommended scheme for serial investigations is as follows, based on ongoing and published research:¹⁰
 - First set at presentation to hospital.
 - If first set is normal, remove first aid (PBI), wait one hour, then repeat all tests.
 - If second set is normal, wait a further 3 hours and repeat all tests (in practice this will be around 6 hours post bite).
 - If third set is normal, repeat all tests at 12 hours post bite. If this last set of tests is normal **and** the patient shows no signs of envenoming (on physical examination), it is generally safe to discharge the patient.*
 - If at any stage the patient develops symptoms or signs suggestive of developing envenoming, consider immediately repeating all blood tests.¹
 - A clinical pathway chart containing additional information is shown on page 69.
- Table 11 on page 68 highlights the key laboratory tests for cases of confirmed or suspected snakebite.

*Recent research indicates that in the majority of envenomed patients, envenoming will develop and is detectable (via laboratory investigations and physical examination) within 12 hours post bite. However, serial monitoring over this period of time may yet potentially miss the small minority of cases where envenoming is delayed beyond 12 hours [10].

Also note that the dataset used to determine the above timings may not adequately represent some geographic regions. Local modification of the pathway may be required [10].

Initial results may be normal – therefore it is essential to conduct repeat tests to capture envenoming that develops later, which can be symptom free

Snakebite: Diagnosis

Table 11. Key laboratory investigations for snakebite^{1,10,12,13,18,19}

Coagulation studies [INR as minimum, but preferably INR, aPTT, fibrin(ogen) degradation products (FDP, d-dimer), and where available, **direct** fibrinogen levels]^{1,10}

Full Blood Count including blood film. [Blood film to be checked for schistocytes if there is evidence of systemic envenoming or if evidence/suspicion of haemolysis – e.g. falling haemoglobin or low platelet count]^{1,10}

Biochemistry (creatinine, urea, electrolytes, CK, bilirubin)^{1,10}

Snake venom detection (perform in selected cases only)

– The SVDK test should be performed when there are signs of systemic envenoming (i.e. if there are clinical or laboratory indicators of envenoming)^{10,12,13,18,19}

– In patients who are asymptomatic at presentation and exhibit no laboratory signs of envenoming, it is appropriate to cut a window in the bandage around the bite, swab the bite site, and store this swab refrigerated (not frozen) for later SVDK testing to be performed if the patient develops clinical or laboratory indicators of systemic envenoming.^{10,13} [The bite site should be rebandaged]

Laboratory investigations request form¹

Laboratory investigation requests for snakebite should always be marked URGENT.

It is advisable to include the following information on the request form to highlight urgency and ensure relevant results are received with haste (see box).

ATTENTION – THIS PATIENT MAY BE ENVENOMED.

Defibrination coagulopathy may have occurred.

Any coagulation tests **exceeding the maximum time** must be reported to the requesting Medical Officer immediately.

Clinical pathway for cases of suspected/confirmed snakebite

This foldout contains an evidence-based clinical pathway chart.¹⁰

The chart is designed to help methodically assess cases of suspected or confirmed snakebite to determine whether or not the patient has developed systemic envenoming (and consequently, the potential requirement for antivenom therapy).

Note: Local modification of the pathway may be required.

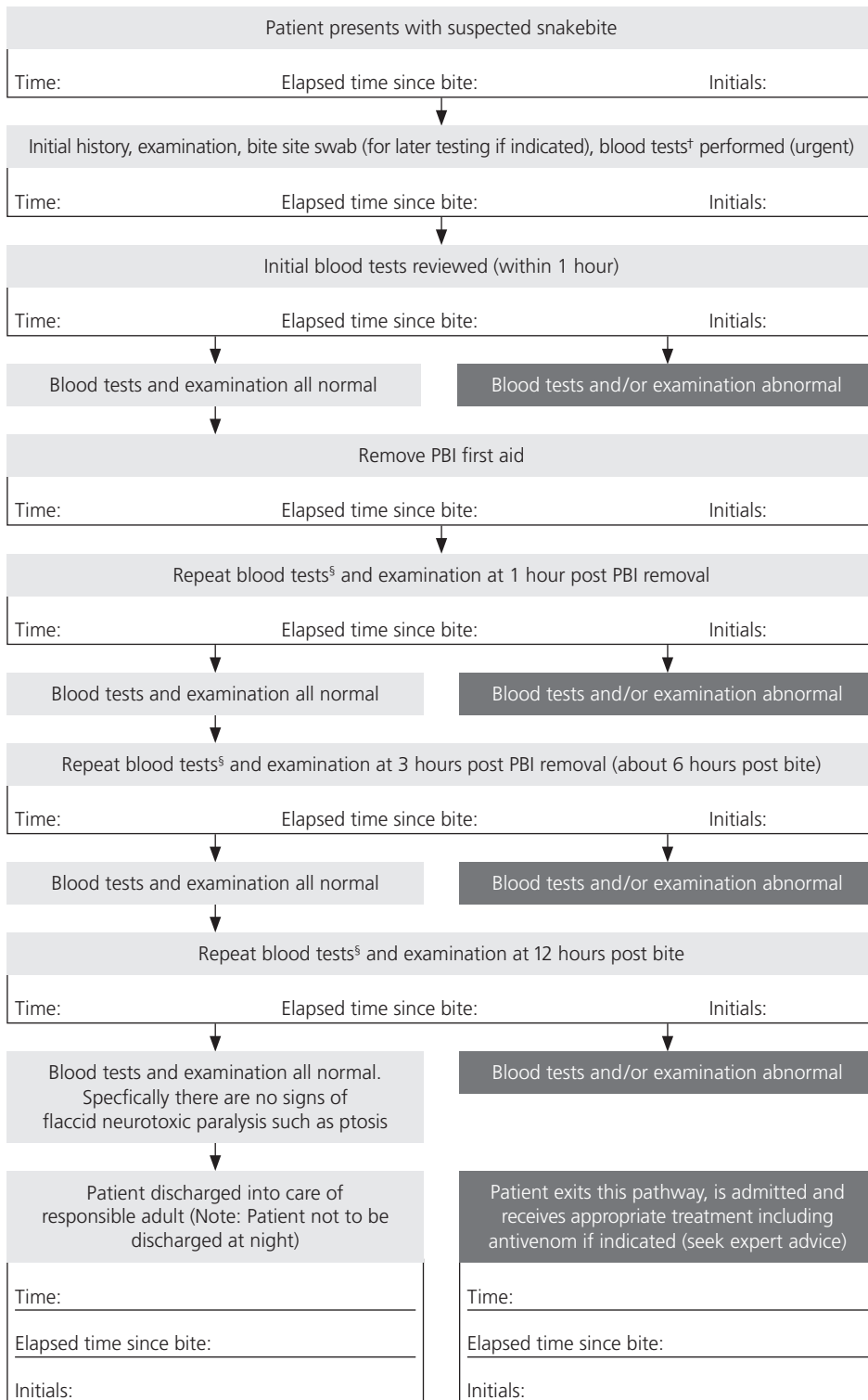
The chart may be copied for use.

Clinical pathway for suspected & confirmed snake bite:

All cases should be observed with serial blood testing and serial examination for 12 hours to exclude systemic envenoming using the following pathway.¹⁰ Frequent routine observations should continue throughout the 12 hour observation period (in addition to the formal testing noted below).

Surname: _____
 First name: _____
 DOB: _____
 UR: _____

Date: _____ Doctor: _____ Signature: _____



† Recommended initial laboratory investigations:
 Coagulation tests (INR, aPTT, FDP/d-dimer), FBC (+ blood film), CK, UEC.
 Swab bite area for snake venom detection (SVDK to be performed if there is clinical/laboratory evidence of envenoming)*

§ Recommended subsequent laboratory investigations:
 Coagulation tests (INR, aPTT, FDP/d-dimer), FBC, CK, UEC

Examination should include testing for flaccid neurotoxic paralysis, especially repeated testing for ptosis, ophthalmoplegia (especially failure of lateral & upward gaze) and vigilance for bulbar weakness (poor cough, gag, drooling), and limb weakness

*Venom detection using the SVDK can assist in the choice of the appropriate AV if AV therapy is clinically indicated. SVDK should not be used as a screening test to determine whether or not a patient has been bitten by a snake. Many health services now have a policy of performing SVDK only if there is clinical or laboratory evidence suggestive of developing envenoming

Snakebite: Diagnosis

Tests in rural hospitals with limited laboratory facilities

Point-of-care INR machines¹

- The increasing availability of point-of-care INR machines in country hospitals and medical practices to assess warfarin treatment has led to the suggestion that these machines may be a useful alternative for diagnosis of snakebite coagulopathy. Indeed many country hospitals rely on these machines to test for coagulopathy in snakebite cases.
- However, new evidence suggests that point-of-care INR machines may fail to diagnose snakebite coagulopathy as the mechanism of measurement is different to that of standard coagulation studies, and consequently, does not detect whether fibrinogen is being consumed. These machines can produce an apparently normal result even in the presence of massive defibrination coagulopathy in snakebite.
- Therefore, at this time, point-of-care INR machines cannot be recommended for measurement of snakebite coagulopathy. This remains an active area of study and longer-term recommendations may be expected in the future.

WBCT¹

- Whole blood clotting time (WBCT), and particularly the 20WBCT (see below) has for many years, been considered a simple test for detecting snakebite coagulopathy that can be performed in the absence of laboratory facilities, e.g. in a small country hospital.
- However, clinical experience in Australia indicates that WBCT may not be particularly reliable and may give false positives for coagulopathy. 20WBCT has not been tested for reliability in Australia, although is used successfully in PNG.

.....continued

Point-of-care INR machines may fail to diagnose snakebite coagulopathy as the mechanism of measurement is different to that of standard coagulation studies

- Nevertheless, if no other alternative is available, 20WBCT or WBCT should at least be considered, and in many cases, should be used.^{1,5}
- The test works on the principle that normal venous blood, when exposed to the surface in a small glass vessel (not plastic), is activated so that a clot will form, usually within 5-10 min.
- If there is snakebite coagulopathy with consumption of fibrinogen (procoagulant/defibrination effect), then clot formation will be impaired or absent.
- Clot formation also may be delayed or absent in the presence of anticoagulant coagulopathy.
- It is essential to use a clean **glass** vessel.
- Depending on the size of the vessel, 2-10 mL of venous blood is placed in it and the sample is checked at 20 minutes (20WBCT) or the time to clot is measured (WBCT). For 20WBCT leave the glass vessel undisturbed until checked at 20 minutes, at which time it should be inverted (if possible) to see if a clot has developed. If there is no clot at 20 minutes, the test is positive – indicating coagulopathy.
- To substantially increase reliability of interpretation, using a similar glass vessel, run a control sample from a normal volunteer at the same time (if available, use a sample from the patient's relative or friend).
- 20WBCT has been validated for coagulopathy from non-Australian snakebite.²⁰

SVDK

Laboratory facilities are not required when using SVDK. The only requirements are:¹¹

- A refrigerator (to store the kit reagents)
and
- A suitable wash liquid (saline or water).

Snakebite: Diagnosis

Interpreting laboratory test results

Coagulation studies^{2,5,14,16,21}

| Abnormal results | Interpretation | Snakes |
|--|---|--|
| Elevated to grossly elevated INR and aPTT and decreased to undetectable fibrinogen and elevated to grossly elevated degradation products of fibrinogen | Defibrination coagulopathy due to procoagulants (Venom Induced Consumption Coagulopathy = VICC) | <ul style="list-style-type: none"> – Brown snakes – Tiger snakes – Taipans – Rough scaled snake – Broad headed snake – Pale headed snake – Stephens' banded snake |
| Elevated to grossly elevated INR and aPTT but normal fibrinogen and degradation products | Anticoagulant coagulopathy | <ul style="list-style-type: none"> – Mulga snakes – Collett's snakes – Black snakes |

Full blood count (including blood film)^{1,3,22}

| Abnormal results | Interpretation | Snakes |
|--|--|--|
| Low haemoglobin, thrombocytopenia, schistocytes (fragmented red cells on blood film) | Microangiopathic haemolytic anaemia (MAHA) | Any snake, but particularly: <ul style="list-style-type: none"> – Brown snakes – Tiger snakes – Mulga snakes – Taipans – Rough scaled snake |

Creatinine/urea^{2,14}

| Abnormal results | Interpretation | Snakes |
|------------------|----------------|--|
| Elevation | Renal damage | Any snake, but particularly: <ul style="list-style-type: none"> – Brown snakes – Tiger snakes – Mulga snakes – Taipans – Rough scaled snake |

Creatine kinase (CK)^{1,2,4,14,15}

| Abnormal results | Interpretation | Snakes |
|---|-----------------------|--|
| Significant to gross elevation (10,000 to > 100,000 IU/L) Some cases with less severe myolysis will show CK in the range 1,500 to \geq 5,000. Rapidly rising CK is a marker for possible developing major myolysis | Systemic myolysis | – Tiger snakes – Mulga snakes – Collett's snakes – Black snakes – Taipans – Rough scaled snakes – Sea snakes |

Electrolytes^{1,2,14,15}

| Abnormal results | Interpretation | Snakes |
|-------------------------|--|---|
| Elevated potassium | Secondary hyperkalaemia associated with major myolysis and renal failure (beware cardiotoxicity) | – Tiger snakes – Mulga snakes – Collett's snake – Black snakes – Taipans – Rough scaled snakes – Sea snakes |
| Low sodium | Secondary hyponatremia | – Tiger snakes – Possibly other snakes |

Bilirubin^{1,3,22}

| Abnormal results | Interpretation | Snakes |
|-------------------------|---|--|
| Elevated | Associated with intravascular haemolysis (MAHA) | Any snake, but particularly: – Brown snakes – Tiger snakes – Mulga snakes – Taipans – Rough scaled snakes |

Snake venom detection

See pages 76-92.

Snakebite: Diagnosis

Determining the type of snake/venom involved: General principles¹

There are four key methods for determining the type of snake or venom involved.

1. Geographic location

Important for considering/excluding snake species based on the range of distribution of different snake species. However, bear in mind that the popularity of reptile collecting means that patients do, at times, present with bites from snakes well outside of the natural range of distribution.

For further information on distribution of venomous snakes in Australia see pages 143-167.

2. Clinical and laboratory features (diagnostic algorithms based on these features)

The pattern of toxic effects (e.g. coagulopathic; myolytic; neurotoxic) can vary for different snake venoms. Consequently, bites from different snake groups or species frequently produce distinguishing clinical/laboratory features of envenoming.

Paying careful attention to the patient's clinical picture is the key to diagnostic decision making in snakebite. To assist with this process, diagnostic algorithms have been developed based on clinical and laboratory features in envenomed patients. These algorithms are shown on pages 94-95. In addition, some key early signs of envenoming may assist in reaching a broad/indicative diagnosis (see pages 65-66).

Additional information on the clinical effects of venoms from Australian venomous snakes is available on pages 143-167.

Paying careful attention to the patient's clinical picture is the key to diagnostic decision making in snakebite

3. Snake venom detection – using bioCSL’s Snake Venom Detection Kit (SVDK)

In envenomed patients, SVDK plays a complementary role in assisting diagnosis. The primary purpose of SVDK is to guide the selection of the most appropriate monovalent antivenom to use if antivenom therapy is clinically indicated.

4. Identification of the actual snake (if available)

While it is **never** appropriate to attempt to catch or kill the snake as this increases the risk of multiple bites or another person being bitten, some patients or bystanders do present to hospital with the killed snake allegedly responsible for the bite. Efforts should be made to educate these individuals and actively discourage the repeat practice of catching/killing snakes.

If the culprit snake is available, this may be useful for determining snake identity. Snake identification should always be performed in consultation with an expert (conversely, it is inadvisable for clinicians to identify snakes without expert assistance).

Notably, even with expert guidance, misidentification can be a problem. Therefore, foremost, the clinician must be guided by the patient’s clinical features and must ensure that the identity of the snake, if determined, correlates with the clinical and laboratory features of the patient.

Importantly, clinical management of the patient should always take priority over snake identification.

Some basic information regarding snake identification is provided on pages 168-174.

The primary purpose of SVDK is to guide the selection of the most appropriate monovalent antivenom to use, if antivenom therapy is clinically indicated

Snakebite: Diagnosis

Snake venom detection: Fundamentals

- bioCSL's Snake Venom Detection Kit (SVDK) is a diagnostic assay used for Australian and a number of PNG snake venoms.¹¹
- In patients with symptoms/signs of envenoming requiring antivenom therapy, the SVDK assists in choosing the most appropriate neutralising monovalent antivenom.^{11,12,18} Importantly however, the test should not be used to confirm or exclude snakebite (i.e. as a screening test), or to determine whether the patient is envenomed.^{1,11,12,18}
- A bite site swab is the best sample for venom detection.¹¹
- In patients without evidence of envenoming at presentation, it is standard practice in many hospitals to perform a bite site swab and store refrigerated (not frozen) for later testing using SVDK if the patient develops clinical or laboratory indicators suggestive of developing envenoming.^{10,12,13,18,19} [Use a sterile saline-soaked swab for this purpose].*¹

*Note: The SVDK Product Leaflet provides instructions on the use of a swab soaked in Yellow Sample Diluent, or alternatively, suggests using a dry cotton swab [11]. The use of a saline-soaked swab is based on expert clinical experience [1].

- Urine may be used if systemic envenoming has occurred (use in non-envenomed patients can sometimes result in false positives).^{12,18} [Note: Venom can only be present in urine after entering the systemic circulation].
- A urine sample taken at presentation is useful for venom detection if the patient has signs of systemic envenoming at presentation.¹
- If a patient with confirmed snakebite does not show clinical or laboratory indicators of systemic envenoming at presentation, but if envenoming develops at a later stage (and if a bite site swab is unavailable):¹
 - A fresh urine sample taken **after** the development of systemic envenoming may be used for venom detection.¹

If the snake venom detection test is conducted on the urine sample taken at presentation, the result may be negative because the patient was not significantly envenomed at the time the sample was taken.¹

Note however that delayed urine sampling in envenomed patients may miss the period of venom clearance in urine and give rise to false negative results.¹⁸

bioCSL's Snake Venom Detection Kit (SVDK)

Purpose of SVDK

- The SVDK uses Enzyme Immunoassay to detect and identify the snake venom immunotype in cases of snakebite exhibiting clinical/laboratory signs of systemic envenoming. This assists in the selection of the most appropriate neutralising monovalent antivenom.¹¹
- Venom concentrations as low as 0.01 ng/mL are detectable via SVDK.¹¹
- A positive result in Wells 1 to 5 of the kit indicates the immunotype of the culprit snake's venom, to help select the most appropriate antivenom to be used if antivenom therapy is indicated.¹¹
- Note: SVDK does not identify the snake species – instead the test identifies the immunotype of the snake venom¹¹ (importantly, different snake species may share a common venom immunotype).
- A positive SVDK result is not an automatic indication to provide antivenom therapy.^{11,12} **Antivenom should only be used if there are clinical or laboratory indicators of systemic envenoming.**^{12,23-29}
- Conversely, a negative SVDK test should not form the basis for discharging the patient.^{12,18}
 - The decision to discharge should be based on adequate monitoring and ensuring that clinical/laboratory signs of systemic envenoming are absent.¹⁰
 - This includes serial investigations at specified times, including repeat laboratory testing and physical examination at 12 hours post bite and evaluation of the results prior to discharge (see 'laboratory investigations' on pages 67-69).¹⁰
- **SVDK test results should not be used in isolation for clinical decision making.**¹² Rather, the results should be used as an adjunct to clinical/laboratory features (i.e. diagnostic algorithms) and geography, to help optimise patient management.¹⁸ If the SVDK test result does not correlate with the clinical picture/geography, seek urgent expert advice.

Snakebite: Diagnosis

bioCSL's Snake Venom Detection Kit (SVDK) ... cont'd

When to use SVDK

- For cases of confirmed snakebite that have developed clinical symptoms/signs or laboratory indicators suggestive of developing systemic envenoming.*^{10,12,13,18,19}

*When a patient appears clinically well on examination, in order to improve efficiency, some hospitals prefer to send the bite site swab to the laboratory alongside the initial blood samples – with clear instructions that the SVDK test should be performed only if the initial laboratory tests indicate evidence of envenoming. If the patient is not envenomed, the laboratory is advised to store the swab refrigerated (not frozen) for testing later, if laboratory evidence of envenoming is noted at any stage during serial monitoring of the patient, or if the clinician advises that the patient has developed clinical symptoms/signs of envenoming [8].

- In patients presenting with signs of systemic envenoming of unknown aetiology and if history/examination/laboratory investigations suggest snakebite as the likely cause.¹

Note: SVDK is not designed to be used to confirm or exclude snakebite or to determine whether a patient is envenomed.¹¹ A negative SVDK result does not exclude a bite from a venomous snake.¹⁸ Conversely, a positive SVDK is not an automatic indication for the provision of antivenom.^{11,12,18} A thorough history and appropriate investigations are key to determining whether snakebite is the likely cause of signs/symptoms and the need for antivenom therapy.¹

What about patients who do not have evidence of systemic envenoming?

- If a patient is non-envenomed at presentation (based on history, examination and laboratory investigations), the bite site swab taken at presentation should be stored refrigerated (not frozen) for later SVDK testing, which is performed only if the patient shows clinical or laboratory indicators suggestive of developing envenoming.^{1,10,12,13,18,19}

This is now standard practice in many hospitals.^{1,19}

Used in this manner, the SVDK test results can be used as a complementary tool (alongside clinical/laboratory features and geography) to guide the choice of appropriate monovalent antivenom when antivenom therapy is clinically indicated.¹⁸

....continued

On the other hand, if the patient has remained non-envenomed for 12 hours post bite (based on serial clinical and laboratory investigations), SVDK testing of the stored bite site swab is not required (and certainly neither is antivenom therapy).¹ The patient may be discharged into the care of a responsible adult (do not discharge patients in the evening or at night).¹

- Some hospitals may still choose to perform the SVDK test on a bite site swab from a patient who appears non-envenomed at initial presentation (as a precautionary measure in the event that envenoming is precipitate upon removal of first aid).³⁰ Note: Many experts do not recommend this practice.^{1,10,12,13,18,19} In this instance:

A negative SVDK result **should not** be used as the basis for excluding envenoming and sending the patient home.^{12,18}

Conversely, a positive result is not an automatic indication for the provision of antivenom.^{11,12,18}

Instead, regardless of the SVDK test result, it is imperative to continue monitoring the patient's clinical and laboratory parameters at requisite intervals¹⁰ (see pages 67-69).

- If systemic envenoming does develop later, the initial positive SVDK result may be used to guide the choice of monovalent antivenom (provided the SVDK test result correlates with the patient's clinical features and with the geographic distribution of snakes within the region).^{1,18}
- If serial monitoring provides no evidence of systemic envenoming at 12 hours post-bite, the patient likely received a dry bite. The patient may be discharged regardless of the initial SVDK result¹⁰ (remember to always discharge patients into the care of a responsible adult and never discharge patients in the evening or at night).

Use SVDK for cases of confirmed snakebite that have developed clinical/laboratory evidence suggestive of developing envenoming

Snakebite: Diagnosis

bioCSL's Snake Venom Detection Kit (SVDK) ... cont'd

SVDK components¹¹

Each kit contains six main components, with enough material to perform 3 separate tests (Table 12). Running water, saline or buffered saline for the washing phase and a container for waste will be required. If the kit is to be used in transit in a medical retrieval plane or ambulance, carry a squeeze bottle of water for washing and a waste liquid container.

Table 12. Components of a single bioCSL Snake Venom Detection Kit¹¹

| Kit component | Purpose | Quantity in kit and how to use |
|-----------------------|---|---|
| Yellow Sample Diluent | For preparing the venom sample to be tested. Any venom sample MUST be mixed in diluent | 3 vials Use one vial for each test |
| Cotton Swabs | For venom sampling (used to swab bite site) | 3 swabs Use one for each test |
| Test Strips | These are the main component of the assay containing the attached antibody to detect venom | 3 strips Use one for each test |
| Strip Holder | Holds strip in place to assist with washing, and importantly, ensures correct orientation of the test strip | 1 strip holder Reuse for all three tests |
| Chromogen Solution | Reagent added during each test | 1 vial Reuse for all three tests |
| Peroxide Solution | Reagent added during each test | 1 vial Reuse for all three tests |



bioCSL's Snake Venom Detection Kit.

Step 1. Sample/specimen selection and preparation

Bite site swab

- If available, this is the best specimen for venom detection.¹¹
- Locate the bite site if there is first aid in place.
- To gain access, cut the bandage over the bite site only. Make sure no one washes the bite site.
- Venom may be detected in a swab from the bite site, from skin surrounding fang puncture marks, or from tissue exudate gently squeezed from the punctures.¹¹
- If the bite site swab is to be kept for later SVDK testing:¹
 - Use a sterile swab moistened with saline and thoroughly swab the bite site. Gently squeeze the bite site and swab any tissue exudate released. Do not squeeze roughly.

Remember to rebandage the bite site.

Store the swab refrigerated (not frozen) until ready for SVDK testing. Do not store the swab in wet or gel transport media.¹¹

When ready to perform SVDK test, prepare the bite site swab sample in Yellow Sample Diluent (see page 82).

Note: The SVDK Product Leaflet provides instructions on the use of a swab soaked in Yellow Sample Diluent, or alternatively, suggests using a dry cotton swab [11]. The use of a saline-soaked swab is based on expert clinical experience [1].

.....continued overleaf



Thoroughly swab the bite site.



Step 1: Prepare sample.

A bite site swab is the best specimen for venom detection

Snakebite: Diagnosis

Step 1. Sample/specimen selection and preparation ... cont'd

Bite site swab ... cont'd

- If the bite site swab will be tested immediately via SVDK:
Carefully remove the lid and dropper from an unused Yellow Sample Diluent vial and moisten the swab in the diluent.¹¹

Thoroughly swab the bite site. Gently squeeze the bite site and swab any tissue exudate released. Do not squeeze roughly.¹¹

Prepare in Yellow Sample Diluent as indicated below.

[Remember to rebandage the bite site].

- Preparing the bite site swab specimen in Yellow Sample Diluent:¹¹
Thoroughly agitate the swab in the diluent. The swab may then be removed and discarded or snapped off leaving the cotton section in the vial.
Replace the dropper and lid, and mix well by inverting several times.
- Alternatively, if there is clear evidence of systemic envenoming and the bite site is of poor quality, test urine.¹

Affected bandage or cloth specimen¹¹

- If the bite site is dry, a valuable sample may be obtained from affected portions of the bandage or clothing over the bite site.
- Cut a small sample of clothing or bandage (1-1.5 cm²) that has blood or tissue exudate on it. Keep refrigerated (not frozen), until ready for SVDK testing if indicated.
- Alternatively, soak the affected material in approximately 1 mL of water or saline to release any venom.
- Preparing the sample for SVDK test:
Remove the lid and dropper from an unused Yellow Sample Diluent vial and place the bandage or cloth specimen into the vial, or use a disposable pipette or syringe to transfer the washings into the vial.
Replace the dropper and lid, and mix the solution well by gently inverting several times.

Urine¹¹

- Carefully remove the lid and dropper from an unused Yellow Sample Diluent vial and without removing any of the vial's contents, fill to the neck with test urine using a disposable pipette or syringe.
- Replace the dropper and lid, and mix well by gently inverting several times.

Plasma or blood¹¹

- Blood should be used only if systemic envenoming is apparent and a bite site swab or urine sample is unavailable.
- Plasma or serum is preferred. However, whole anticoagulated blood is recommended in urgent situations as it does not require centrifugation and is therefore available more rapidly.
- Note: Erroneous reactions resulting in an invalid assay may occur if a whole blood specimen is tested.
- Remove the lid and dropper from an unused Yellow Sample Diluent vial and without removing any of the vial's contents, fill to the neck with serum, plasma or whole blood using a disposable pipette or syringe.
- Replace the dropper and lid, and mix well by gently inverting several times.

.....continued overleaf

Snakebite: Diagnosis

bioCSL's Snake Venom Detection Kit (SVDK) ... cont'd

Step 2. Preparing the Test Strip¹¹

- Place the test strip into the strip holder ensuring correct orientation. The test strip has a matching tag that fits into a slot in the strip holder to ensure correct orientation. Do not force the strip.
- The bottom well should be the Blank Well (well with no blue material) when the handle is pointing to the right hand side and the CSL logo is readable.
- Carefully remove the well sealing strip from the test strip. Avoid disturbing the contents of the wells.

Step 3. Adding the test sample¹¹

- Add **two drops** of the prepared test sample in Yellow Sample Diluent (yellow lid) into each well.
- **Gently** agitate the strip holder to reconstitute and mix the lyophilised conjugate with the test sample.
- Incubate for 10 minutes at room temperature (22° to 24°C).



Step 2: Prepare test strip.



Fit matching tag into slot for correct orientation.



Step 3: Add test sample.

Step 4. Removing the well contents¹¹

- After 10 minutes, flick the contents of the wells into a sink or waste container.

Step 5. Washing the Test Strip*¹¹

- Tap water, purified water, saline or buffered saline may be used. **Washing solutions that are hot, or contain high levels of contaminants (ie. bore water) or chlorine, should not be used.** If in doubt, purified drinking water or irrigation saline are recommended.
- Run the strip through a **gentle** stream of water or saline to wash the wells, ensuring the wells are thoroughly washed.
- Flick out the contents completely into a sink or waste container or tap out the strip onto high quality paper, tissue or Chux™ to ensure all the excess water is removed from the wells. **Paper hand towel must not be used as loose fibres may enter the test strip and may cause false positive reactions.**
- Repeat this procedure a minimum of **7 times for a bite site or urine sample and 15 times for plasma, serum, whole blood or other samples.** Urine samples displaying haematuria should be washed 15 times.
- After the last wash, ensure the washing fluids have been flicked and tapped out to remove any excess washing solution before proceeding.

*Note: Insufficient washing during this step may cause erroneous results.

....continued overleaf



Step 4: Remove well contents.



Step 5: Wash the wells.

Snakebite: Diagnosis

bioCSL's Snake Venom Detection Kit (SVDK) ... cont'd

Step 6. Add Chromogen Solution¹¹

– Add **one drop** of Chromogen Solution (blue lid) to each of the test wells.

Step 7. Add Peroxide Solution¹¹

– Add **one drop** of Peroxide Solution (grey lid) to each of the test wells.

– **Gently** agitate the strip holder to mix the Chromogen and Peroxide Solutions together.

– Place the test strip on the template provided on the product leaflet or on a white background and **observe the wells continuously over the next 10 minutes** whilst the colour develops. **The first well to show visible colour is diagnostic of the venom immunotype.** [If one well changes colour, several others may change colour later]. If snake venom is not detected, Wells 1 to 5 will not change colour.

Note: Strict adherence to the 10 minute observation period after addition of the Chromogen and Peroxide Solutions is essential. Slow development of colour in one or more wells after 10 minutes should not be interpreted as positive detection of snake venom.

Return relevant reagents, etc into the box and refrigerate. The reagents may quickly deteriorate if left out at room temperature for too long.



Step 6: Add chromogen.



Step 7: Add peroxide.

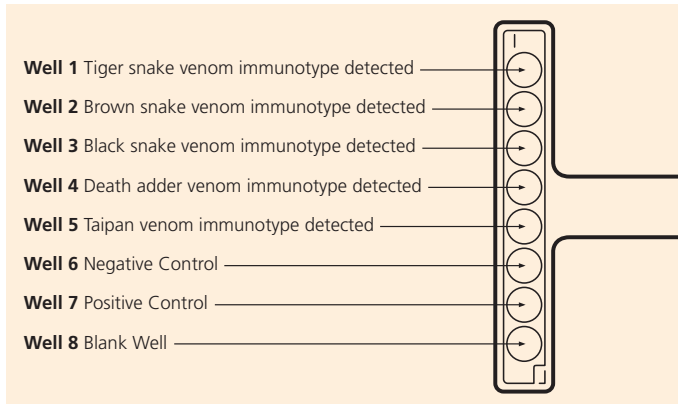
The first well to show visible colour is diagnostic of the venom immunotype

Step 8. Interpreting SVDK results¹¹

Test Validation

The SVDK has an in built Positive and Negative Control to ensure that each test gives a valid result. For the test to be valid the Negative Control (Well 6) should be visually clear, with no blue colour. The Positive Control (Well 7) should show rapid blue colour. This indicates that all SVDK components are active and performing correctly.

SVDK template



SVDK example result



SVDK test on a case of tiger snake bite showing positive colour change in Positive Control (Well 7) and Tiger snake venom immunotype (Well 1). Note also faint colour changes in some other wells.

Snakebite: Diagnosis

Table 13. Interpreting SVDK results – in all cases, for a valid result, Well 7 (Positive Control) must turn blue and Well 6 (Negative Control) must remain clear¹¹

| Colour change seen | Interpretation | Which antivenom to use if patient shows signs of envenoming? |
|--|---|--|
| No Colour – Negative Test [Wells 1 to 5 show no colour change. Only Well 7 (Positive Control) shows colour] | No venom has been detected from the five most clinically important venom immunotypes However, this result does not exclude snakebite | Polyvalent antivenom or a locally relevant monovalent antivenom or a combination of locally relevant monovalent antivenoms (e.g. in Victoria: Tiger Snake Antivenom and Brown Snake Antivenom). ²⁸ Seek expert advice regarding the choice of antivenom(s) in your region |
| Well 1 – Tiger Immunotype [Well 1 changes to blue first] | Venom has been detected of the Tiger Immunotype The SVDK may have detected venom from tiger snake, copperhead or rough scaled snake (also called Clarence River snake). Venom from broad headed, pale headed and Stephens’ banded snakes may occasionally give positive results in this well | Tiger Snake Antivenom |
| Well 2 – Brown Immunotype [Well 2 changes to blue first] | Venom has been detected of the Brown immunotype The SVDK may have detected venom from brown snake, dugite or gwardar | Brown Snake Antivenom |
| Well 3 – Black Immunotype [Well 3 changes to blue first] | Venom has been detected of the Black Immunotype The SVDK may have detected venom from mulga snake (king brown), Papuan black snake, red bellied black snake, spotted (or blue bellied) black snake, Butler’s mulga snake, pygmy mulga snake, or Collett’s snake ^{1,11} | Envenoming from all of these snakes can be treated with Black Snake Antivenom. However, this antivenom is best reserved for bites by mulga snakes (king brown, Butler’s mulga snake, pygmy mulga snake), Collett’s snake and the Papuan black snake ^{1,11} All other snakes mentioned respond well to Tiger Snake Antivenom (which is a lower-volume antivenom) If the species of the culprit snake is unknown, use Black Snake Antivenom |

| Colour change seen | Interpretation | Which antivenom to use if patient shows signs of envenoming? |
|---|--|---|
| Well 1 and 3 (Well 1 is lighter) [Well 3 changes to blue first with well 1 also showing visible blue colour although significantly lighter] | The Black Immunotype and Tiger Immunotype have some venom components in common. As a result, when Black Immunotype venoms are tested in the SVDK, Well 3 changes to blue first, with Well 1 also showing visible blue colour (but significantly less) This pattern indicates that venom has been detected from the Black Immunotype | See instructions for Black Immunotype in the table on the previous page |
| Well 4 – Death Adder Immunotype [Well 4 changes to blue first] | Venom has been detected of the Death Adder Immunotype The SVDK may have detected venom from any snake in the death adder group including the common death adder, desert death adder, Northern death adder, or Pilbara death adder | Death Adder Antivenom |
| Well 5 – Taipan Immunotype [Well 5 changes to blue first] | Venom has been detected of the Taipan Immunotype The SVDK may have detected venom from taipan, inland taipan (also called small-scaled or fierce snake) or Papuan taipan | Taipan Antivenom |
| Combination other than those listed above and on the table in the previous page | | Please contact bioCSL for advice on (03) 9389 2000 |

Note: Some other species of venomous Australian land snakes, not known to cause medically significant envenoming, may occasionally give a positive SVDK result [19]. Notable examples are whip snakes [31].

....continued overleaf

Snakebite: Diagnosis

bioCSL's Snake Venom Detection Kit (SVDK) ... cont'd

Interpreting SVDK results ... cont'd



Negative test – i.e. Wells 1 to 5 show no colour change. Only Well 7 (Positive Control) is positive.



Well 1 and 7 are blue – Tiger Immunotype.



Well 2 and 7 are blue – Brown Immunotype.



Well 3 and 7 are blue – Black Immunotype.



Well 3 and 7 are blue and Well 1 is lighter blue – Black Immunitytype.



Well 4 and 7 are blue – Death Adder Immunitytype.



Well 5 and 7 are blue – Taipan Immunitytype.

SVDK interpretation: Key points

- Always remember that a positive SVDK result for venom from the bite site does not mean the patient has been significantly envenomed.¹¹
- A positive SVDK from the bite site is not an indication to give antivenom. **It is an indication of the type of antivenom to give, if, on clinical grounds, the patient needs antivenom therapy.**¹¹
- For the test to be valid, Well 6 (Negative Control) must not change colour and Well 7 (Positive Control) must turn blue. Well 8 is blank.¹¹
- If Wells 1 to 5 show no colour change, then no venom has been detected.¹¹ A negative SVDK test should not be used in isolation to exclude snakebite. The patient should be monitored carefully (including laboratory testing) and decisions should be made on clinical grounds.^{1,10,12,18}

For the test to be valid Well 6 (Negative Control) must not change colour and Well 7 (Positive Control) must turn blue

Snakebite: Diagnosis

bioCSL's Snake Venom Detection Kit (SVDK) ... cont'd

SVDK: Common pitfalls¹

Operator error can be a problem when performing SVDK and it is important to avoid a number of common mistakes (see box). A training tool (bioCSL's Venom Practice Kit and an on-line instructional video) is available to help develop competency in the use of the SVDK to reduce the risk of operator error.

Common pitfalls of SVDK¹

- Failure to collect an adequate specimen (failure to moisten the swab particularly if the bite site is dry, or rubbing the swab too lightly, or over too small an area, or on the wrong area).
- Failure to use the Yellow Sample Diluent as the test fluid for the system.
- Placing the Test Strip the wrong way in the strip holder – this error will cause the Positive Control to appear to be a positive for Brown Immunotype. There is a tag and slot to help prevent this mistake.
- Failure to adequately wash the test strip after the first incubation phase.
- Use of paper hand towels to remove excess water from wells (loose fibres may enter the test strip and cause false positive reactions)¹¹
- Failure to continuously observe the test strip during the second incubation phase. It is essential to see which well turns blue first if venom is present – as multiple wells may turn blue during the 10 minute period.
- Using out-of-date kits or in-date kits that have either been inappropriately stored at room temperature, or been left out on a warm benchtop for a prolonged period when last used.
- Using the wrong sample (e.g. testing urine in a patient without apparent systemic envenoming).
- Misunderstanding the purpose and limitations of the kit, such as trying to use it as a screening test for snakebite or envenoming, or over reliance on the test results (instead of checking to ensure the SVDK result correlates with the patient's clinical/laboratory features and the geographic distribution of snakes).

Diagnostic algorithms: Determining the most likely type of snake involved¹

When managing a patient with envenoming, seek advice from an experienced clinician.

If skilled help is not available locally, access to experienced clinicians is available through the relevant contacts listed on page 3 of this handbook.

- Diagnostic algorithms for Australian snakebite are based on the patient's clinical and laboratory features, and consequently are designed to work in cases where significant systemic envenoming is present.
- As their only purpose is to help with the choice of antivenom, **the algorithms are only relevant in systemically envenomed patients and cannot be applied to patients who are not envenomed.**
- The algorithms have been formulated to cover common patterns of envenoming for each snake group, but cannot cover every situation.
- Atypical cases/venom effects may cause the algorithm to fail.
- Therefore, diagnostic algorithms are best used in conjunction with geography (i.e. location where bite occurred) and snake venom detection, which then allows a higher degree of confidence in determining the most likely snake venom immunotype involved – and therefore, the choice of an appropriate antivenom.
- Diagnostic algorithms based on clinical and laboratory features in snakebite are provided on pages 94-95.

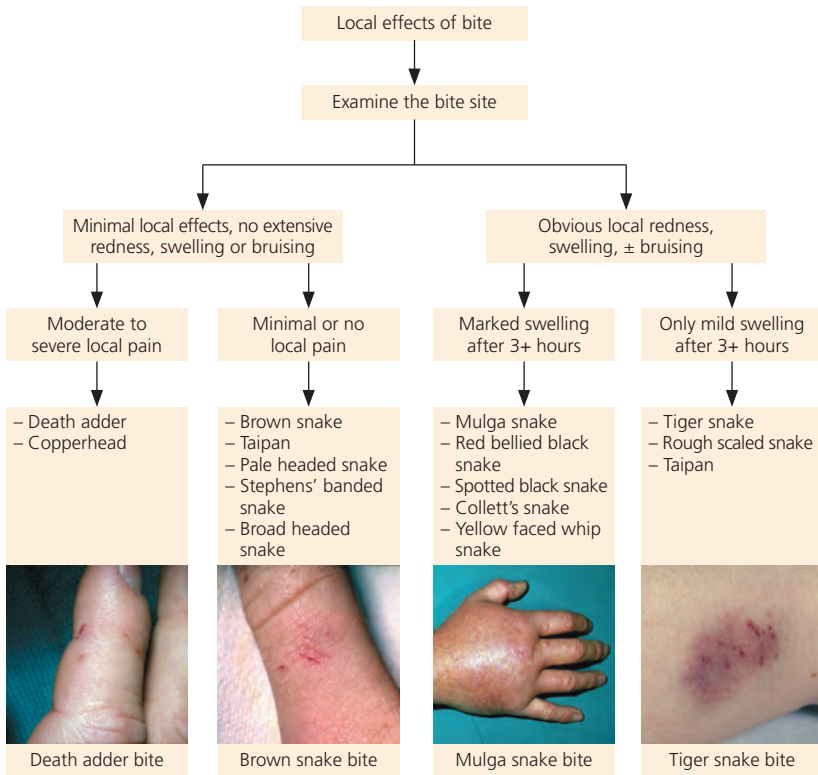
.....continued overleaf

Diagnostic algorithms for Australian snakebite are designed to work in cases where significant systemic envenoming is present their purpose is to help with the choice of antivenom

Snakebite: Diagnosis

Diagnostic algorithms: Determining the most likely type of snake involved¹ ... cont'd

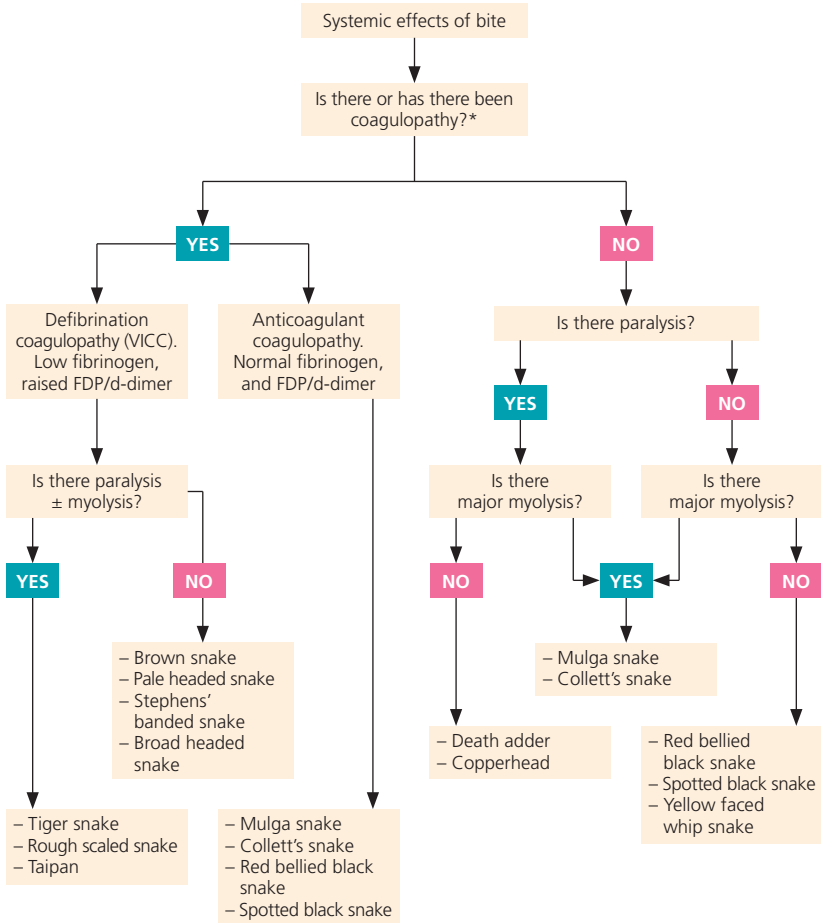
Determining the most likely snake based on local effects of the bite



Note: Combine the information above with the result of the systemic effects chart on page 95 to obtain a best guess for the type of snake most likely to have bitten the patient. The diagnostic process may be aided by matching this with known snake fauna for the geographic region where the bite occurred. In addition, since the patient is systemically envenomed, SVDK may be a useful adjunct test to assist with choosing the type of monovalent antivenom required if antivenom therapy is clinically indicated.

Please note, the chart cannot cover all possible situations and assumes an understanding of the symptoms and signs of local, general and specific envenoming by Australian venomous snakes (see pages 51-52 & 65-66). If in doubt, seek advice from relevant sources listed on page 3 of this handbook.

Determining the most likely snake based on systemic effects of the bite



*Resolving coagulopathy may still be evident through elevated d-dimer/FDP.

Snakebite: Diagnosis

Identifying a snake specimen¹

- Occasionally, the patient or bystanders may have killed the culprit snake.
- This specimen is potentially very valuable as an absolute confirmation of the species involved (however, expert assistance must be sought). If the snake has been killed, always request it be presented with the patient.
- **Note, bioCSL does not in any way condone the practice of capturing and/or killing snakes. Patients and bystanders should always be strongly advised against the practice of catching and killing snakes.**
- However, if the snake is presented with the patient, the availability of the specimen does provide an additional path to diagnosis (with the assistance of an expert).
- To prevent putrefaction, dead snakes can be immersed in a large container of surgical alcohol. Identifying snakes is often difficult and requires considerable expertise.
- However, using some key parameters, a health professional untrained in snake identification may be able to identify a snake by working with an expert in another location (e.g. by phone/email).
- Further information regarding snake identification is provided towards the end of Section 3 (pages 168-174).
- Additionally, for descriptions of venomous snakes in Australia see the overview of Australian venomous snakes (pages 143-167).
- Importantly, even with expert assistance, misidentification is possible. Hence, monitoring the patient's clinical status should always take priority over snake identification.
- If the identification result does not fit with the clinical picture, it is prudent to be guided by the clinical features rather than the purported identity of the snake.

Treating snakebite: Fundamentals

Basic principles of management^{1,32-34}

- Snakebite is a potential **medical emergency**. Cases should always receive high priority assessment and treatment, even if patients appear to be well initially.
- The majority of snakebites will not result in significant envenoming^{33,34} and these cases will not require antivenom therapy.
- Pay particular attention to patients with a history of multiple bites and/or early collapse, as major envenoming will occur in most of these cases – sometimes requiring increased amounts of antivenom.
- Admit all cases of probable snakebite for at least 12 hours after the bite or after removal of effective PBI first aid, or overnight, preferably to a suitable clinical unit such as emergency short stay, high dependency or ICU.
- **Patients with suspected snakebite should never be discharged in the evening or during the night or to a situation where no other adult is able to observe the individual over the following 24 hours.**
- Cases should be managed only in hospitals fully equipped to do so – i.e. with on-site laboratory facilities that can perform the requisite investigations, adequate stocks of appropriate antivenom, and a clinician who is able to manage the patient.

.....continued overleaf

Admit all cases of probable snakebite for at least 12 hours after the bite or after removal of effective PBI first aid, or overnight.... preferably to emergency short stay, high dependency or ICU

Treating snakebite: Fundamentals

Basic principles of management ... cont'd

- If transfer to a higher level of care is required, organise this early in the patient's presentation.
 - If first aid is not in place, apply PBI first aid when planning patient transfer even if the patient is asymptomatic and without laboratory indicators of envenoming.
 - A number of steps must be undertaken when organising patient transfer. These are detailed on pages 115-117.
- Antivenom is a specific antidote for venom and is a key tool when managing significant envenoming.^{14,23-29}
- However, antivenom therapy can be associated with adverse reactions and therefore should only be used when clearly indicated,^{14,23-29} but when indicated, antivenom therapy should never be withheld because of fear of those adverse reactions.^{1,14}
- Prior to administering antivenom, seek expert advice. Access to experts is available through relevant contacts shown on page 3 of this handbook.

Snakebite: Urgent treatment^{1,14}

1. ABC: Ensure adequate respiratory and cardiac function.
2. Assess if effective PBI first aid is in place. If not – apply PBI first aid.
3. Insert I.V. line and provide fluid load.
4. Take blood.
5. Perform key history and examination diagnostic process.
6. Take a bite site swab.
7. Test urine.
8. Urgently obtain and assess blood test results and combine with history and examination to determine if significant systemic envenoming is present.
9. Administer appropriate further treatment.

See pages 99-100 for details.

Antivenom is a specific antidote for venom and is a key tool when managing significant envenoming

Treating snakebite: Urgent treatment

Snakebite: Urgent treatment^{1,10,13,14,18}

1. **ABC: Ensure adequate respiratory and cardiac function¹**

Assess and maintain airway. Provide respiratory support as indicated. Respiratory and cardiac support takes priority over all other interventions including antivenom. However, in cases of significant envenoming, early antivenom therapy is likely to be required.

Note: If the patient already has signs of developing flaccid paralysis, antivenom treatment may be urgently required in an attempt to prevent progression to full respiratory paralysis. In this setting, there may not be enough time to obtain blood test and snake venom detection results before commencing antivenom therapy. If the identity of the snake is uncertain, either a mixture of appropriate monovalent snake antivenoms or bioCSL's Polyvalent Snake Antivenom may be indicated. If possible, consult a clinical toxicologist for advice (see page 3 for contact details).

2. **Assess if effective PBI first aid is in place¹**

If not, immediately apply PBI first aid.

