STUDIES IN CLINICAL TOXINOLOGY

IN SOUTH AUSTRALIA

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A Thesis submitted for admission to the degree of

DOCTOR OF MEDICINE

UNIVERSITY OF ADELAIDE

Department of PATHOLOGY

Faculty of MEDICINE

MAY 1988

This Thesis is dedicated to my wife,
Dr Beata M. BYOK, and my parents,
Dr Ian C.L. WHITE and Betty WHITE.

CONTENTS

STATEMENT OF AUTHENTICITY

This thesis contains no material which has been accepted for the award of any other degree or diploma in any University. To the best of my knowledge and belief, the thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis. I consent to the thesis being made available for photocopying and loan if applicable if accepted for the award of the degree of Doctor of Medicine.

Julian WHITE

May 1988

ACKNOWLEDGEMENTS

I gratefully acknowledge the many people who have assisted in various ways in the development of my interest in herpetology and toxinology, and those friends, colleagues, and others who have provided information or assistance over the years in my management of cases of envenomation, on which the following papers are based.

A number of people have made special contributions and deserve specific mention:

Dr Cassian BON; Mr Joe BREDL; Prof. David CHAPMAN; Dr Kevin CHENEY; Dr Peter CLEMENTS; Mr Geoff COOMBE; Prof. James CORRIGAN; Mr Alan COULTER; Mr Kingsley COULTHARD; Ms Jeanette COVACEVICH; Ms Adrienne EDWARDS; Mr Harry EHMANN; Dr Brian FOTHERINGHAM; Dr David HARDY; Ms Elizabeth HENDER; Mr David HIRST; Dr Terry HOUSTON; Dr David LEE; Prof. Dietrich MEBS; Prof. Sherman MINTON; Ms Beryl MORRIS; Prof. John PEARN; Prof. Derrick POUNDER; Prof. Alistair REID; Prof. Finlay RUSSELL; Dr Terry SCHWANER; Dr Eric SIMS; Mr Neil SMYTH; Dr Ron SOUTHCOTT; Dr Isobel SPEED; Dr David THEAKSTON; Mr Mike TYLER; Prof. David WARRELL; Mr Vaughan WILLIAMS.

There are of course many others who have helped over the years, too numerous to mention here.

In the last five years Mrs Olivia LAMMAS has made a special contribution in preparation of my manuscripts, and the staff of the Photography Department, Adelaide Children's Hospital, have greatly assisted in preparation of illustrations for manuscripts.

Prof. Barry VERNON-ROBERTS is owed a special acknowledgement for his willingness to act as supervisor on preparation of this thesis.

STUDIES IN CLINICAL TOXINOLOGY IN SOUTH AUSTRALIA

Julian White, MB, BS

The accompanying papers, chapters and appended published works and reports document an evolving interest in and knowledge of aspects of clinical toxinology in South Australia, and this is the unifying theme running through all these works.

At the time I commenced these studies, there was no unified work on snakebite in Australia, or on spiderbite, though, particularly for the former, there was a mass of individual publications, which when considered collectively gave a considerable data base on which to build a more comprehensive view embodied in a single major monograph. These previous works by other authors are extensively referenced in the papers, monograph, and chapters which comprise this thesis.

While pursuing my medical studies, and then postgraduate studies, I also undertook extensive work in herpetology, and to a lesser extent arachnology, and this work is documented in part in Appendix 1. The reports listed are those reports still extant of collecting trips made under my leadership or supervision, where reptiles in various parts of the state were sampled for the South Australian Museum. The reports are internal reports of the South Australian Herpetology Group Inc., which were also deposited at the S.A. Museum and the S.A. National Parks and Wildlife Service, and a few were published in newsletters and other herpetological publications. The focus of collecting was on new distribution records of reptiles, and extension of existing collections, and as such venomous snakes do not feature prominently. However, through organisation and leadership of these trips, I was able to build a substantial knowledge of the State's herpetofauna, including venomous snakes, and a good working

knowledge of reptile taxonomy. This has formed a vital base for understanding many aspects of snakebite in South Australia and beyond, and has put me in a unique position amongst medical practitioners interested in snakebite in Australia, as I have commenced my medical studies of snakebite with an already established expertise in snake taxonomy, biology, and biogeography. The purpose of the reports in Appendix 1 is therefore to indicate the extent of this experience and knowledge of herpetology.

While not documented in reports, it should be noted that this field experience also extended to other groups of fauna, particularly spiders and scorpions.

Even before completing my undergraduate medical studies it became apparent to me that in many cases medical management of snakebite was less than optimal, reflecting the lack of interaction between herpetology and medicine, the lack of medical practitioners with a deep commitment to toxinology, and the lack of either teaching or a comprehensive text on this field.

This must be contrasted, to some extent, with the situation in spiderbite, and more particularly marine envenomation, where South Australia did indeed have a medical practitioner with extensive knowledge and commitment, internationally recognised - namely Dr Ron Southcott.

Through personal involvement in managing cases of snakebite in my early postgraduate years, and a wide ranging review of available literature, I built up a personal perspective on snakebite. This was reflected in numerous requests for lectures on this subject, and for a comprehensive reference work. This culminated in my monograph, "Ophidian envenomation; a South Australian perspective". This single author work of some 110 pages, inclusive of title page, foreword and contents index, was conceived as a single reference covering all aspects of snakebite pertaining to South

Australia. This therefore consisted of a synthesis of all my knowledge on the topic of snakebite set against a background of a review of all pertinent current knowledge published by others.

The first section was a review of the evolution and principles of taxonomy of Australian snakes. By definition, this was essentially a summary of the work of others. The second section was a listing, species by species, of all South Australian snakes, together with details of their description, distinguishing features, biology and distribution, the latter as an appendix. Again, by definition, many points on identification were based on recognised taxonomic works of others, supplemented by my own observations. The distribution maps were the most comprehensive ever produced in Australia for reptiles, and have only recently been improved on by an Australian National Parks publication. They combined exact plots of S.A. Museum registered specimens (kindly provided by Museum staff) and my own field experience. To this day I, and others, use this as a prime reference for identifying South Australian snakes.

The third section was on snake venoms, bringing together research data from many sources into a unified text, with a personal perspective on the relationship between research findings on venom actions and components, and clinical problems of snakebite in South Australia.

The fourth section was on envenomation, commencing with a world overview, then sections on epidemiology, clinical envenomation findings, and specific complications. This included, in addition to the review of the literature, significant information on personal experience with snakebite, including detailed information on some cases, especially regarding coagulation disturbances, some of which was amongst the most detailed ever published. The concluding part of this section, on features of envenomation for each snake group, was a presage of future works and a recurrent theme in many of

my papers, that