3. **Insert I.V. line and provide fluid load^{1,14}**

Choice of crystalloid is not critical (e.g. normal saline; Hartmann's solution). Avoid fluid overload, particularly in children and adults with cardiac compromise.

4. **Take blood^{1,14}**

See page 68 for required tests. Mark laboratory investigations request as 'Urgent'.

5. **Perform key history and examination diagnostic process¹⁴**

(see pages 60-66).

.....continued overleaf

Treating snakebite: Urgent treatment

Snakebite: Urgent treatment^{1,10,13,14,18} ... cont'd

6. Take a bite site swab¹⁴

If the location of the bite site is known, cut the bandage around bite site for inspection and swab for venom.¹⁴ If on examination the patient appears significantly envenomed, submit the swab for urgent venom detection. If the patient does not appear to be envenomed, store the swab refrigerated (not frozen), to submit for venom detection if there are laboratory indicators of envenoming or if the patient develops clinical or laboratory signs of envenoming at a later stage.^{1,10,13,18}

7. Urine testing¹

Dipstick test.

Keep urine for possible snake venom detection later if indicated (if delayed envenoming occurs, obtain a new urine sample for venom detection).

8. Urgently obtain and assess blood test results and combine with history and examination to determine if significant systemic envenoming is present¹⁴

If significant systemic envenoming is present, consider antivenom therapy before removal of first aid.¹⁴

If there is no evidence of significant envenoming and antivenom is readily available, remove first aid and re-evaluate over the next few hours.¹⁴ In this latter setting, repeat examination and laboratory investigations will be required at regular intervals (see page 67).¹⁰

9. Administer appropriate further treatment¹

See 'General treatment' of snakebite (pages 107-110).

If indicated provide 'Specific treatment with antivenom' (pages 111-134).

Also manage venom-induced toxicity (see pages 135-142).

Snakebite management charts/algorithms^{1,14,23-29}

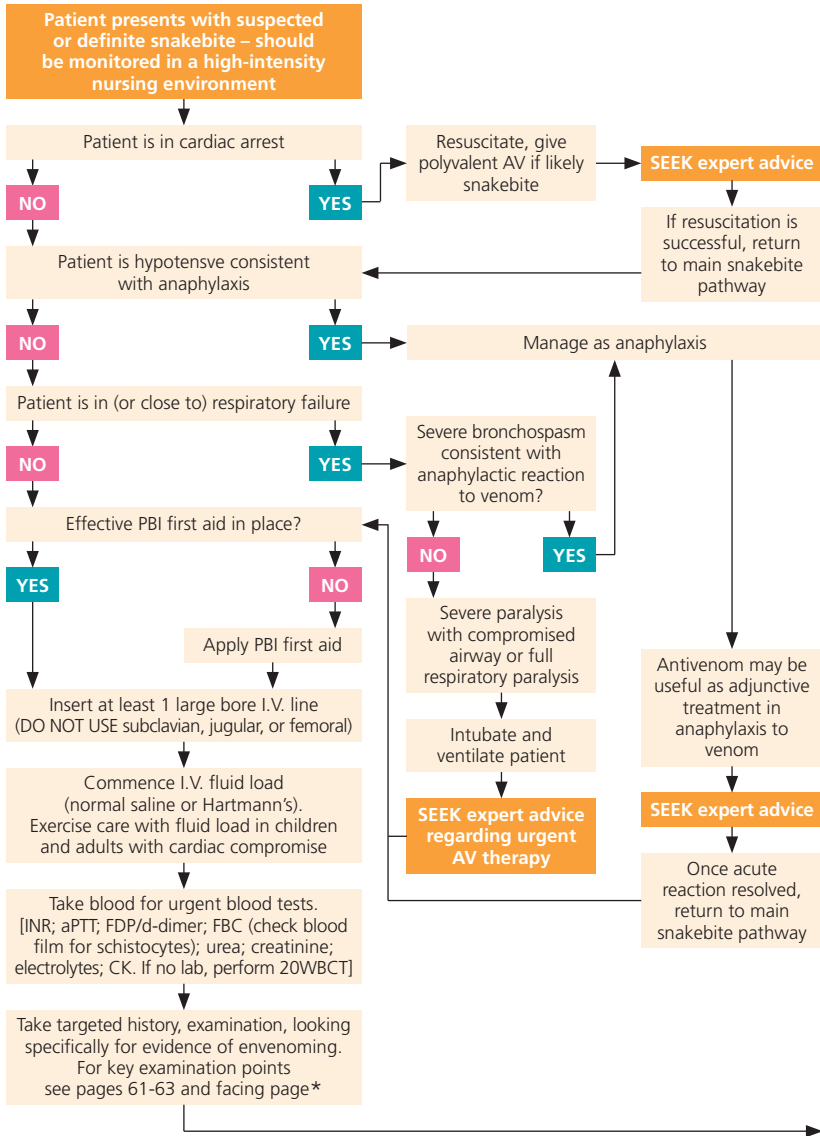
The following pages 102-106 depict a diagrammatic overview of managing snakebite based on clinical features at presentation.¹

The algorithms provide a general summary of the treatment pathway, but do not (and cannot) cover all possible clinical presentations/problems.¹

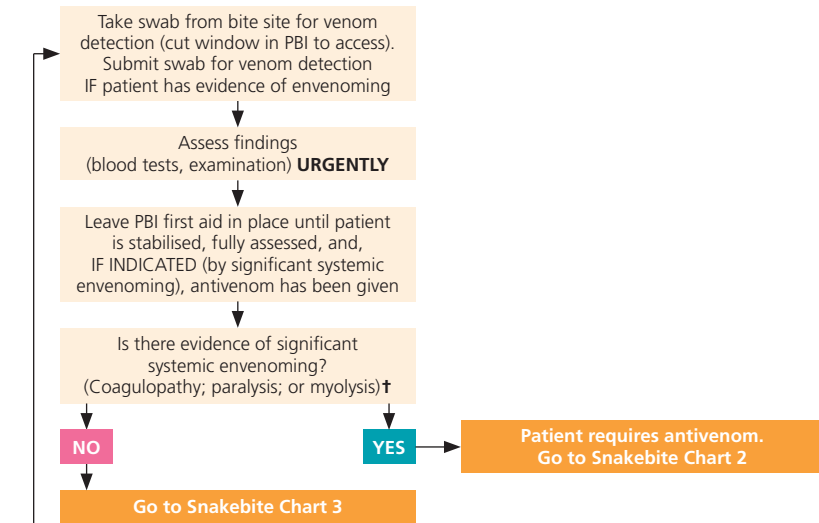
Antivenom is a specific antidote for venom and is a key component in managing significant envenoming. However, antivenom therapy can be associated with adverse reactions, so it should only be used where clearly indicated,^{14,23-29} but when indicated, should never be withheld because of fear of those adverse reactions.^{1,14}

In addition to urgent treatment of snakebite cases, methodical general treatment is required. Detailed information regarding additional general treatment of snakebite and specific treatment with snake antivenom is provided on pages 107-134 following the snakebite management algorithms.

Snakebite management chart 1



Continued on next page

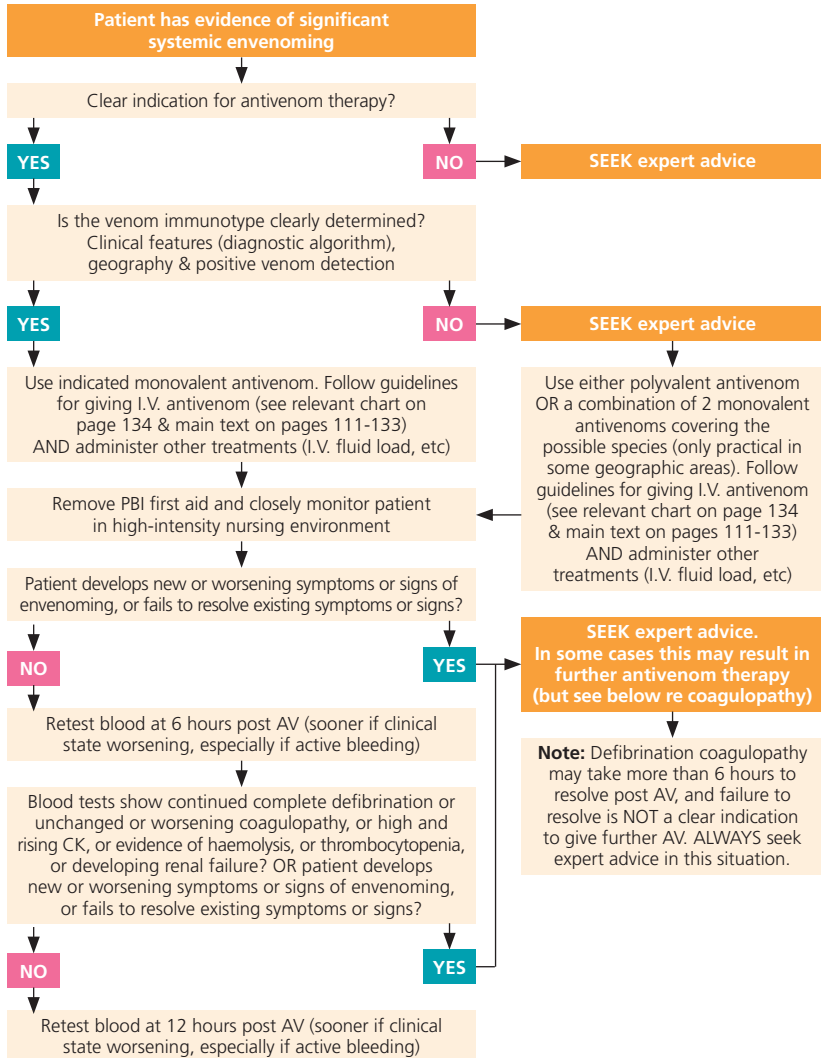


*** Key examination points:**

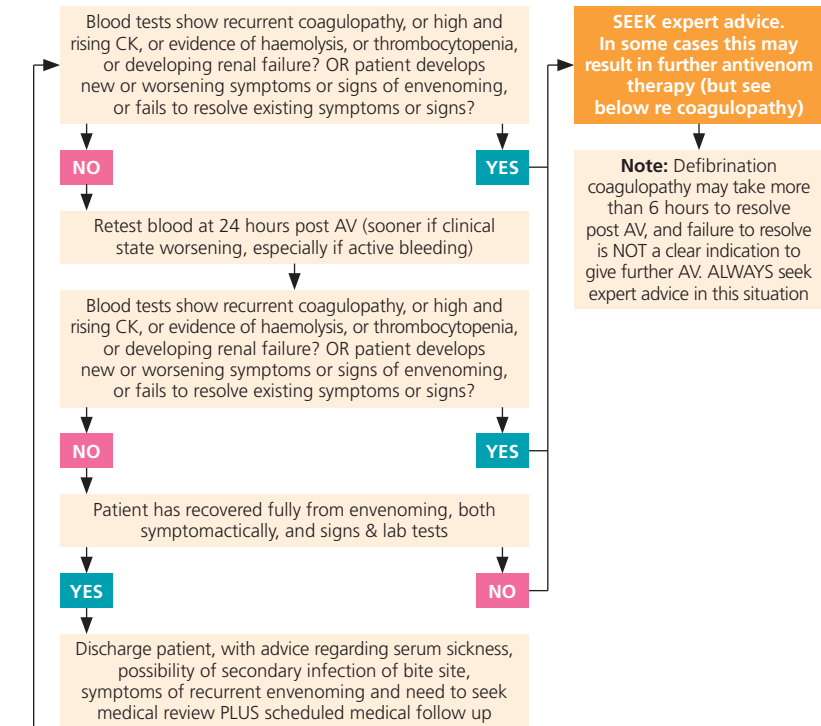
- Paralysis – ptosis; other cranial nerve deficits; limb weakness; respiratory/airway compromise.
- Coagulopathy – persistent oozing blood from bite site, venepuncture sites and/or gums.
- Myolysis – muscle pain, tenderness or weakness.

† **Indications for antivenom** – see page 112 for details.

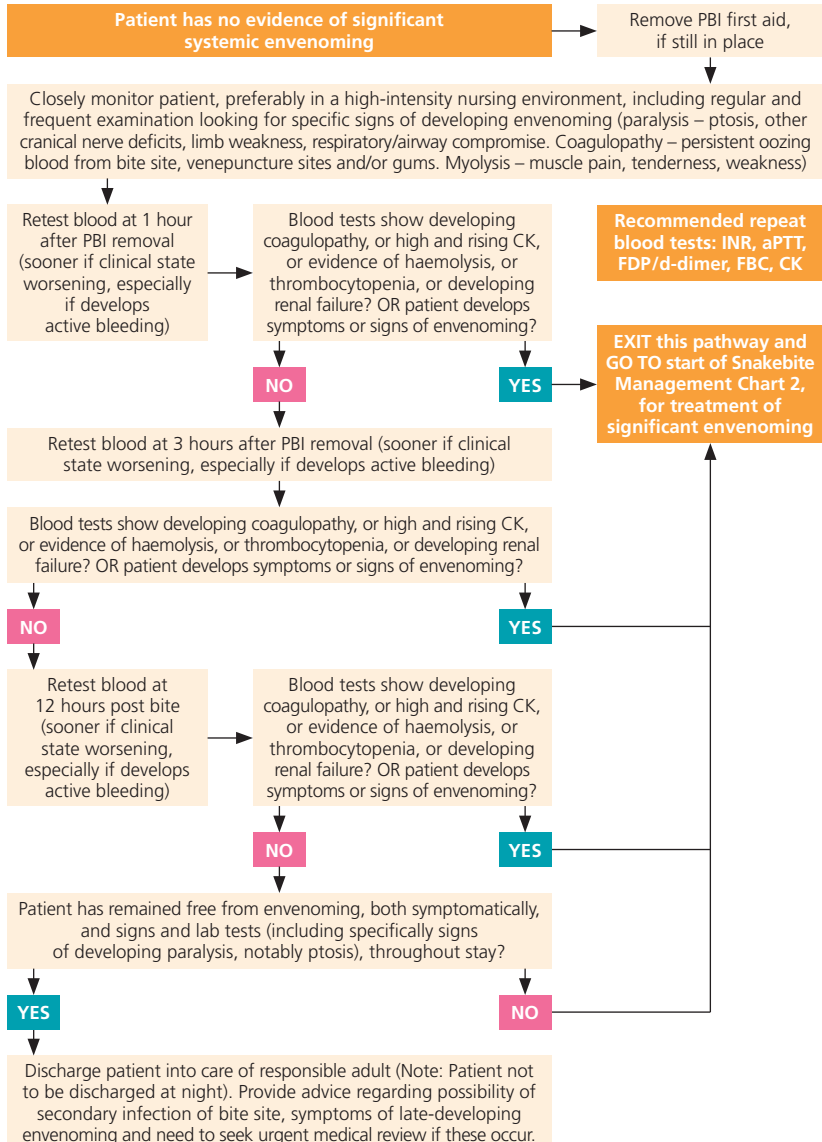
Snakebite management chart 2



Continued on next page



Snakebite management chart 3



Snakebite: General treatment

1. Continue to monitor ABC (Airway; Breathing; Circulation)

Intubate if there is respiratory failure or distress.¹

2. Intravenous line and I.V. fluid load¹

Degree of fluid load will depend on the age and size of the patient and any pre-existing medical conditions. Beware fluid overload in young children and elderly patients with pre-existing cardiac problems.

If there is evidence of renal failure despite adequate I.V. fluid hydration – consider haemodialysis.

Be cautious when selecting I.V. sites and other injection sites^{2,14}

– Snakebite coagulopathy can cause major bleeding from I.V. sites and into I.M. injection sites. Do not give I.M. injections, e.g. for tetanus booster, until any coagulopathy has been reversed.

– Some I.V. sites have a high potential for complications with coagulopathy. In general, avoid using subclavian, jugular or femoral sites.

3. Monitoring^{1,2,10}

As the degree of envenoming can change, sometimes rapidly, it is essential to regularly re-evaluate snakebite patients, even if they seem initially well and non-envenomed.^{2,10}

Key parameters include HR, BP, RR, O₂ saturation (pulse oximeter); PEFR (if neurotoxic paralysis is a potential risk); cardiac monitor (if available); check for ptosis and partial ophthalmoplegia; ensure complete fluid balance charts are maintained.^{1,2}

Laboratory tests are vital for snakebite and must be performed at specific intervals (for details see pages 67-69).¹⁰

.....continued overleaf

Snakebite: General treatment

3. Monitoring^{1,2,10} ... cont'd

Until fully assessed and the patient is evaluated as currently non-envenomed, nil orally (to minimise the risk of venom-induced vomiting causing aspiration pneumonia). Thereafter clear fluids only for at least 6 hours post bite.¹

Encourage patients to micturate voluntarily as catheterisation increases the chance of secondary infection and can be problematic in patients with coagulopathy.¹

4. Patients with myolysis^{1,2}

In patients with myolysis it is particularly important to maintain good renal throughput by providing an ongoing I.V. fluid load.²

Monitor and manage hyperkalaemia if present.¹

Alkalinisation of urine in cases with myolysis from other causes has been suggested. However, it is neither supported, nor refuted by current evidence in snakebite.¹

5. Patients with severe paralysis^{1,2,14}

Particularly in cases requiring mechanical ventilation, it is important to try and establish ongoing communication with the patient, even at the simple "yes/no" level, through movement of some incompletely paralysed part of the body. This is very reassuring for these patients who may seem obtunded, but may actually be awake and fully aware of their surroundings.¹

Progressive neurotoxic flaccid paralysis can result in full respiratory paralysis and loss of airway protection, which endangers respiration much earlier.^{2,14}

– In this setting, it is important to secure the airway, often by intubation, and maintain adequate respiration with mechanical ventilation.

– Tracheostomy is highly **inadvisable** in this setting and is clearly contraindicated if there is any possibility of venom-induced coagulopathy.

6. Analgesia^{1,2,14}

Most patients with snakebite will not have significant pain.² Presence of severe pain in the bite area underneath PBI first aid suggests the possibility that first aid is too tight.¹

Note: If a previously well patient with snakebite develops generalised pain (myalgia), this may indicate developing myolysis, which requires urgent reassessment and appropriate management before considering analgesia.¹

If analgesia is required, avoid narcotic analgesics, as these may interfere with respiration or may induce drowsiness.^{2,14}

7. Antihistamines¹

Rarely indicated in snakebite. If antihistamines are required, avoid sedation.

8. Antibiotics^{1,2,4,14,16,17,35}

Secondary infection after Australian snakebite is uncommon to rare and use of prophylactic antibiotics is inappropriate.^{2,14} If secondary infection of the bite area develops, conduct culture and sensitivity testing prior to commencing appropriate antibiotic therapy.¹

A few species of snake typically cause a venom-induced chemical cellulitis in the bitten limb, with erythema, pain, swelling, and sometimes, tenderness – particularly bites by the black snake group. This chemical cellulitis may peak 1-3 days post bite and slowly resolves over subsequent days.^{4,16,17,35}

Note: Tetanus immunity should be updated in cases of snakebite, but only after coagulopathy has resolved (to avoid muscle haematoma) [2,14].

9. Discharging patients who do not develop evidence of envenoming^{1,10,32,36}

All patients with definite or suspected snakebite, who do not show evidence of envenoming at any stage, should be observed in hospital for a minimum of 12 hours,¹⁰ and should not be discharged in the evening or at night, but kept in hospital until the following morning.³²

.....continued overleaf

Snakebite: General treatment

9. Discharging patients who do not develop evidence of envenoming^{1,10,32,36} ... cont'd

Before discharge, repeat examination and laboratory testing must be performed at or just prior to the 12-hour mark.¹⁰ [Note: Previously, some authors suggested that 6 hours post bite was sufficient time to exclude late envenoming,³⁶ but available evidence (published and unpublished) does not support this view].^{1,10}

Among cases that do develop significant envenoming the vast majority will do so within the 12-hour window.¹⁰ **However, a small minority may not develop any evidence of envenoming until after the 12 hour period – sometimes as long as 24 hours.** Ensure patients discharged before 24 hours will be in an environment where some other responsible adult can observe them.^{1,10}

10. Discharging patients who develop systemic envenoming¹

For patients who have developed evidence of envenoming, even those with minor envenoming not requiring antivenom therapy, a longer period of in-hospital monitoring (i.e. greater than 12 hours) is mandated.

If antivenom has been given, at least 24 hours should elapse after the last antivenom dose before considering discharge.

Patients who developed systemic envenoming should not be discharged until it is clear that envenoming has resolved. However, certain complications of envenoming, such as loss of taste/smell, minor slow-resolving (and isolated) ptosis, may take longer than 24 hours to resolve (sometimes days to weeks, or even months) and it is not appropriate to keep such cases in hospital during this prolonged convalescence.

If antivenom has been given, at least 24 hours should elapse after the last antivenom dose before considering discharge... [discharge only if it is clear that envenoming has resolved]

Specific treatment of snakebite: Antivenom

Antivenom therapy for snakebite^{1,2,14,23-29}

- The key to managing significant systemic envenoming from snakebite is the use of specific antidote – namely antivenom.^{14,23-29}
- There is little doubt that antivenom has been a major factor in reducing snakebite mortality in Australia.¹⁴
- However, antivenom cannot reverse all types of envenoming. It is clearly most efficacious when used early,² but also has an adverse effects profile that prevents it being used in every case of suspected snakebite irrespective of the degree of envenoming.²³⁻²⁹
- It follows that when managing snakebite, the key question is ‘does this patient require antivenom therapy – i.e. is significant envenoming present?’ If yes, the related question is the choice of antivenom and dose.^{1,2}
- Pages 112-134 provide details regarding antivenom administration including indications, timing, choice of antivenom and the management of any adverse reactions.
- When antivenom therapy is indicated, please seek expert advice (see relevant contact details on page 3 of this handbook).

When managing snakebite, the key question is ‘does this patient require antivenom therapy – i.e. is significant envenoming present?’ If yes, the related question is the choice of antivenom and dose



bioCSL's snake antivenoms.

Specific treatment of snakebite: Antivenom

Indications for antivenom^{14,23-29}

- Antivenom therapy is indicated for snakebite if there are signs of significant systemic envenoming (see box).^{14,23-29}
- If there is evidence of significant systemic envenoming, seek expert advice.
- Patients with severe headache, vomiting or abdominal pain, but without any of the indications listed in the box should not normally receive antivenom.¹⁴
- However, antivenom may be considered in cases of intractable vomiting that is not responsive to anti-emetic therapy.¹⁴

Indications for antivenom therapy for snakebite¹⁴

If one or more of the following occur:

- Any degree of flaccid paralysis including ptosis.
- Any degree of significant myolysis (myoglobinuria, or CK > 1500 IU/L).
- Any degree of significant coagulopathy (INR > 2; active bleeding).
- Evidence of renal damage (creatinine/urea above normal range for patient, or renal output abnormality).
- Documented collapse and/or convulsions in a witnessed snakebite.

Note: Patients should receive antivenom therapy at the earliest sign of neurotoxic paralysis to prevent extension of paralysis (pre-synaptic paralysis cannot be reversed with antivenom).^{2,14}

Timing of antivenom therapy¹

- It may never be too late to give antivenom in the presence of severe envenoming.
- The greater the delay between onset of envenoming and antivenom commencement, the less likely a good outcome will be achieved.
- Where more than 24 hours has elapsed since the bite, the risks of continuing severe envenoming need to be weighed against the risks of antivenom therapy and the uncertainty of response to that antivenom. Seek expert advice.

The greater the delay between onset of envenoming and antivenom commencement, the less likely a good outcome will be achieved

Choice of antivenom^{1,14,23-29}

- Wherever possible, it is preferable to use specific monovalent antivenom rather than polyvalent antivenom as the volume given usually will be lower and therefore the risk of adverse effects may be reduced.^{14,23-29}
- However, it is critical that the antivenom administered fully covers the snake species involved. **Thus choosing when to use a specific monovalent antivenom, and which antivenom, is a key management issue for Australian snakebite.¹**
- The possible methods for determining the type of snake or venom have been discussed on pages 58-96.
- The methods include a description of the snake (often unreliable), identification of a dead snake (rarely available and requires expertise), geographic location (assists within limits, towards narrowing the species under consideration to those found in the area), diagnostic algorithms based on clinical features, and snake venom detection (convenient but not infallible).¹

When to use bioCSL's Polyvalent Snake Antivenom¹

1. Where identification of the snake either fails or is impractical (e.g. in a severely envenomed patient requiring urgent antivenom treatment) it may be necessary to use bioCSL's Polyvalent Snake Antivenom, which will cover the five snake venom immunotypes predominating in land snakes in Australia and PNG.
 Alternatively, in some regions, it may be appropriate to use a mixture of two monovalent snake antivenoms, if the range of possible species is limited and the combined volume of the monovalent antivenoms is significantly less than that of bioCSL's Polyvalent Snake Antivenom. For most regions, pre-determining the practicality and choice of such a mix of monovalent antivenoms is advisable, but is beyond the scope of this handbook. Please seek expert advice.

.... continued overleaf

[Where diagnosis is impractical] e.g. in a severely envenomed patient requiring urgent antivenom therapy, it may be necessary to use bioCSL's Polyvalent Snake Antivenom

Specific treatment of snakebite: Antivenom

When to use bioCSL's Polyvalent Snake Antivenom¹ ... cont'd

2. In many parts of Australia, if the identity of the snake remains undetermined, the diversity of snake species in the area will make bioCSL's Polyvalent Snake Antivenom the obvious choice. Conversely, there are regions where the venomous snake fauna is limited and a single monovalent antivenom will cover all species (e.g. Tasmania and Kangaroo Island – use bioCSL's Tiger Snake Antivenom).
3. **bioCSL's Polyvalent Snake Antivenom should always be considered as a backup in situations where the supply of monovalent antivenom has been used up in treating a severe bite and the patient requires additional antivenom therapy. When additional vials of the appropriate monovalent antivenom are unavailable, it is always preferable to use Polyvalent Snake Antivenom rather than failing to use an adequate dose of antivenom.**
4. There are some settings, e.g. a small remote country hospital where it is practical as well as economical to stock bioCSL's Polyvalent Snake Antivenom and perhaps the most-needed monovalent snake antivenom, rather than maintaining stock vials of all types of monovalent antivenom.

Note: Compared with monovalent antivenoms, there is an increased rate of hypersensitivity reactions with bioCSL's Polyvalent Snake Antivenom.²⁸ This is primarily believed to be a volume effect (at least for delayed reactions, e.g. serum sickness), due to the larger volume per vial of Polyvalent Snake Antivenom *versus* each of its monovalent counterparts.^{1,28}

In many parts of Australia, if the identity of the snake remains undetermined, the diversity of snake species in the area will make bioCSL's Polyvalent Snake Antivenom the obvious choice

If antivenom is not immediately available³²

1. First steps

- If a patient presents with definite or possible snakebite and antivenom is not immediately available, it is crucial to:
 - Apply immediate PBI first aid if this is not already in place and
 - Organise immediate medical evacuation.
- Seek expert assistance.
- DO NOT wash the wound. If feasible, swab the bite site before applying PBI first aid. However, do not delay the application of first aid just to swab the wound. If the patient appears to be envenomed and SVDK is available
 - organise to have the bite site swab tested. If SVDK is not available, store the swab and send it with the patient during medical evacuation.
- Keep the patient as still and quiet as possible.
- Fast the patient and be prepared for vomiting.
- Carefully watch for evidence of envenoming and if there is respiratory distress, provide respiratory support.
- Monitor urine output and colour.
- If the killed snake was brought in with the patient, place it in alcohol if feasible and send it with the patient at the time of evacuation.

2. Medical evacuation

- During medical evacuation, provide the retrieval team with the following:
 - Patient's name, age and sex.

Brief history of the suspected bite. Time bite occurred? Was a snake seen? What type of snake? Are there multiple bites? Past history of snakebite or antivenom therapy, allergies, renal or CV disease, and use of anticoagulant or antiplatelet drugs.

Symptoms and signs of envenoming if present (e.g. headache; nausea; abdominal pain; collapse; convulsions; early paralysis such as ptosis; double vision; slurred speech; limb weakness; evidence of coagulopathy such as persistent ooze from the bite site; evidence of muscle damage such as dark red urine; muscle pain).

... continued overleaf

Specific treatment of snakebite: Antivenom

If antivenom is not immediately available³² ... cont'd

3. What to do if there is a delay in medical evacuation

– Certain procedures may assist an envenomed patient if there is a delay in medical evacuation. All efforts should be made to organise speedy evacuation or to access antivenom while any of the following steps are implemented.

– Progressive neurotoxic flaccid paralysis can result in full respiratory paralysis and loss of airway protection, which endangers respiration much earlier.^{2,14}
In this setting, it is important to secure the airway, often by intubation, and maintain adequate respiration with mechanical ventilation.

Tracheostomy is highly **inadvisable** and is clearly contraindicated if there is any possibility of venom-induced coagulopathy.

– There is limited evidence to support the use of anticholinesterase therapy and atropine in selected cases of severe paralysis³⁷⁻³⁹ [Adult dose: neostigmine 2.5 mg with atropine sulphate 0.6 mg I.V.].³⁷

Theoretically, this treatment should only work for snake venoms with purely post-synaptic neurotoxins, which excludes most Australian snake venoms – but anticholinesterases have been used on occasion to treat neurotoxic paralysis from death adder bites in PNG^{37,39} and Australia³⁸ with some success.

For sea snake bites with respiratory paralysis, where only post-synaptic neurotoxins are involved, neostigmine is certainly worth consideration, if antivenom is not available.^{1,14,15}

Importantly anticholinesterase treatment for paralysis should never be used as an alternative to antivenom, but merely as an adjunct or as a stopgap while sourcing antivenom.^{1,14}

... continued

Anticholinesterase treatment should never be used as an alternative to antivenom, but merely as an adjunct or as a stopgap while sourcing antivenom

- In addition to securing respiratory function, it is important to maintain renal function with adequate I.V. fluid therapy, usually above-maintenance in the first 6-12 hours, commencing with an initial fluid load.^{1,14}
 - Caution is required regarding I.V. fluid loading in children and adults with cardiac compromise.¹
 - If there is evidence of renal failure despite adequate I.V. fluid hydration
 - consider haemodialysis.¹
- For coagulopathy with major bleeding problems, use of factor replacement therapy (e.g. FFP, cryoprecipitate) is potentially hazardous and in general should only be considered after appropriate antivenom has been given and after consultation with an expert. However, in the unlikely setting of severe and potentially catastrophic bleeding, where antivenom is unavailable but with availability of FFP, seek expert advice before considering FFP administration.¹

Snake antivenoms: Initial dose

- Ongoing recent clinical research and expert advice indicates that the high doses previously advised by some experts for brown snake and tiger snake antivenoms, are not required.^{1,22,32,40-43}
- The initial doses in Table 14 on pages 118-121, reflect the Product Information as well as expert recommendations (at the time of writing this handbook) based on clinical experience, consensus discussions at scientific meetings and some published evidence.
- Always consult the Product Information and seek expert advice. Please be aware that expert consensus recommendations may change with ongoing research.

... see tables overleaf

Recent research indicates that the high doses previously advised by some experts for brown snake and tiger snake antivenoms, are not required

Specific treatment of snakebite: Antivenom

Snake antivenoms: Initial dose ... cont'd

Before administering the initial dose of antivenom, seek expert advice and also see pages 122-134 for additional details regarding follow-up dosing and the preparatory steps and procedure for administering snake antivenoms.

Table 14a. Initial dose of antivenom – for brown snake bite

| Snake | Preferred antivenom | Initial dose |
|--|------------------------|--|
| Brown snakes (<i>Pseudonaja</i> spp) | Brown Snake Antivenom* | 1 or 2 vials ^{1,23,41,42,44} The PI advises the use of 1 vial. The actual amount needed in clinical practice may be more ²³ The expert panel recommends 2 vials as a starting dose ¹ Please see additional information below |

*If Brown Snake Antivenom is unavailable or if the amount available is inadequate, Polyvalent Snake Antivenom may be substituted at the intended dose of Brown Snake Antivenom [1].

- Brown snake venom contains varied toxins including procoagulants, pre-synaptic neurotoxins and post-synaptic neurotoxins.⁴⁵ Additionally, venom yields from brown snakes can be higher than anticipated.⁴⁶
- The expert panel involved in producing this handbook believes it is important to maximise the potential for neutralising all venom components in all cases, including those where unusually large amounts of venom have been injected, and therefore, advises the use of 2 vials as an initial dose.¹
- Note: 2 vials also is the current consensus recommendation of experts such as the WA Toxicology Service and the Emergency Medicine Expert Group.^{42,44}
- The panel recognises that an initial dose of 1 vial will be sufficient in some cases. However, at the time of administering initial antivenom, it may not be possible to differentiate these cases from those that would benefit from a higher dose.¹
- The treating clinician will make the final decision regarding dosing, based on the circumstances of the individual case.¹

Table 14b. Initial dose of antivenom – for bites by snakes in the tiger snake group

| Snake | Preferred antivenom | Initial dose |
|--|------------------------|---|
| Tiger snakes (<i>Notechis</i> spp) | Tiger Snake Antivenom* | 1 or 2 vials ^{1,22,24,42,44} |
| Rough scaled snake (<i>Tropidechis carinatus</i>) | | For bites by Tasmanian tiger snakes – use at least 2 vials ^{1,47,48} |
| Copperheads (<i>Austrelaps</i> spp) | | For bites by Chappell Island tiger snakes – use at least 4 vials ^{1,47,48} |
| Pale headed, broad headed and Stephens' banded snakes (<i>Hoplocephalus</i> spp) | | The PI advises the use of 1 vial. The actual amount needed in clinical practice may be more ²⁴ The expert panel recommends 2 vials as a starting dose ¹ Please see additional information below |

*If Tiger Snake Antivenom is unavailable or if the amount available is inadequate, Polyvalent Snake Antivenom may be substituted at the intended dose of Tiger Snake Antivenom [1].

- The expert panel involved in producing this handbook believes it is important to maximise the potential for neutralising all venom components in all cases, including those where unusually large amounts of venom have been injected, and therefore, advises the use of 2 vials as an initial dose (and higher doses for Tasmanian and Chappell Island tiger snakes).¹
- Note: 2 vials also is the current consensus recommendation of experts such as the WA Toxicology Service and the Emergency Medicine Expert Group.^{42,44}
- The higher initial doses recommended for Tasmanian and Chappell Island tiger snakes are based on past clinical experience and the significantly higher quantities of venom likely to be injected by these snakes.⁴⁹ These recommendations date back over many decades, to advice in publications such as the CSL Medical Handbook (1979).^{47,48}
- The panel recognises that an initial dose of 1 vial will be sufficient in some cases. However, at the time of administering initial antivenom, it may not be possible to differentiate these cases from those that would benefit from a higher dose.¹
- The treating clinician will make the final decision regarding dosing, based on the circumstances of the individual case.¹

... continued overleaf

Specific treatment of snakebite: Antivenom

Snake antivenoms: Initial dose ... cont'd

Table 14c. Initial dose of antivenoms – for bites by snakes in the black snake group

| Snake | Preferred antivenom | Initial dose |
|---|--|---|
| Mulga snakes (<i>Pseudechis australis</i> ; <i>Pseudechis butleri</i> ; <i>Pseudechis weigeli</i>) Collett's snakes (<i>Pseudechis colletti</i>) | Black Snake Antivenom* | 1 vial ^{1,17,25,42,44} A higher dose is only rarely required ¹ |
| Red bellied black snake and blue bellied/spotted black snakes (<i>Pseudechis spp</i>) | Tiger Snake Antivenom** Note: Tiger Snake Antivenom is preferred for the management of envenoming from red bellied black snakes or blue bellied black snakes ^{14,25} | 1 vial ^{24,48} Many cases may not require antivenom therapy ^{1,14,16} If there is major myolysis (CK > 5,000 IU/L), consider using 1 vial of Black Snake Antivenom instead. ^{1,14} Note however, a recent study indicates that, as recommended in the Product Information, 1 vial of Tiger Snake Antivenom may be sufficient ¹⁶ |

*For envenoming due to bites by mulga snakes or Collett's snakes, if Black Snake Antivenom is unavailable or if the amount available is inadequate, Polyvalent Snake Antivenom may be substituted at the intended dose of Black Snake Antivenom [1].

**For envenoming from red bellied black snakes or blue bellied/spotted black snakes, If Tiger Snake Antivenom is unavailable, Black Snake Antivenom or Polyvalent Snake Antivenom may be used at the intended dose of Tiger Snake Antivenom [1,25,48].

Note regarding antivenom therapy for Collett's snake envenoming:

- The Product Information recommends Tiger Snake Antivenom as the preferred treatment when antivenom therapy is clinically indicated for Collett's snake bite [24,25].
- The table above differs from the Product Information by advising the use of Black Snake Antivenom for envenoming from Collett's snake bite. This advice is based on published evidence demonstrating the potential for severe envenoming from Collett's snake bite, which produces an envenoming syndrome similar to that of mulga snake bite [17]. In addition, Black Snake Antivenom was shown to be significantly more efficacious (*versus* Tiger Snake Antivenom) *in vitro*, in reversing the myotoxic effects of *Pseudechis colletti* venom [50]. Therefore, researchers advise that Black Snake Antivenom appears to be the more appropriate treatment for Collett's snake envenoming, and this is now also reflected in recent published expert consensus and guidelines [17,42,44].

Table 14d. Initial dose of antivenom – for taipan bite

| Snake | Preferred antivenom | Initial dose |
|------------------------------------|--|--|
| Taipans (<i>Oxyuranus</i> spp) | Taipan Antivenom or Polyvalent Snake Antivenom ^{1,14} | 1 vial ²⁷ In severely envenomed cases consider using an initial dose of at least 3 vials ²⁷ |

Table 14e. Initial dose of antivenom – for death adder bite

| Snake | Preferred antivenom | Initial dose |
|---|------------------------|---|
| Death adders (<i>Acanthophis</i> spp) | Death Adder Antivenom* | 1 vial ²⁶ Increased doses may be required in severe cases ^{1,26} |

*If Death Adder Antivenom is unavailable or if the amount available is inadequate, Polyvalent Snake Antivenom may be substituted at the intended dose of Death Adder Antivenom [1].

Table 14f. Initial dose of antivenom – for sea snake bite

| Snake | Preferred antivenom | Initial dose |
|--|---------------------|--|
| Sea snakes (numerous genera and species) | Sea Snake Antivenom | 1 vial ²⁹ 3-4 vials for severe envenoming ²⁹ Please see additional information below on possible options if Sea Snake Antivenom is unavailable |

What if Sea Snake Antivenom is unavailable?

Polyvalent Snake Antivenom is not designed for the purpose of treating envenoming resulting from sea snake bite and efficacy is unknown. If Sea Snake Antivenom is unavailable, it is unclear if any other snake antivenom might be substituted. In the past Tiger Snake Antivenom (TSAV) was recommended as a substitute for Sea Snake Antivenom (SSAV), with a dose ratio of 3 vials of TSAV for each vial of SSAV that might have been used. However, due to the nature of antivenom manufacturing processes, the results of earlier research cannot be extrapolated to the current TSAV product. Hence the efficacy of the current Tiger Snake Antivenom against sea snake venoms, remains unknown [1].

Nevertheless, in desperate circumstances, if SSAV were unavailable, TSAV could be considered for sea snake envenoming [29]. Alternatively, if TSAV also is unavailable, Polyvalent Snake Antivenom could be considered as it contains tiger snake antivenom, but the clinicians and the patient would need to clearly understand the uncertainty regarding antivenom efficacy in this instance and the known risks of using any antivenom product. In this situation always seek expert advice [1].

Specific treatment of snakebite: Antivenom

Evaluating patients after initial antivenom therapy¹

Giving the initial dose of antivenom to an envenomed patient is merely the start of treatment, not the end.

It is essential to monitor patients closely post antivenom administration to determine whether or not envenoming is resolving or if it is worsening, and to observe for any developing complications such as delayed myolysis, renal failure or microangiopathic haemolytic anaemia.

Therefore frequent targeted examination post antivenom administration is crucial.

- Strict fluid balance measurements must be charted (looking for declining urine output) and follow-up blood testing is mandatory.
- The precise timing of repeat blood testing can vary depending on clinical circumstances. As an approximate guide, repeat tests at 6, 12 and 24 hours post antivenom, assuming the patient shows continued improvement, such that, by 24 hours post antivenom therapy, tested parameters are substantially or completely returned to normal values.

If envenoming does not appear to be resolving after the initial dose of antivenom, or if it is worsening, the patient may require a follow-up dose of antivenom (see page 123).^{1,5}

Frequent targeted examination post antivenom administration is crucial. Strict fluid balance measurements must be charted (looking for declining urine output) and follow-up blood testing is mandatory

Follow-up doses of antivenom^{1,2,5,14,40,51-53}

- In most cases, the initial dose of antivenom will be sufficient to neutralise all venom, and typically, follow-up dosing is not required.¹
- In the past, management of snakebite coagulopathy frequently involved administration of large follow-up doses of antivenom,⁴⁰ but recent research suggests this may be unnecessary, as the envenoming process in procoagulant coagulopathy (at least for Australian snakes) involves a very short period of active venom effect (suggesting the coagulopathy-causing procoagulants may be rapidly deactivated in the blood).⁵¹

However, the defibrination coagulopathy (VICC) caused by the procoagulants may take 6 or more hours to begin to show any sign of resolution because it may take that long for depleted coagulation factors to be replenished to a detectable level.⁵² Hence, in cases of defibrination coagulopathy, it is important to wait at least 6 hours (and probably longer) after initial antivenom dosing and to test for coagulation function before making the decision regarding follow-up dosing.¹

Regardless of the above, if the patient has continuing active major or life-threatening bleeding (including intracranial haemorrhage), urgent administration of further antivenom should be considered, in conjunction with coagulation factor replacement therapy (FFP to be administered greater than 1 hour after and within 4 hours of starting antivenom treatment).^{1,53} Seek expert advice.

It is possible that ongoing release of venom from the bite site may cause resurgence of coagulopathy,⁵ and consequently, there would be no evidence of coagulation test improvement at 8 or more hours post antivenom therapy. In this situation, consider repeat antivenom dosing after consultation with a clinical toxicologist.¹

- For anticoagulant coagulopathy, typically follow-up doses of antivenom are not required.¹

.... continued overleaf

The defibrination coagulopathy (VICC) caused by procoagulants may take 6 or more hours to begin to show any sign of resolution

Specific treatment of snakebite: Antivenom

Follow-up doses of antivenom^{1,2,5,14,40,51-53} ... cont'd

- Myolysis (delineated by CK level) may take more than 24 hours to reach a peak, after which there is usually a gradual return to normal over several days.¹

If there is either a second peaking of CK or continued rapid rise to high levels after initial antivenom therapy, then consider providing a follow-up dose of antivenom.¹

While there is no clear evidence at present to support the value of late antivenom therapy in the management of snakebite myolysis, anecdotal cases do exist where late antivenom therapy appears to have been beneficial.¹

- Antivenom as treatment for neurotoxic paralysis can be problematic. Essentially all Australian venomous snakes that are likely to cause major paralysis in humans, contain both pre- and post-synaptic neurotoxins in their venom.

Antivenom can reverse post-synaptic neurotoxin paralysis – but cannot reverse pre-synaptic neurotoxin paralysis, which may take longer to manifest.^{2,14}

Therefore, in a patient with severe paralysis who has not responded to an initial adequate dose of antivenom, there is no justification for giving multiple repeat doses of antivenom in a (futile) attempt to reverse the paralysis.¹

This is why it is critically important to administer adequate antivenom at the earliest sign of neurotoxicity, to prevent extension of the paralysis.¹

- **Monitor patients for at least 24 hours after the last dose of antivenom and until parameters have substantially or completely returned to normal values.¹**

Antivenom can reverse post-synaptic neurotoxin paralysis – but cannot reverse pre-synaptic neurotoxin paralysis... This is why it is critically important to give adequate antivenom at the earliest sign of neurotoxicity, to prevent extension of the paralysis

Preparation prior to commencing antivenom therapy^{32,54,55}

Prior to commencing antivenom therapy, ensure all facilities are ready at hand to treat anaphylaxis, in the event that this should occur.

- Dedicate one small-bore I.V. line (18-20 G in adults) to antivenom administration.
- Dedicate one large bore I.V. line (16-14 G in adults) for emergency resuscitation.
- Prepare 1L normal saline (20 mL/kg in children) ready to administer under pressure.
- Prepare adrenaline (1:1000 – i.e. 1 mg adrenaline in 1 mL) drawn up to a dose of 0.01 mg/kg (maximum 0.5 mg – i.e. 0.5 mL) and label as ‘Adrenaline for **I.M.** injection only (dose in mg)’.
- Ideally, also prepare an I.V. infusion of adrenaline 1 mg in 100 mL, which is controlled by infusion pump or syringe driver and ready to attach by a side arm to the resuscitation line. Anti-reflux valves must be attached above the side arm on any other infusions using this I.V. line, to prevent adrenaline going back up into other fluid bags. To prevent erroneous administration, **do not attach the adrenaline infusion unless it is needed.**
- Record blood pressure on the opposite arm to the fluid/adrenaline infusion – to avoid prolonged cuff inflations and thus, extravasation of infusion fluids.
- See ‘What to do if there is an adverse reaction to antivenom’ on pages 130-131 for method of emergency resuscitation if required.

Specific treatment of snakebite: Antivenom

How to administer snake antivenoms

Table 15. Administering snake antivenoms^{1,8,9,14,23-29,42,56}

| | |
|-----------------------------------|---|
| Treatment location | If possible, patients should receive antivenom in a monitored environment with immediate access to resuscitative equipment and one-to-one nursing |
| What to do about first aid | If adequate PBI first aid was applied, the splint and pressure bandage should not be removed until antivenom is available for infusion, and if required, until after the infusion has commenced, as removal can precipitate significant effects of systemic envenoming See pages 56-57 for timing of removal of PBI first aid for snakebite |
| Route of administration | Intravenous Always administer snake antivenoms I.V. – preferably through drip set |
| Dilution of antivenom | Dilute up to 1 in 10 with an isotonic crystalloid solution (e.g. normal saline; Hartmann’s solution)* To avoid fluid overload, use smaller volumes in small children and in adults with compromised cardiac function – Adults with compromised cardiac function: Up to a 1 in 5 dilution may be more appropriate – Small children: Dilute antivenom to the extent that the total volume delivered does not exceed 10 mL/kg |
| Time period of dosing | Each dose should be run over about 30 minutes (a single dose may comprise multiple vials) Start very slowly and increase the rate gradually to deliver antivenom over approximately 30 minutes |
| Observation | See following section |

*Product Information leaflets for a number of bioCSL’s snake antivenoms recommend dilution using Hartmann’s solution. The use of other isotonic crystalloid such as normal saline to dilute these antivenoms is based on expert clinical experience and is accepted current clinical practice [1,23-28,42].

Observation during antivenom therapy^{1,54,57}

- Carefully observe the patient during antivenom administration and for 1 hour after, to ensure adverse reactions (if they occur) are recognised and treated promptly (adverse reactions are discussed further on pages 130-133).
- In particular, look for the development of symptoms and signs of anaphylaxis. An erythematous rash may be the first sign of developing adverse reactions (often first seen in the axilla or the lower abdomen).
- Also observe for hypotension and bronchospasm.
- Carefully monitor BP, HR and respiratory function, oxygen saturation, with particular attention to development of hypotension and/or bronchospasm.
- Look for additional warning signs of anaphylaxis in children (Table 16).
- See pages 132-133 for further information on potential complications of antivenom therapy.

Table 16. Warning signs of anaphylaxis in children^{1,57}

| | |
|------------------------------------|---|
| Rash; hypotension; or bronchospasm | Profuse sweating |
| Nasal, palatal, or ocular pruritus | Faecal or urinary urgency or incontinence |
| Coughing | Abdominal pain |
| Sneezing | A sense of impending doom |



Antivenom rash in an adult.

Specific treatment of snakebite: Antivenom

Premedication prior to administering antivenom

Premedication prior to administering antivenom remains an issue surrounded by controversy, some conflicting evidence and opinion, and uncertainty about “best practice”.

Studies outside Australia have provided evidence showing premedication using antihistamines and/or hydrocortisone are either ineffective, or possibly effective to a variable extent.^{58,59} Hence, available evidence is unhelpful and contradictory for these medications. Similar contradictory evidence applies to adrenaline premedication within and outside Australia.⁶⁰⁻⁶⁴

Data from recent Australian clinical research reported in 2008 (the ASP study – a multicentre, prospective study of snakebite envenoming from over 60 major tertiary centres and regional hospitals around Australia) suggests that there is no clear benefit in giving premedication prior to antivenom administration, including no clear benefit in using adrenaline as premedication.⁶⁵

Further, the ASP study has shown that in Australia, the use of premedication prior to antivenom therapy is not common practice (adrenaline premedication was provided to 8.7% of patients in whom data about premedication was available).⁶⁵

Additionally, recent expert consensus suggests that premedication to prevent adverse reactions to antivenom therapy is not routinely indicated.⁴²

.... continued

Clearly, there may be specific clinical circumstances, judged on an individual patient basis, where a clinician may consider the use of adrenaline as premedication prior to antivenom administration. For example – in a patient with known major allergy to antivenom where resuscitation facilities may be suboptimal.¹

In such situations the uncertain and unproven benefits of adrenaline premedication should be carefully weighed against the known and documented adverse effects from use of adrenaline, and wherever practical, the patient should be told of this risk-benefit situation so that informed consent can be given.¹

Clearly, in some situations, with severe life-threatening systemic envenoming, informed consent is impractical and should not form a necessary condition of using adrenaline premedication if the clinician deems this is required.¹

Irrespective of whether premedication is used or not, antivenom should only be administered in an environment where rapid detection and appropriate treatment of severe early adverse reactions will occur.¹

- This includes the immediate availability of adrenaline, oxygen and resuscitation equipment and staff competent and prepared to use these treatments effectively.
- In such a setting, a controlled I.V. adrenaline dilute infusion may be the optimal route for administering adrenaline to treat anaphylactic or anaphylactoid reactions (see pages 130-131 for further details).¹

Specific treatment of snakebite: Antivenom

What to do if there is an adverse reaction to antivenom^{1,32,54,57,65}

Adverse reactions may be related to the rate of antivenom infusion – those reactions can include flushing, hypotension or bronchospasm. Hypotension and bronchospasm are hallmarks of major adverse reaction (anaphylaxis).

Adverse reactions may respond to temporarily stopping the antivenom infusion, waiting to ensure that there is no return or worsening of the reaction, and then re-starting at a slower rate.

For anaphylactic reactions, adrenaline is generally the drug of first choice. Importantly, snakebite envenoming may cause severe coagulopathy – consequently, when giving adrenaline, be cautious to avoid blood pressure surges, which may precipitate intracranial haemorrhage.

See the box below and on page 131.

Steps to take if there is either a sudden fall in blood pressure, or bronchospasm after starting antivenom infusion^{1,32,42,54,55,57}

- Suspend the antivenom infusion.
- Lie the patient flat (if not already in this position) and commence high-flow 100% oxygen and support airway/ventilation as required.
- Begin rapid infusion of one litre normal saline (20 mL/kg in children) over 2-3 minutes.
- Administer adrenaline 1:1000 I.M. into the lateral thigh at a dose of 0.01 mg/kg to a maximum of 0.5 mg (i.e. a maximum of 0.5 mL). Note: Adrenaline 1:1000 ampoule is 1 mg adrenaline in 1mL.
- Alternatively, those experienced with I.V. administration of adrenaline can proceed to do this directly instead of I.M injection. See procedure on page 131 in the section 'If adverse reactions do not respond to initial management'.
- Seek expert advice regarding ongoing management.
- In most cases, once the adverse reaction is controlled, cautious reintroduction of antivenom is possible. Note: A patient requiring antivenom therapy prior to the adverse reaction will likely continue to require antivenom after the adverse reaction.

Note: The recommendations above and on page 131 for the management of anaphylactic reactions to antivenom reflect current published anaphylaxis management guidelines and expert advice and may vary from the Product Information for bioCSL's antivenoms [1,54,55,57].

If adverse reactions do not respond to initial management^{1,32,42,54,55,57}

- If hypotensive, repeat normal saline bolus as per box on page 130 (up to 50 mL/kg may be required).
- Commence I.V. infusion of adrenaline (0.5-1 mL/kg/hr of adrenaline 1 mg in 100 mL) and titrate according to response. Monitor BP every 3-5 minutes using the arm opposite to the infusion.
- Be aware that as the adverse reaction to antivenom resolves, adrenaline requirements will fall, the blood pressure will rise and the adrenaline infusion rate will need to be reduced.
- Consider nebulised salbutamol for bronchospasm, nebulised adrenaline for upper airway obstruction, and I.V. atropine for severe bradycardia.
- Seek advice urgently from local/regional ED Consultant and/or ICU Consultant.
- In most cases, once the adverse reaction is controlled, cautious reintroduction of antivenom is possible. Note: A patient requiring antivenom therapy prior to the adverse reaction will likely continue to require antivenom after the adverse reaction.

Antivenom therapy: Commonest mistakes¹

Some of the most common mistakes relating to antivenom therapy are highlighted below.

- Failure to use antivenom when clearly indicated.
- Giving antivenom unnecessarily, i.e. when there are no clear clinical indicators of significant systemic envenoming.
- Choosing the wrong antivenom.
- Choosing the wrong dose.
- Antivenom given too late.
- Administering antivenom by the wrong route.
- Failing to prepare for an adverse reaction.
- Failing to inform the patient about serum sickness.

Specific treatment of snakebite: Antivenom

Complications of antivenom therapy

Essentially, antivenom is whole or modified antibody from an animal. It is obtained by hyperimmunising the animal against a particular venom or group of venoms. The IgG antibody from blood plasma is used, and typically, is fractionated to the F(ab')₂ fragment of IgG. bioCSL's snake antivenoms are derived from horse plasma, with the IgG antibody fractionated to the F(ab')₂ fragment.^{23-29,66}

When making antivenoms, bioCSL undertakes assiduous efforts to filter and discard any extraneous blood components and contaminants. Nevertheless even high-quality antivenoms will cause adverse reactions in some patients. The clinically important adverse reactions can be subdivided into 'early' and 'late'.

Early and late adverse reactions to antivenom

Early reactions are those that occur immediately after commencing antivenom therapy or within the first few hours (Table 17). Late adverse reactions may occur several days later (Table 18 on page 133).

Table 17. Early adverse reactions to antivenom^{1,23-29,42,54,57}

| | |
|---|---|
| Rash | Localised or generalised erythematous, sometimes pruritic rash. May occur as an isolated and generally trivial adverse reaction or it may herald the onset of a more severe adverse reaction |
| Pyrexia | Febrile reactions may potentially occur |
| Anaphylactic/ anaphylactoid reaction | <p>This is a type of potentially life-threatening reaction. Therefore, antivenom should never be given until measures to manage such a reaction are in place. This is why the use of antivenom outside a hospital environment is strongly discouraged. Note however, when antivenom is clinically indicated, it should never be withheld for fear of an adverse reaction. Seek expert advice</p> <p>Anaphylaxis may be preceded by a localised or generalised rash, sometimes first seen in the axilla or lower abdomen, proceeding to hypotension and/or bronchospasm</p> <p>Look for additional warning signs of anaphylaxis in children (Table 16 on page 127)</p> |

Table 18. Late adverse reactions to antivenom^{1,23-29,42}

The principal late adverse reaction to antivenom is serum sickness, a type III delayed hypersensitivity reaction, which most commonly presents 4-14 days post exposure

Serum sickness is characterised by a flu-like illness with fever, joint and muscle pain and general malaise, often preceded by or associated with a maculopapular or erythema multiformae-like rash

Serum sickness rates often increase as the antigen load increases. Therefore, it is more likely to occur in patients who have had a high volume load of antivenom

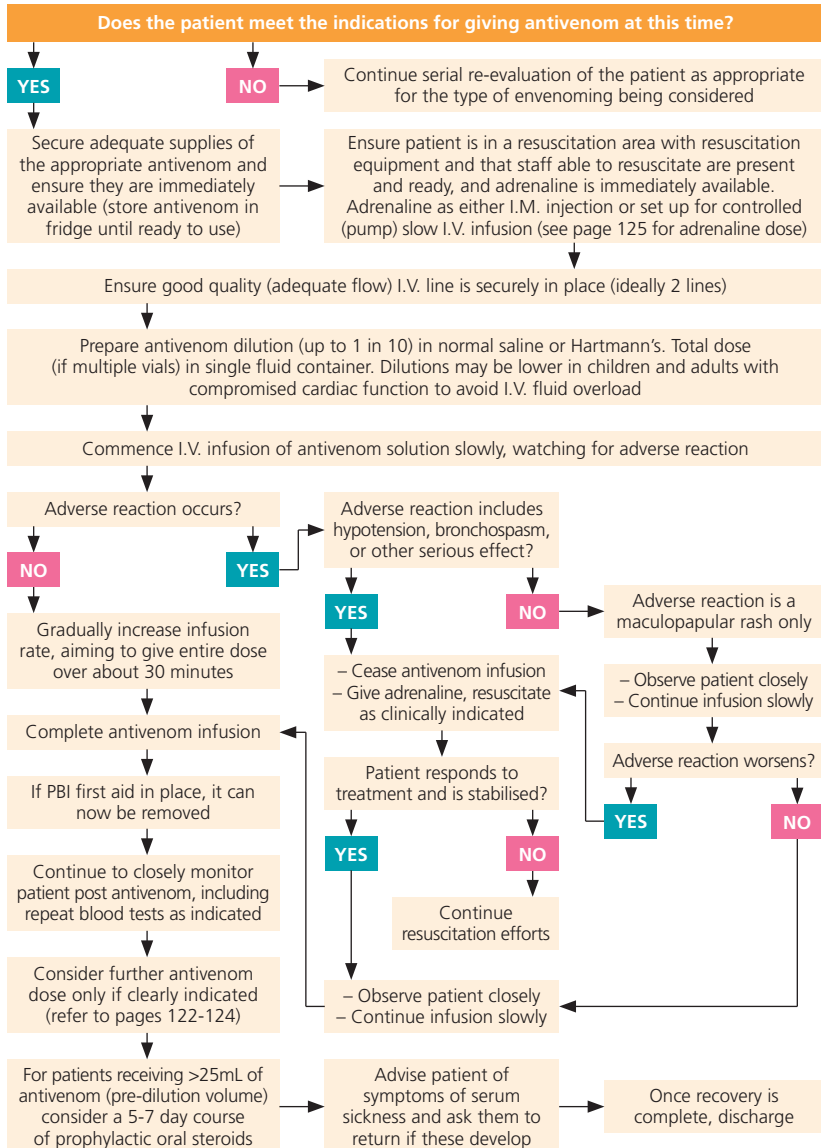
Every patient who receives antivenom should be advised of the symptoms of serum sickness and told to seek medical care if these symptoms arise after discharge from hospital

Serum sickness is more likely to occur in patients who have had a high volume load of antivenom

Management of serum sickness^{1,14,42}

- While serum sickness can be a mild and self-limited disease, it can be distressing for patients and early diagnosis and treatment is advisable.
- There is a diversity of opinion about the approach to treatment for various causes of serum sickness (i.e. not just antivenom) – which may involve first-line use of antihistamines or oral steroids. Serum sickness post antivenom therapy is usually managed with a short course of oral corticosteroids.
- If uncertain about the treatment approach, consult with a clinical immunologist.
- Some experts suggest prescribing a week-long course of oral prednisolone commencing immediately after antivenom, for all patients who have received more than 25 mL of antivenom. This treatment has not yet been tested through clinical trials.

Administering I.V. antivenom



Treating snakebite: Managing venom-induced toxicity

In addition to the general and specific management of patients with snakebite, including provision of antivenom therapy when indicated, in patients with significant envenoming, the toxic effects of snake venom (haematological, myolytic, nephrotoxic, CV and local cytotoxicity) also will require management.

Haematological effects of snake venom

Table 19 depicts the clinical features/observations relating to haemotoxicity of snake venom.

Table 19. Snake venom haematological effects

| Effect of venom | Observations |
|-----------------------------------|--|
| Defibrination coagulopathy (VICC) | <p>These potent coagulants act as prothrombin converters, resulting in consumption of fibrinogen. This may lead to rapid complete defibrination (within 30 minutes of the bite), with resulting bleeding tendency, although platelet count and function are usually normal, at least initially⁵</p> <p>In some species (e.g. brown snakes; taipans) the coagulopathy may be prolonged unless adequate antivenom is given, though it will ultimately resolve¹</p> <p>In others (e.g. tiger snakes) it may be initially severe, yet resolve spontaneously within 12 to 18 hours without antivenom treatment.¹ In this situation there may be residual raised fibrin(ogen) degradation products present up to 24 hours after the bite, giving a clue to what has occurred^{1,67}</p> <p>Major haemorrhage associated with snakebite coagulopathy is not very common, nor is it rare, with intracranial bleeding a particular worry⁵</p> |
| Anticoagulant coagulopathy | Potent anticoagulants in a few snake venoms may cause rapidly reversible coagulopathy, without defibrination ⁵ |
| Haemolytic effects | <p>While some Australian snake venoms contain potent haemolytic toxins, clinical haemolysis is mostly seen in cases of MAHA (Microangiopathic Haemolytic Anaemia)</p> <p>MAHA is most frequently reported following brown snake bite,^{3,14} but brown snake venom has no significant haemolytic activity.⁴⁵ Therefore, the mechanism involved in MAHA following snakebite is currently uncertain</p> |

Treating snakebite: Managing venom-induced toxicity

Managing the haematological effects of snake venom

1. Management of snake venom-induced defibrination coagulopathy (VICC)

Management is evolving as ongoing research increases our understanding of this complex condition.

Currently, the recognised treatment involves:

- Neutralisation of procoagulant with an initial dose of antivenom and a wait of at least 6 hours to allow depleted coagulation factors to return to measurable levels before deciding on the need for further treatment such as a repeat dose of antivenom.^{14,52}
- **It is inappropriate to give repeat antivenom sooner solely because clotting tests have failed to improve within this time.** In the past this type of inappropriate treatment has resulted in some patients receiving very large amounts of antivenom without discernible benefit.¹
- Regardless of the above, if the patient has continuing active major or life-threatening bleeding (including intracranial haemorrhage) consider urgent administration of further antivenom in conjunction with coagulation factor replacement (FFP to be given greater than 1 hour after and within 4 hours of antivenom administration).^{1,43,53}

Current understanding of defibrination coagulopathy (VICC) remains incomplete. But recent research indicates that the process may switch off quite early and the role of antivenom may be uncertain.^{21,43,51} This is absolutely NOT a reason to avoid antivenom therapy as all venoms contain a variety of different toxins and antivenom would still be required to neutralise other venom effects such as neurotoxicity and/or myolysis.^{1,43}

The role of factor replacement therapy in defibrination coagulopathy is discussed on page 137.

.... continued

The role of factor replacement therapy (FFP; cryoprecipitate) remains controversial and may change as new evidence becomes available.

- In the past, giving factor replacement therapy early was thought to worsen coagulopathy and was associated with isolated cases of fatal haemorrhage.^{1,53,68}
- Recent research indicates that FFP may be safely given 1 hour or more (and within 4 hours) after an adequate dose of antivenom and that it may hasten recovery of laboratory parameters^{43,53} although this may not be associated with improved clinical outcome.⁶⁸ A randomized controlled trial is currently underway.
- At this time, routine use of FFP in the management of coagulopathy cannot be recommended.⁶⁸

However, in patients with coagulopathy and potentially catastrophic bleeding, FFP should certainly be considered in addition to antivenom therapy in an attempt to quickly control haemorrhage. In all such situations seek urgent expert advice.¹

2. Management of anticoagulant coagulopathy^{1,5,16}

- In terms of diagnosis, anticoagulant coagulopathy is clearly distinguishable from defibrination coagulopathy by measuring the levels of fibrinogen and its degradation products.

INR/apTT readings are abnormal. However, as there is no consumption of fibrinogen, levels of fibrinogen should essentially be normal and degradation products are absent.⁵

If only INR/aPTT are tested, it may not be possible to distinguish between the two types of coagulopathy.

- Anticoagulant coagulopathy is comparatively easy to manage. Antivenom therapy usually quickly and completely reverses the abnormality, as indicated by return to normal values for INR/aPTT.^{1,16}

This again reflects the difference in mechanism compared with defibrination coagulopathy. In anticoagulant coagulopathy, consumption of key clotting factors does not occur, so there is no delay in factor levels returning to normal.

Treatment with FFP is not required.¹

Treating snakebite: Managing venom-induced toxicity

Managing the haematological effects of snake venom ... cont'd

3. Management of venom-induced haemolysis: MAHA^{1,3}

- The management of MAHA is currently ill defined.
- The role of haemoperfusion/haemofiltration is uncertain when compared to other causes of this clinical syndrome (e.g. TTP, HUS).³
- Where there is severe anaemia, red cell transfusion may be required, but should be used with caution in the presence of active haemolysis.¹
- Renal dialysis may be required to manage kidney failure.³
- Although uncommon, isolated thrombocytopenia or thrombocytopenia as part of MAHA can occur following snakebite. Toxin mechanisms are poorly understood, but the thrombocytopenia usually resolves over several days.³ Sometimes it is severe enough to require platelet transfusions.¹ The role of haemoperfusion in the treatment of these conditions is currently undefined.³
- Early consultation with a haematologist is recommended.^{1,3}

4. Management of other haematological effects of snake venom¹

- Significant envenoming is frequently associated with a high white cell count, usually neutrophilia, often with an acute lymphopenia.
- However, these changes are inconsistent and do not appear to be a viable marker for systemic envenoming. They do not require therapeutic intervention.

Myotoxicity: Snake venom-induced rhabdomyolysis

- Myotoxins are often structurally related to neurotoxins (i.e. phospholipase A₂).¹⁴
- Table 20 depicts the clinical features of venom-induced myolysis and its management.

Table 20. Snake venom-induced myolysis: Clinical features and management^{1,2,14}

| | |
|-----------------------------|--|
| Clinical features | <ul style="list-style-type: none">– Myotoxins bind to muscle fibres, causing progressive destruction of the muscle cells with the release of breakdown products¹⁴– The process may take hours to days to become evident,¹⁴ by which time irreversible damage has been done¹– The result is progressive muscle weakness and pain on movement, with myoglobinuria and possible secondary renal failure and hyperkalaemia (which can be severe, intractable and lethal)¹⁴ |
| Management | <ul style="list-style-type: none">– The management of myolysis is two-fold^{1,2} Firstly, antivenom is used to neutralise circulating myotoxins Secondly, supportive treatments help minimise potential complications. The issues of adequate I.V. fluid load, supporting renal function if impaired, and control of hyperkalaemia have been mentioned earlier (see 'General treatment' pages 107-110)– In patients with myolysis, it is particularly important to maintain good renal throughput by providing an ongoing I.V. fluid load^{1,2}– Reduction of CK levels is indicative of resolving myolysis^{1,2}– Alkalinisation of urine in cases with myolysis has been suggested but is neither supported, nor refuted by current evidence in snakebite¹ |
| Key laboratory tests | Plasma CK; urinary myoglobin (qualitative only; positive Ward test for blood); plasma potassium levels; renal function ^{1,2} |

Treating snakebite: Managing venom-induced toxicity

Renal/nephrotoxic effects^{1,2,14}

- While there are no isolated nephrotoxins in any Australian snake venom, nephrotoxins are known to occur in some exotic snake venoms.
- Kidney damage is not rare in Australian snakebite.
- The mechanisms are unclear, but may include both direct toxic effects on the kidney and secondary damage as a result of hypotension, myolysis or coagulopathy.
- Kidney damage may develop early and it is much more likely to occur if the patient has consumed significant amounts of alcohol prior to the bite.
- Kidney damage is uncommon in children.
- Key laboratory tests: Urea; creatinine; potassium.

Cardiovascular effects¹⁴

- In Australian snakebite, CV effects generally arise as secondary complications of other aspects of envenoming.
- Coagulopathy in the early stages may result in brief clot formation in critical cardiac vessels which can cause cardiac collapse, usually soon after the bite. This mechanism is thought to be the likely cause of cases of cardiac arrest pre-hospital following brown snake bite. Most of these cases are fatal.
- Severe myolysis with secondary renal failure can be associated with marked hyperkalaemia, which can cause severe and even lethal cardiotoxicity.

Kidney damage may develop early and it is much more likely to occur if the patient has consumed significant amounts of alcohol prior to the bite

Neurotoxic effects – pre-synaptic and post-synaptic neurotoxicity^{1,2,14}

- Neurotoxins act at the skeletal neuromuscular junction (NMJ), either pre- or post-synaptically, causing progressive flaccid paralysis of voluntary and respiratory muscles.
- Since the venom must first reach the NMJ and then fix to target tissues, the first effects of these toxins are seen at least 1 hour after the bite and usually the earliest signs take 2-4 hours or more to develop.
- Complete paralysis, which is not common, even in untreated cases, may take from 3 hours to greater than 18 hours to develop. However, respiratory difficulty due to paralysis of the tongue and pharynx may develop much earlier than full respiratory paralysis and will require intubation and ventilation. [This complication also increases the risk of aspiration].
- While laboratory tests are not diagnostic – pulse oximetry and peak flow measurements may be helpful in documenting deteriorating respiratory function.

Management of neurotoxic paralysis^{1,2,14}

- The cornerstone of therapy is the provision of an adequate dose of antivenom at the earliest sign of paralysis, to prevent progression to full pre-synaptic paralysis² (for detailed information on follow-up antivenom therapy for major paralysis see ‘Follow-up dose of antivenom’ on page 124).
- Progressive neurotoxic flaccid paralysis can result in full respiratory paralysis and loss of airway protection, which endangers respiration much earlier.^{2,14}
 In this setting, it is important to secure the airway, often by intubation, and maintain adequate respiration with mechanical ventilation.

 Tracheostomy is highly **inadvisable** and is clearly contraindicated if there is any possibility of venom-induced coagulopathy.

.... continued overleaf

Treating snakebite: Managing venom-induced toxicity

Management of neurotoxic paralysis^{1,2,14} ... cont'd

– It is important to try and establish ongoing communication with patients who develop severe paralysis, particularly those requiring mechanical ventilation. Even at the simple “yes/no” level, through movement of some incompletely paralysed part of the body. This is very reassuring for these patients who may seem obtunded, but may actually be awake and fully aware of their surroundings.¹

Local cytotoxicity^{1,2,4,14,16,17,35}

– The presence of local cytotoxins has not been clearly demonstrated in Australian snake venoms – and major local skin damage at the bite site is rare in Australia.^{2,14}

– However, some local cytotoxic effects in the form of pain or swelling can occur with Australian snakebite (see Table 21).^{2,14}

– As secondary infection after Australian snakebite is uncommon to rare, the use of prophylactic antibiotics is inappropriate.^{2,14}

– If secondary infection of the bite area develops, culture and sensitivity testing should be performed prior to commencing appropriate antibiotic therapy.¹

– Be aware that a few species of snake typically cause venom-induced chemical cellulitis in the bitten limb, with erythema, pain, swelling, and sometimes, tenderness. In particular, this is associated with bites by the black snake group.^{4,16,17,35} This chemical cellulitis may peak at 1-3 days post bite and slowly resolves over subsequent days.³⁵

Table 21. Local cytotoxic effects of Australian snake venoms^{1,4,14,16,17,22,35}

| Snakes | Local cytotoxicity |
|---|--|
| Mulga snakes, red bellied black snakes and other members of genus <i>Pseudechis</i> | Moderate to marked local swelling |
| Brown snakes | Minimal or no local pain, or redness or swelling and bite marks are often hard to spot |
| Tiger snakes | Commonly cause mild local swelling with ecchymosis and pain |
| Taipans | Variable local effects, usually less severe than for tiger snakes |
| Death adders | Mild swelling but marked pain |

Australian venomous snakes overview: Brown snake group

The species^{1,14,45}

- All dangerous Australian venomous snakes are members of the cobra family Elapidae.
- Brown snakes (Genus *Pseudonaja*) are the commonest cause of snakebites and snakebite deaths in Australia.
- The fangs are small (average fang length in adults is only 2.8 mm).
- Compared to many other dangerous snakes, they generally produce little venom, although the venom is very potent.
- There are several species. These have recently seen taxonomic change.
- Medically, the most important are the eastern or common brown (*P. textilis*), the western brown or gwardar (*P. mengdeni*), the tropical brown (*P. nuchalis*), the patch nosed brown (*P. aspidorhyncha*) and the dugite (*P. affinis*) – which vary in length up to 2 m, rarely more.
- Colouration is variable. They may be brown, orange-red, grey, almost black, banded, unbanded, speckled, or black headed.

Snake classification and colour

- The term 'brown snake', 'black snake', etc does not refer to colour.
- Instead, the category, e.g. brown snake group, typically includes snakes of a particular genus but different species. Snakes belonging to the brown snake group can vary substantially in colour. All snakes included within this group share a common venom immunotype (due to evolutionary relationship).
- Some groups, e.g. the tiger snake group include snakes from more than one genus. Once again, all the snakes within this group share a common venom immunotype.
- Black snakes (red-bellied black snake; blue-bellied/spotted black snake) belong to the black snake group (genus *Pseudechis*).

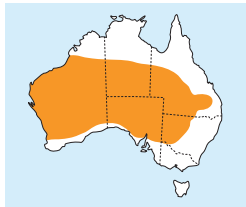


Eastern brown snake, *Pseudonaja textilis*.
Photo copyright A/Prof Julian White.

Australian venomous snakes overview: Brown snake group

Distribution^{1,45}

- Brown snakes are common throughout mainland Australia in essentially all habitats, including urban areas.
- They are not present in Tasmania or the islands off the southern coast of Australia, such as Kangaroo Island (SA).
- They are relatively common in some urban and metropolitan areas – hundreds of brown snakes are removed from houses and other properties each year in some capital cities and >10% of these snakes are found inside the house!



Western brown snake/gwardar, *Pseudonaja mengdeni*.



Patch nosed brown snake, *Pseudonaja aspidorhyncha*.



Tropical brown snake, *Pseudonaja nuchalis*.



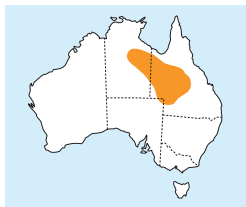
Eastern brown snake, *Pseudonaja textilis*.



Dugite, *Pseudonaja affinis*.



Peninsular brown snake, *Pseudonaja inframacula*.



Spotted brown snake, *Pseudonaja guttata*.



Ingram's brown snake, *Pseudonaja ingrami*.



Five-ringed brown snake, *Pseudonaja modesta*.

Venom composition^{1,45}

- The venom is multicomponent and includes powerful pre-synaptic neurotoxins and post-synaptic neurotoxins and procoagulants, and possibly cardiotoxins and direct nephrotoxins.
- There is no myolytic activity.
- The venom of the eastern/common brown snake is the second most potent snake venom in the world.

[Brown snake venom] includes powerful pre-synaptic neurotoxins and post-synaptic neurotoxins and procoagulants. There is no myolytic activity



Juvenile brown snakes, showing black mark on head and neck, sometimes with body banding.



Colour variations of the western brown snake complex, previously a single species, *Pseudonaja nuchalis*, recently split into 3 distinct species. Note colour is variable within these new split species and determining the species just from colouration may not be practical. Of the 3 snakes shown (formerly *P nuchalis*), photos 1 and 2 are now considered to be *P mengdeni* and the third photo is *P aspidorhyncha*.

Australian venomous snakes overview: Brown snake group

Clinical effects^{1-5,14}

- Perhaps because of their small fangs and relatively small amount of venom available, many brown snake bites are minor (dry bite) and do not require antivenom therapy. However, even a baby brown snake can deliver a lethal bite. Therefore, all bites should be treated as potentially lethal.^{1,14}
- When a brown snake does inject plenty of venom, it is fast acting and very toxic.^{1,14}
- A typical brown snake bite is either painless or almost painless. The tiny fangs often leave only faint puncture or scratch marks, without local redness, bruising or swelling.^{1,14}
- Indeed, the bite can be hard to see without a magnifying glass. Do not be fooled; even such an apparently trivial looking bite can result in lethal envenoming, particularly in young children.¹
- If a significant amount of venom has been injected, the patient may develop local draining lymph node tenderness/swelling, persistent ooze from the bite site, headache, nausea/vomiting, abdominal pain, severe coagulopathy and sometimes, kidney damage.^{2,4}
- Paralysis also is possible, but is only rarely seen in humans,^{4,14} even with severe envenoming. However, paralysis is common in domestic animals.
- Myolysis does not occur.¹⁴

.... continued



Peninsular brown snake,
Pseudonaja inframacula.



Spotted brown snake,
Pseudonaja guttata.



Five-ringed brown snake,
Pseudonaja modesta.

When a brown snake does inject plenty of venom, it is fast acting and very toxic

- In young children and occasionally in adults, early collapse with or without *grand mal* convulsions may occur.¹⁴
- In cases where there has been massive envenoming, e.g. with multiple bites, the early collapse may be associated with cardiac arrhythmias and even cardiac arrest, probably secondary to short-lived coronary vessel occlusion by thrombi generated by venom procoagulants.^{4,5}
- **Defibrination coagulopathy (VICC) is the hallmark of brown snake envenoming** (Table 22 below and on page 148).¹⁴
- Renal impairment is the other major clinical problem of brown snake bite.¹⁴ It is mostly seen in adults, especially if they have consumed alcohol prior to the bite.¹ Causation is uncertain, but may include direct nephrotoxic action of the venom, though as yet this is unproved.¹
 - Early generous I.V. hydration in all cases of brown snake bite may reduce the chance of major kidney damage.¹
- Kidney damage as part of MAHA is also most commonly seen after brown snake bite, compared to other snakes.³
- While the majority of patients with brown snake bites will not need antivenom, if there is any evidence of either coagulopathy or kidney damage, antivenom therapy is required.¹⁴

Table 22. Defibrination coagulopathy (VICC) in envenoming from brown snake bite^{1,2,5,14,21,23,40-44,51-53}

| |
|--|
| There is consumption of circulating fibrinogen and other clotting factors, with production of fibrinogen breakdown products ^{5,21} |
| Total defibrination may occur within 30 minutes of the bite ⁵ |
| In these patients with complete defibrination, INR will be > 12 (actually infinity), aPTT is > 150 seconds, the fibrinogen unmeasurably low and degradation products massively elevated ^{2,5,14,21} |
| Fortunately, platelets are often not affected, at least initially ^{5,14} |
| The patient is at risk of major haemorrhage. Fatal intracranial haemorrhages have occurred ^{2,5} |
| Anything causing hypertension should be avoided ¹ |

....table continued overleaf

Australian venomous snakes overview: Brown snake group

Table 22. Defibrination coagulopathy (VICC) in envenoming from brown snake bite^{1,2,5,14,21,23,40-44,51-53} ... cont'd

Antivenom administration has been the approach to reversing this devastating coagulopathy.¹⁴ However, recent research indicating that venom procoagulants are active for only a short time, has created uncertainty regarding the role of antivenom^{21,43,51}

Importantly, this is NOT a reason to avoid antivenom therapy as all venoms contain a variety of different toxins and antivenom would still be required to neutralise other venom components such as neurotoxins and/or cardiotoxins and nephrotoxins^{1,43}

The large amounts of antivenom recommended in the past⁴⁰ are no longer applicable and 1 or 2 vials of Brown Snake Antivenom is generally an adequate initial dose.^{1,23,41,42,44} [Please see additional explanations regarding expert panel advice in the note below]

Wait at least 6 hours, preferably longer, before definitive retesting of coagulation to determine if more antivenom is required. Such retesting is not based on return to normal clotting, but rather an indication there is even a slight improvement in parameters^{14,52}

However, if after administration of the initial dose of antivenom, the patient has continuing active major or life-threatening bleeding (including intracranial haemorrhage) urgent administration of further antivenom should be considered, in conjunction with coagulation factor replacement therapy. Seek urgent expert advice^{1,43,53}

The role of FFP in managing coagulopathy is discussed on page 137

Note regarding expert panel advice on antivenom dosing:

- Research suggests that a single vial of Brown Snake Antivenom is an adequate initial dose for neutralising the procoagulant effects of brown snake venom [41]. However, aside from procoagulants, brown snake venom comprises additional toxins (e.g. neurotoxins) [45]. Based on this, and the higher-than-anticipated venom yields for brown snakes, [46] the expert panel involved in producing this handbook believes it is important to use an initial dose sufficient to maximise the potential for neutralising all venom components in all cases, including those where unusually large amounts of venom have been injected [1].
- Therefore, the panel recommends the use of 2 vials of antivenom as an initial dose for envenoming from brown snake bite. Note: 2 vials also is the current consensus recommendation of experts such as the WA Toxicology Service and the Emergency Medicine Expert Group [42,44].
- The panel recognises that an initial dose of 1 vial will be sufficient in some cases. However, at the time of administering initial antivenom, it may not be possible to differentiate these cases from those that would benefit from a higher dose [1].
- The treating clinician will make the final decision regarding dosing, based on the circumstances of the individual case [1].
- The above advice may change with the emergence of further data from ongoing research on envenoming from snakebite.

Australian venomous snakes overview: Tiger snake group

The species^{1,49,69-71}

- Tiger snakes, genus *Notechis*, are a common cause of snakebites and the second most common cause of snakebite deaths in Australia.
- Adult fangs in the common tiger snake are about 3.5mm long. They produce a moderate amount of very toxic venom.
- The snakes vary in length up to 1.5 m, though some sub species of black tiger snake may exceed 2 m, with correspondingly longer fangs and more venom.
- Colouration for the common (mainland) species varies, though most specimens show banding. However, unbanded brown or black colour phases are seen. The black tiger snake is usually coloured as its name suggests, though juveniles may show faint banding and in some regions (i.e. Kangaroo Island, SA) they can be coppery in colour rather than black.

.....continued overleaf



Mainland tiger snake, *Notechis scutatus*.



Unbanded brown phase of mainland tiger snake.



Western tiger snake, *Notechis scutatus (ater) occidentalis*.



Black tiger snake, *Notechis scutatus (ater) ater*.



Lowlands copperhead, *Austrelaps superbus*.



Copper coloured black tiger snake from Kangaroo Island, SA.

Australian venomous snakes overview: Tiger snake group

The species^{1,49,69-71} ... cont'd

- The common copperhead, *Austrelaps superbus*, is similar in size to the common tiger snake, but has a russet sheen on the sides of the body and is not banded. There are typical pale cream or white edging to the scales of the upper and lower lips in all copperheads, a useful diagnostic feature.
- The highland copperhead, *Austrelaps ramsayi*, is similar to the common copperhead, though generally smaller. The pygmy copperhead, *Austrelaps labialis*, is smaller and grey in colour.
- The rough scaled snake, *Tropidechis carinatus*, also known as the Clarence River tiger snake, is up to 1.2 m long, with banding on the body.
- The broad headed snake, pale headed snake and Stephens' banded snake, all of the genus *Hoplocephalus*, are generally nocturnal, and small in size. Colouration depends on species.



Pygmy copperhead,
Austrelaps labialis.



Rough scaled snake,
Tropidechis carinatus.



Broad headed snake,
Hoplocephalus bungaroides.



Pale headed snake,
Hoplocephalus bitorquatus.



Stephens' banded snake,
Hoplocephalus stephensi.



Head of pygmy copperhead.
Copperheads typically have
distinctive white edging to lips
(labial scales).

Distribution^{49,69-71}

- Tiger snakes are moderately common in wetter areas of southern and coastal eastern Australia (including Kangaroo Island, SA and Tasmania), and in parts of southwest Western Australia, particularly near water courses or swamps.
- The copperheads are also found in wetter areas of south eastern Australia.
- The rough scaled snake is found in wetter areas in parts of eastern Australia.
- The broad headed snake is increasingly rare, confined to rocky areas of parts of the Great Dividing Range, NSW.
- Pale headed and Stephens' banded snakes cover NE NSW to SE QLD.



Tiger snakes,
Notechis scutatus.



Rough scaled snake,
Tropidechis carinatus.



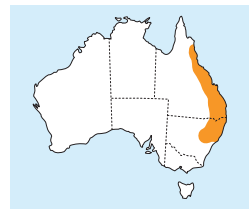
Common copperhead,
Austrelaps superbus.



Highlands copperhead,
Austrelaps ramsayi.



Pygmy copperhead,
Austrelaps labialis.



Pale headed snake,
Hoplocephalus bitorquatus.



Stephens' banded snake,
Hoplocephalus stephensi.



Broad headed snake,
Hoplocephalus bungaroides.

Australian venomous snakes overview: Tiger snake group

Venom composition

Table 23 highlights the composition of venom from snakes belonging to the Tiger snake group, which respond to bioCSL's Tiger Snake Antivenom.

Table 23. Venom composition – Tiger snake group^{1,14,49,69,72,73}

| Venom | Venom composition |
|--|--|
| Tiger snake venom | The venom is very potent and contains pre- and post-synaptic neurotoxins, myolysins, and procoagulants. Kidney damage may occur, but is probably a secondary phenomenon. Occasionally, anosmia may occur |
| Copperhead venom | Copperhead venom is less well characterised but contains pre- and post-synaptic neurotoxins and may cause myolysis. Coagulopathy is less certain |
| Rough scaled snake venom | Has effects very similar to those of tiger snake venom |
| Broad headed snakes, pale headed snakes and Stephens' banded snake venom | Contain a procoagulant, but not neurotoxins or myolysins of clinical significance |

Note: A single case of envenoming with myolysis due to *Rhinoplocephalus nigrescens* – a potentially lethal tiger type snake from eastern Australia, has been reported. There is limited evidence regarding this snake in Australia [1,30].

Clinical effects of tiger snake bite

- Unlike brown snake bites, most tiger snake bites will result in systemic envenoming, so most patients will need antivenom therapy.^{1,14,22}
- Prior to the development of antivenom, approximately 40% of all tiger snake bites were fatal.^{14,74} However, a recent large Australian case series of tiger snake envenoming showed that only 1 of 23 cases with significant envenoming, died – indicating that rapid antivenom treatment and modern intensive care management ensures survival of the majority of cases of envenoming.⁷⁵

.....continued

Most tiger snake bites will result in systemic envenoming, so most patients will need antivenom therapy

- A typical tiger snake bite is felt. It is also locally painful and often there is local redness, swelling, and bruising at the bite site. Occasionally there is a small area of skin damage. Fang puncture or scratch marks are usually visible to the naked eye.^{14,22}
- The venom will cause the usual features of systemic envenoming, such as headache, nausea/vomiting, abdominal pain and sometimes collapse.⁷⁵
- In children early collapse and *grand mal* convulsions may occur.¹
- Draining lymph nodes may be tender.¹
- Paralysis is an important feature of tiger snake bites.^{22,75}
 - The earliest sign is usually ptosis, which develops from 1 to several hours after the bite. It may be followed by progressive paralysis of ocular, facial, peripheral and glossopharyngeal muscles, the latter imperiling the airway.
 - Full paralysis of respiratory muscles may eventually develop, if no antivenom is given.
- Pre-synaptic paralysis by this venom is not reversed by antivenom, therefore it is essential that early signs of paralysis be noted and antivenom given then, prior to development of major paralysis.^{2,14,67}
- If the patient presents many hours after the bite, with major paralysis requiring intubation and ventilation, it may be necessary to continue this for days, weeks, and very occasionally, even over a month!^{1,67}
- Severe myolysis can occur with tiger snake bites, particularly if treatment is delayed or inadequate. The usual signs may occur, including muscle weakness and movement pain, myoglobinuria (red/brown urine that looks like haematuria and tests positive for blood) and later development of muscle wasting.^{14,22}

.....continued overleaf

Paralysis is an important feature of tiger snake bites... it is essential that early signs of paralysis be noted and antivenom given then, prior to development of major paralysis

Australian venomous snakes overview: Tiger snake group

Clinical effects of tiger snake bite ... cont'd

- Secondary kidney failure may occur, as may severe hyperkalemia – a potentially fatal complication requiring heroic measures, including dialysis.^{1,14,22}
- Defibrination coagulopathy is a common feature of tiger snake bites.^{14,22,75}
It may be profound as with brown snake bites, and lethal intracranial haemorrhages have occurred.^{1,22}

However, unlike brown snake bite defibrination, which is slow to resolve without antivenom therapy, tiger snake bite defibrination often completely resolves spontaneously after 15 to 18 hours, even without antivenom therapy.^{1,67}

Thus a tiger snake bite seen very late may have apparently normal clotting, but degradation products will still be raised, as evidence of the earlier coagulopathy.⁶⁷

- **Importantly however, given the danger of major haemorrhage, letting the coagulopathy resolve spontaneously is definitely not a clinical option, and, if present, coagulopathy should be treated with antivenom.**^{1,75}
The PI advises the use of 1 vial (or more if required).²⁴ Expert opinion favours the use of 2 vials* of bioCSL's Tiger Snake Antivenom.^{1,42,44}
- Use of FFP is controversial. See page 137 for further discussion regarding FFP.
- Kidney damage does occur, though less commonly than with brown snake bites, and causation is multifactorial, due to secondary factors such as myolysis and coagulopathy.¹
- MAHA can occur.²²

*Regarding the dose of bioCSL's Tiger Snake Antivenom:

- Venom yields from tiger snakes can be higher than anticipated [46].
- Therefore, in the opinion of the expert panel involved in producing this handbook, it is important to use an initial dose of 2 vials to maximise the potential for neutralising all venom components in all cases, including those where unusually large amounts of venom have been injected [1].
- Note: 2 vials also is the current consensus recommendation of experts such as the WA Toxicology Service and the Emergency Medicine Expert Group [42,44].
- The panel recognises that the initial dose of 1 vial will be sufficient in some cases. However, at the time of administering initial antivenom, it may not be possible to differentiate these cases from those that would benefit from a higher dose [1].
- The treating clinician will make the final decision regarding dosing, based on the circumstances of the individual case [1].
- The above advice may change with the emergence of further data from ongoing research on envenoming from snakebite.

Clinical effects: Copperheads and other snakes in the tiger snake group

- Copperhead bites are infrequently reported and there is a paucity of good clinical data.¹

From available information, it appears that the common copperhead can cause severe envenoming, with paralysis (pre- and post-synaptic) and possibly myolysis, but defibrination type coagulopathy is unlikely, though secondary kidney damage might occur.

Bites are likely to cause local pain and swelling.

Bites by the highland copperhead are likely to be similar to those of the common copperhead.

For the pygmy copperhead in South Australia, all bites recorded thus far have been minor, with minimal local pain or swelling and no significant systemic effects. However, this does not imply that every bite will be minor.

- Bites by the broad headed snake, pale headed snake and Stephens' banded snake can cause severe defibrination coagulopathy, but not paralysis or myolysis. Clinically they behave similarly to brown snake bites, with which they may be confused.⁷³
- Bites by rough scaled snakes can be severe, with local pain and swelling, paralysis, myolysis, defibrination coagulopathy and secondary kidney damage.^{1,72}

The common copperhead can cause severe envenoming, with paralysis (pre- and post-synaptic) and possibly myolysis

Australian venomous snakes overview: Black snake group

The species^{1,76}

- The mulga snake or king brown, *Pseudechis australis*, is a large land snake, which may exceed 2.5 m in length.

It has moderately large fangs and produces more venom than any other Australian snake. Fortunately, this venom is less toxic than venoms produced by some of the other species.

It is brown to red brown in body colour, with cream coloured belly scales. The body scales have distinctive colouration, being paler brown or cream on the inner aspect, darkening towards the margin of each scale. This gives the snake a subtle reticulated appearance, but is easily mistaken for a uniform brown colour.

The head is large and triangular in shape, compared to the true brown snake.

- Butler's mulga snake, *Pseudechis butleri*, is similar except it has more distinct yellow or cream markings on the body, producing a speckled pattern in some specimens.
- Pygmy mulga snake, *Pseudechis weigeli*, is smaller than *P australis* (maximum length about 1.2 m). Other differences include a contrasting head and neck pattern, relatively narrower head, and all subcaudal scales being single.
- Collett's snake, *Pseudechis colletti*, may exceed 2.5 m in length. It is brown with pinkish specks on the body, which is almost banded in some specimens.
- The red bellied black snake, *Pseudechis porphyriacus*, is a uniformly black coloured snake with a deep red belly.
- The blue bellied or spotted black snake, *Pseudechis guttatus*, has a black or dark grey-brown body with black specks and a dark to black belly.



Mulga snake ("King Brown"), *Pseudechis australis*.



Collett's snake, *Pseudechis colletti* (two specimens shown).



Distribution^{1,76,77}

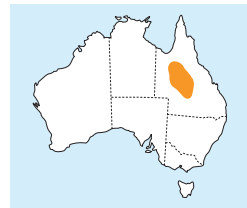
- The mulga snake is common in arid and semi arid areas through to tropical areas in northern Australia. It is found in some urban areas in central and northern Australia.
- Butler's mulga snake is restricted to inland areas of the southern half of Western Australia.
- Pygmy mulga snake is restricted to the wet-dry tropics of northern Australia, from the Kimberley region east across to Winton in Queensland, usually in close association with freshwater creeks and rivers. Seemingly increasingly present in the northern portions of its range, possibly reflecting the corresponding decrease in mulga snakes and other species due to the recent arrival of poisonous cane toads, which the pygmy mulga snake apparently avoids.⁷⁷
- Collett's snake is found in a restricted area of central Queensland.
- The black snakes are found in a variety of habitats in eastern Australia. The commonest species, the red bellied black snake prefers wetter areas, often near water. The blue bellied black snake is more common in dryer habitats, notably in rocky areas.



Mulga snake,
Pseudechis australis.



Butler's mulga snake,
Pseudechis butleri.



Collett's snake,
Pseudechis colletti.



Red bellied black snake,
Pseudechis porphyriacus.



Spotted/blue bellied black snake,
Pseudechis guttatus.

Australian venomous snakes overview: Black snake group

Venom composition

- Mulga snake venom contains several potent phospholipase toxins that are either myotoxins, neurotoxins, or haemolysins. It also contains an anticoagulant toxin, but no procoagulant.⁷⁶
- Butler's mulga snake, Collett's snake and the pygmy mulga snake are presumed to have similar venom components to that of the mulga snake.^{1,76}
- Red bellied and blue bellied/spotted black snakes similarly contain neurotoxins and myolysins, though possibly not as potent as related species and minor myolysis may occur as may anticoagulant coagulopathy (and occasionally, anosmia may also occur). In domestic animals, paralysis can occur, but this is not recorded in humans.^{1,16,35,76,78}



Pygmy mulga snake,
Pseudechis weigeli.



Red bellied black snake,
Pseudechis porphyriacus.



Blue bellied or spotted black snake, *Pseudechis guttatus*
(two colour phases shown).



Mulga snake venom contains several potent toxins that are either myotoxins or neurotoxins. It also contains an anticoagulant toxin, but no procoagulant

Clinical effects

- It is not known what proportion of mulga snake bites would be lethal if no treatment were given, although a figure of 30% has been estimated.⁶⁷ Due to the size of mulga snakes and the large amount of venom available, envenoming is expected to develop in most cases.
- Locally, mulga snake bites cause pain and extensive swelling in the majority of cases,^{4,14} which may involve much of the bitten limb. However tissue damage is rare and the swelling usually subsides over 2-4 days.¹
- **The principal clinical problem with mulga snake bites is myolysis,⁴ which may be severe, with potential for secondary kidney failure and hyperkalaemia.** The usual clinical features of myolysis may be expected, including myoglobinuria (red to brown urine testing positive for blood), skeletal muscle weakness and pain on movement.^{4,14}
- Defibrination coagulopathy is not seen,⁴ but there may be slight elevation of clotting times due to anticoagulants in the venom.¹⁴
- Occasionally, there may be an obvious anticoagulant coagulopathy, with prolonged prothrombin time and aPTT, but normal fibrinogen and no fibrin(ogen) degradation products.¹⁴
- As there are neurotoxins in the venom, paralysis might be predicted, but in clinical practice, neuromuscular paralysis of clinical significance is rarely seen⁴ (except for ptosis), though there may be detectable muscle weakness secondary to muscle damage.
- Collett's snake and probably Butler's mulga snake cause similar clinical effects
 - i.e. early generalised systemic effects (nausea, vomiting, abdominal pain, diarrhoea and headache) and an anticoagulant coagulopathy, followed in some cases by rhabdomyolysis and acute renal failure in untreated patients within 24 hours.^{1,17}
- Bites by red bellied and blue bellied/spotted black snakes are rarely lethal and most commonly produce marked local envenoming and generalised systemic symptoms, but do not cause paralysis or defibrination coagulopathy.^{1,16,35,78} Anticoagulant coagulopathy occurs frequently, and occasionally, major myolysis is seen. Antivenom therapy may be clinically indicated in envenomed patients.¹⁶ Some recent evidence suggests that administering antivenom early (within 6 hours) to patients with prolonged aPTT may be associated with a reduced incidence of significant myolysis.¹⁶

Australian venomous snakes overview: Death adder group

The species^{1,79}

- Death adders, Genus *Acanthophis*, are distinctive snakes with triangular heads, squat bodies and thin tails, sometimes used as a lure.
- They are the predominant dangerous Australian venomous snake that are habitually nocturnal, although most other species will be active on hot nights.
- They are also known as “deaf” adders, with good reason. Like all snakes, they lack external ears and so rely on vibration rather than hearing to detect the approach of large animals, including humans.
- All other dangerous Australian snakes will usually try and move away from a potential threat. Instead, death adders often rely on camouflage, burrowing deeper into leaf litter or other ground debris, rather than moving out of the way.
- If stepped on they will bite rapidly and effectively.
- Death adders seem to have adjusted poorly to human encroachment on their environment. In many parts of southern Australia their range is contracting. They are a rather infrequent cause of bites now.



Common death adder,
Acanthophis antarcticus.



Desert death adder,
Acanthophis pyrrhus.



Northern death adder,
Acanthophis praelongus.

Distribution^{1,79}

- Death adders are found in a variety of habitats, both arid and temperate to tropical, but in most parts of their current range, they are encountered infrequently.
- In some areas this is due to contracting populations of these snakes, but in other areas it is more likely due to their cryptic habits.
- They are not found in Tasmania or the islands off the southern coast.



Death adders, *Acanthophis* spp.

Venom composition^{14,79}

- Death adders have large fangs, producing a considerable quantity of toxic venom.
- The venom does not contain either procoagulants or myolysins of significance.
- In cases of death adder bite in Australia* the only major effect of the venom appears to be pre- and post-synaptic neurotoxic paralysis.

*Death adder species in PNG are sometimes associated with an anticoagulant coagulopathy and mild myolysis [30].

In cases of death adder bite in Australia, the only major effect of the venom appears to be pre- and post-synaptic neurotoxic paralysis

Australian venomous snakes overview: Death adder group

Clinical effects

- Death adder bites were greatly feared 50-100 years ago – carrying a high rate of mortality due to profound paralysis.⁷⁴
- With current medical facilities, paralysis should be a non-lethal complication of snakebite in Australia in all but the most exceptional cases. However, deaths do still occur (and this is particularly a problem in PNG, where bites often occur in remote localities and respiratory paralysis may be inadequately treated).^{1,14,30}
- Many bites should result in systemic envenoming, but in practice, at least in urban areas in Australia, nearly all bites are from captive specimens and often do not result in significant envenoming. However, in PNG, death adder bites remain a major problem and threat to life.^{1,14,30}
- Where venom has been injected, there is often local pain and mild swelling, followed by progressive development of paralysis over the next few hours, starting with ptosis, then affecting other cranial nerves, peripheral muscles, and finally, if untreated, respiratory muscles.¹⁴
- Major respiratory paralysis can occur within 6 hours of the bite, but occasionally onset of paralysis may be delayed to 24 hours post bite.¹⁴
- In Australia, defibrination coagulopathy, myolysis and major kidney damage do not occur,^{4,14} although the latter might conceivably occur as a secondary complication. However, as noted previously, in PNG, death adder bites tend to be severe and life threatening, and in addition to paralysis, anticoagulant coagulopathy and mild myolysis may be seen.³⁰

Where [death adder] venom has been injected, there is often local pain and mild swelling, followed by progressive development of paralysis over the next few hours

Australian venomous snakes overview: Taipans

The species⁸⁰

- The taipans, genus *Oxyuranus*, are (at least in theory), amongst the most deadly snakes in the world, combining very potent venom, large amounts of venom and long fangs with accurate strike.
- The common taipan, *Oxyuranus scutellatus* may exceed 2.5 m in length and is found in mixed habitats. It often has a pale head with olive to brown body and is a swift moving snake.
- The common taipan may have fangs greater than 1cm in length, capable of penetrating a leather boot! This, coupled with the large quantity of venom produced, has helped fuel the fearsome reputation of these snakes.
- The inland taipan, *Oxyuranus microlepidotus* (small scaled snake; fierce snake) has a more restricted natural distribution, but is also a large snake, sometimes exceeding 2.5 m. Colour is variable, some specimens have a shiny black sheen to the head at some times of the year.
- The recently described Central Ranges taipan, *Oxyuranus temporalis*, is currently known from five specimens from Western Australia – near the border with South Australia and Northern Territory and several hundred kilometres south of this point.¹



Coastal taipan,
Oxyuranus scutellatus.



Inland taipan,
Oxyuranus microlepidotus.



Central Ranges taipan,
Oxyuranus temporalis.

Australian venomous snakes overview: Taipans

Distribution^{1,80}

- The common taipan is found across the top of Australia and part way down the east coast, and may enter urban areas.
- The inland taipan is restricted to arid areas, mostly in black soil plains country in part of central inland Australia, including some of the Lake Eyre drainage basin. It is only rarely encountered, but is possibly common within its range, where it may spend much of the time beneath the surface, in the deep cracks in the soil.
- A third species of taipan (Central Ranges taipan) has been described from inland Australia, but is only known from five specimens.¹
- Because of their reputation, taipans are much sought after by reptile collectors, and a considerable number of these snakes are kept alive in both public and private collections in capital cities and some rural towns, thus bites may present well outside of the natural range for these snakes.^{1,4}



Common taipan,
Oxyuranus scutellatus.



Inland taipan,
Oxyuranus microlepidotus.



Central Ranges taipan,
Oxyuranus temporalis.

Venom composition^{14,80}

- Taipan venom is amongst the most potent of all snake venoms.
- Inland taipan venom is the most toxic snake venom known.
- Taipan venom components include pre- and post-synaptic neurotoxins, powerful procoagulants, myolysins, and clinically, kidney damage may occur, though this may be secondary.

Clinical effects

- Prior to the development of specific taipan antivenom by CSL in 1956, taipan bite was nearly always fatal.¹⁴ There were only two reported survivors of taipan bite in the years before 1956.¹
- Nearly all taipan bites are likely to result in life-threatening envenoming, with the exception of bites by juveniles in captivity, where current experience suggests that the rate of major bites may be lower.¹
- There is often pain and swelling at the bite site, though this is not always so and a trivial looking bite site does not imply a trivial bite.¹⁴
- There may be rapid development of major systemic envenoming, including headache, nausea/vomiting, collapse, convulsions (especially in children), paralysis, defibrination coagulopathy, myolysis and kidney damage.^{1,4,14}
- The paralysis may be severe, with major respiratory paralysis developing within 2 to 6 hours of the bite in some cases.¹
- The coagulopathy is often profound, with complete defibrination within an hour of the bite and a potential for major haemorrhage, including cerebral haemorrhage.^{1,5}
- Myolysis, if present, is often not as severe as seen with some tiger snake and mulga snake bites.¹⁴
- Kidney damage, while not a constant feature of taipan bite, does occur sometimes, possibly as a secondary phenomenon.¹⁴ There is one case of renal cortical necrosis following taipan bite.¹

Nearly all taipan bites are likely to result in life-threatening envenoming....paralysis may be severe [defibrination] coagulopathy is often profound

Australian venomous snakes overview: Sea snakes

The species^{1,15}

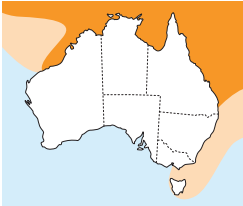
- There are 31 species of sea snake in northern Australian waters alone. All are possibly dangerous to humans, but relatively few of these have caused bites of significance.
- Sea snakes are closely related to the venomous Australian land snakes of the family Elapidae, and are currently classified in a subfamily, Hydrophiinae.
- Two subfamilies have been listed in the past, the sea kraits, “Laticaudinae”, and the true sea snakes, “Hydrophiinae”, though recent research suggests this previous subfamilial division may be inappropriate. All spend some of their life (sea kraits) or all of their life (sea snakes) in the sea.
- Most are fish eaters.
- They may be inquisitive but usually are not aggressive unless threatened, such as when caught in a fishing net.
- A description of all species is beyond the scope of this publication.



Sea snake.

Distribution

- Sea snakes are found predominantly in the northern waters of Australia, though storms may carry the occasional specimen southward, with bites authenticated from as far south as Sydney.¹⁵
- They are not likely to be found in waters off the southern coast of Australia, where alleged sea snake bites are essentially always due to some other organism, usually an eel.



Sea snakes: Distribution.

Venom composition¹⁵

- Sea snake venoms have been the subject of much research, because of their post-synaptic neurotoxins, many of which have been sequenced.
- The other important component of some sea snake venoms is myotoxin, which may dominate the clinical picture.

Clinical effects¹⁵

- Sea snake bite is usually felt, with small but distinct teeth marks visible, which may be multiple, mostly from non-fang teeth.
- Pain at the bite site is not a major feature – neither is swelling.
- The important effects, seen only in some cases, are systemic – either paralysis and/or myolysis.
- If envenoming has occurred, then either paralysis or myolysis may be expected to occur within 4 hours in most cases, manifested as either early paralysis (e.g. ptosis, ophthalmoplegia, limb or respiratory weakness) or myolysis (e.g. myoglobinuria, muscle pain and weakness).
- Secondary kidney damage may occur if there is major myolysis and there may also be severe hyperkalaemia.
- Coagulopathy is not seen.

If envenoming has occurred, then either paralysis or myolysis may be expected to occur within 6 hours in most cases

Snake identification

Scale counting¹

On occasion, bystanders may kill the culprit snake, and present it with the patient. The practice of catching or killing a snake should **be actively discouraged** and patients/bystanders should be advised to never repeat this practice. However, when a snake is presented, it may be possible to perform scale counting on the dead snake to assist with identification.

While a dead snake is essentially harmless, care must be taken when handling to avoid the possibility of finger-prick envenoming from contaminated fangs.

Performing scale counts is not simple and it is vital to seek assistance from an expert.

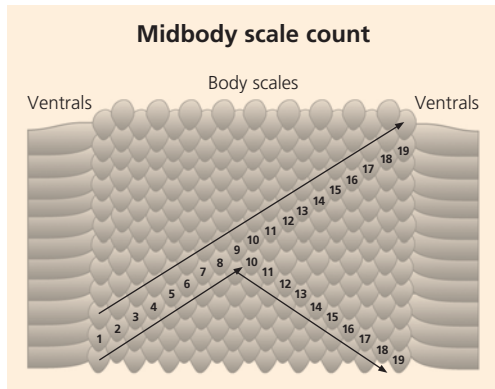
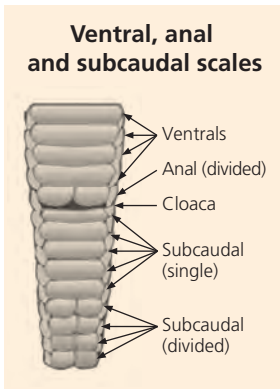
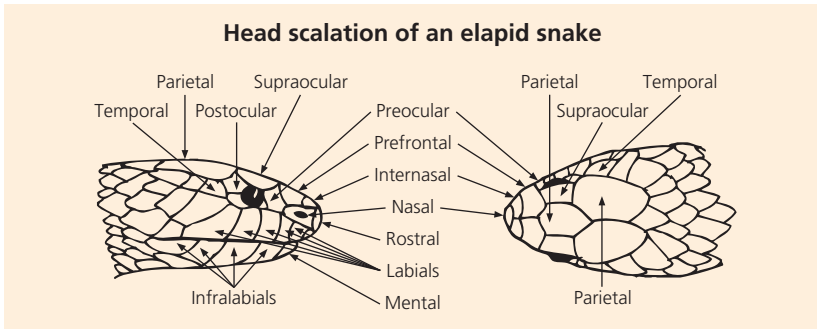
Needless to say, a snake that has been caught alive and brought in (miraculously) without having bitten anyone else – should never be handled by healthcare professionals for scale counting or any other purpose. Professional snake handlers should be brought in to deal with the situation.

Counting snake scales: Key points¹

- Managing the patient should always take priority over snake identification.
- Always confer with an expert regarding the procedure for scale counting. If discussing over the phone, send a photo of the snake.
- Remember, despite best efforts, errors can occur and misidentification is a distinct possibility even when guided by an expert.
- Importantly, the clinician **should not rely on scale count alone** for diagnostic decision making.
 - Ensure that the snake identity determined via scale counting is consistent with the clinical features of the patient.
 - If there is any doubt/ambiguity, management decisions should be based on the patient's clinical signs/symptoms rather than scale count results.
- Finally, consider whether or not the snake presented is the actual snake that caused the bite (see page 172 for further information).

Tip

When performing scale counts, use a coloured texta/marker to mark each scale as it is counted. This avoids the problem of losing one's 'spot' while counting.¹



Underside (ventral) of cloacal region and tail of brown snake.

Scale counts: Australian venomous snakes

| Snake | Mid-body | Ventral | Anal | Subcaudals | Adult length |
|---|----------|---------|---------|-------------------|--------------|
| Eastern brown snake <i>Pseudonaja textilis</i> | 17 | 192-231 | Divided | 57-72; divided | 1.5m |
| Tropical brown snake <i>Pseudonaja nuchalis</i> | 17 | 194-207 | Divided | 57-66; divided | 1.5m |
| Patch nosed brown snake <i>Pseudonaja aspidorhyncha</i> | 17 | 207-226 | Divided | 47-63; divided | 1.4m |
| Gwardar <i>Pseudonaja mengdeni</i> | 17 | 193-224 | Divided | 49-64; divided | 1.2m |
| Dugite <i>Pseudonaja affinis</i> | 17-19 | 204-226 | Divided | 51-63; divided | 1.2m |
| Peninsular brown snake <i>Pseudonaja inframacula</i> | 17 | 194-208 | Divided | 55-66; divided | 1.4m |
| Speckled brown snake <i>Pseudonaja guttata</i> | 19-21 | 190-220 | Divided | 45-70; divided | 0.9m |
| Ingram's brown snake <i>Pseudonaja ingrami</i> | 17 | 190-220 | Divided | 55-70; divided | 1.2m |
| Ringed brown snake <i>Pseudonaja modesta</i> | 17 | 145-175 | Divided | 35-55; divided | 0.6m |
| Common tiger snake <i>Notechis scutatus</i> | 17-19 | 140-190 | Single | 35-65; single | 1.2m |
| Black tiger snake, <i>Notechis (scutatus) ater</i> (several subspecies) | 15-21 | 140-185 | Single | 35-60; single | 0.7 to 1.4m |
| Rough scaled snake <i>Tropidechis carinatus</i> | 23 | 160-185 | Single | 50-60; single | 0.6m |
| Pale headed snake <i>Hoplocephalus bitorquatus</i> | 19-21 | 190-225 | Single | 40-65; single | 0.5m |
| Broad headed snake <i>Hoplocephalus bungaroides</i> | 19-21 | 200-230 | Single | 40-65; single | 0.5m |
| Stephens' banded snake <i>Hoplocephalus stephensii</i> | 21 | 220-250 | Single | 50-70; single | 0.5m |
| Lowland copperhead <i>Austrelaps superbus</i> | 15-17 | 140-165 | Single | 35-55; single | 1.0m |
| Highlands copperhead <i>Austrelaps ramsayi</i> | 15-17 | 150-165 | Single | 35-55; single | 0.7m |

Scale counts: Australian venomous snakes

| Snake | Mid-body | Ventral | Anal | Subcaudals | Adult length |
|---|----------|---------|---------|-------------------------|--------------|
| Pygmy copperhead <i>Austrelaps labialis</i> | 15 | 140-155 | Single | 35-55; single | 0.45m |
| Mulga snake <i>Pseudechis australis</i> | 17-19 | 185-225 | Divided | 50-75; first few single | 2.3m |
| Butler's mulga snake <i>Pseudechis butleri</i> | 17-19 | 200-220 | Divided | 55-65; first few single | 1.2m |
| Collett's snake <i>Pseudechis colletti</i> | 19-21 | 215-235 | Divided | 50-70; first few single | 2.0m |
| Red bellied black snake <i>Pseudechis porphyriacus</i> | 17-19 | 170-215 | Divided | 40-65; first few single | 1.4m |
| Blue bellied black snake <i>Pseudechis guttatus</i> | 19-21 | 175-205 | Divided | 45-65; first few single | 1.5m |
| Papuan black snake <i>Pseudechis papuanus</i> | 19-21 | 220-230 | Divided | 45-75; first few single | 1.4m |
| Common taipan <i>Oxyuranus scutellatus</i> | 21-23 | 220-250 | Single | 45-80; divided | 1.8m |
| Inland taipan <i>Oxyuranus microlepidotus</i> | 23-25 | 220-270 | Single | 55-70; divided | 1.8m |
| Central Ranges taipan* <i>Oxyuranus temporalis</i> | 21* | 250* | Single | 60*; divided | 1.2m* |
| Common death adder <i>Acanthophis antarcticus</i> | 21-23 | 110-135 | Single | 35-60; first few single | 0.6m |
| Desert death adder <i>Acanthophis pyrrhus</i> | 19-21 | 136-158 | Single | 43-63; first few single | 0.5m |
| Northern death adder <i>Acanthophis praelongus</i> | 21-23 | 120-140 | Single | 39-57; first few single | 0.45m |
| Pilbara death adder <i>Acanthophis wellsii</i> | 17-21 | 123-141 | Single | 41-55; first few single | 0.35m |
| Yellow faced whip snake <i>Demansia psammophis</i> | 15 | 165-230 | Divided | 60-105; divided | 1.0m |
| Pythons (various species): non-venomous | >35 | – | – | – | – |

*Based on single specimen.

Table copyright A/Prof Julian White.

Snake identification

Is the bite from a venomous snake or a non-venomous creature?

When a patient claims to have been bitten by a snake, the clinician must assume that it was a venomous snake and approach investigations accordingly.

Nevertheless, while the majority of snakebites in Australia are likely caused by potentially dangerous venomous species – there are a number of snake species which can attain significant length, which are either completely non-venomous (pythons, some colubrids), or are rear-fanged venomous and are not able to cause significant envenoming in humans (some colubrids).¹

In particular, pythons are easily distinguished from venomous snakes and determining that the snake was a python at an early stage can obviate the need for expensive and time consuming investigations in a case of suspected snakebite. However, this is only possible where an identifiable snake is presented with the patient, or a series of good quality photographs of the snake are readily available, and expert assistance is available. [The caveat stands, regarding the possibility of mis-identification even with the help of an expert].¹

It is essential to confirm that the snake specimen/photos are of the actual snake that was seen biting the patient.¹

Beware of identifying a snake found later in the location where a snakebite was suspected to have occurred, as it may not be the culprit snake. Ask direct questions such as 'is this the snake that was definitely seen biting the person?' and 'when and how was the snake caught?'

It is essential to confirm that the snake specimen/photos are of the actual snake that was seen biting the patient

Pythons

Pythons have a different scale structure to venomous snakes and are readily distinguished in Australia by a significantly higher mid-body scale count. Also many species have a very different head structure to venomous snakes and have heat sensing pits in the nasal region and along the lower jaw. Their colouration can also be distinctive, such as carpet pythons and diamond pythons.¹

Colubrid snakes

Colubrid snakes, either non-fanged or rear-fanged/back-fanged can be more difficult to distinguish from medically important venomous species and identification requires the services of an expert herpetologist. The scalation around the head, notably the side of the head, can be important in the identification process.¹

The scalation around the head [of colubrids], notably the side of the head, can be important in the identification process



Head of carpet python, with infralabial heat sensing pits visible.



Carpet python.



Woma python.

Snake identification

Legless lizards¹

Australia is home to variety of legless lizard species, which are sometimes mistaken for snakes. At least one species has a colouration similar to that of a juvenile brown snake. However, legless lizards have a number of quite clear identification features that allow them to be readily distinguished from snakes provided the specimen is available for examination. The presence of an external ear opening, a fleshy rather than forked tongue, a long tail in comparison to body and vestigial hind limbs (large flap-like scales) in the anal area are all distinguishing features.



Legless lizard.



Fleshy (as opposed to forked) tongue.



Legless lizard, external ear.



Legless lizard showing tail longer than body (in snakes, the body is much longer than the tail).



Legless lizard, hind limb (residual) 'flaps'.

Section 4

Spiders

- Background
- Spiderbite: Clinical presentation
- Spiderbite: First aid
- Spiderbite: Diagnosis
- Management of envenoming due to spiderbite
- Antivenom therapy
- Necrotic arachnidism

In this section

Spiderbite: Background

Overview 177

Spiderbite: Clinical presentation

Local signs/symptoms 178
General systemic effects 178
Specific systemic effects 179
Spiderbite in children 179
Problem presentations 180
Specific presentations 181
Funnel web spider bite 182
Red back spider bite 183

Spiderbite: First aid

General principles 184
Funnel web spider bite: Timing of removal of PBI first aid 184

Spiderbite: Diagnosis

Principles of diagnosis and management 186
Spiderbite: Observation 187
History 188
Examination 189
Laboratory investigations 189
Key symptoms/signs of envenoming based on spider species 190
Diagnostic algorithms 191

Spiderbite decision tree based on clinical presentation (flowchart) 192

Spider identification

Identifying a spider 193

Management of envenoming due to spiderbite

Basic principles 195
Urgent treatment 196
Management of suspected or confirmed funnel web spider bite (flowchart) 197
Management of suspected or confirmed red back spider bite (flowchart) 198

Specific treatment for funnel web spider bite: Antivenom

Indications for antivenom therapy 199
Timing of antivenom therapy 199
Initial dose of bioCSL's Funnel Web Spider Antivenom 200
Follow-up dosing: bioCSL's Funnel Web Spider Antivenom 200

Route of administration of bioCSL's Funnel Web Spider Antivenom 201
Monitoring patients after antivenom therapy 201
What if bioCSL's Funnel Web Spider Antivenom is not immediately available? 202

Specific treatment for red back spider bite: Antivenom

Envenoming from red back spider bite 203
Indications for bioCSL's Red Back Spider Antivenom 203
Timing of bioCSL's Red Back Spider Antivenom therapy 203
Dose of bioCSL's Red Back Spider Antivenom 204
Route of administration of bioCSL's Red Back Spider Antivenom 205
What if bioCSL's Red Back Spider Antivenom is not immediately available? 206

Spider antivenoms: Preparation, administration and complications

Preparation prior to commencing antivenom therapy 207
How to administer spider antivenoms:
 bioCSL's Funnel Web Spider Antivenom 208
 bioCSL's Red Back Spider Antivenom 209
Observation during antivenom therapy 210
Premedication prior to administering antivenom 211
What to do if there is an adverse reaction to antivenom 213
Antivenom therapy: Commonest mistakes 214
Complications of antivenom therapy 215
Management of serum sickness 216

Necrotic arachnidism

Necrotic arachnidism: Evidence *versus* mythology 217
Necrotic skin lesions: Approach to investigations (flowchart) 220

Australian spiders overview

Funnel web spider group 221
Red back spiders 223
Mouse spiders 226
Other spiders 227

Spiderbite: Background

Overview

- Australia is home to a vast array of spider species, most of which are small and unlikely to bite or cause significant effects in humans.^{1,2}
- Clinical effect profiles have been described for bites by only a limited number of spider species.¹⁻³
- For the majority of reported symptomatic spider bites, the predominant feature is local pain, usually of short duration.^{2,3}
- A few species can cause significant envenoming, either local or systemic, and one group, the **funnel web spider group, can cause potentially lethal envenoming in humans.**⁴⁻⁶
 - Consequently, the funnel web spider group drives the diagnostic process for spiderbite. It is essential to rapidly assess and treat any bite by a spider that may be a funnel web species.⁶
 - Unfortunately, confounding the diagnostic process for the funnel web spider group are a significant number of similar looking spiders that are broadly grouped as ‘big black spiders’.^{6,7}
- A second group of medical significance includes the red back spiders. While unlikely to cause lethal envenoming, bites from these spiders can result in effects, which frequently require medical treatment.^{3,4}
- To assist in appropriate triage and management of patients with spiderbite a diagnostic algorithm has been developed based on clinical presentation (see page 192).⁸

The funnel web spider group drives the diagnostic process for spiderbite. It is essential to rapidly assess and treat any bite by a spider that may be a funnel web species

Spiderbite: Clinical presentation

Presenting symptoms following spiderbite can be highly variable, depending on the type of spider involved. Importantly, bites from some medically important spiders can cause a fairly specific and diagnostic set of symptoms.^{2,7}

Local signs/symptoms^{2,7,9,10-13}

- No symptoms to minor swelling; erythema.
- Bites may be painless through to exquisitely painful with pain being either immediate or delayed.
- Bite marks are frequently invisible, but larger species (e.g. funnel web spiders/ big black spiders, and huntsman spiders) can cause obvious punctures, occasionally with local short-lived bleeding.
- Lymph nodes draining the bite area may sometimes be swollen and/or tender, indicating venom movement.
- ‘Necrotic arachnidism’, i.e. skin damage/ulceration/necrosis caused by suspected spiderbite, is generally an inappropriate diagnosis in Australia as evidence supporting the association between spider bites in Australia and skin necrosis, is lacking^{2,12,13} (see pages 217-220 for further explanation).

General systemic effects^{2,3,4,9}

- Non-specific general systemic effects following spiderbite are uncommon.
- Symptoms may include nausea/vomiting, headache, abdominal pain.



Sydney funnel web spider (male), *Atrax robustus*.



Fangs of Sydney funnel web spider *A. robustus*.



Red back spider (female), *Latrodectus hasseltii*.

Specific systemic effects

Envenoming from spiderbite can lead to a number of specific systemic effects with varied presentations (Table 24).

Table 24. Specific systemic effects of spider venoms: Presenting signs/symptoms^{4,8,9,14}

| Systemic effect of spiderbite | Presentation |
|-------------------------------|--|
| Neuroexcitatory envenoming | <ul style="list-style-type: none">– Hypertension/hypotension– Tachycardia/bradycardia– Piloerection– Increased sweating– Hypersalivation/lacrimation– Muscle spasm/pain– Respiratory distress/pulmonary oedema– Muscle fasciculation– Severe abdominal or chest pain |
| Systemic myolysis | In general does not occur, although rare cases of envenoming from red back spider bite can be associated with a mild rise in CK |
| Coagulopathy | Does not occur* (except in rare cases as a secondary complication) |
| Renal damage | Does not occur* (except in rare cases as a secondary complication) |

*Important diagnostic feature.

Spiderbite in children^{3,8,9,10,14}

In children, especially young children, spiderbite can lead to more severe and rapidly developing envenoming (funnel web spiders; red back spiders). Young children may present with any of above symptoms but without a clear history of spiderbite, necessitating a high index of suspicion for accurate diagnosis.

Spiderbite: Clinical presentation

Problem presentations

- Generally, in adults, problem/cryptic presentations of spiderbite are of lesser relevance to clinical practice (than for snakebite).
- This is primarily because clinical experience suggests that spiderbite may be overdiagnosed given the tendency in the general community to associate any sort of localised skin reaction with spiderbite, and in particular, the inappropriate association between skin damage and the white tail spider.^{2,3,8,12,13}
 - Many cases of alleged spiderbite may not be associated with a witnessed bite – resulting in a speculative diagnosis of spiderbite.⁸
 - On the other hand, the most important spiderbite, i.e. that from funnel web spiders, is mostly associated with a clear episode of a bite being felt and a spider seen.^{2,6}
- Red back spider bite, though commonly presenting without a clearly witnessed bite, frequently presents with clear symptoms and signs pointing to a diagnosis.^{8,9}
 - The patient may present with abdominal or chest pain in the absence of a noticed bite.^{4,8,9}
 - In such cases, careful questioning usually will elicit a history of possible exposure to spiderbite and initial localised pain, indicating the true diagnosis.^{4,8}
- Consequently, unlike snakebite, medically significant spiderbite will present with clear symptomatology in most cases.⁸

Unlike snakebite, medically significant spiderbite will present with clear symptomatology in most cases

Specific presentations

Funnel web spider group

- Funnel web spiders only occur naturally in parts of eastern and southeastern Australia (see map on page 221).^{5,7,14,15} Bites by the numerous species within this group are potentially rapidly lethal. The bites are often painful and a bite mark is usually seen.^{7,9,14}
- Medically significant bites have thus far been confined to eastern NSW and southeast QLD.^{9,14}
- Bites by a variety of related spiders (big black spiders) may be confused with those of true funnel web spiders.⁷
- However, the majority of funnel web spider bites will prove to be minor and will not lead to systemic envenoming.^{7,9}
- Table 25 (page 182) depicts features relating to systemic envenoming from funnel web spider bite.

Mouse spiders

- Mouse spiders of the genus *Missulena* have venom similar to funnel web spiders and potentially can cause a similar envenoming syndrome.^{7,16}
- However, clinical experience indicates that bites are uncommon and significant envenoming occurs very rarely.^{2,16}

Red back spider group

- Red back spiders are part of the widow spider group (genus *Latrodectus*).
- These spiders are common as well as a common cause of bites.^{2,3}
- The bite site may be painless or it may feel like a pinprick.¹⁰
- In one study, the majority of definite red back spider bites caused significant effects including severe persistent pain in two-thirds of cases.⁴
- While often distressing for the patient, red back spider envenoming is not likely to be lethal.³ There have been no reports of death from a red back spider bite since the introduction of antivenom.⁹
- Clinical features of systemic envenoming due to red back spider bite are shown in Table 26 on page 183.

Spiderbite: Clinical presentation

Funnel web spider bite

Table 25. Funnel web spider bite: Clinical presentation^{3,7-9,14}

| | |
|---|---|
| Bite and bite site | <ul style="list-style-type: none">– The bite itself is painful– Usually, fang marks are obvious– The spider may remain attached to the patient until shaken off or otherwise removed |
| Timing of development of systemic envenoming | <ul style="list-style-type: none">– Where systemic envenoming develops, it will do so within 4 hours of the bite¹⁴– Initial symptoms may develop as early as 10-15 min post bite |
| Systemic envenoming symptoms | <ul style="list-style-type: none">– Earlier symptoms are perioral tingling with or without tongue fasciculation– Headache, nausea, vomiting and abdominal pain may all occur– Autonomic excitation causes increased sweating, salivation and lacrimation, and piloerection– The patient initially is hypertensive and tachycardic, although bradycardia and later hypotension is sometimes observed– There may be rapid progression to pulmonary oedema and consequent dyspnoea and cyanosis. Resultant hypoxia may produce irritability, decreased consciousness, or even coma |
| Diagnostic tests | <ul style="list-style-type: none">– None available |

Red back spider bite

Table 26. Red back spider bite: Clinical presentation^{4,8-10,17,18}

| | |
|--|--|
| Bite and bite site | <ul style="list-style-type: none">– The majority of patients will feel the bite, but perhaps only as a pinprick. In some cases, the bite may be painless– Local sweating at the bite site is common. Sometimes local erythema or blanching, or piloerection may occur |
| Timing of development and symptoms of systemic envenoming | <ul style="list-style-type: none">– Within minutes to an hour or more after the bite, significant local pain develops around the bite area, often with increased sweating– Over the following hours, the pain may become more severe and spread proximally, often with pain and/or swelling of the draining lymph nodes. As the pain spreads proximally it may lessen in the bite area– Sweating may become regionalised and the patient may develop nausea, become hypertensive, and suffer general malaise– In more severe cases generalised symptoms may develop, involving most or all of the body, and the pain may mimic acute abdomen or cardiac chest pain– If left untreated, this pain syndrome may last hours to days |
| Classic presentations | <ul style="list-style-type: none">– A triad of progressive severe pain, marked sweating and hypertension– A triad of local bite site pain, sweating and piloerection¹⁷– Gravitation of symptoms to the lower limbs in delayed presentations – i.e. burning sensation in the soles of the feet and pain and profuse sweating of both lower legs even if the bite was elsewhere |
| Diagnostic tests | <ul style="list-style-type: none">– None available |

Spiderbite: First aid

General principles^{9,19}

- The majority of spider bites do not require specific first aid.
- Pressure Bandaging & Immobilisation (PBI) first aid should be applied for bites by funnel web spiders and related big black spiders (see pages 38-43 in Section 2 for details of PBI application).¹⁹
- Red back spider bites do not require specific first aid other than perhaps intermittent application of ice or a cold compress. The PBI technique is contraindicated as it may worsen pain.¹⁹

PBI first aid should be applied for bites by funnel web spiders... [but the] PBI technique is contraindicated for red back spider bite as it may worsen pain

Funnel web spider bite: Timing of removal of PBI first aid^{5,8}

In patients bitten by funnel web spider, the removal of first aid may precipitate severe systemic envenoming.

Consequently, for cases of suspected funnel web spider bite or bite by any other big black spider, first aid measures **should not be removed** until the patient has been admitted to an intensive care unit or emergency department that holds bioCSL's Funnel Web Spider Antivenom and has access to resuscitation facilities and skilled clinicians.

When patients bitten by funnel web spider/other big black spider present to an appropriately resourced medical facility, a number of steps must be undertaken prior to the removal of PBI first aid (see Table 27 on page 185).

Table 27. Timing and process of PBI first aid removal for funnel web spider bite^{3,5,7-9,14}

| | |
|---|---|
| Key principle | Funnel web spider bite should be managed as a medical emergency |
| Removal of PBI first aid in funnel web spider bite: General principles | <p>Patient must be stable – if the patient is unwell, or exhibits unstable observations or is obviously envenomed, do not remove PBI first aid</p> <p>If the patient does not show signs of envenoming at presentation, PBI first aid should be removed only if Funnel Web Spider Antivenom is immediately available along with a clinician experienced in resuscitation</p> |
| Process prior to PBI first aid removal | <p>If a patient with suspected/confirmed funnel web spider bite presents to hospital without effective PBI first aid in place, immediately apply PBI first aid before proceeding further</p> <p>First aid measures for funnel web spider bite should remain undisturbed until all of the following steps have been undertaken (in most cases this will take 15 to 30+ minutes)</p> <ul style="list-style-type: none"> – The patient is admitted to hospital – An I.V. line is in place – The patient has been fully assessed including history and examination, noting symptoms present if any – at which point an assessment can be made about the need for antivenom – The doctor has assembled Funnel Web Spider Antivenom (preferably 4 vials) and the drugs that may need to be administered when the bandage and splints are removed – Adrenaline and resuscitation equipment are immediately available in case of a severe adverse reaction to antivenom. [Anaphylaxis, though unlikely due to the catecholamine storm in funnel web spider envenoming, remains a potential risk] |
| After completing the above steps | <p>If the patient has significant envenoming requiring antivenom therapy, antivenom should be commenced and symptoms should begin to subside prior to removal of first aid. After removal of PBI first aid, observe the patient carefully in case systemic envenoming recurs</p> <p>Alternatively, if the patient does not have signs of systemic envenoming at presentation and antivenom is readily available, remove PBI first aid and fully reassess 1 hour later (or sooner if symptoms/signs of envenoming develop more rapidly). A patient who remains symptom free at 4 hours post bite with first aid removed for at least 2 hours, may be discharged^{7,14}</p> |

Spiderbite: Diagnosis

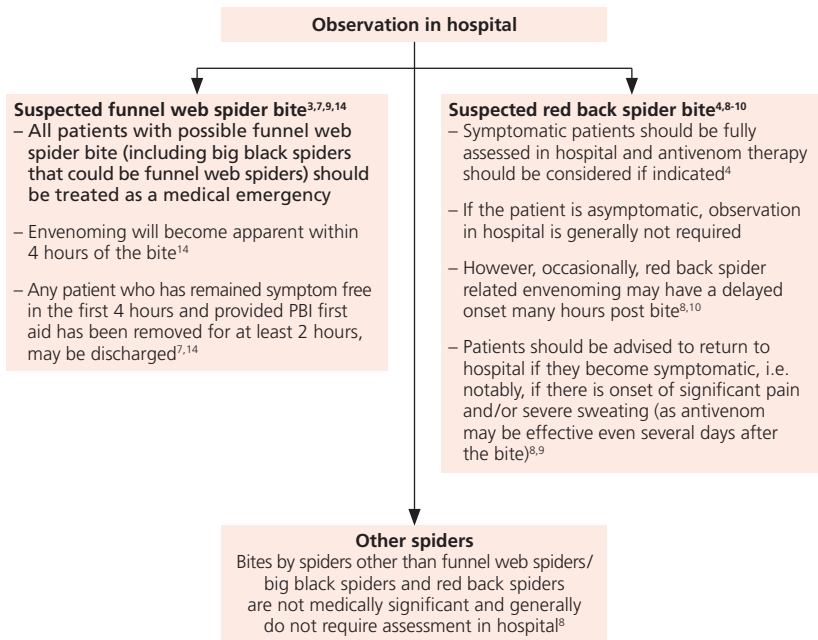
Principles of diagnosis and management^{3,8,9}

- Diagnosis of spiderbite is based on recognising the characteristic features of envenoming elicited through history and examination.
- In contrast to snakebite, laboratory tests are of no relevance for spiderbite.
- When managing spiderbite, the key question is whether there is evidence of significant envenoming, and if present, whether the pattern of envenoming reflects that of red back spider or funnel web spider, as each requires the appropriate antivenom.
- Key indications for each type of antivenom are provided in forthcoming sections covering funnel web spiders and red back spiders (pages 199 & 203).
- Non-specific local and general symptoms can arise as a consequence of any of the following.
 - Minor reactions to venom
 - Allergy
 - Infection
 - Anxiety
- Consequently, non-specific symptoms are **not** an indication for the use of antivenom.

When managing spiderbite, the key question is whether there is evidence of significant envenoming, and if present, whether the pattern reflects that of red back spider or funnel web spider

Spiderbite: Observation^{3,6}

For cases of suspected spiderbite, prolonged observation in hospital generally is not warranted.



Note: Any bite or sting can introduce secondary infection. Therefore, **all patients** should be advised of the symptoms of secondary infection and the need to seek medical attention, should these symptoms occur [8]. Research indicates that secondary infection following spiderbite is a rare occurrence [2].

Any patient [with suspected funnel web spider bite] who has remained symptom free in the first 4 hours and provided PBI first aid has been removed for at least 2 hours, may be discharged

Spiderbite: Diagnosis

History

Obtaining a thorough history is key to the diagnostic process in spiderbite (Table 28).

Table 28. Spiderbite cases: Taking a detailed history^{8,9}

| | |
|--|--|
| Circumstances of the bite | <ul style="list-style-type: none">– Time of day– Activity/what the patient was doing at the time of bite– Geographic location to limit the range of spiders to be considered– Was the spider actually seen biting the patient?– Number of bites – because multiple bites are potentially more severe |
| Details of the spider if seen | <ul style="list-style-type: none">– Size– Colouration– Distinguishing features |
| Symptoms, including time of onset | <ul style="list-style-type: none">– See Table 30 on page 190 for key symptoms/signs of envenoming based on spider species |
| Details of first aid | <ul style="list-style-type: none">– Type of first aid– Any delay in application |
| Medical history and medications | <ul style="list-style-type: none">– Past exposure to spiderbite or antivenom– Allergy history– Significant pre-existing medical conditions such as cardiac or renal disease– Medications |

Examination

Table 29. Spiderbite cases: Physical examination^{8,9}

| | |
|--|--|
| ABC | Check airway, breathing and circulation and assess the presence of any immediate life threats |
| Bite site | Look for: swelling; sweating; piloerection; erythema; multiple bites Obvious fang marks (funnel web spider bites) |
| Draining lymph nodes | Tenderness or swelling |
| General signs of envenoming | HR (tachycardia or bradycardia) BP (hypotension or hypertension) SpO ₂ – falling Respiratory rate – increased? GCS (Glasgow Coma Scale) – impaired state of consciousness |
| Specific signs of systemic envenoming | Refer to Table 30 on key early signs on page 190 |

Laboratory investigations^{8,9}

- Typically, laboratory investigations are of no assistance in diagnosing spiderbite or assessing the degree of envenoming.
- Laboratory testing would play a role only in cases where the cause of the bite is unknown and snakebite also needs consideration.



Red back spider bite.
Photo copyright A/Prof Julian White.

Spiderbite: Diagnosis

Key symptoms/signs of envenoming based on spider species

Obtaining a thorough history is key to the diagnostic process in spiderbite (Table 28).

Table 30. Key symptoms/signs for diagnostic decision making in spiderbite^{4,5,8-10,14,17}

| Key symptoms | Key signs | Species to consider* |
|---|--|---|
| Immediate significant local pain | Two distinct fang punctures separated by several millimeters | Funnel web spider group (if within geographic range)* A number of other spider species may cause immediate pain and/or distinct fang marks |
| Paraesthesiae in lips ± twitching/fasciculation of the tongue | | Funnel web spider group (if within geographic range)* |
| Respiratory distress | Increased respiratory rate, cyanosis, pulmonary oedema | Funnel web spider group (if within geographic range)* |
| | Increased salivation/lacrimation | Funnel web spider group (if within geographic range)* |
| | Marked local sweating ± piloerection | Funnel web spider group (if within geographic range)* Red back spider |
| | Hypertension | Funnel web spider group (if within geographic range)* Red back spider |
| Progressive severe pain in the bite site moving to the rest of the bitten limb/region | | Red back spider |
| Severe abdominal/chest pain | No evidence of acute abdomen or myocardial ischaemia | Red back spider |
| Miserable, inconsolable infant | No specific local signs but may have erythematous rash | Red back spider |
| Triad of progressive severe local to regional pain, marked sweating and hypertension, or a triad of local bite site pain, sweating and piloerection | | Red back spider |

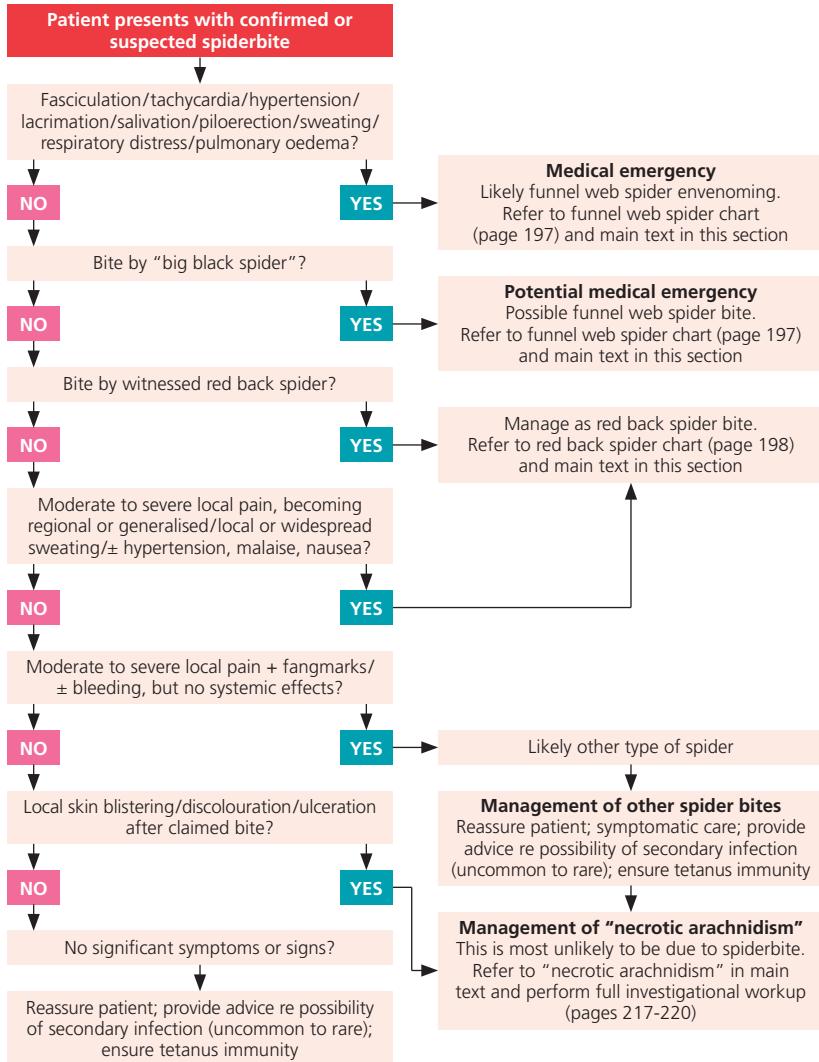
*With an increasing range of spiders being sold through pet shops, it is worthwhile bearing in mind that cases of spiderbite may at times present outside the range of distribution of the spider species.

Diagnostic algorithms⁸

- Diagnostic algorithms for bites from Australian spiders are designed to separate the two medically important groups (funnel web spider bite and red back spider bite) from all other cases, to ensure specific treatment, if indicated, can be offered at the earliest opportunity.
- Importantly, as always, the algorithms only cover common situations and cannot be expected to be accurate in every circumstance.
- Algorithms should be used only as a guide and in combination with good clinical judgment.
- A diagnostic algorithm for spiderbite based on the patient's clinical features is shown on page 192.
- In addition, Isbister and Sibbritt have developed diagnostic information based on geography and time of year.⁶
- While geography is clearly important given the limited range of distribution of the most medically significant spider species (funnel web spider) – it is important to bear in mind that with an increasing range of spiders being sold through pet shops, cases of spiderbite may at times present outside the range of distribution of the spider species.

Diagnostic algorithms for bites from Australian spiders are designed to separate the two medically important groups (funnel web spider bite and red back spider bite) from all other cases, to ensure specific treatment can be offered at the earliest opportunity if indicated

Spiderbite decision tree based on clinical presentation



Spider identification

Identifying a spider⁸

If the culprit spider is brought in with the patient, identifying the spider to at least a basic level can be valuable as it may help distinguish a potential funnel web spider bite (big black spider), *versus* some other type of spider not requiring further medical attention (the exception being the red back spider where diagnosis may be made on symptoms in the absence of a witnessed spider).

Spiders fall into two broad groups, which can be distinguished by external body characteristics (see page 194).

1. Mygalomorphs (funnel web spiders, trapdoor spiders, tarantulas, etc).^{7,9}
2. Araneomorphs (red back spiders, orb-weaving spiders, huntsman, wolf spiders, white tail spiders, black house spiders, etc).⁹

Consult with an expert when attempting to identify a spider – a photo may assist. Importantly, even with an expert's guidance, errors may occur during identification. Consequently the clinician should foremost be guided by the clinical picture. Do not rely on physical identification alone – ensure that the identity of the spider, if determined, correlates with the clinical features of the patient. If in doubt, rely on the clinical features.

Expert identification may be facilitated through the National Poisons Information Centre (Tel: 13 11 26), but will probably involve a clinical toxinology service and/or a State museum spider expert.

Finally, as always, clinical management of the patient should always take precedence over identification of the spider.

The clinician should foremost be guided by the clinical picture... ensure that the identity of the spider, if determined, correlates with the clinical features of the patient

Spider identification

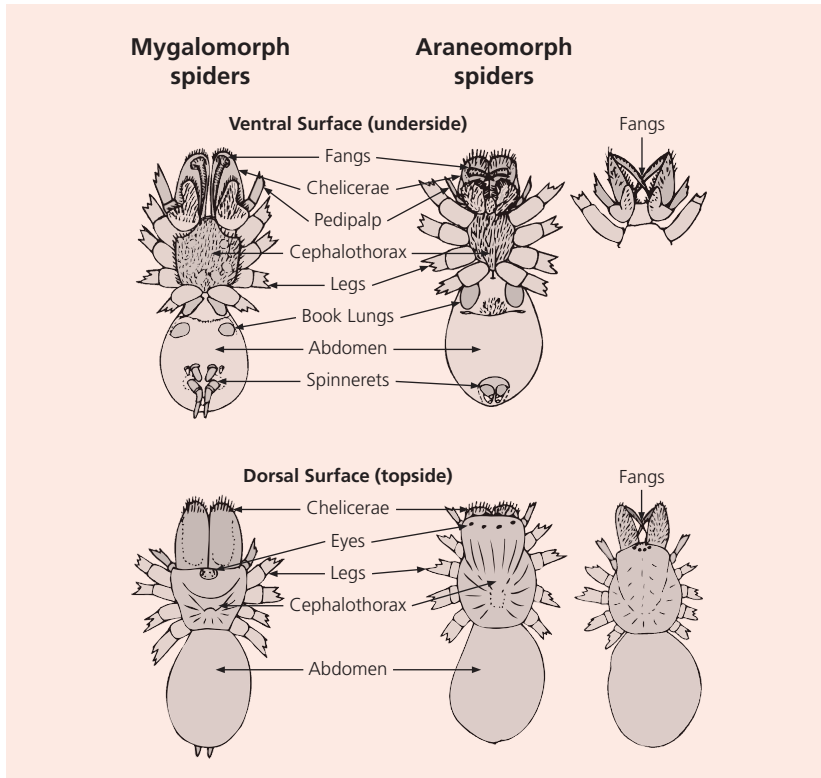
Mygalomorph fangs



Araneomorph fangs



Identifying a spider



Photos and diagram copyright A/Prof Julian White.

Management of envenoming due to spiderbite

Basic principles^{4,8,13,14}

- At the outset, it is crucial to identify cases that could potentially be severe (i.e. bites from the funnel web spider group) so that these patients are placed in a position where urgent specific treatment can be administered if indicated.
- Antivenom is the cornerstone of therapy for cases of systemic envenoming due to funnel web spider bite. Since the availability of antivenom, there have been no deaths from funnel web spider envenoming.
- Cases of suspected or confirmed red back spider bite also should be identified so that appropriate treatment can be offered if and when indicated. Antivenom therapy will be indicated for some cases.
- All other spiderbites require reassurance and appropriate advice to the patient regarding the potential (if rare) of problems such as secondary infection.
- Patients presenting with a putative diagnosis of “necrotic arachnidism” require complete work up for causes of skin damage, leaving “necrotic arachnidism” as an uncertain diagnosis of last resort only (see page 220).

At the outset, it is crucial to identify cases that could potentially be severe (i.e. bites from the funnel web spider group)

Management of envenoming due to spiderbite

Urgent treatment^{5,7,8,14}

In general, urgent treatment will only apply to definite and suspected funnel web spider bite.

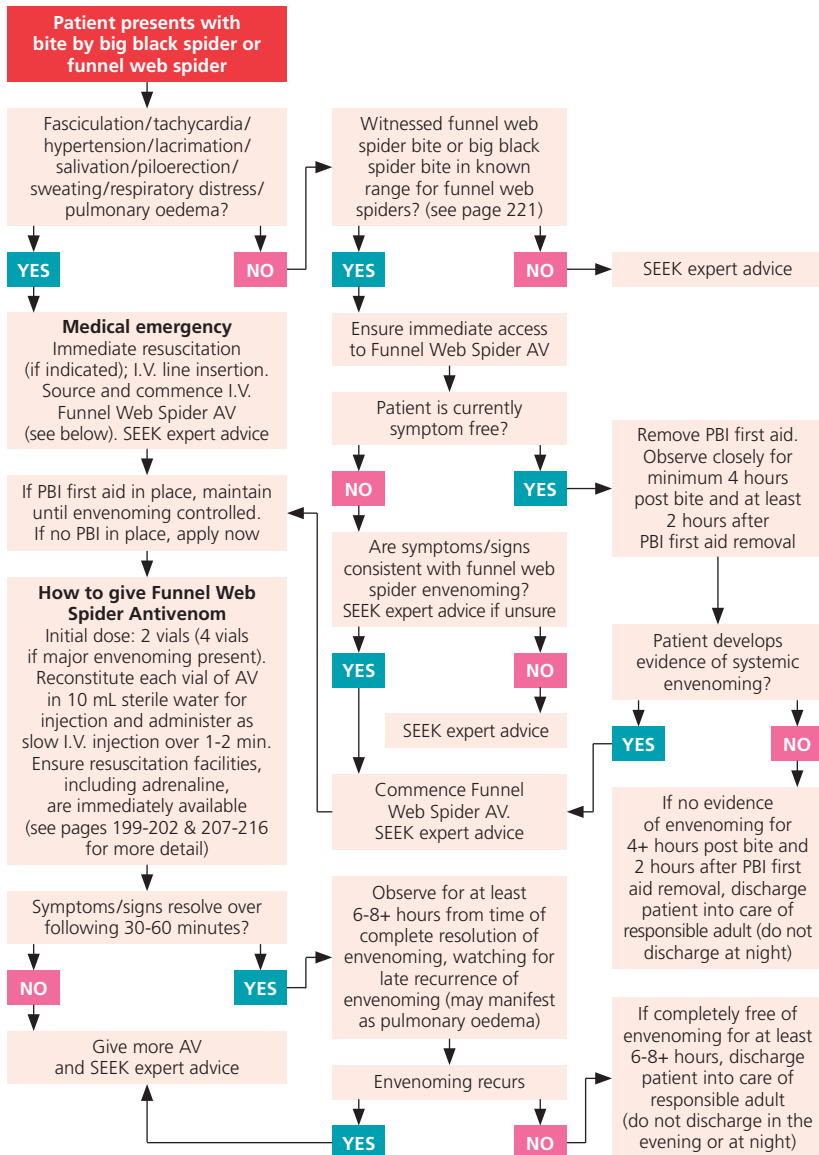
1. ABC: Ensure adequate respiratory and cardiac function.
Assess and maintain airway.
Provide respiratory support as indicated.
2. Assess if effective PBI first aid is in place. If not, apply PBI.
3. Insert I.V. line and provide fluid load.
Choice of crystalloid is not critical (e.g. normal saline, Hartmann's solution, etc).
4. Perform key history and examine patient as part of the diagnostic process (see pages 188-189).
5. Urgently assess history and examination findings to determine if systemic envenoming is present (see page 182 for symptoms and signs of envenoming).
If systemic envenoming has occurred, antivenom therapy with bioCSL's Funnel Web Spider Antivenom is indicated before removal of first aid.

If there is no evidence of significant envenoming and antivenom is readily available, remove first aid and re-evaluate over the next few hours to at least 4 hours from time of bite and at least 2 hours after removal of first aid.

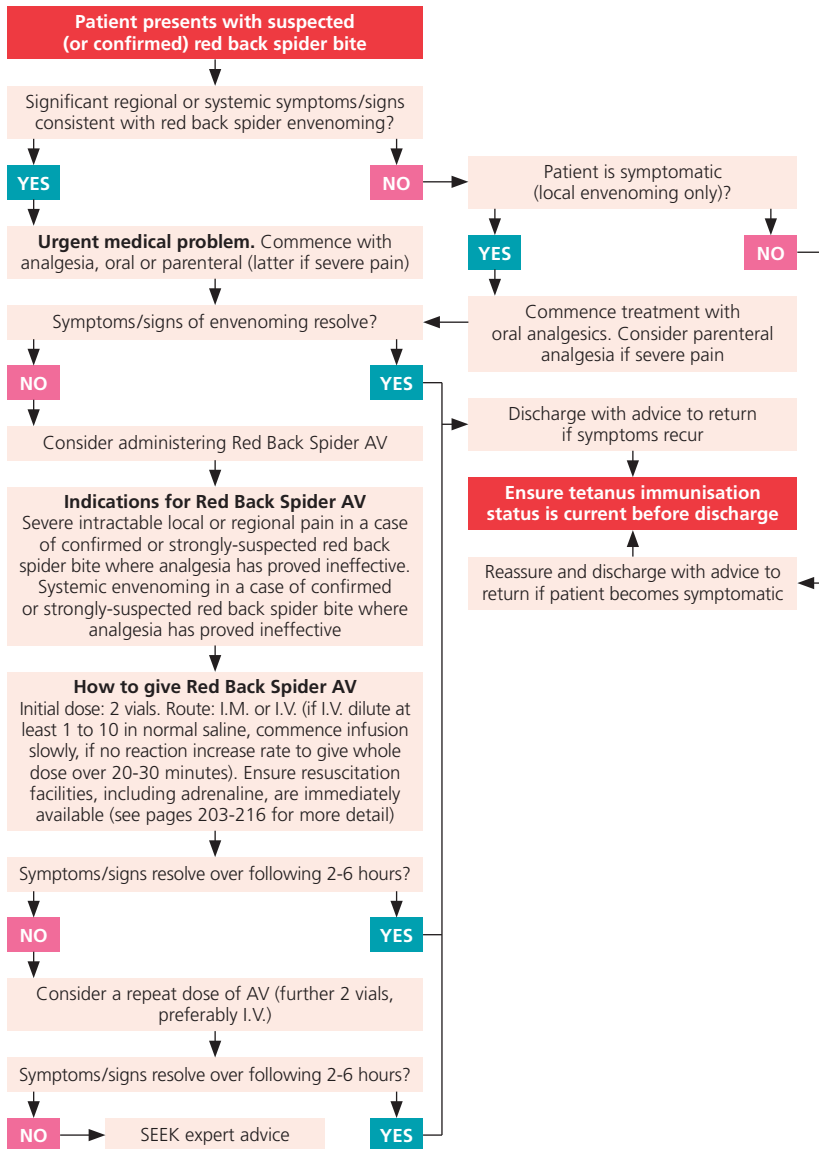
Always seek expert advice when managing a patient who may have been bitten by a funnel web spider or requires antivenom therapy.

Urgently assess findings. If systemic envenoming is present, antivenom therapy with bioCSL's Funnel Web Spider Antivenom is indicated before removal of first aid

Management of suspected or confirmed funnel web spider bite



Management of suspected or confirmed red back spider bite



Specific treatment for funnel web spider bite: Antivenom

Indications for antivenom therapy

For funnel web spider bite, specific treatment involves administration of antivenom provided this is clinically indicated – i.e. if there is clear evidence of systemic envenoming (see box).²⁰ If systemic envenoming has occurred, seek expert advice.

Indications for funnel web spider antivenom therapy⁵

- Any degree of systemic envenoming consistent with funnel web spider bite, even just early symptoms (oral paraesthesia; tongue fasciculation) in a case of suspected or confirmed funnel web spider or big black spider bite – **except** where more than 4 hours have elapsed post bite, first aid has been removed for at least 2 hours, and the symptoms have not progressed beyond mild envenoming for at least 2 hours.^{7,14}
- Any patient who has been bitten by a spider that is possibly a funnel web spider, and who develops excessive salivation or lacrimation, or twitching of the tongue, piloerection, significant tachycardia, respiratory distress, hypertension if previously normotensive (patient may become hypotensive late in the syndrome), or disorientation, confusion or depressed level of consciousness, should be assumed to have systemic envenoming and should receive antivenom.

Timing of antivenom therapy

Funnel web spider envenoming can progress quickly from the first signs of envenoming to life-threatening envenoming, which could potentially occur in 30 minutes or less, especially in children.⁵

Clinical experience indicates that antivenom can reverse even advanced envenoming,¹⁴ so it is almost never too late to try antivenom, but clearly the sooner it is given once systemic envenoming develops, the greater the likelihood of a good outcome.⁸

Since the majority of bites by funnel web spiders do not result in significant envenoming and therefore do not require antivenom,^{7,9} there is no justification for routine antivenom administration in every case of funnel web spider bite. Antivenom should be reserved only for those cases that develop systemic envenoming.²⁰

Specific treatment for funnel web spider bite: Antivenom

Initial dose of bioCSL's Funnel Web Spider Antivenom

- Initial dose is 2 vials.²⁰
- Use 4 vials if envenoming is already severe.^{5,21-23}

The Product Information for bioCSL's Funnel Web Spider Antivenom does not specifically list this recommendation.²⁰

The advice regarding a higher initial dose of 4 vials for severe cases is based on published expert clinical experience and spiderbite management guidelines.^{5,21-23}
- Be prepared to give more antivenom if required, especially for severe cases.^{20,22,23}

The requirement for and timing of follow-up antivenom are determined on a case-by-case basis.²⁴

Typically, repeat dosing is performed within 15 minutes of the initial dose.^{20,24}

Follow-up dosing: bioCSL's Funnel Web Spider Antivenom

- Further antivenom may be required if there is continued or progressive systemic envenoming.^{5,20}
- If there has been an initial response, followed by later return of respiratory distress without other features of envenoming (such as salivation and lacrimation), it is quite possible this is due to further venom entering the circulation, and more antivenom may be needed.⁸
- Another possible explanation in this situation, at least in children, is that they are developing pulmonary oedema due to overload of I.V. fluids.^{8,22,23}

Unless overload of I.V. fluids is clearly the explanation, err on the side of further venom effect and administer more antivenom.⁸

If the respiratory impairment is mild, it might be reasonable to try a diuretic, such as frusemide as first-line therapy, but if this does not work, or respiratory distress worsens or is severe, use antivenom.⁸

Route of administration of bioCSL's Funnel Web Spider Antivenom

bioCSL's Funnel Web Spider Antivenom should be administered I.V. (as slow I.V. injection – see page 208).²⁰

Monitoring patients after antivenom therapy

- While clinical experience indicates that funnel web spider antivenom is efficacious, with a low risk of relapse provided an adequate dose of antivenom has been administered,²⁴ relapse has been known to occur more than 6 hours after antivenom therapy.^{22,23}
- Additionally, expert opinion suggests that the nature of funnel web spider envenoming including its rapidity of development and multi-system effects, necessitates prolonged observation prior to discharge. Therefore, patients with systemic envenoming should be admitted overnight post antivenom therapy.²⁴
- A systematic review of cases of funnel web spider bite presenting to hospitals in Australia indicates that for cases of severe envenoming who received antivenom therapy, the median stay in hospital was 1.8 days.¹⁴

Expert opinion suggests that the nature of funnel web spider envenoming necessitates prolonged observation prior to discharge patients should be admitted overnight post antivenom therapy

Important note

Prior to administering bioCSL's Funnel Web Spider Antivenom seek expert advice and please see additional information on pages 207-216 regarding preparatory procedures prior to antivenom therapy, administering antivenom, patient observation, and potential complications.

Specific treatment for funnel web spider bite: Antivenom

What if bioCSL's Funnel Web Spider Antivenom is not immediately available?

Antivenom is the key treatment for envenoming from funnel web spider bite. The availability of antivenom has dramatically altered outcomes and management *versus* the pre-antivenom era.¹⁴

In major funnel web spider envenoming, time is of the essence for administering antivenom therapy.^{3,5,8,17}

- If a patient bitten by a funnel web spider is asymptomatic, apply PBI first aid and transfer the patient to a hospital that stocks bioCSL's Funnel Web Spider Antivenom and has access to skilled clinicians.
- If a patient who is bitten exhibits symptoms of envenoming it may be preferable to move Funnel Web Spider Antivenom (and if possible, the skilled clinician) to the patient.

The following alternatives to antivenom treatment are based on past experience, prior to the availability of bioCSL's Funnel Web Spider Antivenom. Please bear in mind that during the pre-antivenom era, despite best efforts, survival was far from guaranteed. Seek expert assistance.⁸

- Severe intractable pulmonary oedema may be managed in ICU with intubation and mechanical ventilation (IPPV; PEEP), oxygen and possibly diuretics.
- Early hypertension and tachycardia, if severe enough, could be managed with sympathetic blockade (alpha blockade, not beta blockade).
- Later developing hypotension may respond to volume replacement and pressor agents such as isoprenaline. Atropine has been used with some success.
- There is limited evidence that PBI may allow local destruction of venom if left in place for a prolonged period.²⁵

In major funnel web spider envenoming, time is of the essence for administering antivenom therapy

Specific treatment for red back spider bite: Antivenom

Envenoming from red back spider bite

In Australia, red back spider bite is by far the most common cause of significant envenoming resulting in the administration of antivenom therapy.²⁶

Indications for bioCSL's Red Back Spider Antivenom

- Despite the frequency of red back spider envenoming and use of antivenom in Australia, recent controversial research has confounded the perception of the effectiveness of this antivenom,^{4,27,28} and therefore, the indications for the antivenom.
- Until the situation becomes clearer as the result of further research, the following indications (see box) should be considered broadly advisory only, and subject to clinical judgement on a case-by-case basis.⁸

Indications for bioCSL's Red Back Spider Antivenom

- Severe intractable local or regional pain in a case of confirmed or strongly suspected red back spider bite where analgesia has proved ineffective.^{4,8}
- Systemic envenoming in a confirmed or strongly suspected red back spider bite where analgesia has proved ineffective.^{8,29}

Timing of bioCSL's Red Back Spider Antivenom therapy⁸

- Clinical experience indicates that even parenteral analgesia with strong analgesics such as morphine may prove ineffective for the severe pain of red back spider envenoming.
- However, sufficient time should be allowed for analgesic therapy to show effectiveness before concluding it has failed and moving to antivenom therapy.

Specific treatment for red back spider bite: Antivenom

Dose of bioCSL's Red Back Spider Antivenom

Table 31. Dosing: bioCSL's Red Back Spider Antivenom

| | |
|-------------------------|--|
| Initial dose | <p>2 vials^{22,23,30,31}</p> <p>One vial will neutralise 5 mg of venom <i>in vitro</i>. The actual amount needed in clinical practice may be more.²⁹</p> <p>Current treatment guidelines recommend 2 vials as a starting dose^{22,23,30,31}</p> <p>Please see additional information regarding dosing in the section immediately below this table</p> |
| Follow-up dosing | <p>Each follow-up dose is 2 vials^{22,23,30,31}</p> <p>Only severe cases are likely to require follow-up doses⁸</p> <p>A significant period (at least 2 hours) should be allowed between doses to evaluate effectiveness of the preceding dose^{22,23,29}</p> <p>Do not exceed a total of 4 vials (including the initial dose) without seeking expert advice^{22,23}</p> <p>Please see additional information in the section immediately below this table</p> |

Note the doses in the above table differ from the Product Information for bioCSL's Red back Spider Antivenom.

- The Product Information for bioCSL's Red Back Spider Antivenom recommends:²⁹
An initial dose of 1 vial.

A follow-up dose of 1 vial (to be administered if envenoming has not reversed in 2 hours after the initial dose).

- The Product Information also states that further antivenom may be needed for some cases, but it is unusual to require more than 3 vials.²⁹
- The dosing advice in the above table is based on current (published) treatment guidelines, which recommend a greater number of vials for initial and follow-up dosing than that recommended in the Product Information.^{22,23,30,31}

Route of administration of bioCSL's Red Back Spider Antivenom

- Traditionally, bioCSL's Red back Spider Antivenom has been given I.M. However, in recent years, there has been a trend towards I.V. use.
- While there is a body of evidence substantiating the efficacy of the I.M. route,^{8,10} recent research contradicts this experience by indicating that I.M. antivenom is slow to achieve measurable circulating levels.²⁷
- Theoretically, I.V. is now the preferred route, as high blood levels will be achieved more rapidly and I.V. antivenom appears to have a low adverse reaction rate.^{26,27}
- Randomised controlled trials indicate comparable efficacy of I.M and I.V routes at 2 hours post antivenom but that more patients experience significantly improved pain at 24 hours post I.V. antivenom.^{28,32}
- Therefore, the expert panel involved in producing this handbook recommends administering bioCSL's Red Back Spider Antivenom I.V., preferably diluted in normal saline, Hartmann's solution* or similar, starting the infusion slowly and increasing the rate if no adverse reaction is observed.⁸
 - One method of I.V. administration involves using a 100 mL burette or similar, diluting the antivenom to 100 mL (a lower volume may be required in infants and small children), and administering via a pump.⁸
- However, given the body of past work, it remains acceptable to use the I.M. route (as recommended in the Product Information).^{8,29}

*The Product Information for bioCSL's Red Back Spider Antivenom recommends dilution using Hartmann's solution. The use of other isotonic crystalloid such as normal saline is based on expert clinical experience and is accepted current clinical practice [8,29,30].

Important note

Prior to administering bioCSL's Red back Spider Antivenom seek expert advice and please see additional information on pages 207-216 regarding preparatory procedures prior to antivenom therapy, administering antivenom, patient observation, and potential complications.

Specific treatment for red back spider bite: Antivenom

What if bioCSL's Red Back Spider Antivenom is not immediately available?

- While envenoming from red back spider bite can be unpleasant/distressing for the patient, it is not likely to be fatal.³
- The role of antivenom is to ameliorate symptoms and reduce the period of incapacity.
- In the absence of antivenom, there are no pharmacologic alternatives of proven efficacy. This explains the popularity of antivenom as a treatment.⁸
 - Some patients will respond to analgesia, most likely parenteral analgesia.
 - Pharmacologic agents such as diazepam have been used outside Australia for widow spider bite with mixed response.
 - Intravenous calcium infusions enjoyed limited popularity for widow spider bite in North America but their effectiveness has not survived clinical trials, and this treatment is not advised.
 - Patients should be reassured that symptoms will eventually resolve although sometimes it may take many days or longer to do so.

In the absence of antivenom, there are no pharmacologic alternatives of proven efficacy. This explains the popularity of antivenom as a treatment

Spider antivenoms: Preparation, administration and complications

Preparation prior to commencing antivenom therapy^{23,33,34}

Prior to commencing antivenom therapy, ensure all facilities are ready at hand to treat anaphylaxis, in the event that this should occur.

- Dedicate one small-bore I.V. line (18-20 G in adults) to antivenom administration.
- Dedicate one large bore I.V. line (16-14 G in adults) for emergency resuscitation.
- Prepare 1L normal saline (20 mL/kg in children) ready to administer under pressure.
- Prepare adrenaline (1:1000 – i.e. 1 mg adrenaline in 1 mL) drawn up to a dose of 0.01 mg/kg (maximum 0.5 mg – i.e. 0.5 mL) and label as 'Adrenaline for **I.M.** injection only (dose in mg)'.
- Ideally, also prepare an I.V. infusion of adrenaline 1 mg in 100 mL, which is controlled by infusion pump or syringe driver and ready to attach by a side arm to the resuscitation line. Anti-reflux valves must be attached above the side arm on any other infusions using this I.V. line, to prevent adrenaline going back up into other fluid bags. To prevent erroneous administration, **do not attach the adrenaline infusion unless it is needed.**
- Record blood pressure on the opposite arm to the fluid/adrenaline infusion – to avoid prolonged cuff inflations and thus, extravasation of infusion fluids.
- See 'What to do if there is an adverse reaction to antivenom' on pages 213-214 for method of emergency resuscitation if required.



bioCSL's Funnel Web Spider Antivenom.



bioCSL's Red Back Spider Antivenom.

Spider antivenoms: Preparation, administration and complications

How to administer spider antivenoms

Table 32. Administering bioCSL's Funnel Web Spider Antivenom^{5,8,20,24,30}

| | |
|-----------------------------------|--|
| Treatment location | If possible, patients should receive Funnel Web Spider Antivenom in a monitored environment with immediate access to resuscitative equipment and one-to-one nursing |
| What to do about first aid | <p>If adequate PBI first aid was applied, the splint and pressure bandage should not be removed until after antivenom has been administered, as removal can precipitate significant effects of systemic envenoming</p> <p>See pages 184-185 on the timing of removal of PBI first aid for funnel web spider bite</p> |
| Route of administration | <p>Intravenous</p> <p>Always administer Funnel Web Spider Antivenom as a slow I.V. injection</p> |
| Dilution of antivenom | bioCSL's Funnel Web Spider Antivenom is formulated as a freeze-dried preparation. Reconstitute each vial to up to 10 mL with sterile water for injection |
| Time period of I.V. dosing | Administer each vial of reconstituted antivenom by slow I.V. injection (over about 1 to 2 minutes) |

Table 33. Administering bioCSL's Red Back Spider Antivenom^{8,17,19,29,30,35}

| | |
|-----------------------------------|---|
| Treatment location | If possible, patients should receive Red Back Spider Antivenom in a monitored environment with immediate access to resuscitative equipment and one-to-one nursing |
| What to do about first aid | First aid is not required for red back spider bite other than perhaps intermittent application of ice or a cold compress Note: PBI first aid is contraindicated for red back spider bite as it may worsen pain ¹⁹ |
| Route of administration | Intravenous through drip set Alternatively, the intramuscular route also is an option (as per the Product Information) |
| Dilution of antivenom | For I.V. administration: <ul style="list-style-type: none"> – Dilute Red Back Spider Antivenom up to 1 in 10 or more with an isotonic crystalloid solution (e.g. normal saline; Hartmann's solution)* – dilution to 100 mL is often used in adults – To avoid fluid overload, use smaller volumes in small children and in adults with compromised cardiac function <li style="padding-left: 20px;">Adults with compromised cardiac function: Up to a 1 in 5 dilution may be more appropriate <li style="padding-left: 20px;">Small children: Dilute antivenom to the extent that the total volume delivered does not exceed 10 mL/kg For I.M. administration inject each vial undiluted |
| Time period of I.V. dosing | For I.V. administration, each dose of antivenom should be run over about 30 minutes (a single dose may comprise multiple vials). Start very slowly and increase the rate gradually to deliver antivenom over approximately 30 minutes |

*The Product Information for Red Back Spider Antivenom recommends dilution using Hartmann's solution. The use of other isotonic crystalloid such as normal saline is based on expert clinical experience and is accepted current clinical practice [8,29,30].

Spider antivenoms: Preparation, administration and complications

Observation during antivenom therapy^{8,33,36}

- Carefully observe the patient during antivenom administration and for 1 hour after, to ensure adverse reactions (if they occur) are recognised and treated promptly (adverse reactions are discussed further on pages 213-216).
- In particular, look for the development of symptoms and signs of anaphylaxis. An erythematous rash may be the first sign of developing adverse reactions (often first seen in the axilla or the lower abdomen).
- Also observe for hypotension and bronchospasm.
- Carefully monitor BP, HR and respiratory function, oxygen saturation, with particular attention to development of hypotension and/or bronchospasm.
- Look for additional warning signs of anaphylaxis in children (Table 34).
- See pages 215-216 for further information on potential complications of antivenom therapy.

Table 34. Warning signs of anaphylaxis in children^{8,36}

| | |
|------------------------------------|---|
| Rash; hypotension; or bronchospasm | Profuse sweating |
| Nasal, palatal, or ocular pruritus | Faecal or urinary urgency or incontinence |
| Coughing | Abdominal pain |
| Sneezing | A sense of impending doom |



Antivenom rash in an adult.

Premedication prior to administering antivenom

Premedication prior to administering antivenom remains an issue surrounded by controversy, some conflicting evidence and opinion, and uncertainty about “best practice”.

Studies outside Australia have provided evidence showing premedication using antihistamines and/or hydrocortisone are either ineffective, or possibly effective to a variable extent.^{37,38} Hence, available evidence is unhelpful and contradictory for these medications. Similar contradictory evidence applies to adrenaline premedication within and outside Australia.³⁹⁻⁴³

Data from recent Australian clinical research reported in 2008 (the ASP study – a multicentre, prospective study of snakebite envenoming from over 60 major tertiary centres and regional hospitals around Australia) suggests that there is no clear benefit in giving premedication prior to antivenom administration, including no clear benefit in using adrenaline as premedication.⁴⁴

Further, the ASP study has shown that in Australia, the use of premedication prior to antivenom therapy is not common practice (adrenaline premedication was provided to 8.7% of patients in whom data about premedication was available).⁴⁴

Additionally, recent expert consensus suggests that premedication to prevent adverse reactions to antivenom therapy is not routinely indicated.³⁰

.... continued overleaf

Spider antivenoms: Preparation, administration and complications

Premedication prior to administering antivenom ... cont'd

Clearly, there may be specific clinical circumstances, judged on an individual patient basis, where a clinician may consider the use of adrenaline as premedication prior to antivenom administration. For example – in a patient with known major allergy to antivenom where resuscitation facilities may be suboptimal.⁸

In such situations the uncertain and unproven benefits of adrenaline premedication should be carefully weighed against the known and documented adverse effects from use of adrenaline, and wherever practical, the patient should be told of this risk-benefit situation so that informed consent can be given.⁸

Clearly, in some situations, with severe life-threatening systemic envenoming, informed consent is impractical and should not form a necessary condition of using adrenaline premedication if the clinician deems this is required.⁸

Irrespective of whether premedication is used or not, antivenom should only be administered in an environment where rapid detection and appropriate treatment of severe early adverse reactions will occur.⁸

- This includes the immediate availability of adrenaline, oxygen and resuscitation equipment and staff competent and prepared to use these treatments effectively.
- In such a setting, a controlled I.V. adrenaline dilute infusion may be the optimal route for administering adrenaline to treat anaphylactic or anaphylactoid reactions (see pages 213-214 for further details).⁸

Adrenaline premedication is considered redundant when treating significant envenoming from funnel web spider bite, due to the release of catecholamines during the process of envenoming.^{8,45}

What to do if there is an adverse reaction to antivenom^{23,33,36,44}

Adverse reactions may be related to the rate of antivenom infusion – those reactions can include flushing, hypotension or bronchospasm. Hypotension and bronchospasm are hallmarks of major adverse reaction (anaphylaxis).

Adverse reactions may respond to temporarily stopping the antivenom infusion, waiting to ensure that there is no return or worsening of the reaction, and then re-starting at a slower rate.

For anaphylactic reactions, adrenaline is generally the drug of first choice.

See the box below and on page 214.

Steps to take if there is either a sudden fall in blood pressure, or bronchospasm after starting antivenom infusion^{8,23,30,33,34,36}

- Suspend the antivenom infusion.
- Lie the patient flat (if not already in this position) and commence high-flow 100% oxygen and support airway/ventilation as required.
- Begin rapid infusion of one litre normal saline (20 mL/kg in children) over 2-3 minutes.
- Administer adrenaline 1:1000 I.M. into the lateral thigh at a dose of 0.01 mg/kg to a maximum of 0.5 mg (i.e. a maximum of 0.5 mL). Note: Adrenaline 1:1000 ampoule is 1 mg adrenaline in 1mL.
- Alternatively, those experienced with I.V. administration of adrenaline can proceed to do this directly instead of I.M injection. See procedure on page 214 in the section 'If adverse reactions do not respond to initial management'.
- Seek expert advice regarding ongoing management.
- In most cases, once the adverse reaction is controlled, cautious reintroduction of antivenom is possible. [Note: A patient requiring antivenom therapy prior to the adverse reaction will likely continue to require antivenom after the adverse reaction].

Note: The recommendations above and on page 214 for the management of anaphylactic reactions to antivenom reflect current published anaphylaxis management guidelines and expert advice and may vary from the Product Information for bioCSL's antivenoms [8,33,34,36].

.... continued overleaf

Spider antivenoms: Preparation, administration and complications

What to do if there is an adverse reaction to antivenom ... cont'd

If adverse reactions do not respond to initial management^{8,23,30,33,34,36}

- If hypotensive, repeat normal saline bolus as per box on page 213 (up to 50 mL/kg may be required).
- Commence I.V. infusion of adrenaline (0.5-1 mL/kg/hr of adrenaline 1 mg in 100 mL) and titrate according to response. Monitor BP every 3-5 minutes using the arm opposite to the infusion.
- Be aware that as the adverse reaction to antivenom resolves, adrenaline requirements will fall, the blood pressure will rise and the adrenaline infusion rate will need to be reduced.
- Consider nebulised salbutamol for bronchospasm, nebulised adrenaline for upper airway obstruction, and I.V. atropine for severe bradycardia.
- Seek advice urgently from local/regional ED Consultant and/or ICU Consultant.
- In most cases, once the adverse reaction is controlled, cautious reintroduction of antivenom is possible. [Note: A patient requiring antivenom therapy prior to the adverse reaction will likely continue to require antivenom after the adverse reaction].

Antivenom therapy: Commonest mistakes⁸

Some of the most common mistakes relating to antivenom therapy are highlighted below.

- Failure to use antivenom when clearly indicated.
- Giving antivenom unnecessarily, i.e. when there are no clear clinical indicators of significant systemic envenoming.
- Choosing the wrong antivenom.
- Choosing the wrong dose.
- Antivenom given too late. [However, for envenoming from red back spider bite, delayed antivenom therapy (by a day or more) may still be effective].⁹
- Administering antivenom by the wrong route.
- Failing to prepare for an adverse reaction.
- Failing to inform the patient about serum sickness.

Complications of antivenom therapy

Essentially, antivenom is whole or modified antibody from an animal. It is obtained by hyperimmunising the animal against a particular venom or group of venoms. The IgG antibody from blood plasma is used, and typically, is fractionated to the F(ab')₂ fragment of IgG, and on occasion, whole IgG. bioCSL's Funnel Web Spider Antivenom is a purified whole IgG preparation fractionated from rabbit plasma. bioCSL's Red Back Spider Antivenom is derived from horse plasma, with the IgG antibody fractionated to the F(ab')₂ fragment.^{20,29,46}

When making antivenoms, bioCSL undertakes assiduous efforts to filter and discard any extraneous blood components and contaminants. Nevertheless even high-quality antivenoms will cause adverse reactions in some patients. The clinically important adverse reactions can be subdivided into 'early' and 'late'.

Early and late adverse reactions to antivenom

Early reactions are those that occur immediately after commencing antivenom therapy or within the first few hours (Table 35). Late adverse reactions may occur several days later (Table 36 on page 216).

Table 35. Early adverse reactions to antivenom^{8,20,29,30,33,36}

| | |
|---|---|
| Rash | Localised or generalised erythematous, sometimes pruritic rash. May occur as an isolated and generally trivial adverse reaction or it may herald the onset of a more severe adverse reaction |
| Pyrexia | Febrile reactions may potentially occur |
| Anaphylactic/ anaphylactoid reaction | <p>This is a type of potentially life-threatening reaction. Therefore, antivenom should never be given until measures to manage such a reaction are in place. This is why the use of antivenom outside a hospital environment is strongly discouraged. Note however, when antivenom is clinically indicated, it should never be withheld for fear of an adverse reaction. Seek expert advice</p> <p>Anaphylaxis may be preceded by a localised or generalised rash, sometimes first seen in the axilla or lower abdomen, proceeding to hypotension and/or bronchospasm</p> <p>Look for additional warning signs of anaphylaxis in children (see Table 34 on page 210)</p> |

Spider antivenoms: Preparation, administration and complications

Table 36. Late adverse reactions to antivenom^{8,20,29,30}

The principal late adverse reaction to antivenom is serum sickness, a type III delayed hypersensitivity reaction, which most commonly presents 4-14 days post exposure

Serum sickness is characterised by a flu-like illness with fever, joint and muscle pain and general malaise, often preceded by or associated with a maculopapular or erythema multiformae-like rash

Serum sickness rates often increase as the antigen load increases. Therefore, it is more likely to occur in patients who have had a high volume load of antivenom

Every patient who receives antivenom should be advised of the symptoms of serum sickness and told to seek medical care if these symptoms arise after discharge from hospital

Serum sickness is more likely to occur in patients who have had a high volume load of antivenom

Management of serum sickness^{8,30,47}

- While serum sickness can be a mild and self-limited disease, it can be distressing for patients and early diagnosis and treatment is advisable.
- There is a diversity of opinion about the approach to treatment for various causes of serum sickness (i.e. not just antivenom) – which may involve first-line use of antihistamines or oral steroids. Serum sickness post antivenom therapy is usually managed with a short course of oral corticosteroids.
- If uncertain about the treatment approach, consult with a clinical immunologist.
- Some experts suggest prescribing a week-long course of oral prednisolone commencing immediately after antivenom, for all patients who have received more than 25 mL of antivenom. This treatment has not yet been tested through clinical trials.

Necrotic arachnidism

Necrotic arachnidism: Evidence *versus* mythology

The problem of necrotic lesions or ulcers and their purported association with bites by the white tail spider is clouded by much speculation and mythology. Although necrotic lesions attributed to white tail spider bites have been reported in the literature, the reports lack evidence of an identified white tail spider biting the patient.¹²

In contrast, in recent years, prospective studies of confirmed spiderbite provide strong evidence that in Australia, spiderbite in general and the white tail spider specifically, do not cause skin injury/necrosis.^{2,12}

- In one study of 750 cases of definite spiderbite, not a single case of necrotic ulcers was seen.²
- In another study of 130 patients with confirmed white tail spider bite, three characteristic patterns of clinical effects were associated with the bites (pain only; pain and red mark for less than 24 hours; a persistent painful or irritating red lesion). However, no necrotic ulcers developed as a consequence of white tail spider bite.¹²

Furthermore, in a study of eleven patients with skin lesions or necrotic ulcers referred to the Hunter Area Toxicology Service, a diagnosis other than spiderbite was made in all 9 cases where appropriate investigations and follow up was possible (see box on page 218). Only one patient recalled witnessing a spider biting (although all cases had been initially referred as white tail spider bites or necrotic arachnidism).¹³

.... continued overleaf

In a study of 130 patients with confirmed white tail spider bite... no necrotic ulcers developed as a consequence of white tail spider bite

Necrotic arachnidism

Necrotic arachnidism: Evidence *versus* mythology ... cont'd

Non-spiderbite causes of necrotic skin lesions or ulcers¹³

In 9 patients with skin lesions/ulcers thought to be 'white tail spider bite or necrotic arachnidism' appropriate investigations and follow up led to the following diagnoses:

- Dermatophytoses [2 cases]
- Staphylococcal infections [3 cases]
- Pyoderma gangrenosum [1 case]
- Cutaneous polyarteritis nodosa [1 case]
- *Nocardia braziliensis* [1 case]
- Infected diabetic ulcer [1 case]

It is apparent that in Australia, necrotic ulcers are unlikely to be caused by white tail or other spiders. Hence, all patients presenting with ulcers or necrotic lesions should be properly investigated (bacterial, fungal and mycobacterial cultures and skin biopsy for histopathology) – see chart on page 220.^{2,12,13}

Other types of spiders, particularly those outside Australia, can cause primary skin necrosis. Foremost among these is the recluse spider group (loxoscelism), including the fiddlbeback spider, *Loxosceles rufescens*. *Loxosceles rufescens* is now clearly implicated in two South Australian cases of necrotic arachnidism.^{2,3} However, these appear to be isolated cases, with no subsequent cases noted in recent years. Therefore, it is unreasonable to invoke loxoscelism as a likely cause of necrotic skin lesions following suspected bites in Australia.⁸

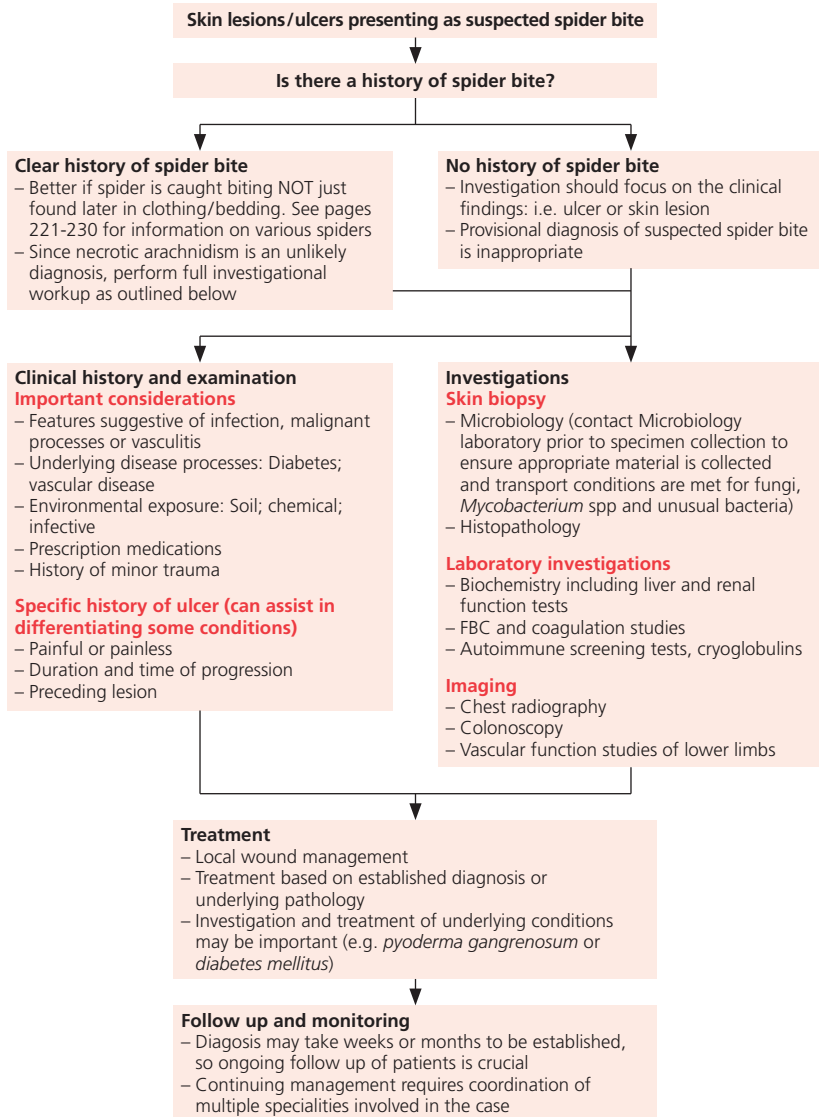
All patients presenting with ulcers or necrotic lesions should be properly investigated (bacterial, fungal and mycobacterial cultures and skin biopsy for histopathology)

Consider necrotic arachnidism only if there is initial pain, then blistering and dusky colouration of the skin, developing over 2-7 days, with later darkening of the skin, preferably with a witnessed spiderbite – **but only after all other causes have been excluded**.²³ At this time, necrotic arachnidism is an unsubstantiated diagnosis in the Australian clinical setting.^{2,12,13}

Although necrotic arachnidism has been a problem in the US for some time, treatment approaches remain controversial. Early surgical intervention, steroids or antibiotics appear to confer little benefit. Nevertheless, if secondary infection develops, this will clearly require treatment. There is anecdotal evidence supporting the use of hyperbaric oxygen therapy. However, firm evidence is lacking and use is limited by the impracticality of treating every case in this manner. In Brazil, where loxoscelism is considered 'common', antivenom is routinely used, but the evidence for effectiveness is unclear.^{8,9,23}

At this time, necrotic arachnidism is an unsubstantiated diagnosis in the Australian clinical setting

Necrotic skin lesions: Approach to investigations



Australian spiders overview: Funnel web spider group

The species^{7,9}

- There are at least 35 species of funnel web spiders in 2 genera, *Atrax* and *Hadronyche*.
- All are medium to large, robust spiders, mostly dark or black in colour, with stout legs and large fangs. Both males and females build silk tube retreats, usually in the ground.
- Males leave these retreats in search of female mates, a process which may increase the chance of adverse interaction with people, as the wandering males may get underfoot, into shoes, boots or clothing left on or near the floor.

Distribution^{5,7,9,14,15}

- Funnel web spiders have a wide distribution throughout south east Australia, including Tasmania (see map).
- However, thus far, the species proven to be dangerous to humans have a more limited range, largely limited to eastern parts of NSW and SE Queensland.
- The only proven killer, the Sydney funnel web spider *Atrax robustus*, is restricted to the Sydney region and adjacent areas, approximately 160 km radius north, south and west of the city, including Gosford and Newcastle. However, clinical experience and venom studies indicate at least several other species are potentially lethal and these cover, collectively, a far wider range than the Sydney species alone. [A case of funnel web spider envenoming in inner-city Brisbane has been described recently, which has wider implications regarding stocking of antivenom].¹⁵



Female Sydney funnel web spider *Atrax robustus*.



Female tree funnel web spider, *Hadronyche formidabili*.



Australian funnel web spiders: distribution.

Australian spiders overview: Funnel web spider group

Venom composition^{9,48}

- Funnel web spider venom is multicomponent, but a protein toxin, δ -hexatoxin-Ar1a (previously named robustoxin or atracatoxin), is the principal component responsible for severe envenoming in humans.
- Interestingly, while many mammals are relatively unaffected by funnel web spider venom, humans are very sensitive to it.
- δ -hexatoxin-Ar1a causes stimulation of the nervous system at a variety of synapses, with rapid and devastating effect.

Clinical effects

- Despite its fearsome reputation, there are only 13 recorded deaths due to funnel web spider bite,^{14,49} and experience indicates that many bites do not result in significant envenoming.^{2,7}
- However, when the spider does inject a dangerous quantity of venom, the effects can be rapid and severe, and death may occur within an hour.^{5,14}
- Consequently, all cases of suspected funnel web spider bite must be treated as a potential medical emergency.^{3,9}
- Typically the bite site is painful (due to large fangs), and the spider is usually seen.^{7,9}
- If envenoming occurs, it will develop early, starting with tingling around the lips, twitching of the tongue, then profuse salivation, lacrimation, sweating, piloerection and muscle twitching/spasms.^{5,9,14}
- Hypertension and tachycardia are features, as is respiratory distress due to rapid development of pulmonary oedema, which may be very severe and potentially lethal. In the early stages this may be mistaken for organo-phosphate poisoning. Convulsions may occur.^{5,8,9,14}

Australian spiders overview: Red back spiders

The species^{3,4}

- Red back spiders usually build webs under objects, with drop lines to the ground or other flat surface. Thus, they are commonly found under lower shelves, the bottom rails of fence lines, in refuse areas, especially old car bodies, even on the underside of seats left outside and in the recesses of cupboards indoors!
- Only the female is dangerous to humans, although on rare occasions, the male may bite and cause at least local envenoming.
- As the spiders tend not to wander, most bites occur when the person comes in contact with the web structure.
- Cupboard spiders *Steatoda sp* are close relatives of the red back spider and are commonly found in and around human habitation. They are mentioned here because they can also bite and cause symptoms similar to red back spiders, but generally less severe. Physically they look very similar to red back spiders, but lack red markings on the abdomen.

Distribution¹⁸

Red back spiders are found throughout Australia, in essentially all habitats, from very arid through to tropical, and they are common in urban areas, where most bites occur.

Venom composition¹⁸

The venom is multicomponent, with a family of protein toxins, the latrotoxins, most prominent. One of these, α -latrotoxin, stimulates neural synapses throughout the body with wide-ranging effects.



Red back spider (female),
Latrodectus hasselti.



Cupboard spider, *Steatoda grossa*
(looks similar to red back spider
but without red markings)

Australian spiders overview: Red back spiders

Clinical effects

- The clinical features of major envenoming by red back spiders are sometimes wide ranging in extent and may mimic other conditions, but rarely are lethal.^{3,9,10}
- Antivenom therapy should be considered for cases of significant envenoming.⁴
- The classic effective red back spider bite is felt as a mild sting only, with little to see at the bite site.¹⁰
- Between 10-40 minutes later (sometimes longer), the bite site becomes painful.^{10,18}
- The pain becomes severe over a variable timeframe, extending proximally and involving draining lymph nodes in the axilla or groin.^{9,10,18}
- There is often local sweating.^{9,10,18}
- The pain may then spread to the abdomen, chest, neck or head, often associated with profuse sweating, either localised or general, mild to severe hypertension, and malaise with nausea.^{9,10,18}
- Classic presentations include:
 - A triad of progressive severe pain, marked sweating and hypertension.^{4,8,9}
 - A triad of local bite site pain, sweating and piloerection.¹⁷
 - Gravitation of symptoms to the lower limbs in delayed presentations – i.e. burning sensation in the soles of the feet and pain and profuse sweating of both lower legs even if the bite was elsewhere.^{8,10}
- Many other symptoms or signs may occur, but the above are clinically most consistent and useful for diagnosis.⁸
- The progression from local to generalised pain may occur within 1 to 3 hours, or take up to 24 hours.⁸⁻¹⁰

.... continued

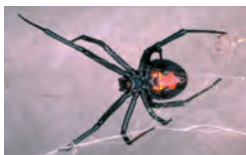
Antivenom therapy should be considered for cases of significant envenoming

- In infants, general miserableness, refusal of feeds, inconsolable crying, and sometimes a non-specific erythematous rash are key features of red back spider bite.⁹
- Occasionally the initial bite may not have been noticed and the patient may present with abdominal or chest pain. In such cases, careful questioning will usually elicit a history of possible exposure to spiderbite and initial localised pain, pointing to the true diagnosis.^{4,8,9}
- In pregnancy, the generalised abdominal pain of red back spider bite may appear similar to the onset of premature labour. Usually a clear history of a bite with initial local pain will be available.⁸
- Bites by cupboard spiders (*Steatoda sp*) can cause similar symptomatology, although generally not severe.³

In infants, general miserableness, refusal of feeds, inconsolable crying, and sometimes a non-specific erythematous rash are key features of red back spider bite

bioCSL's Red Back Spider Antivenom

- bioCSL's Red Back Spider Antivenom has been developed purely for use against bites by the Australian red back spider.²⁹
- However, there is some evidence of the efficacy of this antivenom in neutralising the *in vitro* effects of venom from other black widow spiders, and in the management of significant envenoming by cupboard spiders (*Steatoda spp*).^{50,51}



Red back spider (female),
Latrodectus hasselti.

Photo copyright A/Prof Julian White.

Australian spiders overview: Mouse spiders

- Mouse spiders (*Missulena spp*) are ground-dwelling mygalomorph spiders with large fangs and venom which is similar to that of the funnel web spider.
- A single severe (but non-fatal) paediatric case from SE Queensland has been reported (with symptoms/signs similar to those of envenoming by funnel web spider).¹⁶
- A study of 40 confirmed cases (including the one in Queensland) found that:¹⁶
 - Only the QLD case developed significant envenoming (with hypertension, muscle spasms, opisthoclonus and unconsciousness), which appeared to respond to bioCSL's Funnel Web Spider Antivenom.
 - Of the remaining 39 cases, 6 showed local neurotoxic effects (paraesthesiae, numbness and diaphoresis) and 5 had minor systemic effects (headache, nausea).
- Therefore, mouse spiders, while technically capable of causing major envenoming, in practice are unlikely to do so.¹⁶
- In practical terms, based on the similarity of envenoming signs/symptoms between these big black spiders and funnel web spiders, cases of mouse spider bite in eastern Australia (i.e. within the range of distribution of funnel web spiders) should be observed in an emergency department for 4 hours after the bite (and at least 2 hours after PBI first aid removal) to exclude major envenoming (see page 197).^{7,16}
- In areas outside the range of distribution of funnel web spiders, a shorter (1-2 hours) observation period would be appropriate.¹⁶
- Should severe envenoming occur, consider the use of bioCSL's Funnel Web Spider Antivenom, although this is not yet an approved indication.^{8,16}



Male mouse spider,
Missulena spp.

Australian spiders overview: Other spiders

White tail spider *Lampona cylindrata*

- This common hunting spider, found in houses, has been erroneously linked with necrotic arachnidism.
- A large published case series of 130 definite bites by white tail spider with expert identification of the spiders, described a pattern of clinical effects including local short-lived pain and erythema, sometimes with mild swelling or lump formation that can persist for a few days, but no instances of either secondary infection or ulceration/necrosis.¹²
- Research on white tail spider venom also has failed to confirm the association with skin damage.¹²
- Thus, the white tail spider can now be excluded as a cause of necrotic arachnidism.^{2,12,13}
- Suspected bites by unknown organisms, causing ulceration should NOT be labelled as “white tail” spider bites.¹³
- Other, non-bite causes of skin ulceration/necrosis should be considered in these cases with possible spiderbite left as a diagnosis of last resort, once all else is excluded (see pages 217-220).¹³



White tail spider,
Lampona cylindrata.

Australian spiders overview: Other spiders

The black house spider *Badumna insignis*⁵²

- This is a robust black spider of family Desidae, found in untidy webs with a tube retreat.
- It is common in urban habitats.
- The bite can cause moderate, and sometimes severe local pain of short duration, with erythema and swelling, and occasionally mild systemic symptoms.
- A recent series of 25 prospectively identified definite cases confirms that the black house spider is not a likely cause of “necrotic arachnidism”.

Huntsman spiders¹¹

- There are many species of huntsman spiders, family Sparassidae, which are common inside houses.
- Bites are generally very mild, with short-lived pain (approximately 5 min), reportedly severe in 27% of cases.
- Bite marks and local bleeding are more common than for bites by other spiders.
- In a minority of cases, local mild swelling, itchiness and erythema can all occur.
- A few species can occasionally also cause mild systemic symptoms, notably headache and nausea.
- A large series of 168 definite cases of huntsman spider bite has confirmed that these spiders **do not** cause skin damage/ulceration/necrosis.



Black house spider,
Badumna insignis.



Huntsman spider,
Isopeda spp.

Wolf spiders⁵³

- Many species of these ground hunting spiders, family Lycosidae, are common in gardens, sometimes entering houses.
- A reported series of 45 confirmed cases described bites by these spiders as generally minor, causing short-lived local pain, sometimes severe, local erythema, with occasional swelling and itchiness.
- Puncture marks/bleeding occurs in about a third of cases.
- Very few cases develop systemic symptoms, all temporary and minor (nausea, headache, malaise).
- There are no cases of skin damage/ulceration/necrosis associated with bites by wolf spiders.

Tarantula spiders⁸

- The common name ‘tarantula’ covers many spiders across a number of genera. These often large, hairy mygalomorph spiders of the family Theraphosidae, are increasingly common as indoor “pets”.
- They have powerful fangs and bites can cause moderate to severe pain, usually short-lived, with occasional non-specific systemic symptoms.
- While initially unpleasant, bites do not appear dangerous to humans, although bites are usually lethal to dogs.



Wolf spider, *Lycosa* spp.



Bird-eating spider (“tarantula”)
Selenocosmia stirlingi.

Australian spiders overview: Other spiders

Orb weaving spiders^{8,54}

- These common spiders of the family Aranaeidae, with many species, build “typical” spider webs in the garden at night.
- People walking into the web and crushing the spider against their body may be bitten, resulting in mild local pain of short duration and a small red lump, lasting about 24 hours.
- These spiders also may hide in clothing left outside overnight, on washing lines.
- The next person to put the clothing on may be bitten!



Garden orb weaver,
Eriophora spp.



Golden orb weaver,
Nephila spp.

Trapdoor spiders^{7,55}

- Includes a number of species of robust, ground dwelling, burrowing mygalomorph spiders, with very large fangs (families Idiopidae; Nemisiidae).
- They are often dug up in gardens.
- Despite the spider's size, bites appear to cause mostly mild pain, and occasionally, severe pain of short duration. There are rare cases of minor non-specific systemic effects.
- These spiders are frequently misidentified as funnel web spiders and therefore are part of the big black spider group (bites by these spiders should be regarded as potential funnel web spider bites and managed accordingly – see page 197).



Trapdoor spider,
Aganippe subtristis.

Section 5

Other arthropods

- Insects, centipedes and scorpions
- Paralysis ticks

In this section

Insects, centipedes and scorpions

| | |
|------------------|-----|
| Insects | 233 |
| Centipedes | 234 |
| Scorpions..... | 234 |

Paralysis ticks

| | |
|---|-----|
| Background | 235 |
| Paralysis tick envenoming: | |
| Clinical presentation | 236 |
| Tick paralysis in children..... | 237 |
| Problem presentations..... | 237 |
| First aid for tick bite | 238 |
| Diagnosis of paralysis tick envenoming..... | 238 |
| Treatment | 239 |

Insects, centipedes and scorpions

Insects¹

- A variety of insects can bite or sting humans, but in Australia, medically significant venomous species are typically *Hymenoptera* (bees; wasps; stinging ants).
- Injuries from exposure to other insects, particularly certain types of caterpillars do occur but are beyond the scope of this handbook. For advice on clinical cases contact the Poisons Information Centre 13 11 26.
- Medically significant envenoming, (i.e. massive stings from bees and wasps, usually with sting numbers measured in the hundreds or higher), is rare. Rather, it is potentially severe allergic reactions (particularly anaphylaxis) on repeat stings that are of medical concern.
- The topic of insect sting allergy is beyond the scope of this handbook.
- Nevertheless it is important to note some key stinging insects causing major allergy problems.
 - Honeybees.
 - A few species of native bees.
 - The European wasp.
 - Paper wasp (including *Polistes* and *Ropalidia* sp).
 - Large stinging ants (e.g. jumper ant; inch ant) – significant cause of major insect sting allergy.



Honeybee, *Apis mellifera*.



European wasp, *Vespa germanica*.



Jumper ant, *Myrmecia pilosula*.



Inch ant, *Myrmecia pyriformis*.

Insects, centipedes and scorpions

Centipedes¹

- These have fangs and venom glands at the head and bites may cause severe local pain, usually of short duration. Severe swelling at the bite site is often seen.
- Infection may occur, which is occasionally severe.
- Systemic envenoming does not occur.

Scorpions^{1,2}

- None of the many species of Australian scorpions is dangerous to humans but most cause intense local pain when they sting, often with local erythema, sometimes local tenderness, numbness or paraesthesia – usually lasting only a few hours.
- Systemic symptoms rarely occur and are never severe.
- Smaller Buthid scorpions, particularly *Lychas* spp cause the majority of stings, which generally occur indoors at night.
- The effects of stings by the larger non-Buthid scorpions tend to be milder.
- Of note, scorpions are now increasingly common in pet shops.



Red legged centipede.



Marbled scorpion,
Lychas marmoreus.



Sand scorpion,
Urodachus yashenkoi.

Paralysis ticks

Background

- There are numerous species of ticks within Australia but only one genus (*Ixodes*) includes ticks whose saliva produces a form of envenoming.
- These paralysis ticks produce a potent pre-synaptic neurotoxin in their saliva.³
- Only female ticks (including larval and nymph forms) feed on and introduce saliva into human hosts.^{3,4}
- These ticks may attach and feed over a number of days, and envenoming may take several days to develop.^{3,4}
- However, the vast majority of medically important human encounters with paralysis ticks relate to allergic reactions to bites, and not to envenoming. Allergic reactions may vary from urticaria to regional swelling to anaphylaxis.⁵
- Problems that are unrelated to envenoming (including infections relating to tick bite or granuloma formation from retained mouth parts) are not the subject of further discussion within this handbook.
- Paralysis ticks are found in eastern mainland Australia and in Tasmania.^{3,6}

Paralysis ticks produce a potent pre-synaptic neurotoxin in their saliva ... the ticks may attach and feed over a number of days, and envenoming may take several days to develop

Paralysis ticks

Paralysis tick envenoming: Clinical presentation

Local signs/symptoms

- Presence of tick attached to the patient. This may often be in a cryptic position such as on the scalp, or in the external ear or in body folds, and skin encrusting around the tick may occasionally make it hard to see it, as it looks like some form of 'sore'.³
- There may be local effects of the venom, particularly for ticks attached close to the facial nerve, which may cause a unilateral facial palsy.^{7,8}
- There may be evidence of past tick bites, such as from immature ticks, leaving multiple small sores.

General systemic effects

- Typically none. Some patients may develop generalised malaise and/or headache.^{1,9}

Specific systemic effects

- Specific systemic effects of paralysis tick envenoming is restricted to neurotoxic paralysis and/or allergic reactions (Table 37).

Table 37. Specific systemic effects of paralysis tick envenoming^{3,4,5,7}

| | |
|-----------------------------|---|
| Neurotoxic paralysis | Initial presentation is frequently an ataxic gait Progressive flaccid muscle paralysis Loss of deep tendon reflexes Respiratory paralysis Cranial nerve involvement |
| Systemic myolysis | Does not occur |
| Coagulopathy | Does not occur |
| Renal damage | Does not occur |
| Allergy | Local and systemic allergic reactions to tick saliva may occur |

Tick paralysis in children

Children appear to be more susceptible to severe tick envenoming than are adults. Progress from ataxic gait to full respiratory paralysis may take days, but potentially can be more rapid, particularly to the point where bulbar paralysis may threaten respiration directly, or through aspiration.^{1,4}

Problem presentations

- By definition, tick envenoming is usually cryptic, with the patient generally being unaware of the embedded, feeding tick.
- **Progressive onset of ataxia, followed by ascending paralysis may suggest a variety of neurologic diagnoses. Unless tick envenoming is included in the differential diagnosis, the true cause may be missed, with fatal results.**^{4,10}
- The embedded tick may be easily missed, or one tick may be found and others missed.
- Even after removal of all ticks, the paralysis may continue or worsen for up to 48 hours or more before starting to resolve, causing diagnostic confusion.^{4,7,11}

Tick envenoming is usually cryptic, with the patient generally being unaware of the embedded feeding tick.... The embedded tick may be easily missed, or one tick may be found and others missed

Paralysis ticks

First aid for tick bite^{1,3}

- If there is evidence of general major allergic reaction (anaphylaxis) – administer adrenaline **urgently**.
- As soon as possible, carefully remove each tick – pry it off the patient using tweezers on either side of the mouth parts (alternatively, a specific tick-removal tool may be used – many veterinarians in tick-prone areas stock these tools).
- Do not hold the tick by the body when pulling it off as this may leave mouth parts embedded in the skin.
- Carefully examine the whole body (including creases of the skin, the ears and hair/scalp) for other ticks.
- If there are any symptoms of paralysis, such as gait disturbance or other muscle weakness, or if these symptoms develop, the patient requires immediate medical assistance (see ‘treatment’ on pages 239-240).
- Venom from paralysis ticks may cause the patient to stop breathing. Extended respiratory support may be required.
- Advise the patient to:
 - Seek a tetanus immunisation booster from the local doctor if not received within the last 5 years.
 - Seek medical help, if after 24 hours, the wound becomes more red and painful (possible secondary infection).

Diagnosis of paralysis tick envenoming^{1,10}

- There are no specific diagnostic tests or a venom detection test for paralysis tick envenoming.
- Diagnosis is based on the typical history, clinical findings of progressive flaccid paralysis, and the discovery of one or more embedded ticks.
- In particular, carefully document neurological function, notably limb muscle strength, deep tendon reflexes, and cranial nerve signs.
- Include tick paralysis in the differential diagnosis for any cases of paralysis of non-traumatic onset.

.... continued

- Consider tick paralysis in the differential diagnosis of anyone presenting with acute onset of ataxia, lower limb weakness or generalised paralysis and in cases presenting in extremis following a convulsion with a history of respiratory or speech difficulties or generalised weakness preceding the convulsion.¹⁰
- Laboratory investigations are of little value for diagnosis of tick envenoming, but may help assess the extent of envenoming, e.g. if there is respiratory difficulty due to severe paralysis, arterial blood gas analysis may be useful for monitoring respiratory function.¹⁰

Treatment^{1,4,8,10,11}

Where possible, cases of tick paralysis should be managed in an ICU.

- Attend to ABC (Airway, Breathing, Circulation) and address any immediate life threats.
 - Assess and maintain airway.
 - Provide respiratory support as indicated.
- Insert I.V. line and provide fluid load.
 - Choice of crystalloid is not critical (e.g. normal saline; Hartmann's solution).
- Perform key history and examination.¹⁰
 - Obtain history of possible exposure to tick bite, including recent visit to tick-infested areas.
 - Enquire about symptom progression – e.g. interval between exposure to ticks and onset of symptoms.
 - In children – was there irritability prior to symptom onset?
 - Ask specifically about unsteady gait or reduced mobility in children (ataxia is a common feature in children).
- Perform a thorough search for and remove all embedded ticks (ensuring that no mouth parts are left behind in the skin).
- It is advisable to seek expert advice.

.... continued overleaf

Paralysis ticks

Treatment^{1,4,8,10,11} ... cont'd

Cases needing respiratory support will likely require intubation and ventilation for at least 3 days following removal of all ticks – and in some cases, a longer period of respiratory support may be needed.

There is no antivenom available for tick-related envenoming of human beings. CSL Paralysis Tick Antivenom is no longer produced. Experience over a number of years indicated that severe tick envenoming could be managed without antivenom therapy. [However, a number of veterinary products are available].

Due to the possibility of worsening paralysis after the removal of tick(s), patients presenting with symptomatic infestation by species of paralysis tick in Australia should be admitted and observed for 2-4 days after removal of ticks with serial meticulous neurological examination during this time. Provided the patient remains asymptomatic, discharge with advice to return immediately if symptoms recur. Do not discharge at night.^{1,4,11}

For patients who present with paralysis tick infestation and are asymptomatic, a shorter period of observation may sometimes be indicated after tick removal.

Consider admitting children with any signs of neurotoxicity for observation over several days.^{8,11}

Cases needing respiratory support will likely require intubation and ventilation for at least 3 days following removal of all ticks...
[and in some cases] for longer

Section 6

Jellyfish

- Box jellyfish
- Irukandji jellyfish
- Other jellyfish

In this section

| | |
|---|-----|
| Jellyfish stings: Overview | 243 |
| Jellyfish triage algorithm (flowchart) | 244 |
| Box Jellyfish stings: Overview & clinical issues | |
| Box Jellyfish stings..... | 245 |
| Box jellyfish stings: Clinical presentation | |
| Distribution..... | 246 |
| Box jellyfish stings: Clinical presentation | 246 |
| Clinical effects (and timing) of box jellyfish envenoming..... | 247 |
| Box jellyfish stings: First aid & diagnosis | |
| First aid for box jellyfish sting | 248 |
| Diagnosis..... | 249 |
| Box jellyfish stings: Urgent treatment | 250 |
| Box jellyfish stings: Clinical issues | 252 |
| Box jellyfish stings: Management chart | 253 |
| Box jellyfish stings: Antivenom therapy | |
| Indications for antivenom therapy..... | 254 |
| Antivenom dose..... | 255 |
| What if bioCSL's Box Jellyfish Antivenom is not immediately available?..... | 256 |
| Monitoring the patient after antivenom therapy..... | 256 |
| Preparation prior to commencing antivenom therapy..... | 257 |
| Administering bioCSL's Box Jellyfish Antivenom | 258 |
| Observation during antivenom therapy | 259 |
| Premedication prior to administering antivenom | 260 |
| What to do if there is an adverse reaction to antivenom..... | 260 |
| Antivenom therapy: Commonest mistakes... .. | 261 |
| Complications of antivenom therapy..... | 262 |
| Management of serum sickness | 263 |
| Irukandji jellyfish and Irukandji syndrome | |
| Background | 264 |
| Distribution..... | 265 |
| Venom composition | 265 |
| Irukandji syndrome: Clinical issues..... | 265 |
| Irukandji stings: Clinical presentation | |
| Local signs/symptoms | 266 |
| General systemic effects..... | 266 |
| Irukandji stings: First aid & diagnosis | |
| First aid for Irukandji syndrome | 267 |
| Diagnosing Irukandji syndrome | 267 |
| Management of Irukandji syndrome | |
| Irukandji syndrome: Urgent management | 268 |
| General management: Analgesia..... | 269 |
| General management: Hypertension | 271 |
| General management: Other issues..... | 272 |
| Irukandji syndrome management guidelines (flowchart) | 273 |
| Other jellyfish stings | |
| Key principles..... | 274 |
| Clinical presentation | 274 |
| First aid for jellyfish stings | 275 |
| Diagnosis..... | 276 |
| Management | 276 |
| Overview: Other jellyfish of medical importance | 277 |

Jellyfish stings: Overview

- Jellyfish stings occur principally in a marine environment, particularly in beach and coastal areas.
- The vast majority of jellyfish stings will be of minor nuisance value and may not require medical attention.
- However, there are three types of medically important jellyfish stings, which need to be distinguished from all other jellyfish stings at an early stage. These are:¹
 - Box jellyfish/chirodropid jellyfish stings
 - Irukandji/Carybdeid type stings
 - Blue bottle (*Physalia*) stings
- Significant allergic reactions to stings also may occur and require appropriate medical management.¹
- The jellyfish triage algorithm on page 244 may assist in differentiating these categories from other jellyfish stings.

243

There are three types of medically important jellyfish stings, which need to be distinguished from all other jellyfish stings at an early stage



Box jellyfish, *Chironex fleckeri*.



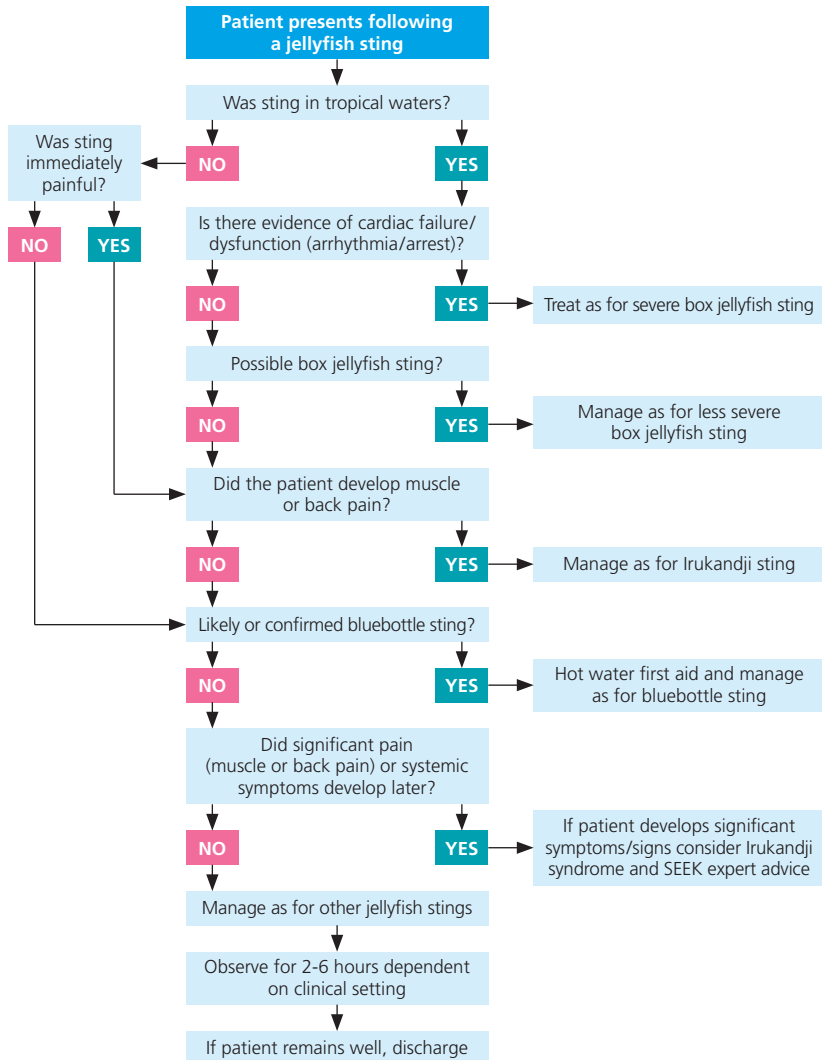
Irukandji jellyfish, *Carukia barnesi*.



Bluebottle, *Physalia* sp.

Box jellyfish photo copyright Dr Lisa-ann Gershwin, Australian Marine Stinger Advisory Services. Irukandji jellyfish photo copyright A/Prof Jamie Seymour. Bluebottle jellyfish photo copyright Morgan Talbot (photo obtained courtesy of Australian Museum).

Jellyfish triage algorithm



Box jellyfish stings: Overview & clinical issues

Box jellyfish stings

- Box jellyfish (e.g. *Chironex fleckeri*) are cubozoan jellyfish from the group Chirodropidae – the jellyfish are large and virtually transparent with a box-like body and numerous tentacles draping from each of the four corners.²
- Each tentacle contains millions of individual stinging cells with specialised stinging organelles (nematocysts), each of which can deliver venom via an everting stinging device.² A proportion of this venom may be injected into capillaries just beneath the skin surface.
- This explains the very rapid development of severe systemic envenoming in major box jellyfish stings.
- The venom is incompletely understood.
 - It contains components that can cause local pain and necrosis, and those that can affect cardiac function and/or respiratory function.³⁻⁵
 - The venom likely causes abnormalities in ionic transport across membranes, resulting in altered membrane permeability.⁵
 - The composition of *Chironex fleckeri* venoms varies considerably between western and eastern Australia, which may explain the geographic variation in reported deaths due to box jellyfish envenoming (numerous fatalities in NT and QLD waters, but no reported deaths in WA).⁶
- Severe envenoming due to box jellyfish sting is relatively rare. The majority of stings are not severe, but a small number of stings will present with moderate to severe effects, which may be lethal.^{5,7,8}
- **The rapidity of development of severe and life-threatening envenoming is unique among venomous animals – with the possibility of death within minutes of a box jellyfish sting.**^{5,8}
- Therefore, it is critically important to urgently recognise and appropriately manage these stings while still on the beach.

It is critically important to urgently recognise and appropriately manage these [box jellyfish] stings while still on the beach

Box jellyfish stings: Clinical presentation

Distribution

Box jellyfish are confined to Australian tropical waters of Australia's north (from Gladstone in Queensland to Broome in Western Australia), including some offshore islands. *Chironex fleckeri* stings can occur year round, although they are most common during the summer months.^{2,8,9}

Box jellyfish stings typically occur close to the shore.⁸

Box jellyfish stings: Clinical presentation^{5,8}

Local signs/symptoms⁸

- Tentacle contact marks (typically a 'ladder' pattern) often with adherent tentacle on the skin.
- Immediate intense local pain in areas of contact.

General systemic effects⁸

- Non-specific symptoms of distress secondary to the intense local pain.
- In very severe cases, collapse with cardiac arrhythmia or cardiac arrest.
- In severe cases, respiratory distress.

Box jellyfish stings in children^{5,10}

- Severity of envenoming is dose dependent and small children are more likely to receive a potentially lethal dose.
- In recent years, most fatalities from box jellyfish stings have been in children.



Box jellyfish, *Chironex fleckeri*.

Clinical effects (and timing) of box jellyfish envenoming^{1,5,7,8}

- The extent of envenoming by chirodropids (e.g. *Chironex fleckeri*) is essentially dependent on the area of discharging tentacle contact.⁸
- Involvement of > 10% of total skin area is potentially lethal envenoming, especially in children.¹
- In such cases, systemic envenoming can occur within minutes of the sting, with cardiac failure or cardiac arrest possible within 5 minutes of a major sting.^{5,8} This emphasises the importance of rapid, effective first aid.
- At every point of tentacle contact with skin there will be immediate, often excruciating pain, usually with linear red welts.⁸
- These affected areas of skin may develop blistering and/or necrosis.⁸
- Incoherence due to pain is a feature in major stings.⁸
- Shortly after, in severe stings, cardiac problems may develop.^{5,8}
- Respiratory dysfunction may occur later, sometimes associated with pulmonary oedema, but a central respiratory depressant effect also has been suggested.⁵
- However, recent studies indicate that the majority of box jellyfish stings are mild to moderate, with severe stings representing about a quarter of cases.^{7,8}

At every point of tentacle contact with skin there will be immediate, often excruciating pain, usually with linear red welts



Ladder pattern of box jellyfish sting.

Photo copyright A/Prof Jamie Seymour.

Box jellyfish stings: First aid & diagnosis

First aid for box jellyfish sting^{1,11,12}

- Remove the person from the water while being careful to avoid stings to rescuers – restrain if necessary.
- Call for an ambulance (dial Triple Zero – 000) and seek assistance from a lifesaver/lifeguard if available.
- ABC – check respiratory and cardiac status and if required, administer CPR (if the patient is in cardiac arrest) – see pages 32-36 for Basic (critical care) first aid. Do not cease CPR until the patient has reached a medical facility.
- Immediately flood the entire stung area with copious amounts of vinegar for at least 30 seconds, to neutralise any unfired nematocysts (stinging organelles). [Do not use fresh water, alcohol or methylated spirits].^{1,11,12}
- Gently pick off any tentacles with tweezers, forceps or gloved fingers.
- If vinegar is unavailable, pick off any remnants of the tentacles (this is not shown to be harmful to the rescuer) and rinse the stung area well with seawater (not freshwater as this may cause unfired nematocysts to discharge).
- If pain relief is required, provide this only after vinegar has been applied.
- Apply a cold pack or dry ice in a plastic bag for pain relief.
- Reassure and keep the person still until medical assistance arrives.

PBI first aid is contraindicated for box jellyfish stings

In the past, the PBI technique was recommended first aid for box jellyfish stings. However, recent research indicates that PBI first aid may actually increase the extent of envenoming following box jellyfish sting and therefore, is not only discarded as a recommendation but considered a contraindicated technique by experts.¹³

Diagnosis

The diagnosis of box jellyfish stings is based principally on the classical clinical presentation, specifically, immediate intense pain, ladder track tentacle marks, often with adherent tentacle, and in severe cases, cardiac or respiratory collapse.

For major stings, confirmation of the type of jellyfish can be made via nematocyst identification from tentacle contact marks.* This is performed by using sticky tape to remove nematocysts from the skin.

Place a strip of sticky tape (sticky side down) onto the bite area.

Peel tape off skin – nematocysts attached to the skin will be retained on the tape.

Place the tape on a slide for microscopic examination.

Contact the Poisons Information Centre on 13 11 26 for assistance with finding an expert to examine the sticky tape, or contact A/Prof Jamie Seymour, James Cook University, Cairns Campus on 07 4042 1229.

There are no diagnostic blood tests or venom detection tests for box jellyfish stings.

However, the majority of box jellyfish stings are likely to be only mild to moderate in severity.⁸ Thus, at times, it may be less easy to distinguish these box jellyfish stings from those by other types of jellyfish. As this has no implications for management, confirmation of these less severe stings is not important.

*For major jellyfish stings where the organism is unknown – determining the identity of the organism may not impact on the clinical management of the current case, but could potentially be very useful for future management and to gain a better understanding of the species causing major stings in the region.

The classical clinical presentation [of box jellyfish sting is] immediate intense pain, ladder track tentacle marks, often with adherent tentacle, and in severe cases, cardiac or respiratory collapse

Box jellyfish stings: Urgent treatment

Urgent treatment: First steps^{1,8}

1. Attend to ABC (Airway, Breathing, Circulation) and address any immediate life threats
 - Assess and maintain airway
 - Provide respiratory support as indicated
 - Cardiac support as indicated (should be sustained)
2. Apply vinegar if not already applied
3. Insert I.V. line and provide fluid load (if indicated)
 - Choice of crystalloid is not critical (e.g. normal saline; Hartmann's solution)
4. Commence ECG monitoring (baseline ECG is useful for all except minor stings)

5. Assess the patient's clinical status and manage accordingly⁸

Life-threatening cardiac or respiratory decompensation/failure or if in cardiac arrest

See below

Cardiac failure/arrhythmia or if the patient is unconscious

See page 251

Non-life threatening envenoming (no cardiopulmonary decompensation)

See page 251

Life-threatening cardiac or respiratory decompensation/failure or patient in cardiac arrest⁸

- Administer 1-3 vials of diluted bioCSL's Box Jellyfish Antivenom as an I.V. push, with I.V. magnesium sulphate (0.2 mmol/kg up to 10 mmol adult dose) as a bolus over 5-15 minutes (if the patient is in cardiac arrest consider the maximal dose of 6 vials of undiluted antivenom administered as a rapid I.V. push).^{1,8,14}
- See pages 254-263 for detailed information regarding antivenom therapy.
- If the patient is in continued cardiac arrest and provided the maximal antivenom dose has not been used, consider using further antivenom up to a total of 6 vials and repeat the I.V. magnesium sulphate dose, and consider other cardioactive drugs, before ceasing CPR.

Cardiac failure/arrhythmia or if the patient is unconscious⁸

Administer a minimum of 1 vial of diluted bioCSL's Box Jellyfish Antivenom as an I.V. push (see pages 254-263 for detailed information regarding antivenom therapy).

Non-life threatening envenoming (no cardiopulmonary decompensation)^{1,8}

- Use ice packs for initial pain relief, together with oral, I.M., or I.V. analgesia, if necessary.
- If this proves ineffective, give I.V. magnesium sulphate (0.2 mmol/kg, up to 10 mmol adult dose) as a bolus over 15 minutes and/ or 1 vial of (diluted) bioCSL's Box Jellyfish Antivenom I.V.
- See pages 254-263 for detailed information regarding antivenom therapy.
- If pain persists consider further narcotic analgesia and/or a further dose of I.V. magnesium sulphate.
- Take sticky tape sample from tentacle contact mark for later nematocyst identification, if required (see page 249 for further details).¹
- Apply wound dressing to any areas of damaged or necrotic skin (areas of tentacle contact) and then manage as for burns.¹

Note: The information above and on page 250 is adapted from the updated management protocol for box jellyfish envenoming, i.e. the Northern Territory revised protocol (with additional input from this handbook's expert panel). Alongside differing antivenom therapy recommendations based on the patient's clinical status, the Northern Territory revised protocol also advises the use of magnesium sulphate in appropriate cases [8].

The Northern Territory revised protocol was written for managing severe *Chironex fleckeri* envenoming wherein antivenom therapy may save a life – i.e. a severe sting with cardiorespiratory arrest near a health centre or hospital, where immediate resuscitation and rapid use of large volumes of I.V. antivenom is possible [8].

Therefore, the updated management protocol devised by experts contains antivenom therapy recommendations that differ from the Product Information for bioCSL's Box Jellyfish Antivenom.

Box jellyfish stings: Urgent treatment

Box jellyfish stings: Clinical issues

There has been some question/uncertainty regarding the ability of antivenom to act rapidly enough to reverse life-threatening envenoming.^{5,8} Recent research evidence in a small animal model indicates that antivenom may not be able to neutralise venom rapidly enough to save life in catastrophic envenoming.¹⁵ However, this is not a reason at this time to abandon antivenom therapy in such a setting in human beings.

There has been some suggestion that prolonged on-beach cardiac resuscitation can result in a successful outcome.^{8,16} Therefore early antivenom therapy may still assist in recovery from other venom effects.

Despite the dramatic nature of severe envenoming, by far the majority of *Chironex fleckeri* stings are mild to moderate, with the initial severe pain controlled with ice packs or simple analgesia and, for moderate stings, a single dose of parenteral narcotic analgesia.^{7,8}

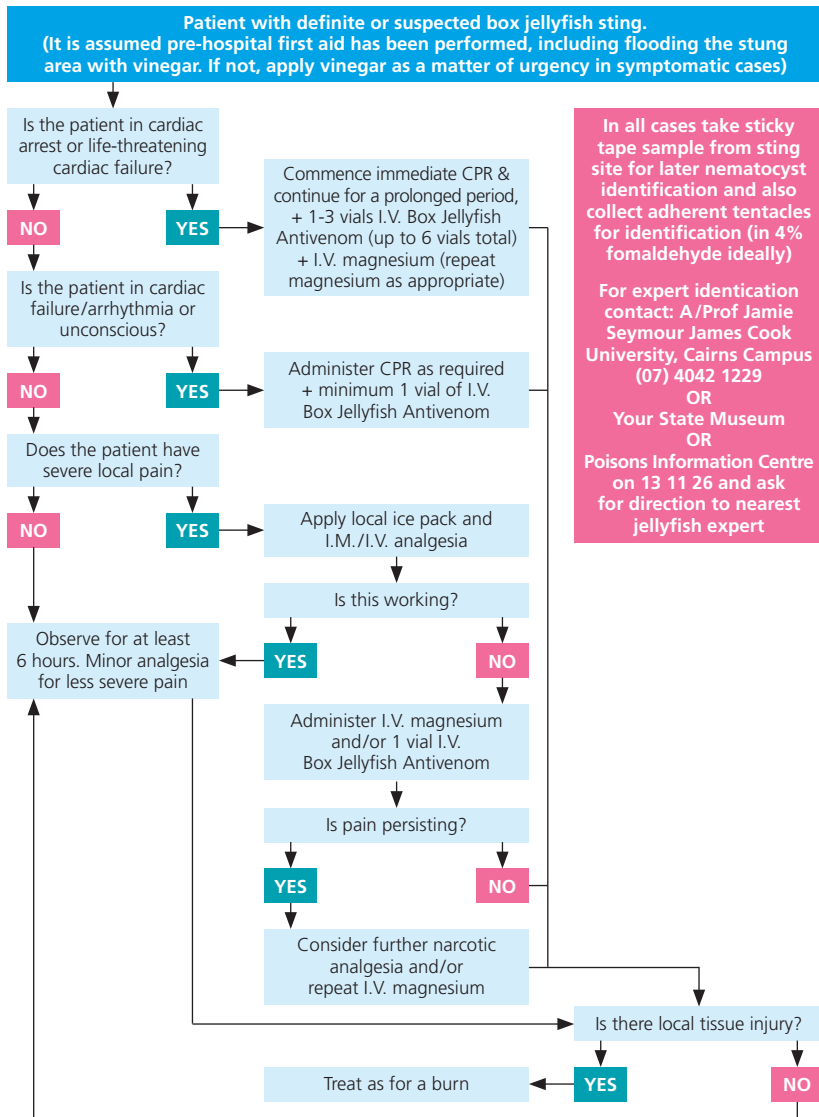
The requirement for parenteral opioid analgesia is far less than in Irukandji syndrome^{8,17,18} and was needed in only 1 of 23 nematocyst-positive cases in one series.⁷

[In severe envenoming] prolonged on-beach cardiac resuscitation can result in a successful outcome. Therefore early antivenom therapy may still assist in recovery from other venom effects



Nematocysts on sticky tape.

Box jellyfish stings: Management chart



Box jellyfish stings: Antivenom therapy

Before administering the initial dose of antivenom, seek expert advice and refer to pages 257-263 for additional details regarding the preparatory steps and procedure for administering bioCSL's Box Jellyfish Antivenom.

Indications for antivenom therapy

- The role of antivenom as specific treatment for severe and life-threatening box jellyfish envenoming remains controversial.^{5,8}
- Recent research using animal models appears to indicate that bioCSL's Box Jellyfish Antivenom effectively targets the cardiotoxins in the venom. However, the potency and rapidity of action of these toxins makes antivenom rescue problematic.¹⁵
- Therefore, it is unclear if antivenom can rescue a patient in severe cardiac failure/decompensation or cardiac arrest after a box jellyfish sting. While this is not a reason for abandoning antivenom treatment in such cases, caution should be exercised when discussing the possible benefits of the antivenom with a patient's relatives.
- Indications for antivenom therapy are outlined in the box.

Indications for bioCSL's Box Jellyfish Antivenom^{8,9,19}

- Cardiac arrest.
- Cardiac failure.
- Unconscious patient.
- Respiratory failure.
- Severe local pain that is unresponsive to parenteral narcotic analgesia and I.V. magnesium sulphate.



bioCSL's Box Jellyfish Antivenom.

Antivenom dose

Antivenom dosing varies depending on the circumstance (Table 38).

Table 38. bioCSL's Box Jellyfish Antivenom dose⁸

| Circumstance | Antivenom dose |
|--|--|
| Life-threatening cardiac or respiratory failure/decompensation, or if in cardiac arrest | 1-3 vials I.V. If in cardiac arrest, consider maximal dose (6 vials undiluted as a rapid I.V. push) ^{1,8,14} |
| Cases with cardiac failure/arrhythmia or if the patient is unconscious | Minimum of 1 vial I.V. |
| In patients with severe local pain unresponsive to parenteral narcotic analgesia and I.V. magnesium sulphate | 1 vial I.V. |

Note: The antivenom therapy recommendations in Table 38 differ from the Product Information for bioCSL's Box Jellyfish Antivenom. The PI recommends the use of 1 vial of diluted Box Jellyfish Antivenom I.V. (or if the I.V. route is impractical, 3 vials undiluted given I.M. at 3 different sites).¹⁹

- The updated antivenom therapy recommendations in Table 38 represent information within the Northern Territory revised protocol for managing *Chironex fleckeri* envenoming, with input from this handbook's expert panel.^{1,8}
- Due to the rapidity of fatal *Chironex fleckeri* envenoming, the critical window of opportunity for potentially life-saving use of antivenom is possibly only minutes, i.e. much smaller than that for envenoming from snakebite. Additionally, large amounts of antivenom may be required.⁸
- The Northern Territory revised protocol was written for managing severe *Chironex fleckeri* envenoming wherein antivenom therapy may save a life – i.e. a severe sting with cardiorespiratory arrest near a health centre or hospital, where immediate resuscitation and rapid use of large volumes of I.V. antivenom is possible.⁸
- Therefore, the updated antivenom therapy recommendations devised by experts differ from the Product Information for bioCSL's Box Jellyfish Antivenom.

Box jellyfish stings: Antivenom therapy

What if bioCSL's Box Jellyfish Antivenom is not immediately available?

- All the non-antivenom management strategies documented earlier remain applicable in the absence of antivenom.^{1,8}
- Specifically, in severe and life-threatening envenoming, cardiopulmonary support is a cornerstone of management.
- Additionally, preventing continuing envenoming by washing the tentacle contact tracks with vinegar and removing adherent tentacles remain important measures.
- The use of I.V. magnesium sulphate and appropriate analgesia are similarly still applicable in the absence of antivenom.

Monitoring the patient after antivenom therapy

Patients should remain under observation for at least 6 hours after receiving bioCSL's Box Jellyfish Antivenom.¹⁹

Preparation prior to commencing antivenom therapy²⁰⁻²²

Prior to commencing antivenom therapy, ensure all facilities are ready at hand to treat anaphylaxis, in the event that this should occur.

- Dedicate one small-bore I.V. line (18-20 G in adults) to antivenom administration.
- Dedicate one large bore I.V. line (16-14 G in adults) for emergency resuscitation.
- Prepare 1L normal saline (20 mL/kg in children) ready to administer under pressure.
- Prepare adrenaline (1:1000 – i.e. 1 mg adrenaline in 1 mL) drawn up to a dose of 0.01 mg/kg (maximum 0.5 mg – i.e. 0.5 mL) and label as 'Adrenaline for **I.M.** injection only (dose in mg)'.
- Ideally, also prepare an I.V. infusion of adrenaline 1 mg in 100 mL, which is controlled by infusion pump or syringe driver and ready to attach by a side arm to the resuscitation line. Anti-reflux valves must be attached above the side arm on any other infusions using this I.V. line, to prevent adrenaline going back up into other fluid bags. To prevent erroneous administration, **do not attach the adrenaline infusion unless it is needed.**
- Record blood pressure on the opposite arm to the fluid/adrenaline infusion – to avoid prolonged cuff inflations and thus, extravasation of infusion fluids.
- See 'What to do if there is an adverse reaction to antivenom' on pages 260-261 for method of emergency resuscitation if required.

Box jellyfish stings: Antivenom therapy

Administering bioCSL's Box Jellyfish Antivenom^{1,8,14,19,23,24}

| | |
|--------------------------------|--|
| Treatment location | <p>If possible, patients should receive antivenom in a monitored environment with immediate access to resuscitative equipment (e.g. a well resourced emergency department or ICU) with one-to-one nursing. However, cases of box jellyfish envenoming with cardiac decompensation or cardiac arrest require immediate treatment at the beach</p> |
| Route of administration | <p>Intravenous</p> <p>Always administer Box Jellyfish Antivenom I.V. – preferably through drip set</p> |
| Dilution of antivenom | <p>Dilute up to 1 in 10 with an isotonic crystalloid solution (e.g. normal saline; Hartmann's solution)</p> <p>To avoid fluid overload, use smaller volumes in small children and in adults with compromised cardiac function</p> <ul style="list-style-type: none"> – Adults with compromised cardiac function: Up to a 1 in 5 dilution may be more appropriate – Small children: Dilute the antivenom to the extent that the total volume delivered does not exceed 10 mL/kg <p>In a catastrophic setting of cardiac arrest on the beach, consider administering Box Jellyfish Antivenom as an undiluted I.V. push</p> |
| Time period of dosing | <p>In a non-critical situation, start slowly and increase the rate gradually to deliver antivenom over 10-20 minutes (or more rapidly in some circumstances). If available, consider using an infusion pump^{1,8,14}</p> <p>In a critical situation, diluted or undiluted antivenom may need to be delivered rapidly as an I.V. push. The treating clinician will make the final decision based on the circumstances of the individual case^{1,8,14}</p> |
| Observation | <p>See page 259</p> |

Observation during antivenom therapy^{1,21,25}

- Carefully observe the patient during antivenom administration and for 1 hour after, to ensure adverse reactions (if they occur) are recognised and treated promptly (adverse reactions are discussed further on pages 260-263).
- In particular, look for the development of symptoms and signs of anaphylaxis. An erythematous rash may be the first sign of developing adverse reactions (often first seen in the axilla or the lower abdomen).
- Also observe for hypotension and bronchospasm.
- Carefully monitor BP, HR and respiratory function, oxygen saturation, with particular attention to development of hypotension and/or bronchospasm.
- Look for additional warning signs of anaphylaxis in children (Table 39).
- See pages 262-263 for further information on potential complications of antivenom therapy.

Table 39. Warning signs of anaphylaxis in children^{1,25}

| | |
|------------------------------------|---|
| Rash; hypotension; or bronchospasm | Profuse sweating |
| Nasal, palatal, or ocular pruritus | Faecal or urinary urgency or incontinence |
| Coughing | Abdominal pain |
| Sneezing | A sense of impending doom |



Antivenom rash in an adult.

Box jellyfish stings: Antivenom therapy

Premedication prior to administering antivenom

The use of premedication prior to antivenom is controversial but is not relevant in this life-threatening situation with box jellyfish antivenom.

What to do if there is an adverse reaction to antivenom^{20,21,25,26}

Adverse reactions may be related to the rate of antivenom infusion – those reactions can include flushing, hypotension or bronchospasm. Hypotension and bronchospasm are hallmarks of major adverse reaction (anaphylaxis).

Adverse reactions may respond to temporarily stopping the antivenom infusion, waiting to ensure that there is no return or worsening of the reaction, and then re-starting at a slower rate.

For anaphylactic reactions, adrenaline is generally the drug of first choice.

See the box below and on page 261.

Steps to take if there is either a sudden fall in blood pressure, or bronchospasm after starting antivenom infusion^{1,14,20-22,25}

- Suspend the antivenom infusion.
- Lie the patient flat (if not already in this position) and commence high-flow 100% oxygen and support airway/ventilation as required.
- Begin rapid infusion of one litre normal saline (20 mL/kg in children) over 2-3 minutes.
- Administer adrenaline 1:1000 I.M. into the lateral thigh at a dose of 0.01 mg/kg to a maximum of 0.5 mg (i.e. a maximum of 0.5 mL). Note: Adrenaline 1:1000 ampoule is 1 mg adrenaline in 1mL.
- Alternatively, those experienced with I.V. administration of adrenaline can proceed to do this directly instead of I.M. injection. See procedure on page 261 in the section 'If adverse reactions do not respond to initial management'.
- Seek expert advice regarding ongoing management.
- In most cases, once the adverse reaction is controlled, cautious reintroduction of antivenom is possible. [Note: A patient requiring antivenom therapy prior to the adverse reaction will likely continue to require antivenom after the adverse reaction].

Note: The recommendations above and on page 261 for the management of anaphylactic reactions to antivenom reflect current published anaphylaxis management guidelines and expert advice and may vary from the Product Information for bioCSL's antivenoms [1,21,22,25].

If adverse reactions do not respond to initial management^{1,14,20-22,25}

- If hypotensive, repeat normal saline bolus as per box on page 260 (up to 50 mL/kg may be required).
- Commence I.V. infusion of adrenaline (0.5-1 mL/kg/hr of adrenaline 1 mg in 100 mL) and titrate according to response. Monitor BP every 3-5 minutes using the arm opposite to the infusion.
- Be aware that as the adverse reaction to antivenom resolves, adrenaline requirements will fall, the blood pressure will rise and the adrenaline infusion rate will need to be reduced.
- Consider nebulised salbutamol for bronchospasm, nebulised adrenaline for upper airway obstruction, and I.V. atropine for severe bradycardia.
- Seek advice urgently from local/regional ED Consultant and/or ICU Consultant.
- In most cases, once the adverse reaction is controlled, cautious reintroduction of antivenom is possible. [Note: A patient requiring antivenom therapy prior to the adverse reaction will likely continue to require antivenom after the adverse reaction].

Antivenom therapy: Commonest mistakes¹

Some of the most common mistakes relating to antivenom therapy are highlighted below.

- Failure to use antivenom when clearly indicated.
- Giving antivenom unnecessarily, i.e. when there are no clear clinical indicators of significant systemic envenoming.
- Choosing the wrong antivenom.
- Choosing the wrong dose.
- Antivenom given too late.
- Administering antivenom by the wrong route.
- Failing to prepare for an adverse reaction.
- Failing to inform the patient about serum sickness.

Box jellyfish stings: Antivenom therapy

Complications of antivenom therapy

Essentially, antivenom is whole or modified antibody from an animal. It is obtained by hyperimmunising the animal against a particular venom or group of venoms. The IgG antibody from blood plasma is used, and typically, is fractionated to the F(ab')₂ fragment of IgG, and on occasion, whole IgG. bioCSL's Box Jellyfish Antivenom is a purified whole IgG preparation fractionated from sheep plasma.^{19,27}

When making antivenoms, bioCSL undertakes assiduous efforts to filter and discard any extraneous blood components and contaminants. Nevertheless even high-quality antivenoms will cause adverse reactions in some patients. The clinically important adverse reactions can be subdivided into 'early' and 'late'.

Early reactions are those that occur immediately after commencing antivenom therapy or within the first few hours (Table 40). Late adverse reactions may occur several days later (Table 41 on page 263).

Table 40. Early adverse reaction to antivenom^{1,14,19,21,25}

| | |
|--|--|
| Rash | Localised or generalised erythematous, sometimes pruritic rash. May occur as an isolated and generally trivial adverse reaction or it may herald the onset of a more severe adverse reaction |
| Pyrexia | Febrile reactions may potentially occur – although there have been no reported cases of pyrexia with bioCSL's Box Jellyfish Antivenom ¹⁹ |
| Anaphylactic / anaphylactoid reaction | <p>This is a type of potentially life-threatening reaction. Therefore, antivenom should never be given until measures to manage such a reaction are in place. This is why the use of antivenom outside a hospital environment is strongly discouraged. However, for Box Jellyfish Antivenom, in cases of on-beach cardiac arrest, antivenom may need to be given in suboptimal circumstances</p> <p>Anaphylaxis may be preceded by a localised or generalised rash, sometimes first seen in the axilla or lower abdomen, proceeding to hypotension and/or bronchospasm</p> <p>Look for additional warning signs of anaphylaxis in children (see Table 39 on page 259)</p> <p>Note, when antivenom therapy is clinically indicated, it should never be withheld for fear of an adverse reaction. Seek expert advice</p> |

Table 41. Late adverse reactions to antivenom^{1,14,19}

The principal late adverse reaction to antivenom is serum sickness, a type III delayed hypersensitivity reaction, which most commonly presents 4-14 days post exposure

Serum sickness is characterised by a flu-like illness with fever, joint and muscle pain and general malaise, often preceded by or associated with a maculopapular or erythema multiformae-like rash

Serum sickness rates often increase as the antigen load increases. Therefore, it is more likely to occur in patients who have had a high volume load of antivenom

Every patient who receives antivenom should be advised of the symptoms of serum sickness and told to seek medical care if these symptoms arise after discharge from hospital

Serum sickness is more likely to occur in patients who have had a high volume load of antivenom

Management of serum sickness^{1,14,28}

- While serum sickness can be a mild and self-limited disease, it can be distressing for patients and early diagnosis and treatment is advisable.
- There is a diversity of opinion about the approach to treatment for various causes of serum sickness (i.e. not just antivenom) – which may involve first-line use of antihistamines or oral steroids. Serum sickness post antivenom therapy is usually managed with a short course of oral corticosteroids.
- If uncertain about the treatment approach, consult with a clinical immunologist.
- Some experts suggest prescribing a week-long course of oral prednisolone commencing immediately after antivenom, for all patients who have received more than 25 mL of antivenom. This treatment has not yet been tested through clinical trials.

Irukandji jellyfish and Irukandji syndrome

Background^{8,17,18,29-35}

- Stings from a variety of tropical jellyfish species (the best known being *Carukia barnesi*) can cause a pattern of symptoms known as Irukandji syndrome.²⁹⁻³¹
- Other species now known to cause Irukandji syndrome include: *Alatina nr mordens*; *Carybdea alata*; *Malo maxima*; *Carybdea xaymacana*; *Carybdea rastoni* and the as yet unnamed 'fire jelly'.³²⁻³⁴
- While *Carukia barnesi* is a very small jellyfish, other species can be moderate in size.
- Unlike box jellyfish stings, which occur almost exclusively close to shore, Irukandji stings can occur either close to shore or as far offshore as the outer Barrier Reef.^{8,30}
- Nematocysts (stinging organelles) are found on the tentacles and body of all Irukandji jellyfish³⁵ (unlike box jellyfish which have nematocysts exclusively on the tentacles).
- Although more common during warmer months, Irukandji stings can occur at any time of the year.^{17,18}
- Only a small area (sometimes only a few square centimetres) of skin contact is required to cause major envenoming.¹⁷
- Consequently, even a fully suited scuba diver can develop Irukandji syndrome from a small sting on the exposed cheek area.

Only a small area (sometimes only a few square centimetres) of skin contact is required to cause major envenoming



Irukandji jellyfish, *Carukia barnesi*.

Distribution

- *Carukia barnesi* appears to be limited to Queensland waters, but other species of jellyfish capable of causing Irukandji syndrome have been found in northern tropical waters from northwest Western Australia across to Queensland Pacific waters.^{17,18,35}
- Irukandji jellyfish are found in coastal waters off beaches and out to deeper waters including the outer edge of the Great Barrier Reef and further offshore.³⁰
- There are limited case reports suggesting that Irukandji jellyfish may occur in subtropical and temperate waters in the southern half of Australia's maritime environment.^{33,36} This requires further confirmation.

Venom composition^{34,35,37}

- Due to the diversity of species involved and the difficulty in collecting sufficient venom for research, Irukandji-causing venoms are currently poorly characterised.
- However, it is clear that the venoms contain toxins with some similarities to certain arthropod toxins (such as venom from the funnel web spider) which are capable of causing a catecholamine storm.

Irukandji syndrome: Clinical issues^{17,18,29,30,37-39}

Irukandji syndrome is essentially a manifestation of venom-induced catecholamine storm, and unlike other jellyfish stings, the symptoms are delayed. Consequently, an often-minor sting may be ignored until the patient develops symptoms of significant systemic envenoming.



Irukandji jellyfish (*C. barnesi*) sting.
Note sting is barely visible.

Irukandji stings: Clinical presentation

Local signs/symptoms

The area of sting contact can be very small and local pain may be insignificant, though sometimes the sting is immediately painful.³⁸

General systemic effects

After a variable interval, most commonly around 20-40 minutes post sting, there is onset of systemic envenoming and associated symptoms (Table 42).^{17,29,38}

Irukandji syndrome can be fatal.³¹ A case of intracranial haemorrhage and death after envenoming by *Carukia barnesi* has been described recently.⁴⁰

Table 42. Irukandji syndrome: Systemic effects (20-40 minutes post sting)^{17,18,30,37-39}

| |
|--|
| Myalgic limb pain |
| Severe back pain |
| Severe headache |
| Nausea/vomiting |
| Pallor |
| Profuse sweating |
| Prostration |
| Generalised erythematous rash |
| Agitation/restlessness |
| A feeling of panic or 'impending doom' |
| General vasoconstriction |
| Severe hypertension |
| Variable cardiac dysfunction (reduced cardiac function on echocardiography – particularly left ventricular dysfunction, occasional elevated troponin, pulmonary oedema). Severe cardiac dysfunction may require intubation and inotropic support |

Irukandji stings: First aid & diagnosis

First aid for Irukandji syndrome^{1,11,17,29,38}

Current recommendations for first aid for Irukandji stings are the same as first aid for box jellyfish stings. This includes:¹¹

- Dialing Triple Zero – 000 for an ambulance.
- Seeking assistance from a lifesaver/lifeguard.
- Assessing ABC (Airway, Breathing, Circulation) and performing CPR if required.
- Irrigation of the sting area with copious amounts of vinegar.

However, in reality, the sting is often not felt, or if felt, is mild (localised erythema or welts or mild skin pain).^{17,38}

As a consequence, many cases of Irukandji sting will not become obvious until systemic envenoming has developed.^{29,38} Although vinegar should be applied, it may be impractical for some cases at this stage, particularly if the area of tentacle or bell contact is very small and the location of the sting site is unclear.¹

Most patients will not require CPR.¹⁷

If systemic envenoming develops, the key first aid manoeuvre should be to transport the patient to definitive medical care as rapidly as is safely possible.^{1,11}

Diagnosing Irukandji syndrome

- Diagnosis is based on the classical clinical presentation, specifically, the delayed onset of severe back and limb pain (as opposed to pain at the sting site), sweating, hypertension, and in severe cases, evidence of cardiac dysfunction.^{17,37-39}

.... continued overleaf

The classical clinical presentation is delayed onset of severe back and limb pain (as opposed to pain at the sting site), sweating, hypertension, and in severe cases, evidence of cardiac dysfunction

Management of Irukandji syndrome

Diagnosing Irukandji syndrome ... cont'd

- Generally, the severity of pain experienced in Irukandji syndrome is greater than that seen with box jellyfish stings. Parenteral opioid analgesia is frequently required.^{8,17,18}
- Nematocyst identification from the tentacle contact marks may help to confirm the type of jellyfish. However, the timeframe within which this is performed means it is unlikely to impact on diagnosis or management of the current patient.
- There are no diagnostic blood tests or venom detection tests.

Irukandji syndrome: Urgent management⁴¹

Urgent management of Irukandji syndrome

- Check ABC (Airway, Breathing, Circulation) and administer CPR if indicated
- Secure cardiac and respiratory function
- Apply vinegar if not already applied and sting location is known.
- Apply high-flow oxygen
- Cardiac (ECG, chest X-Ray, cardiac troponin) and oxygen saturation monitoring
- Monitor BP
- Establish I.V. access
- Commence analgesia
- Control hypertension
- On arrival, all suspected cases of Irukandji syndrome should have the following investigations:
 - Pathology: FBC, UEC, Mg, cardiac troponin
 - 12-lead ECG
 - Chest X-Ray
 - Echocardiogram if there is clinical or radiographic evidence of cardiovascular instability
- For overt cardiac failure, if phentolamine infusions are required, or if there is evidence of neurological dysfunction, transfer/admit the patient to an ICU for aggressive management.
- There is no antivenom for Irukandji syndrome and management is confined to treating symptoms of envenoming as described on pages 269-273.
- **If the patient is unstable or unresponsive to treatment – call the Poisons Information Centre on 13 11 26.**

General management: Analgesia⁴¹

Adequate analgesia is vital in cases of Irukandji syndrome.

Opioids

- Most cases will require parenteral opioid analgesia.
- Generally morphine 0.05 mg/kg or fentanyl 0.5 µg/kg, repeated every 5 minutes until adequate analgesia has been achieved or 4 doses have been delivered.
- Consider opioid infusions.
- Avoid pethidine.

Suggested adjuncts to opioids

- Midazolam 25 µg/kg I.V. up to 4 doses
or
- Chlorpromazine 0.3 mg/kg I.V. over 10 min
or
- Promethazine 0.3 mg/kg I.V. over 10 min.

Magnesium sulphate (I.V.)

- Magnesium sulphate (MgSO₄), a peripheral inhibitor of catecholamine release and effect, may be used for controlling pain. (Note: Setup is described on page 270).

Initial bolus 0.15 mmol/kg over 15 min.

Repeat if necessary.

Then add infusion of 0.1 - 0.15 mmol/kg/hr.

- Monitor to clinical response.
If tendon reflexes are preserved, titrate to clinical response.
Reflexes to be assessed hourly before any bolus MgSO₄ administration.

.... continued overleaf

Adequate analgesia is vital in cases of Irukandji syndrome

Management of Irukandji syndrome

General management: Analgesia⁴¹ ... cont'd

Magnesium sulphate (I.V.) ... cont'd

– Weaning.

Once symptoms are controlled for 4-6 hours, the infusion is reduced by 1-2 mmol/hr every hour.

If breakthrough symptoms occur, give a 0.04 mmol/kg (adult 2.5-3 mmol) dose as a bolus, recommence the infusion at the previous rate, then recommence weaning 4 hours later.

– A number of precautions are applicable when using MgSO₄ (see box below).

– Setup may be performed by one of two methods.

Option 1: Neat Magnesium Sulfate for Injection (2 mmol/mL) 50 mL in syringe driver. Requires side-line fluid to ensure MgSO₄ is flushed in and to reduce injection pain.

Option 2: MgSO₄ 50 mmol added to 1000 mL Normal Saline (0.05 mmol/mL; 200 mL = 10mmol). An infusion controller (pump) is required.

– Ampoules of Calcium gluconate 20 mL must be kept near the patient if on a magnesium infusion.

Precautions when using MgSO₄⁴¹

- Take extreme care in the presence of renal failure or neuromuscular disorders.
- To avoid toxicity it is imperative to monitor tendon reflexes. If there is loss of tendon reflexes, cease the infusion and reassess with a view to recommencement at a lower rate.
- ECG monitoring is highly recommended for all patients with Irukandji syndrome and should be continued throughout the infusion.
- Hypotension due to vasodilation may occur. This is more common in patients who are dehydrated and/or on antihypertensive medications. Treat hypotension by discontinuing the infusion, administering a fluid bolus (Hartmann's 10mL/kg) and once BP has settled, recommencing at the maintenance rate without a further bolus.
- Calcium gluconate will reverse the CV and neuromuscular effects of MgSO₄ and should be available whenever MgSO₄ is used for any indication.
- Injection pain may be reduced by slowing the loading dose or further diluting the MgSO₄.

General management: Hypertension⁴¹

- Control of hypertension may be life saving – fatalities related to Irukandji sting reported in northern Queensland succumbed to intracerebral haemorrhages.
- Use short-acting antihypertensive agents as severe cases may develop venom-induced cardiac dysfunction and consequent hypotension.
- Nitrates should be used first line for severe hypertension (**contraindicated in patients taking Viagra®, Levitra® or other selective phosphodiesterase inhibitors**).
- For SBP > 200 mm Hg and DBP > 120 mm Hg use glyceryl trinitrate (GTN).
 - Initially use GTN inhaler (2 puffs every 5 minutes while infusion is being set up). Prepare GTN 50 mg in 500 mL Normal Saline or 5% dextrose = 100 µg/mL.
 - Follow with I.V. GTN starting at 3 mL/hr (5 µg/min), and double the rate every 5 min until SBP < 160 mm Hg and DBP < 100 mm Hg.
 - Continue non-invasive BP monitoring every 5 minutes.
 - Aim for SBP 100-160 mm Hg and DBP 50-100 mm Hg.
- If GTN is contraindicated.
 - Commence I.V. MgSO₄ early. Contact the Poisons Information Centre on 13 11 26 for further details.
 - I.V. Phentolamine may be used – contact the Poisons Information Centre.
- After BP and pain are controlled for 4 hours, halve the GTN rate every 20 minutes if BP remains within the target range. Cease GTN if BP remains controlled at a dose of 3 mL/hr.

Precautions when managing hypertension⁴¹

- Patients receiving selective phosphodiesterase inhibitor therapy (e.g. Viagra®, Levitra®, etc) will experience a life-threatening hypotensive episode if GTN is administered. Early recognition and aggressive management is required, which involves:
 - Ceasing GTN infusion.
 - I.V. volume loading.
 - Adrenaline boluses.
- Other causes of hypotension typically respond to cessation of infusion and fluid loading alone.

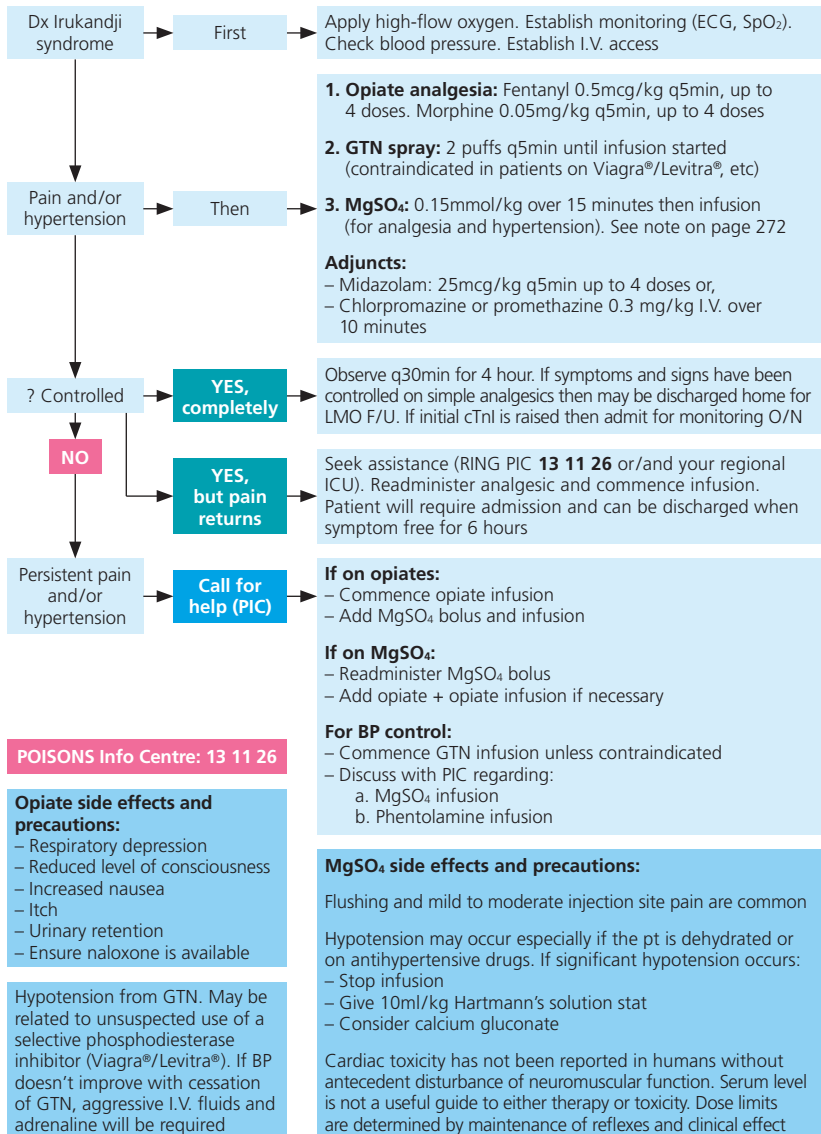
Management of Irukandji syndrome

General management: Other issues⁴¹

| | |
|------------------------------------|--|
| Cardiac function monitoring | <ul style="list-style-type: none">– Due to the risk of severe cardiac dysfunction, ECG monitoring is essential– Also perform baseline chest X-ray (pulmonary oedema), cardiac troponin measurements, and where indicated, echocardiography |
| Overt cardiac failure | <ul style="list-style-type: none">– For overt cardiac failure or the need for phentolamine infusions, or if there is evidence of neurological dysfunction, transfer/admit the patient to an ICU for aggressive management |
| Pulmonary oedema | <ul style="list-style-type: none">– Cases with severe envenoming may develop pulmonary oedema and usually will require intubation and inotropic support. This may include adrenaline³⁷ |
| Observations and discharge | <ul style="list-style-type: none">– Many patients with less severe envenoming may obtain symptom relief after initial boluses of opioids, and if other investigations are normal (including no sign of cardiac dysfunction), it may be reasonable to discharge the patient after 4 hours and provide simple oral analgesia– Patients requiring infusions of opioid analgesic or magnesium or with any evidence of cardiac dysfunction should be admitted for observations and management<ul style="list-style-type: none">Observe for at least 6 hours after cessation of infusionsIf opioid or magnesium have not been required for 6 hours, and symptoms have resolved, the patient may be discharged– In any patients with ongoing severe pain, or cardiac abnormalities on ECG, chest X-Ray or raised troponin, there is a risk of further deterioration. These patients require high-dependency monitoring with serial ECGs, chest X-Ray and cardiac troponin measurements |

The protocol for the management of Irukandji syndrome outlined on pages 268-273 is based on local experience and may change with the results of further research. Emerging research (McCullagh *et al.* *Emerg Med Australas* 2012;24(5):560-5) has questioned the efficacy of MgSO₄ in the management of Irukandji syndrome. Experts now suggest considering the use of adjunct treatments (e.g. midazolam, chlorpromazine or promethazine) before using MgSO₄. If the patient's condition is not improving, it is important to seek urgent expert advice [24].

Irukandji syndrome management guidelines



Other jellyfish stings

Key principles¹

- Australia harbours a vast array of jellyfish species capable of stinging humans, most of which will cause local discomfort or pain and may leave tentacle marks on the skin.
- These jellyfish do not cause significant systemic envenoming, and in many cases, the stings are trivial and do not require medical treatment.
- However, any sting has the potential to sensitise the patient and make them susceptible to allergic reactions on further stings.
- While allergic responses to jellyfish stings are thought to be uncommon, on rare occasions, jellyfish stings cause severe allergic reactions including anaphylaxis.
- In particular, severe allergic reactions have been reported for bluebottle (Portugese Man of War) stings, although this may simply reflect the commonness of stings by these jellyfish.
- The key management principle is ensuring that cases of likely box jellyfish or Irukandji jellyfish sting are not misdiagnosed as a sting by a minor species.

Clinical presentation

- Clinical effects of jellyfish stings can be highly variable, depending on the species of jellyfish, the area of tentacle contact, and the size and health of the patient.
- The following list includes some key symptoms/signs, although not all of these will be present in every case.³⁵
 - Local discomfort or pain (mild to severe).
 - Tentacle or bell contact marks on the skin.
 - Local erythema.
 - Local blistering.
 - Generalised erythema/rash.
 - Non-specific systemic symptoms.
 - Painful corneal lesions due to jellyfish stings to the eye.⁴²

First aid for jellyfish stings

In tropical waters where life-threatening stings can occur from box jellyfish and Irukandji jellyfish, vinegar is the appropriate first aid (unless it is certain that the person was stung by a bluebottle, the sting is localised, and the person is stable).¹¹

In non-tropical waters, for bluebottle and other jellyfish stings, hot water is the appropriate first aid.¹¹

- Keep the person at rest. Reassure and keep under constant observation.
- Prevent rubbing of the stung area.
- For bluebottle and other non-tropical jellyfish stings use a hot shower (prior to this, if possible, pick off any adherent tentacles and rinse the bitten area in sea water to remove invisible stinging organelles).^{11,43}
- Alternatively, place the stung hand or foot in hot water (no hotter than the rescuer can tolerate and no hotter than 45°C). If possible, also place the non-stung limb into the hot water.^{24,43-45}
- Don't immerse the stung hand or foot for more than 20 minutes.^{1,24}
- Remove briefly before re-immersing.^{1,24}
- Continue this cycle if pain persists, but for no longer than 2 hours.^{1,24}
- If the pain persists or is generalised, if the sting area is large (half of a limb or more), or involves sensitive areas (e.g. the eye) call an ambulance (Dial Triple Zero – 000) and seek assistance from a lifesaver/lifeguard if available.¹¹

For bluebottles and other non-tropical jellyfish
[if available] use a hot shower

Other jellyfish stings

Diagnosis¹

- Diagnosis is usually based on a history of likely exposure to a jellyfish in a marine environment, together with examination revealing likely tentacle/bell marks in the stung area.
- Wherever possible, sticky tape retrieval of adherent nematocysts from the skin at the sting site should be performed so that later identification of the jellyfish may be possible.
- There are no diagnostic blood tests or venom detection tests.

Management¹

- For most jellyfish stings, management will consist of first aid on or near the beach, such as the use of hot water to relieve short-lived pain.
- In cases with more severe or persistent pain or pain unresponsive to initial first aid, medical assessment is required and appropriate analgesia should be considered.
- If there is evidence of an allergic response then consider antihistamines.
- If a severe allergic reaction (anaphylaxis) occurs, this should be treated in the standard manner.

Overview: Other jellyfish of medical importance

Jimble, *Carybdea rastoni*³⁵

A small (maximum size 2 cm) 4-tentacled box jellyfish found in all Australian waters. It is common in Australian waters in summer, often in swarms. The sting is painful, with an angry red mark. Significant systemic effects are recently being reported from tropical waters.¹

Vinegar over the adherent tentacles is appropriate first aid.³⁵

Morbakka, *Tamoya sp*^{*35}

A large 4-tentacled box jellyfish from tropical waters (Moreton Bay to Port Douglas). It causes a very painful sting (immediate severe burning pain, which lasts 24 hours). The sting forms a white wheal (approximately 1 cm wide) surrounded by a red flare. Ladder markings similar to *Chironex fleckeri* stings may be seen.

Systemic envenoming may occur, including collapse, respiratory distress and throbbing lumbar pain (reminiscent of Irukandji syndrome) but there are no recorded deaths. Skin necrosis may occur. There is no antivenom. Vinegar is effective as first aid.

*Taxonomy may change.

Bluebottle, *Physalia sp*^{11,35}

A medium sized 'jellyfish' (actually a hydrozoan colony organism) found in all Australian waters, often in swarms. There is immediate pain lasting an hour or more, with typical elliptical blanched wheals and surrounding erythema. Usually, systemic symptoms range from none to mild, but a muscle-pain syndrome may occur.³⁵ There is no antivenom. Hot water is effective first aid.¹¹



Jimble, *Carybdea rastoni*.



Morbakka, *Tamoya sp*.



Bluebottle, *Physalia sp*.

Other jellyfish stings

Overview: Other jellyfish of medical importance ... cont'd

Sea nettle, *Chrysaora sp*¹

Moderate sized jellyfish, which causes mild to moderate local pain, lasting up to several hours. Allergic reactions can occur. There is no antivenom and there is currently no substance that appears useful as first aid to inactivate nematocysts. Vinegar is not effective.

Mauve stinger, *Pelagia sp*³⁵

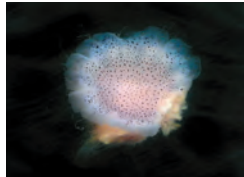
Large jellyfish, sometimes found in swarms throughout Australian waters. It causes immediate local pain, wheals, pruritus and swelling. Dyspnoea has been reported after massive stings. Skin eruptions may recur without repeat contact with the jellyfish. Appears to have allergenic venom. There is no antivenom.

Hair jelly, *Cyanea sp*³⁵

Widespread in Australian coastal waters. Very large jellyfish, which causes short-lived, moderate pain and redness, typically without systemic envenoming. There is no antivenom. Vinegar is **ineffective** as first aid and should not be used.³⁵ Wash the stung area with copious amounts of sea water (not fresh water).¹



Sea nettle, *Chrysaora sp.*



Hair jelly, *Cyanea sp.*

Section 7

Stinging fish

- Stonefish and other stinging fish

In this section

Stinging fish: Background & clinical presentation

| | |
|-----------------------------|-----|
| Background | 281 |
| Clinical presentation | 281 |

Stinging fish: First aid & diagnosis

| | |
|---------------------------------|-----|
| First aid for fish stings | 282 |
| Diagnosis | 282 |

Fish stings: General treatment.....283

Specific treatment for stonefish stings: Stonefish antivenom

| | |
|---|-----|
| bioCSL's Stonefish Antivenom: Specificity.... | 284 |
| Indications for bioCSL's Stonefish Antivenom..... | 284 |
| Initial dose | 284 |
| bioCSL's Stonefish Antivenom: Follow-up dosing | 285 |
| Antivenom: Route of administration..... | 285 |
| Patient follow up after antivenom therapy | 285 |
| What if bioCSL's Stonefish Antivenom is not immediately available?..... | 285 |
| Preparation prior to commencing antivenom therapy..... | 286 |
| Administering bioCSL's Stonefish Antivenom..... | 287 |
| Observation during antivenom therapy | 288 |
| Premedication prior to administering antivenom..... | 289 |
| What to do if there is an adverse reaction to antivenom | 291 |
| Antivenom therapy: Commonest mistakes | 292 |
| Complications of antivenom therapy..... | 293 |
| Management of serum sickness | 294 |

Stonefish: Overview 295 |

Stinging fish: Background & clinical presentation

Background¹⁻³

- Numerous species of fish across many families have evolved venomous spines, which may be located on different parts of the fish depending on the species and family.
- In most cases, a primitive venom gland envelops the sharp spines and when pressure is applied on the spine, e.g. when it enters the skin, it may force venom beneath the skin surface.
- In some species, the venom apparatus is under some pressure and can more effectively inject venom.
- In general, these fish spines result in localised envenoming, with negligible or mild systemic effects and little prospect of lethality.
- The common feature of all cases is local pain, which is frequently intense.
- The other common feature is the susceptibility of this pain syndrome to hot water treatment.

Clinical presentation¹⁻³

| Local signs/symptoms | Systemic effects |
|---|--|
| <ul style="list-style-type: none">– There may be obvious sting penetration marks on the skin– Usually, there is immediate, intense local severe pain, which can be incapacitating– This is followed by marked swelling– There may be tenderness and local skin discolouration around the penetration mark, sometimes characterised as bluish discolouration– Local infection and minor skin necrosis around sting entry points can occur, but appear to be uncommon to rare | <ul style="list-style-type: none">– No specific systemic effects– Non-specific symptoms such as dizziness, nausea/vomiting, sweating and abdominal pain are occasionally reported for stings by the more toxic species such as the stonefish and lionfish– Hypotension, collapse, cyanosis and pulmonary oedema have been described – but are uncommon |

Stinging fish: First aid & diagnosis

First aid for fish stings¹⁻⁶

- Place the stung hand or foot in hot water (no hotter than the rescuer can tolerate and no hotter than 45°C).^{1,2,4,5} If possible, also place the non-stung limb into the hot water.^{3,6}
- Do not immerse stung limb for more than 20 minutes. Do not immerse if local anesthetic has been used.^{3,6}
- Remove briefly before re-immersing.^{3,6}
- If pain persists continue this cycle, but for no longer than 2 hours.^{3,6}
- If the pain is unresponsive to hot water treatment, transport the person to a medical facility (antivenom is available for stonefish envenoming).¹
- Do not use PBI first aid, because for fish stings the venom tends to stay in the stung area.⁴

Place the stung hand or foot in hot water
(no hotter than 45°C) for up to 20 minutes.
Remove briefly before re-immersing

Diagnosis³

Diagnosis of fish stings is generally based on a clear history of likely or witnessed exposure to a stinging fish, such as holding a stinging fish caught by a line or net, or stepping on a bottom-dwelling fish such as the stonefish.

The classic history of immediate intense pain with a clear puncture mark(s) from the sting is generally diagnostic. There are no diagnostic blood tests or venom detection tests. However, X-ray or possibly ultrasound, may be useful in identifying retained sting deep in the wound.

Stingray injuries may sometimes cause similar symptoms and signs, but as the treatment for non-lacerated simple stingray punctures is generally the same as for fish stings, differentiation is of little medical consequence.

Fish stings: General treatment

General treatment for fish stings^{1-3,5,7}

The principal medical problem in most fish stings is intense local pain and control of this pain is central to medical management. In many cases, the use of simple hot water immersion (no hotter than 45°C) will quickly reduce pain. However, the stung region cannot be left in water for prolonged periods (usually immersed for 15-20 minutes then removed, and if pain returns, repeat the cycle for no longer than 2 hours – i.e. up to 4 times).

Persistent significant pain resistant to hot water treatment requires effective analgesia, which in most cases will necessitate parenteral opioid analgesics or consideration of a regional nerve block. In cases of confirmed or likely stonefish sting, consider antivenom therapy for severe pain or severe oedema that has been unresponsive to hot water first aid (see details on pages 284-294).

Local infection is always possible, though uncommon, and it is important to disinfect the wound and ensure tetanus immunisation is current. Prophylactic antibiotics should be avoided, but if infection develops over the following days, broad-spectrum antibiotic cover may be required. Consult with a clinical microbiologist.

In cases of confirmed or likely stonefish sting, consider antivenom therapy for severe pain or severe oedema that is unresponsive to hot water first aid



Stonefish.



Lionfish.

Specific treatment for stonefish stings: Stonefish antivenom

bioCSL's Stonefish Antivenom: Specificity

bioCSL's Stonefish Antivenom is prepared from the plasma of horses immunised with the venom of stonefish (*Synanceia verrucosa* and/or *Synanceia horrida*).⁷

bioCSL's Stonefish Antivenom is specific only for stings by true stonefish (*Synanceia* spp).⁷ There also is anecdotal evidence for some success using stonefish antivenom for stings by related scorpionid fish and recent studies in Brazil indicate effectiveness for stings by *Scorpaena plumieri*.³

Additionally, there is some evidence that bioCSL's Stonefish Antivenom can neutralise the effects of venom from lionfish (*Pterois volitans*),⁸ and the cobbler (*Gymnapistes marmoratus*).⁹ Anecdotal reports indicate that some experts are now using Stonefish Antivenom for the treatment of suspected bullrout envenoming that has not responded to hot water or analgesics.^{6,10} However, there is no firm evidence for this practice and efficacy is unknown.

Importantly, the treatment of envenoming from non-stonefish stings is not an approved indication for this antivenom.

Indications for bioCSL's Stonefish Antivenom

Confirmed stonefish sting with systemic symptoms (see page 281) or significant local symptoms (intense pain or severe oedema not responsive to hot water treatment).⁷

bioCSL's Stonefish Antivenom: Initial dose⁷

| Number of puncture wounds | Dose of bioCSL's Stonefish Antivenom |
|---------------------------|--------------------------------------|
| 1 to 2 | 1 vial |
| 3 to 4 | 2 vials |
| 5 or more | 3 vials |

bioCSL's Stonefish Antivenom: Follow-up dosing

After the initial dose of antivenom, if the patient's symptoms subside significantly with at most, the need for supplementary over-the-counter analgesics, no further antivenom is indicated.⁶

In contrast, if the symptoms have not subsided significantly within about 2 hours after the initial dose of antivenom (i.e. there is continued need for analgesia using prescription medications such as morphine, tramadol or paracetamol-codeine), and the diagnosis of stonefish envenoming is assured, repeat the initial dose of antivenom.^{6,7}

Antivenom: Route of administration

The Product Information recommends administering bioCSL's Stonefish Antivenom I.M. with the I.V. route used for extreme cases.⁷ However, some clinicians may choose to use the I.V. route for all cases of stonefish envenoming.^{3,6} The Product Information suggests the I.V. route is more likely to precipitate anaphylactoid reactions, although this has not been observed in clinical practice.^{6,7} For further information on antivenom administration see pages 286-294.

Patient follow up after antivenom therapy

Observe the patient for a minimum of 4-6 hours post antivenom and until symptoms have resolved.^{6,7}

What if bioCSL's Stonefish Antivenom is not immediately available?

If antivenom is not available for a confirmed stonefish sting, management is directed towards alternative methods of pain relief, i.e. hot water immersion and parenteral analgesia or a regional nerve block.²

Important note

Before administering the initial dose of bioCSL's Stonefish Antivenom seek expert advice and refer to pages 286-294 for additional details regarding the preparatory procedures prior to antivenom therapy, administering antivenom, patient observation, and potential complications.

Specific treatment for stonefish stings: Stonefish antivenom

Preparation prior to commencing antivenom therapy¹¹⁻¹³

Prior to commencing antivenom therapy, ensure all facilities are ready at hand to treat anaphylaxis, in the event that this should occur.

- Dedicate one small-bore I.V. line (18-20 G in adults) to antivenom administration.
- Dedicate one large bore I.V. line (16-14 G in adults) for emergency resuscitation.
- Prepare 1L normal saline (20 mL/kg in children) ready to administer under pressure.
- Prepare adrenaline (1:1000 – i.e. 1 mg adrenaline in 1 mL) drawn up to a dose of 0.01 mg/kg (maximum 0.5 mg – i.e. 0.5 mL) and label as 'Adrenaline for **I.M.** injection only (dose in mg)'.
- Ideally, also prepare an I.V. infusion of adrenaline 1 mg in 100 mL, which is controlled by infusion pump or syringe driver and ready to attach by a side arm to the resuscitation line. Anti-reflux valves must be attached above the side arm on any other infusions using this I.V. line, to prevent adrenaline going back up into other fluid bags. To prevent erroneous administration, **do not attach the adrenaline infusion unless it is needed.**
- Record blood pressure on the opposite arm to the fluid/adrenaline infusion – to avoid prolonged cuff inflations and thus, extravasation of infusion fluids.
- See 'What to do if there is an adverse reaction to antivenom' on pages 291-292 for method of emergency resuscitation if required.



bioCSL's Stonefish Antivenom.

Administering bioCSL's Stonefish Antivenom^{3,6,7,14,15}

| | |
|--------------------------------|---|
| Treatment location | If possible, patients should receive antivenom in a monitored environment with immediate access to resuscitative equipment and one-to-one nursing |
| Route of administration | Intramuscular or intravenous |
| Dilution of antivenom | <p>For I.V. administration, dilute up to 1 in 10 with an isotonic crystalloid solution (e.g. normal saline; Hartmann's solution)</p> <p>To avoid fluid overload, use smaller volumes in small children and in adults with compromised cardiac function</p> <ul style="list-style-type: none">– Adults with compromised cardiac function: Up to a 1 in 5 dilution may be more appropriate– Small children: Dilute antivenom to the extent that the total volume delivered does not exceed 10 mL/kg <p>For I.M. administration, inject antivenom undiluted</p> |
| Time period of dosing | <p>For I.V. administration, each dose should be run over about 30 minutes (a single dose may comprise multiple vials)</p> <p>Start very slowly and increase the rate gradually to deliver antivenom over approximately 30 minutes</p> |
| Observation | See page 288 |

Specific treatment for stonefish stings: Stonefish antivenom

Observation during antivenom therapy^{3,12,16}

- Carefully observe the patient during antivenom administration and for 1 hour after, to ensure adverse reactions (if they occur) are recognised and treated promptly (adverse reactions are discussed further on pages 291-294).
- In particular, look for the development of symptoms and signs of anaphylaxis. An erythematous rash may be the first sign of developing adverse reactions (often first seen in the axilla or the lower abdomen).
- Also observe for hypotension and bronchospasm.
- Carefully monitor BP, HR and respiratory function, oxygen saturation, with particular attention to development of hypotension and/or bronchospasm.
- Look for additional warning signs of anaphylaxis in children (Table 43).
- See pages 293-294 for further information on potential complications of antivenom therapy.

Table 43. Warning signs of anaphylaxis in children^{3,16}

| | |
|------------------------------------|---|
| Rash; hypotension; or bronchospasm | Profuse sweating |
| Nasal, palatal, or ocular pruritus | Faecal or urinary urgency or incontinence |
| Coughing | Abdominal pain |
| Sneezing | A sense of impending doom |



Antivenom rash in an adult.

Premedication prior to administering antivenom

Premedication prior to administering antivenom remains an issue surrounded by controversy, some conflicting evidence and opinion, and uncertainty about “best practice”.

Studies outside Australia have provided evidence showing premedication using antihistamines and/or hydrocortisone are either ineffective, or possibly effective to a variable extent.^{17,18} Hence, available evidence is unhelpful and contradictory for these medications. Similar contradictory evidence applies to adrenaline premedication within and outside Australia.¹⁹⁻²³

Data from recent Australian clinical research reported in 2008 (the ASP study – a multicentre, prospective study of snakebite envenoming from over 60 major tertiary centres and regional hospitals around Australia) suggests that there is no clear benefit in giving premedication prior to antivenom administration, including no clear benefit in using adrenaline as premedication.²⁴

Further, the ASP study has shown that in Australia, the use of premedication prior to antivenom therapy is not common practice (adrenaline premedication was provided to 8.7% of patients in whom data about premedication was available).²⁴

Additionally, recent expert consensus suggests that premedication to prevent adverse reactions to antivenom therapy is not routinely indicated.¹⁴

.... continued overleaf

Specific treatment for stonefish stings: Stonefish antivenom

Premedication prior to administering antivenom ... cont'd

Clearly, there may be specific clinical circumstances, judged on an individual patient basis, where a clinician may consider the use of adrenaline as premedication prior to antivenom administration. For example – in a patient with known major allergy to antivenom where resuscitation facilities may be suboptimal.³

In such situations the uncertain and unproven benefits of adrenaline premedication should be carefully weighed against the known and documented adverse effects from use of adrenaline, and wherever practical, the patient should be told of this risk-benefit situation so that informed consent can be given.³

Clearly, in some situations, with severe life-threatening systemic envenoming, informed consent is impractical and should not form a necessary condition of using adrenaline premedication if the clinician deems this is required.³

Irrespective of whether premedication is used or not, antivenom should only be administered in an environment where rapid detection and appropriate treatment of severe early adverse reactions will occur.³

- This includes the immediate availability of adrenaline, oxygen and resuscitation equipment and staff competent and prepared to use these treatments effectively.
- In such a setting, a controlled I.V. adrenaline dilute infusion may be the optimal route for administering adrenaline to treat anaphylactic or anaphylactoid reactions (see pages 291-292 for further details).³

What to do if there is an adverse reaction to antivenom^{11,12,16,24}

Adverse reactions may be related to the rate of antivenom infusion – those reactions can include flushing, hypotension or bronchospasm. Hypotension and bronchospasm are hallmarks of major adverse reaction (anaphylaxis).

Adverse reactions may respond to temporarily stopping the antivenom infusion, waiting to ensure that there is no return or worsening of the reaction, and then re-starting at a slower rate.

For anaphylactic reactions, adrenaline is generally the drug of first choice.

See the box below and on page 292.

Steps to take if there is either a sudden fall in blood pressure, or bronchospasm after starting antivenom infusion^{3,11-14,16}

- Suspend the antivenom infusion.
- Lie the patient flat (if not already in this position) and commence high-flow 100% oxygen and support airway/ventilation as required.
- Begin rapid infusion of one litre normal saline (20 mL/kg in children) over 2-3 minutes.
- Administer adrenaline 1:1000 I.M. into the lateral thigh at a dose of 0.01 mg/kg to a maximum of 0.5 mg (i.e. a maximum of 0.5 mL). Note: Adrenaline 1:1000 ampoule is 1 mg adrenaline in 1mL.
- Alternatively, those experienced with I.V. administration of adrenaline can proceed to do this directly instead of I.M injection. See procedure on page 292 in the section 'If adverse reactions do not respond to initial management'.
- Seek expert advice regarding ongoing management.
- In most cases, once the adverse reaction is controlled, cautious reintroduction of antivenom is possible. [Note: A patient requiring antivenom therapy prior to the adverse reaction will likely continue to require antivenom after the adverse reaction].

Note: The recommendations above and on page 292 for the management of anaphylactic reactions to antivenom reflect current published anaphylaxis management guidelines and expert advice and may vary from the Product Information for bioCSL's antivenoms [3,12,13,16].

Specific treatment for stonefish stings: Stonefish antivenom

If adverse reactions do not respond to initial management^{3,11-14,16}

- If hypotensive, repeat normal saline bolus as per box on page 291 (up to 50 mL/kg may be required).
- Commence I.V. infusion of adrenaline (0.5-1 mL/kg/hr of adrenaline 1 mg in 100 mL) and titrate according to response. Monitor BP every 3-5 minutes using the arm opposite to the infusion.
- Be aware that as the adverse reaction to antivenom resolves, adrenaline requirements will fall, the blood pressure will rise and the adrenaline infusion rate will need to be reduced.
- Consider nebulised salbutamol for bronchospasm, nebulised adrenaline for upper airway obstruction, and I.V. atropine for severe bradycardia.
- Seek advice urgently from local/regional ED Consultant and/or ICU Consultant.
- In most cases, once the adverse reaction is controlled, cautious reintroduction of antivenom is possible. [Note: A patient requiring antivenom therapy prior to the adverse reaction will likely continue to require antivenom after the adverse reaction].

Antivenom therapy: Commonest mistakes³

Some of the most common mistakes relating to antivenom therapy are highlighted below.

- Failure to use antivenom when clearly indicated.
- Giving antivenom unnecessarily, i.e. when there are no clear clinical indicators of significant systemic envenoming.
- Choosing the wrong antivenom.
- Choosing the wrong dose.
- Antivenom given too late.
- Administering antivenom by the wrong route.
- Failing to prepare for an adverse reaction.
- Failing to inform the patient about serum sickness.

Complications of antivenom therapy

Essentially, antivenom is whole or modified antibody from an animal. It is obtained by hyperimmunising the animal against a particular venom or group of venoms. The IgG antibody from blood plasma is used, and typically, is fractionated to the F(ab')₂ fragment of IgG. bioCSL's Stonefish Antivenom is derived from horse plasma, with the IgG antibody fractionated to the F(ab')₂ fragment.^{7,25}

When making antivenoms, bioCSL undertakes assiduous efforts to filter and discard any extraneous blood components and contaminants. Nevertheless even high-quality antivenoms will cause adverse reactions in some patients. The clinically important adverse reactions can be subdivided into 'early' and 'late'.

Early and late adverse reactions to antivenom

Early reactions are those that occur immediately after commencing antivenom therapy or within the first few hours (Table 44). Late adverse reactions may occur several days later (Table 45 on page 294).

Table 44. Early adverse reactions to antivenom^{3,7,12,14,16}

| | |
|---|---|
| Rash | Localised or generalised erythematous, sometimes pruritic rash. May occur as an isolated and generally trivial adverse reaction or it may herald the onset of a more severe adverse reaction |
| Pyrexia | Febrile reactions may potentially occur – although there have been no reported cases of pyrexia with bioCSL's Stonefish Antivenom ⁷ |
| Anaphylactic/ anaphylactoid reaction | <p>This is a type of potentially life-threatening reaction. Therefore, antivenom should never be given until measures to manage such a reaction are in place. This is why the use of antivenom outside a hospital environment is strongly discouraged. Note however, when antivenom is clinically indicated, it should never be withheld for fear of an adverse reaction. Seek expert advice</p> <p>Anaphylaxis may be preceded by a localised or generalised rash, sometimes first seen in the axilla or lower abdomen, proceeding to hypotension and/or bronchospasm</p> <p>Look for additional warning signs of anaphylaxis in children (see Table 43 on page 288)</p> |

Specific treatment for stonefish stings: Stonefish antivenom

Table 45. Late adverse reactions to antivenom^{3,7,14}

The principal late adverse reaction to antivenom is serum sickness, a type III delayed hypersensitivity reaction, which most commonly presents 4-14 days post exposure

Serum sickness is characterised by a flu-like illness with fever, joint and muscle pain and general malaise, often preceded by or associated with a maculopapular or erythema multiformae-like rash

Serum sickness rates often increase as the antigen load increases. Therefore, it is more likely to occur in patients who have had a high volume load of antivenom

Every patient who receives antivenom should be advised of the symptoms of serum sickness and told to seek medical care if these symptoms arise after discharge from hospital

Serum sickness is more likely to occur in patients who have had a high volume load of antivenom

Management of serum sickness^{3,14,26}

- While serum sickness can be a mild and self-limited disease, it can be distressing for patients and early diagnosis and treatment is advisable.
- There is a diversity of opinion about the approach to treatment for various causes of serum sickness (i.e. not just antivenom) – which may involve first-line use of antihistamines or oral steroids. Serum sickness post antivenom therapy is usually managed with a short course of oral corticosteroids.
- If uncertain about the treatment approach, consult with a clinical immunologist.
- Some experts suggest prescribing a week-long course of oral prednisolone commencing immediately after antivenom, for all patients who have received more than 25 mL of antivenom. This treatment has not yet been tested through clinical trials.

Stonefish: Overview

The stonefish is a rather unattractive squat fish with a most irregular 'skin' accounting for its superb camouflage as it sits on old coral or debris. A series of erectile dorsal spines with associated venom glands are the stonefish's means of envenoming potential predators.²

Distribution²

Stonefish are found throughout northern Australian waters (Brisbane onwards to the north and around to approximately 500 km north of Perth), especially in association with coral reefs. Mostly, they are encountered in shallow water, where, owing to their excellent camouflage, they may be stepped on by accident, or picked up by the unwary.

Venom composition²

The venom is multicomponent, with neurotoxic, myotoxic, cardiotoxic and cytotoxic effects in experimental animals. A pre-synaptic neurotoxin has been described from the venom. While the venom can cause haemolysis, this is not a significant problem in stonefish envenoming. The venom may also cause vascular leakage.

Clinical effects^{2,3,27}

Most stonefish stings occur when a person steps on the fish, or less commonly, when a stonefish is picked up incautiously. A single death believed to be due to stonefish sting has been recorded in Australia, but deaths are more frequently reported for some stonefish in the Indo-Pacific. Instant and severe pain is a feature of these stings, followed by local swelling, which may be marked, along with tenderness and a blue discolouration of skin surrounding the sting penetration site. Dizziness, nausea, hypotension, collapse, cyanosis and pulmonary oedema have been described, though are uncommon to rare. Tissue necrosis at the sting site can occur but also is uncommon to rare.



Stonefish.

Section 8

Other marine animals

- Stingrays
- Blue ringed octopus
- Cone snails

In this section

Stingray stings

Background 299

Management 300

Blue ringed octopus,

Hapalochlaena spp 301

Cone snails, *Conus* spp 302

Stingray stings

Background¹⁻³

- There are numerous species of stingrays in Australian coastal waters, not all of which have venom-enshrouded tail stings.
- The primitive venom gland around a stingray tail delivers venom during the act of stinging.
- The venom principally causes local pain, and sometimes, tissue destruction. In rare cases of major stingray stings, patients may exhibit systemic effects that theoretically could include one or more of nausea, vomiting, abdominal pain, sweating, local muscle cramps, collapse.¹
- However, it is the mechanical damage caused by the whipping effect of the stingray tail that is generally of greater medical significance.²
- In most cases, the person steps on or close to a stingray, usually in shallow sandy-bottomed water, or tries to grab a stranded stingray, resulting in the tail being whipped around and the spine penetrating and sometimes, lacerating the skin.
- Occasionally, the laceration can be deep and can sever vital structures such as tendons, nerves, and blood vessels.¹
- Severed blood vessels (particularly if the injury occurs while still in the water) have the potential to cause massive blood loss and even exsanguination.¹
- Rarely, the spine may penetrate the abdomen or chest where it can cause severe and even lethal injury. In particular, penetrations of the chest wall in the cardiac region may result in either immediate death through laceration of the heart, or delayed death where the venom causes necrosis in and around the heart resulting in later cardiac rupture. Any wound to the chest or abdomen should be treated as a medical emergency and requires hospital assessment.¹
- Stingrays are found in coastal waters throughout Australia.³



Stingray.

Stingray stings

Management^{1,2,4,5}

- First aid involves removing the person from the water, attending to ABC as required, followed by hot water immersion (as for fish stings) with the added need to provide appropriate wound care.^{1,2,4,5}
 - Immerse the stung hand or foot in water no hotter than 45°C for up to 20 minutes, remove and re-immerses if pain persists.
 - Repeat the cycle for up to 2 hours.
- In cases with major bleeding, priority should be given to staunch the bleeding (with the application of proximal pressure), and to resuscitating the patient if required.¹
- Any wound to the chest or abdomen requires investigation to exclude a significant penetrating injury.¹
- Penetrating barbs **should not** be removed pre-hospital, especially those affecting the chest and abdomen.²
- Due to the potential for both secondary infection and venom-induced local necrosis, lacerated wounds should be carefully and vigorously inspected for foreign bodies and cleaned, and where appropriate, debrided, and allowed to heal by secondary intention.¹
- Consider prophylactic antibiotics targeting marine organisms.² Also ensure tetanus prophylaxis.¹
- The severe local pain may require parenteral analgesia or regional nerve block.^{1,2}
- There is no antivenom for stingray envenoming.
- Any stingray wound to the trunk, even in the absence of apparently significant injury, should be treated as a medical emergency.²



Stingray wound.

Blue ringed octopus, *Hapalochlaena* spp

Background and management^{2,6-8}

- There are seven species of blue ringed octopus in Australia (*Hapalochlaena fasciata* and *Hapalochlaena maculosa* and 5 unnamed species)⁶ which are commonly found in all Australian coastal waters.
- The animal is small, and when alarmed, develops vivid blue to purple rings on the tentacles and body.
- Blue ringed octopus saliva contains a potent neurotoxin, tetrodotoxin.⁷
- Bites virtually never occur unless the octopus is removed from the water and placed on exposed skin. The bite may not be painful.⁷
- Not every bite results in systemic envenoming, but flaccid paralysis, including respiratory paralysis, may develop quickly in severe cases, requiring urgent respiratory support on the beach. Typically however, the envenoming is less severe, with tingling around the mouth and mild weakness.⁷
- Where severe paralysis occurs it is likely to be of short duration, measured in a few hours.⁷
- First aid is directed towards providing basic life support, maintaining vital functions, particularly, giving respiratory support, and rapid application of PBI first aid and seeking urgent medical assistance.^{7,8}
- PBI first aid if applied should only be removed once the patient is in a health facility equipped to provide immediate full respiratory support and cardiopulmonary resuscitation.²
- There is no antivenom for blue ringed octopus envenoming.
- Once in hospital, the patient requires supportive care only and generally only for a few hours as envenoming can reverse within hours or up to 1-2 days in some circumstances. [The venom toxins while rapid acting, have relatively short-lived effects].^{2,7}



Blue ringed octopus.

Cone snails, *Conus* spp

Background and management^{2,8,9}

- Only a few cone snails (from tropical waters) are known to be hazardous to humans.
- Their venom contains a complex mixture of varied toxins, notably the conopeptides (previously known as conotoxins).⁹
- Envenoming occurs when the shell is picked up and the snail fires a venom-coated harpoon dart into the skin, which may result in local pain, potentially followed by systemic envenoming, with progressive paralysis and collapse requiring respiratory support.⁹
- First aid is directed towards maintaining vital functions, particularly, the provision of respiratory support and applying PBI first aid.^{8,9}
- If applied, PBI first aid should only be removed once the patient is in a health facility equipped to provide immediate full respiratory support and cardiopulmonary resuscitation.²
- Treatment is supportive, particularly respiration, if impaired.⁹
- There is no antivenom for cone snail envenoming.

Envenoming occurs when the shell is picked up, and may result in local pain, potentially followed by systemic envenoming, with progressive paralysis and collapse



Conus consors.



Conus geographus.

Section 9

Antivenoms: Practical information

In this section

bioCSL's antivenoms

Specificity of bioCSL's antivenoms 305

bioCSL's antivenoms:

Vial presentations 306

bioCSL's antivenoms: Initial dose

Snake antivenoms 307

Spider antivenoms 312

Marine antivenoms 313

Stocking antivenoms in hospitals

Principles of stocking antivenoms 314

bioCSL's antivenoms

Australia is home to ten of the world's most venomous creatures.¹ bioCSL is the only Australian company that manufactures antivenoms to protect against some of our venomous snakes, spiders, jellyfish and stonefish. Antivenoms have been manufactured locally by CSL since the 1930s.

All bioCSL snake antivenoms as well as bioCSL's Stonefish Antivenom and Red Back Spider Antivenom are derived from horses. bioCSL's Funnel Web Spider Antivenom is derived from rabbit while Box Jellyfish Antivenom is derived from sheep. The immunoglobulin in bioCSL's antivenoms is either whole IgG (Funnel Web Spider Antivenom and Box Jellyfish Antivenom) or the F(ab')₂ fragment of IgG (all other bioCSL antivenoms).²⁻¹³

Specificity of bioCSL's antivenoms

Snake antivenoms

Each of the 5 monovalent snake antivenoms (bioCSL's Brown Snake Antivenom; Tiger Snake Antivenom; Black Snake Antivenom; Death Adder Antivenom; and Taipan Antivenom) may contain varied levels of the other snake antivenoms¹⁴ due to the nature of manufacturing processes. Importantly however, the stated minimum neutralising potency applies only to the 'monovalent' snake group listed on the product.²⁻⁶ No assumptions should be made about non-listed neutralising potency.

Additionally, bioCSL manufactures Polyvalent Snake Antivenom, which is prepared with neutralising potency covering all five snake venom immunotypes predominating in land snakes in Australia and PNG.⁷

Other antivenoms

For all other antivenoms, it is imperative to use only the antivenom specific for that group of organisms – e.g. bioCSL's Red Back Spider Antivenom is indicated for the treatment of envenoming from red back spider bite only¹⁰ and **should not** be used to treat envenoming from funnel web spider bites.

Before administering any bioCSL antivenom, please see the relevant Product Information (available at www.bio CSL.com.au/PI) and ensure adrenaline is prepared and resuscitation facilities are on hand in the event of an anaphylactic reaction to antivenom therapy.

bioCSL's antivenoms: Vial presentations



bioCSL's snake antivenoms.



All bioCSL antivenoms: Snakes; funnel web spider; red back spider; box jellyfish; stonefish.

bioCSL's antivenoms: Initial dose

The volume of antivenom is potency dependent. Consequently, antivenom volume per vial may vary from batch to batch.^{2-6,8,10-12}

Snake antivenoms

bioCSL's Brown Snake Antivenom – 1,000 units per vial

| Animal covered | Initial dose |
|--|---|
| Brown snakes (<i>Pseudonaja</i> spp) | 1 or 2 vials ^{2,15-18} The PI recommends the use of 1 vial and advises that the actual amount needed in clinical practice may be more ² The expert panel recommends 2 vials as a starting dose ¹⁶ Please see additional information below |

If Brown Snake Antivenom is unavailable or if the amount available is inadequate, Polyvalent Snake Antivenom may be substituted at the intended dose of Brown Snake Antivenom [16].

- Brown snake venom contains varied toxins including procoagulants, pre-synaptic neurotoxins and post-synaptic neurotoxins.¹⁹
- Additionally, venom yields from brown snakes can be higher than anticipated.²⁰
- The expert panel involved in producing this handbook believes it is important to maximise the potential for neutralising all venom components in all cases, including those where unusually large amounts of venom have been injected, and therefore advises the use of 2 vials as an initial dose.¹⁶

Note: 2 vials also is the current consensus recommendation of experts such as the WA Toxicology Service and the Emergency Medicine Expert Group.^{17,18}

- The panel recognises that the initial dose of 1 vial will be sufficient in some cases. However, at the time of administering initial antivenom, it may not be possible to differentiate these cases from those that would benefit from a higher dose.¹⁶
- The treating clinician will make the final decision regarding dosing, based on the circumstances of the individual case.¹⁶

bioCSL's antivenoms: Initial dose

bioCSL's Tiger Snake Antivenom – 3,000 units per vial

1. bioCSL's Tiger Snake Antivenom for bites by snakes in the tiger snake group

| Animal covered | Initial dose |
|---|--|
| Tiger snakes (<i>Notechis</i> spp) | 1 or 2 vials ^{6,16-18,21} |
| Tasmanian tiger snakes | At least 2 vials ^{16,22,23} |
| Chappell Island tiger snakes | At least 4 vials ^{16,22,23} |
| Rough scaled snake (<i>Tropidechis carinatus</i>) | 1 or 2 vials ^{6,16-18} |
| Copperheads (<i>Austrelaps</i> spp) | 1 or 2 vials ^{6,16-18,23} |
| Pale headed, broad headed and Stephens' banded snakes (<i>Hoplocephalus</i> spp) | 1 or 2 vials ^{6,16-18} |
| | For the snakes covered by Tiger Snake Antivenom, the Product Information recommends an initial dose of 1 vial and advises that the actual amount needed in clinical practice may be more ⁶ The expert panel recommends an initial dose of 2 vials ¹⁶ Please see additional information below |

If Tiger Snake Antivenom is unavailable or if the amount available is inadequate, Polyvalent Snake Antivenom may be substituted at the intended dose of Tiger Snake Antivenom [16].

- Venom yields from tiger snakes can be higher than anticipated.²⁰
- The expert panel involved in producing this handbook believes it is important to maximise the potential for neutralising all venom components in all cases, including those where unusually large amounts of venom have been injected, and therefore advises the use of 2 vials as an initial dose (with higher doses recommended for Tasmanian and Chappell Island tiger snakes).¹⁶

Note: 2 vials also is the current consensus recommendation of experts such as the WA Toxicology Service and the Emergency Medicine Expert Group.^{17,18}

.... continued

- The higher initial doses recommended for Tasmanian and Chappell Island tiger snakes are based on past clinical experience and the significantly higher quantities of venom likely to be injected by these snakes.²⁴ These recommendations date back over many decades, to advice in publications such as the CSL Medical Handbook (1979).^{22,23}
- The panel recognises that an initial dose of 1 vial will be sufficient in some cases. However, at the time of administering initial antivenom, it may not be possible to differentiate these cases from those that would benefit from a higher dose.¹⁶
- The treating clinician will make the final decision regarding dosing, based on the circumstances of the individual case.¹⁶

2. bioCSL's Tiger Snake Antivenom – for bites by red bellied black snakes and blue bellied/spotted black snakes

| Animal covered | Initial dose |
|---|--|
| Red bellied black snakes and blue bellied/spotted black snakes (<i>Pseudechis</i> spp) | 1 vial ^{6,23} Tiger Snake Antivenom is the preferred monovalent antivenom for managing envenoming from red bellied black snakes and blue bellied black snakes ^{3,6} |

Tiger Snake Antivenom is active against venom from red bellied black snakes and blue bellied/spotted black snakes (*Pseudechis* spp), and Tiger Snake Antivenom is successfully used in the clinical practice setting for the treatment of envenoming by these black snakes [6,16,25,26].

If Tiger Snake Antivenom is unavailable, Black Snake Antivenom or Polyvalent Snake Antivenom may be used at the intended dose of Tiger Snake Antivenom [3,16,23].

Before administering any snake antivenom please refer to pages 111-134 for detailed information regarding indications, preparatory procedures, and administration of antivenom therapy.

bioCSL's antivenoms: Initial dose

bioCSL's Black Snake Antivenom – 18,000 units per vial

| Animal covered | Initial dose |
|--|--|
| Mulga snakes (<i>Pseudechis australis</i> ; <i>Pseudechis butleri</i> ; <i>Pseudechis weigeli</i>) | 1 vial ^{3,16,17} |
| Collett's snakes (<i>Pseudechis colletti</i>) | 1 vial ^{17,18,27} |
| Red bellied black snakes and blue bellied/spotted black snakes (<i>Pseudechis</i> spp) | 1 vial ^{3,23} Tiger Snake Antivenom is preferred for envenoming from bites by red bellied black snakes or blue bellied black snakes. ^{3,28} However, if there is major myolysis (CK > 5000 IU/L), some experts suggest the use of 1 vial of Black Snake Antivenom instead ^{16,28} (although, as per the PI, a recent study indicates that 1 vial of Tiger Snake Antivenom may be sufficient) ²⁶ |

If Black Snake Antivenom is unavailable or if the amount available is inadequate, Polyvalent Snake Antivenom may be substituted at the intended dose of Black Snake Antivenom [16].

Note regarding antivenom therapy for Collett's snake envenoming:

- The Product Information recommends Tiger Snake Antivenom as the preferred treatment when antivenom therapy is clinically indicated for Collett's snake bite [3,6].
- The table above differs from the Product Information by advising the use of Black Snake Antivenom for envenoming from Collett's snake bite. This advice is based on published evidence demonstrating the potential for severe envenoming from Collett's snake bite, which produces an envenoming syndrome similar to that of mulga snake bite [27]. In addition, Black Snake Antivenom was shown to be significantly more efficacious (*versus* Tiger Snake Antivenom) *in vitro*, in reversing the myotoxic effects of *Pseudechis colletti* venom [25]. Therefore, researchers advise that Black Snake Antivenom appears to be the more appropriate treatment for Collett's snake envenoming, and this is now also reflected in recent published expert consensus and guidelines [17,18,27].

bioCSL's Taipan Antivenom – 12,000 units per vial

| Animal covered | Initial dose |
|---------------------------------|--|
| Taipans (<i>Oxyuranus</i> spp) | 1 vial ⁵ In severely envenomed cases consider using an initial dose of at least 3 vials ⁵ |

If Taipan Antivenom is unavailable or if the amount available is inadequate, Polyvalent Snake Antivenom may be used at the intended dose of Taipan Antivenom [16].

bioCSL's Death Adder Antivenom – 6,000 units per vial

| Animal covered | Initial dose |
|---|--|
| Death adders (<i>Acanthophis</i> spp) | 1 vial ⁴ Increased doses may be required in severe cases ^{4,16} |

If Death Adder Antivenom is unavailable or if the amount available is inadequate, Polyvalent Snake Antivenom may be substituted at the intended dose of Death Adder Antivenom [16].

bioCSL's Polyvalent Snake Antivenom – 40,000 units per vial

| Animal covered | Initial dose |
|--|--|
| All land snakes listed on page 307-311, but not sea snakes | The PI advises the use of 1 vial and states that the actual amount needed in clinical practice may be more. ⁷ The expert panel recommends using Polyvalent Snake Antivenom at the same dose as intended for the corresponding monovalent antivenom ¹⁶ |

bioCSL's Sea Snake Antivenom – 1,000 units per vial

| Animal covered | Initial dose |
|--|---|
| Sea snakes (numerous genera and species) | 1 vial ⁸ 3-4 vials for severe envenoming ⁸ |

Polyvalent Snake Antivenom is not designed for the purpose of treating envenoming resulting from sea snake bite and efficacy is unknown.

If Sea Snake Antivenom is unavailable, it is unclear if any other snake antivenom might be substituted. In the past Tiger Snake Antivenom (TSAV) was recommended as a substitute for Sea Snake Antivenom (SSAV), with a dose ratio of 3 vials of TSAV for each vial of SSAV that might have been used. However, due to the nature of antivenom manufacturing processes, the results of earlier research cannot be extrapolated to the current TSAV product. Hence the efficacy of the current Tiger Snake Antivenom against sea snake venoms, remains unknown [16].

However, in desperate circumstances, if SSAV were unavailable, TSAV could be considered for sea snake envenoming [8]. Alternatively, if TSAV is also unavailable, Polyvalent Snake Antivenom could be considered as it contains tiger snake antivenom, but the clinicians and the patient would need to clearly understand the uncertainty regarding antivenom efficacy in this instance, and the known risks of using any antivenom product. In this situation always seek expert advice [16].

Before administering any snake antivenom please refer to pages 111-134 for detailed information regarding indications, preparatory procedures, and administration of antivenom therapy.

bioCSL's antivenoms: Initial dose

Spider antivenoms

bioCSL's Funnel Web Spider Antivenom – 125 units per vial

| Animal covered | Initial dose |
|--|--|
| Funnel web spiders (numerous species) | 2 vials ⁹ 4 vials for severe envenoming ²⁹⁻³² |

The Product Information does not specifically list the recommendation for severe cases. The advice regarding a higher initial dose of 4 vials for severe cases is based on published expert clinical experience and snakebite management guidelines [29-32].

bioCSL's Red Back Spider Antivenom – 500 units per vial

| Animal covered | Initial dose |
|------------------|--|
| Red back spiders | 2 vials ^{17,18,30,31} The PI recommends 1 vial, which will neutralise 5mg of venom <i>in vitro</i> . The actual amount needed in clinical practice may be more ¹⁰ Current treatment guidelines recommend 2 vials as a starting dose ^{17,18,30,31} |

Marine antivenoms

bioCSL's Stonefish Antivenom – 2,000 units per vial

| Animal covered | Initial dose |
|----------------|--|
| Stonefish | 1 vial (1-2 puncture wounds) ¹² 2 vials (3-4 puncture wounds) ¹² 3 vials (5 or more puncture wounds) ¹² |

bioCSL's Box Jellyfish Antivenom – 20,000 units per vial

| Animal covered | Initial dose |
|----------------|--|
| Box jellyfish | 1 vial (for severe local pain unresponsive to parenteral narcotic analgesia and I.V. magnesium sulphate) ³³ Minimum 1 vial (for cardiac failure/arrhythmia or if unconscious) ³³ 1-3 vials (for life-threatening cardiac or respiratory failure, or if the patient is in cardiac arrest. If in cardiac arrest consider the maximal dose of 6 vials undiluted as a rapid I.V. push) ^{16,17,33} |

Note: The antivenom therapy recommendations in the above table differ from the Product Information for bioCSL's Box Jellyfish Antivenom. The PI recommends the use of 1 vial of diluted Box Jellyfish Antivenom I.V. (or if the I.V. route is impractical, 3 vials undiluted given I.M. at 3 different sites) [11].

The updated antivenom therapy recommendations in the table above represent information within the Northern Territory revised protocol for managing *C fleckeri* envenoming, with input from this handbook's expert panel [16,33].

The Northern Territory revised protocol was written for managing severe *C fleckeri* envenoming wherein antivenom therapy may save a life – i.e. a severe sting with cardiorespiratory arrest near a health centre or hospital, where immediate resuscitation and rapid use of large volumes of I.V. antivenom is possible [33].

Therefore, the updated antivenom therapy recommendations devised by experts differ from the Product Information for bioCSL's Box Jellyfish Antivenom.

Before administering spider antivenoms, bioCSL's Stonefish Antivenom, or bioCSL's Box Jellyfish Antivenom, please refer to relevant sections of this handbook for detailed information regarding indications, preparatory procedures, and administration of antivenom therapy. For spider antivenoms see pages 199-216. For bioCSL's Stonefish Antivenom go to pages 284-294. For bioCSL's Box Jellyfish Antivenom go to pages 254-263.

Stocking antivenoms in hospitals

Principles of stocking antivenoms¹⁶

It is beyond the scope of this handbook to provide the highly detailed and selective advice required to cover every hospital in Australia.

Some States (SA, NSW, WA) have undertaken detailed reviews of antivenom stock requirements on an individual hospital basis, and these State-based guidelines should be followed by hospitals. In general, the guidelines are based on a multifactorial assessment of each hospital, considering the geographic location, range of dangerous venomous fauna in the hinterland, past rate of venomous bites, past rate of antivenom usage, level of medical, nursing and laboratory resources available, and distance and time to the nearest major/regional hospital.

For small, poorly-resourced rural hospitals situated close to well-resourced regional/base hospitals, it may be better to transfer all suspected cases of envenoming, and therefore, antivenom may not be required in these small hospitals.

Conversely, isolated small rural hospitals that are likely to experience significant delays in transferring patients may be best advised to stock appropriate antivenoms.

.... continued

For small, poorly-resourced rural hospitals situated close to well-resourced regional/base hospitals, it may be better to transfer all suspected cases of envenoming Conversely, isolated small rural hospitals that are likely to experience significant delays in transferring patients, may be best advised to stock appropriate antivenoms

Principles of stocking antivenoms¹⁶ ... cont'd

In some settings, such as a small remote country hospital, with regards to venomous snakebite, it may be practical as well as economical to stock bioCSL's Polyvalent Snake Antivenom and perhaps the most needed monovalent snake antivenom, rather than stocking vials of all types of monovalent antivenom.

Ideally, hospitals should at a minimum hold sufficient quantities of most needed antivenom(s) to cover 1-2 serious cases each (for remote/isolated hospitals, at least 2 cases), with access to other regional centres established in the event of additional need. Additionally, ensure there is access to sufficient antivenom for at least one case of bite by less common, but potentially lethal, local snake species.

Hospital pharmacists should review with doctors, the hospital's past usage requirements – including stocks and species involved. Consultation with nearby hospitals regarding their experience and usage patterns is advisable. In this manner, a regional stocking plan could be developed that balances coverage requirements with frequency of use. Importantly, each hospital should consult the relevant State health authorities to seek guidance, and where appropriate, this may include consulting an expert clinical toxinologist for advice.

Ideally, hospitals should at a minimum hold sufficient quantities of most needed antivenom(s) to cover 1-2 serious cases each (for remote/isolated hospitals, at least 2 cases), with access to other regional centres established in the event of additional need

References

References

Section 1

1. A/Prof Julian White personal communication.
2. Isbister GK, Currie BJ. *Emerg Med (Fremantle)*. 2003;15(2):160-169.
3. Jelinek GA, Breheny FX. *Med J Aust*. 1990;53:658-661.
4. Hughes A. *Emerg Med (Fremantle)*. 2003;5:511-517.
5. Isbister GK, Gray MR. *Toxicon*. 2004;43:133-40.
6. Ireland G, *et al*. *Med J Aust*. 2010;193:285-90.
7. Sutherland SK, *et al*. *Pathology*. 1981;13:13-27.
8. White J. Clinical toxicology of snakebite in Australia and New Guinea. In: Meier J & White J, editors. *Handbook of Clinical Toxicology of Animal Venoms and Poisons*. Informa Healthcare USA, Inc, New York, USA. 2008. p. 595-617.
9. White J. Clinical toxicology of tick bites. In: Meier J & White J, editors. *Handbook of Clinical Toxicology of Animal Venoms and Poisons*. Informa Healthcare USA, Inc, New York, USA. 2008. p. 191-203.
10. White J. Clinical toxicology of blue ringed octopus bites. In: Meier J & White J, editors. *Handbook of Clinical Toxicology of Animal Venoms and Poisons*. Informa Healthcare USA, Inc, New York, USA. 2008. p. 171-75.
11. Isbister GK, Gray MR. *Med J Aust*. 2003;179:88-91.
12. Flecker H. *Med J Aust*. 1952;2:89-91.
13. Little M, Mulcahy R. *Med J Aust*. 1998;169:638-41.
14. Isbister GK, *et al*. *Med J Aust*. 2005;182:407-11.
15. White J. Clinical toxicology of *Conus* snail stings. In: Meier J & White J, editors. *Handbook of Clinical Toxicology of Animal Venoms and Poisons*. Informa Healthcare USA, Inc, New York, USA. 2008. p. 117-27.
16. Cameron RJ, Rowley MP. *Med J Aust*. 1999;171:334-35.
17. Currie BJ, Jacups SP. *Med J Aust*. 2005;183:631-36.
18. White J. Poisonous and venomous animals: the physician's view. In: Meier J & White J, editors. *Handbook of Clinical Toxicology of Animal Venoms and Poisons*. Informa Healthcare USA, Inc, New York, USA. 2008.
19. bioCSL's Snake Venom Detection Kit (SVDK) Product Leaflet, February 2007.
20. Currie BJ. *Emerg Med Australas*. 2004;16:384-386.
21. Mead HJ, Jelinek GA. *Med J Aust*. 1996;164:467-470.
22. CSL Brown Snake Antivenom Product Information, 14 December 2009.
23. CSL Black Snake Antivenom Product Information, 14 December 2009.
24. CSL Death Adder Antivenom Product Information, 14 December 2009.
25. CSL Taipan Antivenom Product Information, 15 December 2009.
26. CSL Tiger Snake Antivenom Product Information, 21 January 2010.
27. CSL Polyvalent Snake Antivenom Product Information, 20 September 2004.
28. CSL Sea Snake Antivenom Product Information, 15 December 2009.
29. CSL Funnel Web Spider Antivenom Product Information, 11 January 2010.

30. CSL Red Back Spider Antivenom Product Information, 15 December 2009.
31. CSL Box Jellyfish Antivenom Product Information, 14 December 2009.
32. CSL Stonefish Antivenom Product Information, 13 April 2011.
33. O'Leary MA, Isbister GK. *Toxicol* 2009;54:192-95.
34. Australian Resuscitation Council. Guideline 13.7 – Medication or fluids for the resuscitation of the newborn infant. Accessed 1 July 2012 from www.resus.org.au
35. Dr Mark Little personal communication.
36. Murray L, *et al*. Antivenoms. In: *Toxicology Handbook*. Second edition. Elsevier Australia, NSW, Australia. 2011. p. 470-89.

Section 2

1. A/Prof Julian White personal communication.
2. Australian Resuscitation Council. Guideline 8: CPR. Accessed 1 July 2012 from www.resus.org.au
3. Australian Resuscitation Council. Guideline 3: Unconsciousness. Accessed 1 July 2012 from www.resus.org.au
4. Australian Resuscitation Council. Guideline 4: Airway. Accessed 1 July 2012 from www.resus.org.au
5. Australian Resuscitation Council. Guideline 5: Breathing. Accessed 1 July 2012 from www.resus.org.au
6. Australian Resuscitation Council. Guideline 6: Compressions. Accessed 1 July 2012 from www.resus.org.au
7. Australian Resuscitation Council. Guideline 9.4.8. Envenomation – Pressure Immobilisation Technique. Accessed 1 July 2012 from www.resus.org.au
8. Australian Resuscitation Council. Guideline 9.4.5. Envenomation – Jellyfish stings. Accessed 1 July 2012 from www.resus.org.au
9. Seymour J, *et al*. *Toxicol*. 2002;40:1503-5.
10. Flecker H, *Med J Aust*. 1952;2:89–91.
11. Barnes JH. *Med J Aust*. 1964;14:897-904.
12. Little M, Mulcahy R. *Med J Aust*. 1998;169:638-41.
13. Australian Resuscitation Council. Guideline 9.4.7. Envenomation – Fish stings. Accessed 1 July 2012 from www.resus.org.au
14. Loten C, *et al*. *Med J Aust*. 2006;184:329-33.
15. Taylor JG. *Med J Aust*. 2007;186:43
16. Australian Resuscitation Council. Guideline 9.4.2: Envenomation – Spiderbite. Accessed 1 July 2012 from www.resus.org.au
17. Sutherland SK, *et al*. *Lancet*. 1979;1:183-5.
18. Sutherland SK, *et al*. *Pathology*. 1981;13:13-27.
19. Canale E, *et al*. *Emerg Med Australas*. 2009;21:184-90.
20. Sutherland S, Tibballs J. First aid for bites and stings. In: *Australian animal toxins: The creatures, their toxins and care of the poisoned patient*. Second edition. Oxford University Press, 5th Melbourne, Australia. 2001. p. 28-47.

References

21. Currie BJ, *et al.* 2008;20:267-70.
 22. Hartwick R, *et al.* Med J Aust. 1980;1(1):15-20.
 23. Atkinson PRT, *et al.* Emerg Med J. 2006;23:503-8.
 24. Dr Mark Little personal communication.
- Section 3**
1. A/Prof Julian White personal communication.
 2. White J. Clinical toxicology of snakebite in Australia and New Guinea. In: Meier J & White J, editors. Handbook of Clinical Toxicology of Animal Venoms and Poisons. Informa Healthcare USA, Inc, New York, USA. 2008. p. 595-617.
 3. Isbister GK, *et al.* Intern Med J. 2007;37:523-8.
 4. Currie BJ. Med J Aust. 2004;181:693-7.
 5. White J. Toxicon. 2005; 45:951-67.
 6. Australian Resuscitation Council. Guideline 9.4.8. Envenomation – Pressure Immobilisation Technique. Accessed 1 July 2012 from www.resus.org.au
 7. Murray L, *et al.* Specific Considerations. In: Toxicology Handbook. Second edition. Elsevier Australia, NSW, Australia. 2011. p. 36-126.
 8. Dr Mark Little personal communication.
 9. Sutherland SK, *et al.* Pathology. 1981;13:13-27.
 10. Ireland G, *et al.* Med J Aust. 2010;193:285-90.
 11. bioCSL's Snake Venom Detection Kit (SVDK) Product Leaflet, February 2007.
 12. Mead HJ, Jelinek GA. Med J Aust. 1996;164:467-70.
 13. Isbister GK. Snakebite. In: Cameron P, *et al.*, eds. Textbook of adult emergency medicine. Third edition. Churchill Livingstone, Sydney, Australia. 2009. p. 983-9.
 14. White J. Envenomation Prevention and Treatment in Australia. In: Mackessy SP, ed. Handbook of venoms and toxins of reptiles. CRC Press Boca Raton FL, USA. 2010. p. 423-51.
 15. White J. Clinical toxicology of sea snake bites. In: Meier J & White J, editors. Handbook of Clinical Toxicology of Animal Venoms and Poisons. Informa Healthcare USA, Inc, New York, USA. 2008. p. 159-69.
 16. Churchman A, *et al.* Med J Aust. 2010;193:696-700.
 17. Isbister GK, *et al.* QJM. 2006;99:109-15.
 18. Currie BJ. Emerg Med Australas. 2004;16:384-6.
 19. Jelinek GA, *et al.* Emerg Med Australas. 2004;16:459-64.
 20. Sano-Martins IS, *et al.* Toxicon. 1994;32:1045-50.
 21. Isbister GK, *et al.* J Thromb Haemost. 2010;8:2504-13.
 22. Isbister GK, *et al.* Med J Aust. 2012;197:73-7.
 23. CSL Brown Snake Antivenom Product Information, 14 December 2009.
 24. CSL Tiger Snake Antivenom Product Information, 21 January 2010.
 25. CSL Black Snake Antivenom Product Information, 14 December 2009.
 26. CSL Death Adder Antivenom Product Information, 14 December 2009.

27. CSL Taipan Antivenom Product Information, 28. CSL Polyvalent Snake Antivenom Product Information, 20 September 2004.
29. CSL Sea Snake Antivenom Product Information, 15 December 2009.
30. Dr Ken Winkel personal communication.
31. Williams V, White J. *Toxicon*. 1990;28:1351-4.
32. White J. Snakebite & Spiderbite Management Guidelines South Australia. 2006. Department of Health, Adelaide, South Australia.
33. Isbister GK, Currie BJ. *Emerg Med (Fremantle)*. 2003;15:160-9.
34. Jelinek GA, Breheny FX. *Med J Aust*. 1990;153:658-61.
35. Jansen M, *et al*. *Med J Aust*. 2007;186:41-2.
36. Hughes A. *Emerg Med (Fremantle)*. 2003;15:511-7.
37. Currie BJ, *et al*. *Med J Aust*. 1988;148:522-5.
38. Little M, Pereira P. *Emerg Med*. 2000;12:241-5.
39. Hudson BJ. *Aust N Z J Med*. 1988;18:792-4.
40. Yeung JM, *et al*. *Med J Aust*. 2004;181:703-5.
41. Isbister GK, *et al*. *Toxicon*. 2007;49:57-67.
42. Murray L, *et al*. Antivenoms. In: *Toxicology Handbook*. Second edition. Elsevier Australia, NSW, Australia. 2011. p. 470-89.
43. Isbister GK, *et al*. *QJM*. 2009;102:563-8.
44. Emergency Medicine Expert Group. *Therapeutic Guidelines: Toxicology & Wilderness*. Version 1. Therapeutic Guidelines Limited, Melbourne, 2008.
45. Sutherland S, Tibballs J. Genus *Pseudonaja*, Brown Snakes. In: *Australian animal toxins: The creatures, their toxins and care of the poisoned patient*. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 99-129.
46. Mirtschin PJ, *et al*. *Ecotoxicology*. 2006;15:531-8.
47. CSL Medical Handbook. Ed: Trinca J. Contributors: Feery BJ, *et al*. Commonwealth Serum Laboratories, VIC, Australia. 1979.
48. Sutherland S, Tibballs J. Treatment of snakebite in Australia. In: *Australian animal toxins: The creatures, their toxins and care of the poisoned patient*. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 296-342.
49. Sutherland S, Tibballs J. Genus *Notechis*, Tiger Snakes. In: *Australian animal toxins: The creatures, their toxins and care of the poisoned patient*. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 74-98.
50. Ramasamy S, *et al*. *Toxicon*. 2004;44:837-45.
51. Tanos PP, *et al*. *Toxicon*. 2008;52:769-80.
52. Isbister GK, *et al*. *Pathology*. 2006;38:568-72.
53. Brown SG, *et al*. *Intensive Care Med*. 2009;35:1532-8.
54. Brown SGA. *Emerg Med Australas*. 2006;18:155-69.

References

55. Dr Simon Brown personal communication.
56. Australian Resuscitation Council. Guideline 13.7 – Medication or fluids for the resuscitation of the newborn infant. Accessed 1 July 2012 from www.resus.org.au
57. Brown SGA. *Med J Aust.* 2006;185:283-9.
58. Fan HW, *et al.* *Br Med J.* 1999;318:1451-3.
59. Gawarammana IB, *et al.* *Med J Aust.* 2004;180:20-3.
60. Arunanth S, Hertzberg SR. *Med J Aust.* 1998;169:257-8.
61. Williams DJ, *et al.* *Toxicol.* 2007;49:780-92.
62. Premawardhena AP, *et al.* *Br Med J.* 1999; 318:1041-3.
63. Soh SY, Rutherford G. *Trop Pediatr.* 2006;52:155-7.
64. Dassanayake AS, *et al.* *Ceylon Med J.* 2002;47:48-9.
65. Isbister GK, *et al.* *Med J Aust.* 2008;188:473-6.
66. Keiran Ragas, bioCSL Pty Ltd, Production Manager, Products of National Significance, personal communication Sept 2012.
67. Nocera A, *et al.* *Med J Aust.* 1998;168:69-71.
68. White J. *Intensive Care Med.* 2009;35:1503-4.
69. Sutherland S, Tibballs J. Genus *Austrelaps*, the Copperheads. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p.130-6.
70. Sutherland S, Tibballs J. *Trophidechis carinatus*, the Rough-scaled or Clarence River snake. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 194-202.
71. Sutherland S, Tibballs J. Genus *Hoplocephalus*. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 209-19.
72. Gan M, *et al.* *Med J Aust.* 2009;191:183-6.
73. Isbister GK, *et al.* *Toxicol.* 2011;58:634-40.
74. Hamilton Fairely N. *Med J Aust.* 1929;1:296-313.
75. Scop J, *et al.* *Anaesth Intensive Care.* 2009;37:613-8.
76. Sutherland S, Tibballs J. Genus *Pseudechis*, Black Snakes. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 137-159.
77. John Weigel personal communication July 2012.
78. Pearn J, *et al.* *Toxicol.* 2000;38:1715-29.
79. Sutherland S, Tibballs J. Genus *Acanthophis*, Death Adders. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 160-6.

80. Sutherland S, Tibballs J. Genus *Oxyuranus*, the Taipan and the Small-scaled or Fierce Snake. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 167-93.

Section 4

1. Sutherland S, Tibballs J. Spiders. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 343-82.
2. Isbister GK, Gray MR. QJM. 2002;95:723-31.
3. Isbister GK, White J. Toxicon. 2004;43:477-92
4. Isbister GK, Gray MR. Med J Aust. 2003;179:88-91
5. Sutherland S, Tibballs J. The genera *Atrax* and *Hadronyche*, Funnel web spiders. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 402-64.
6. Isbister GK, Sibbritt D. Emerg Med Australas. 2004;16:161-6.
7. Isbister GK and Gray MR. Toxicon. 2004;43:133-40.
8. A/Prof Julian White personal communication.
9. White J, *et al.* Clinical toxicology of spider bite. In: Meier J & White J, editors. Handbook of Clinical Toxicology of Animal Venoms and Poisons. Informa Healthcare USA, Inc, New York, USA. 2008. p. 259-329.
10. Sutherland SK, Trinca JC. Med J Aust. 1978;2:620-3.
11. Isbister GK, Hirst D. Toxicon. 2003;42:163-71.
12. Isbister GK, Gray MR. Med J Aust. 2003;179:199-202.
13. Isbister GK, Whyte IM. Intern Med J. 2004;34:38-44.
14. Isbister GK, *et al.* Med J Aust. 2005;182:407-11.
15. Rosengren D, *et al.* Emerg Med Australas. 2008;20:164-6.
16. Isbister GK. Med J Aust. 2004;180:225-7.
17. Dr Mark Little personal communication.
18. Sutherland S, Tibballs J. *Latrodectus hasselti*, the Red back or Jockey Spider. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 383-401.
19. Australian Resuscitation Council. Guideline 9.4.2: Envenomation – Spiderbite. Accessed 1 July 2012 from www.resus.org.au
20. CSL Funnel Web Spider Antivenom Product Information, 11 January 2010.
21. Hartman LJ, Sutherland SK. Med J Aust 1984;141:796-99.
22. NSW Health. Snakebite & Spiderbite Clinical Management Guidelines – NSW. May 2007.
23. White J. Snakebite & Spiderbite Management Guidelines South Australia. 2006. Department of Health, Adelaide, South Australia.
24. Dr John Morgan personal communication.

References

25. Sutherland SK, *et al.* *Med J Aust.* 1980;2:435-7.
26. Isbister GK. *Intern Med J.* 2007;37:820-2.
27. Isbister GK, *et al.* *Br J Clin Pharmacol.* 2008;65:139-43.
28. Isbister GK, *et al.* *QJM.* 2008;101:557-65.
29. CSL Red Back Spider Antivenom Product Information, 15 December 2009.
30. Murray L, *et al.* Antivenoms. In: *Toxicology Handbook.* Second edition. Elsevier Australia, NSW, Australia. 2011. p. 470-89.
31. Emergency Medicine Expert Group. *Therapeutic Guidelines: Toxicology & Wilderness.* Version 1. Therapeutic Guidelines Limited, Melbourne, 2008.
32. Ellis RM, *et al.* *Emerg Med Australas.* 2005;17:152-6.
33. Brown SGA. *Emerg Med Australas.* 2006;18:155-69.
34. Dr Simon Brown personal communication.
35. Australian Resuscitation Council. Guideline 13.7 – Medication or fluids for the resuscitation of the newborn infant. Accessed 1 July 2012 from www.resus.org.au
36. Brown SGA. *Med J Aust.* 2006;185:283-9.
37. Fan HW, *et al.* *Br Med J.* 1999;318:1451-3.
38. Gawarammana IB, *et al.* *Med J Aust.* 2004;180:20-3.
39. Arunanth S, Hertzberg SR. *Med J Aust.* 1998;169:257-8.
40. Williams DJ, *et al.* *Toxicol.* 2007;49:780-92.
41. Premawardhena AP, *et al.* *Br Med J.* 1999;318:1041-3.
42. Soh SY, Rutherford G. *Trop Pediatr.* 2006;52:155-7.
43. Dassanayake AS, *et al.* *Ceylon Med J.* 2002;47:48-9.
44. Isbister GK, *et al.* *Med J Aust.* 2008;188:473-6.
45. Duncan AW, *et al.* *Med J Aust.* 1980;2:429-35.
46. Keiran Ragas, bioCSL Pty Ltd, Production Manager, Products of National Significance, personal communication Sept 2012.
47. White J. Envenomation Prevention and Treatment in Australia. In: Mackessy SP, ed. *Handbook of venoms and toxins of reptiles.* CRC Press Boca Raton FL, USA. 2010. p. 423-51.
48. ArachnoSERVER Spider Toxin Database. Accessed 1 Oct 2012 from <http://www.arachnoserver.org/toxincard.html?id=AS000305>
49. Australian Museum. Funnel web spiders. Accessed 1 Oct 2012 from <http://australianmuseum.net.au/Funnel-web-Spiders-group>
50. Daly FF, *et al.* *J Toxicol Clin Toxicol.* 2001;39:119-23.
51. South M, *et al.* *Med J Aust.* 1998;169:642.
52. Isbister GK, Gray MR. *Intern Med J.* 2004;34:287-9.
53. Isbister GK, Framenau VW. *J Toxicol Clin Toxicol.* 2004;42:153-61.

54. Australian Museum. Garden orb weaving spiders. Accessed 1 Oct 2012 from <http://australianmuseum.net.au/Garden-Orb-Weaving-Spiders>
55. Australian Museum. Trapdoor spiders. Accessed 1 Oct 2012 from <http://australianmuseum.net.au/Trapdoor-Spiders>

Section 5

1. A/Prof Julian White personal communication.
2. Sutherland S, Tibballs J. Venomous arthropods of medical importance, other than spiders and ticks. Scorpions. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 516-19.
3. Sutherland S, Tibballs J. Ticks. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 465-88.
4. Pearn J. Med J Aust. 1977;2:313-8.
5. Brown AF, Hamilton DL. J Accid Emerg Med. 1998;15:111-3.
6. Tibballs J, Cooper SJ. Med J Aust. 1986;145:37-8.
7. Pearn J. J Neurol Sci. 1977;34:37-42.
8. Miller MK. Med J Aust. 2002;176:264-5.
9. Dr Ken Winkel personal communication.
10. White J. Clinical toxicology of tick bites. In: Meier J & White J, editors. Handbook of Clinical Toxicology of Animal Venoms and Poisons. Informa Healthcare USA, Inc, New York, USA. 2008. p. 191-203.
11. Cameron RJ, Rowley MP. Med J Aust. 1999;171:334-5.

Section 6

1. A/Prof Julian White personal communication.
2. Sutherland S, Tibballs J. *Chironex fleckeri*, the Box Jellyfish. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 570-81.
3. Baxter EH, Marr AGM. Toxicol. 1969;7:195.
4. Ramasamy S, et al. Toxicol 2004;43:685-90.
5. Currie BJ. J Toxicol Clin Toxicol. 2003;41:301-8.
6. Winter KL et al. Toxicol Lett. 2010;192 419-24.
7. O'Reilly GM, et al. Med J Aust. 2001;175:652-5.
8. Currie BJ, Jacups SP. Med J Aust. 2005;183:631-6.
9. Bailey et al. Med J Aust. 2003;178:34-7.
10. Northern Territory Government. Centre for Disease Control. *Chironex fleckeri* (Box jellyfish). Accessed Sept 2012 from: [http://www.health.nt.gov.au/library/scripts/objectifyMedia.aspx?file=pdf/26/02.pdf&siteID=1&str_title=Box Jellyfish.pdf](http://www.health.nt.gov.au/library/scripts/objectifyMedia.aspx?file=pdf/26/02.pdf&siteID=1&str_title=Box%20Jellyfish.pdf)

References

11. Australian Resuscitation Council Guideline 9.4.5. Envenomation – Jellyfish stings. Accessed 1 July 2012 from www.resus.org.au
12. Hartwick R, *et al.* *Med J Aust.* 1980;1:15-20.
13. Seymour J, *et al.* *Toxicom.* 2002;40:1503-5.
14. Murray L, *et al.* *Antivenoms.* In: *Toxicology Handbook.* Second edition. Elsevier Australia, NSW, Australia. 2011. p. 470-89.
15. Winter KL, *et al.* *Toxicol Lett.* 2009;187:94-8.
16. Williamson JA, *et al.* *Med J Aust.* 1980;1:13-6.
17. Nickson CP, *et al.* *Ann Emerg Med.* 2009;54:395-403.
18. Macrokakis CJ. *Med J Aust.* 2004;181:699-702.
19. CSL Box Jellyfish Antivenom Product Information. 14 December 2009.
20. White J. *Snakebite & Spiderbite Management Guidelines South Australia.* 2006. Department of Health, Adelaide, South Australia.
21. Brown SGA. *Emerg Med Australas.* 2006;18:155-69.
22. Dr Simon Brown personal communication.
23. Australian Resuscitation Council. Guideline 13.7 – Medication or fluids for the resuscitation of the newborn infant. Accessed 1 July 2012 from www.resus.org.au
24. Dr Mark Little personal communication.
25. Brown SGA. *Med J Aust.* 2006;185:283-9.
26. Isbister GK, *et al.* *Med J Aust.* 2008;188:473-6.
27. Keiran Ragas, bioCSL Pty Ltd, Production Manager, Products of National Significance, personal communication Sept 2012.
28. White J. Envenomation Prevention and Treatment in Australia. In: Mackessy SP, ed. *Handbook of venoms and toxins of reptiles.* CRC Press, Boca Raton FL, USA. 2010. p. 423-51.
29. Barnes JH. *Med J Aust.* 1964;14: 897-904.
30. Huynh TT, *et al.* *Med J Aust.* 2003;178:38-41.
31. Fenner PJ, Hadok JC. *Med J Aust.* 2002;177:362-3.
32. Gershwin L. *Zootaxa.* 2005;1084:1-30.
33. Gershwin L. *QJM.* 2006;99:801-2.
34. Little M, *et al.* *QJM.* 2006;99:425-7.
35. Sutherland S, Tibballs J. Other jellyfish of medical importance. In: *Australian animal toxins: The creatures, their toxins and care of the poisoned patient.* Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 582-623.
36. Cheng AC, *et al.* *Aust N Z J Med.* 1999; 29:835.
37. Little M, *et al.* *Anaesth Intensive Care* 2003;31:642-7.
38. Flecker H. *Med J Aust.* 1952;2: 89-91.
39. Little M, Mulcahy R. *Med J Aust.* 1998;169:638-41.
40. Pereira P, *et al.* *Clin Toxicol (Phila).* 2010;48:390.

41. Pereira P, *et al* (Queensland Irukandji Taskforce). Irukandji Taskforce Guidelines for the Emergency Management of Irukandji Syndrome. 2007.
42. Winkel K, *et al*. Wilderness Environ Med. 2002;13:203-5.
43. Atkinson PRT, *et al*. Emerg Med J. 2006;23:503-8.
44. Loten C, *et al*. Med J Aust. 2006;184:329-33.
45. Taylor JG. Med J Aust. 2007;186(1):43.
9. Church JE, Hodgson WC. Toxicon. 2001;39:319-24.
10. Dr Jamie Seymour and Dr Peter Pereira personal communication, 29 July 2010.
11. White J. Snakebite & Spiderbite Management Guidelines South Australia. 2006. Department of Health, Adelaide, South Australia.
12. Brown SGA. Emerg Med Australas. 2006;18:155-69.
13. Dr Simon Brown personal communication.

Section 7

1. Sutherland S, Tibballs J. Venomous fish other than stonefish. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 656-700.
2. Sutherland S, Tibballs J. Genus *Synanceia*, Stonefish. In: Australian animal toxins: The creatures, their toxins and care of the poisoned patient. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 639-655.
3. A/Prof Julian White personal communication.
4. Australian Resuscitation Council Guideline 9.4.7. Envenomation – Fish stings. Accessed 1 July 2012 from www.resus.org.au
5. Atkinson PRT, *et al*. Emerg Med J. 2006;23:503-8.
6. Dr Mark Little personal communication.
7. CSL Stonefish Antivenom Product Information. 13 April 2011.
8. Church JE, Hodgson WC. Toxicon. 2002;40(6):787-96.
14. Murray L, *et al*. Antivenoms. In: Toxicology Handbook. Second edition. Elsevier Australia, NSW, Australia. 2011. p. 470-89.
15. Australian Resuscitation Council. Guideline 13.7 – Medication or fluids for the resuscitation of the newborn infant. Accessed 1 July 2012 from www.resus.org.au
16. Brown SGA. Med J Aust. 2006;185:283-9.
17. Fan HW, *et al*. Br Med J. 1999;318:1451-3.
18. Gawarammana IB, *et al*. Med J Aust. 2004;180:20-3.
19. Arunanthy S, Hertzberg SR. Med J Aust. 1998;169:257-8.
20. Williams DJ, *et al*. Toxicon. 2007;49:780-92.
21. Premawardhena AP, *et al*. Br Med J. 1999;318:1041-3.
22. Soh SY, Rutherford G. Trop Pediatr. 2006;52:155-7.
23. Dassanayake AS, *et al*. Ceylon Med J. 2002;47:48-9.
24. Isbister GK, *et al*. Med J Aust. 2008;188:473-6.

References

25. Keiran Ragas, bioCSL Pty Ltd, Production Manager, Products of National Significance, personal communication Sept 2012.
26. White J. Envenomation Prevention and Treatment in Australia. In: Mackessy SP, ed. Handbook of venoms and toxins of reptiles. CRC Press, Boca Raton FL, USA. 2010. p. 423-51.
27. Ngo, SYA, *et al.* Singapore Med J. 2009;50:507-9.
8. Australian Resuscitation Council. Guideline 9.4.8. Envenomation – Pressure Immobilisation Technique. Accessed 1 July 2012 from www.resus.org.au
9. Cruz LJ, White J. Clinical toxicology of *Conus* snail stings. In: Meier J & White J, editors. Handbook of Clinical Toxicology of Animal Venoms and Poisons. Informa Healthcare USA, Inc, New York, USA. 2008. p. 117-27.

Section 8

1. Acott C, Meier J. Clinical toxicology of venomous stingray injuries. In: Meier J & White J, editors. Handbook of Clinical Toxicology of Animal Venoms and Poisons. Informa Healthcare USA, Inc, New York, USA. 2008. p. 135-140.
2. A/Prof Julian White personal communication.
3. Australian Venom Research Unit (AVRU) website. Melbourne. Stingrays (Dasyatidae): Distribution Map. Accessed 19 September 2012 from http://www.avru.org/general/distrib_ray.html
4. Australian Resuscitation Council Guideline 9.4.7. Envenomation – Fish stings. Accessed 1 July 2012 from www.resus.org.au
5. Atkinson PRT, *et al.* Emerg Med J. 2006;23:503-8.
6. Dr Mark Norman, Museum Victoria, personal communication, July 2010.
7. White J. Clinical toxicology of blue ringed octopus bites. In: Meier J & White J, editors. Handbook of Clinical Toxicology of Animal Venoms and Poisons. Informa Healthcare USA, Inc, New York, USA. 2008. p. 171-5.

Section 9

1. Australian Venom Research Unit (AVRU) website. Melbourne, Australia. Facts & Figures: World's Most Venomous Snakes. Accessed 10 July 2012 from www.avru.org/general/general_mostvenom.html
2. CSL Brown Snake Antivenom Product Information, 14 December 2009.
3. CSL Black Snake Antivenom Product Information, 14 December 2009.
4. CSL Death Adder Antivenom Product Information, 14 December 2009.
5. CSL Taipan Antivenom Product Information, 15 December 2009.
6. CSL Tiger Snake Antivenom Product Information, 21 January 2010.
7. CSL Polyvalent Snake Antivenom Product Information, 20 September 2004.
8. CSL Sea Snake Antivenom Product Information, 15 December 2009.
9. CSL Funnel Web Spider Antivenom Product Information, 11 January 2010.
10. CSL Red Back Spider Antivenom Product Information, 15 December 2009.
11. CSL Box Jellyfish Antivenom Product Information, 14 December 2009.

12. CSL Stonefish Antivenom Product Information, 13 April 2011.
13. Keiran Ragas, bioCSL Pty Ltd, Production Manager, Products of National Significance, personal communication Sept 2012.
14. O'Leary MA, Isbister GK. *Toxicol.* 2009;54:192-95.
15. Isbister GK, *et al.* *Toxicol.* 2007;49:57-67.
16. A/Prof Julian White personal communication.
17. Murray L, *et al.* *Antivenoms*. In: *Toxicology Handbook*. Second edition. Elsevier Australia, NSW, Australia. 2011. p. 470-89.
18. Emergency Medicine Expert Group. *Therapeutic Guidelines: Toxicology & Wilderness*. Version 1. Therapeutic Guidelines Limited, Melbourne, 2008.
19. Sutherland S, Tibballs J. Genus *Pseudonaja*, Brown Snakes. In: *Australian animal toxins: The creatures, their toxins and care of the poisoned patient*. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 99-129.
20. Mirtschin PJ, *et al.* *Ecotoxicology*. 2006;15:531-8.
21. Isbister GK, *et al.* *Med J Aust.* 2012;197:173-7.
22. CSL Medical Handbook. Ed: Trinca J. Contributors: Feery BJ, *et al.* 1979. Commonwealth Serum Laboratories, VIC.
23. Sutherland S, Tibballs J. Treatment of snakebite in Australia. In: *Australian animal toxins: The creatures, their toxins and care of the poisoned patient*. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 286-342.
24. Sutherland S, Tibballs J. Genus *Notechis*, Tiger Snakes. In: *Australian animal toxins: The creatures, their toxins and care of the poisoned patient*. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 74-98.
25. Ramasamy S, *et al.* *Toxicol.* 2004;44: 837-5.
26. Churchman A, *et al.* *Med J Aust.* 2010;193:696-700.
27. Isbister GK, *et al.* *QJM.* 2006;99:109-15.
28. White J. Envenomation Prevention and Treatment in Australia. In: Mackessy SP, ed. *Handbook of venoms and toxins of reptiles*. CRC Press, Boca Raton FL, USA. 2010. p. 423-51.
29. Hartman LJ, Sutherland SK. *Med J Aust.* 1984;141:796-9.
30. NSW Health. *Snakebite & Spiderbite Clinical Management Guidelines 2007 – NSW*. May 2007.
31. White J. *Snakebite & Spiderbite Management Guidelines South Australia*. 2006. Department of Health, Adelaide, South Australia.
32. Sutherland S, Tibballs J. The genera *Atrax* and *Hadronyche*, Funnel web spiders. In: *Australian animal toxins: The creatures, their toxins and care of the poisoned patient*. Second edition. Oxford University Press, Sth Melbourne, Australia. 2001. p. 402-64.
33. Currie BJ, Jacups SP. *Med J Aust.* 2005;183:631-6.

Serial observations template chart

The adjacent foldout contains a template chart for recording serial observations and results (including details of antivenom therapy, if administered) for patients bitten/stung by Australian venomous fauna.

The chart may be copied for use.

Note: The chart is designed for use alongside information within this handbook. Additional investigations/observations (to those mentioned in the chart) may be required for specific bites/stings. Consult the relevant section of this handbook for further information.

Surname: _____
 First name: _____
 DOB: _____
 UR: _____

Snakebite/spiderbite/marine stings observation chart

| | | |
|---------------------------------------|----------------------------|---------------|
| Date of bite: | Time of bite: | No. of bites: |
| Geographic location: | | |
| Culprit creature seen (Yes/No): | Culprit identity if known: | |
| SVDK sample (circle one): bite/urine* | SVDK result:* | |

*SVDK to be performed only for snakebite & only if there are clinical/laboratory indicators of envenoming.

Serial observations and results

| | | | | | | | | | | | | |
|---|--|--|--|--|--|--|--|--|--|--|--|--|
| Initials of person recording | | | | | | | | | | | | |
| Date: | | | | | | | | | | | | |
| Time: | | | | | | | | | | | | |
| Time after bite: | | | | | | | | | | | | |
| Local/Regional | | | | | | | | | | | | |
| Pain (patient score 0-10) | | | | | | | | | | | | |
| Sweating (Yes/No) | | | | | | | | | | | | |
| Bleeding/oozing (Yes/No) | | | | | | | | | | | | |
| Node tenderness/swelling (Yes/No) | | | | | | | | | | | | |
| Local necrosis/ulceration (Yes/No) | | | | | | | | | | | | |
| General | | | | | | | | | | | | |
| Pulse rate | | | | | | | | | | | | |
| Blood pressure | | | | | | | | | | | | |
| Respiratory rate | | | | | | | | | | | | |
| Temperature | | | | | | | | | | | | |
| Oxygen saturation SpO ₂ | | | | | | | | | | | | |
| Headache (Yes/No) | | | | | | | | | | | | |
| Nausea/vomiting (Yes/No) | | | | | | | | | | | | |
| Abdominal pain (score 0-10) | | | | | | | | | | | | |
| Back/generalised pain (score 0-10) | | | | | | | | | | | | |
| Increased salivation (Yes/No) | | | | | | | | | | | | |
| Tongue fasciculation (Yes/No) | | | | | | | | | | | | |
| Paralytic signs | | | | | | | | | | | | |
| Ptosis (Yes/No) | | | | | | | | | | | | |
| Ophthalmoplegia (Yes/No) | | | | | | | | | | | | |
| Fixed dilated pupils (Yes/No) | | | | | | | | | | | | |
| Drooling (Yes/No) | | | | | | | | | | | | |
| Limb weakness (Yes/No) | | | | | | | | | | | | |
| Ataxia (Yes/No) | | | | | | | | | | | | |
| Loss of deep tendon reflexes (Yes/No) | | | | | | | | | | | | |
| Respiratory paralysis or failure (Yes/No) | | | | | | | | | | | | |
| Peak flow rate PEFr (L/min) | | | | | | | | | | | | |

.....See overleaf for additional observations

Surname: _____

First name: _____

DOB: _____

UR: _____

Serial observations and results ... cont'd

| | | | | | | | | | | | | | | | | | | | |
|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|
| Initials of person recording | | | | | | | | | | | | | | | | | | | |
| Date: | | | | | | | | | | | | | | | | | | | |
| Time: | | | | | | | | | | | | | | | | | | | |
| Time after bite: | | | | | | | | | | | | | | | | | | | |
| Coagulopathic signs: snakebite only | | | | | | | | | | | | | | | | | | | |
| Persistent bleeding (e.g. I.V. sites; gums etc) (Yes/No) | | | | | | | | | | | | | | | | | | | |
| Haematuria (Yes/No) | | | | | | | | | | | | | | | | | | | |
| Major active bleeding (Yes/No) | | | | | | | | | | | | | | | | | | | |
| Myolytic signs: snakebite only | | | | | | | | | | | | | | | | | | | |
| Muscle tenderness/pain (Yes/No) | | | | | | | | | | | | | | | | | | | |
| Myoglobinuria (Yes/No) | | | | | | | | | | | | | | | | | | | |
| Renal signs: snakebite only | | | | | | | | | | | | | | | | | | | |
| Urine output (mL) | | | | | | | | | | | | | | | | | | | |
| Laboratory tests: snakebite only | | | | | | | | | | | | | | | | | | | |
| INR/PT | | | | | | | | | | | | | | | | | | | |
| aPTT | | | | | | | | | | | | | | | | | | | |
| Fibrinogen (direct fibrinogen) | | | | | | | | | | | | | | | | | | | |
| d-dimer/FDP | | | | | | | | | | | | | | | | | | | |
| Platelet count | | | | | | | | | | | | | | | | | | | |
| Haemoglobin | | | | | | | | | | | | | | | | | | | |
| White cell count | | | | | | | | | | | | | | | | | | | |
| Creatinine | | | | | | | | | | | | | | | | | | | |
| Urea | | | | | | | | | | | | | | | | | | | |
| CK | | | | | | | | | | | | | | | | | | | |
| Potassium | | | | | | | | | | | | | | | | | | | |

| | | | | | | | | | | | | | | | | | | | |
|-------------------------------------|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|
| Antivenom | | | | | | | | | | | | | | | | | | | |
| Type/time/amount | | | | | | | | | | | | | | | | | | | |
| Adverse reactions – minor or major* | | | | | | | | | | | | | | | | | | | |

*Minor = rash; Major reaction = hypotension/bronchospasm/angioneurotic oedema.
 Copyright 2013 A/Prof Julian White; permission is granted for this chart to be copied and used in any hospital or health centre for the purpose of recording serial observations and results for individual patients bitten/stung by Australian venomous fauna.

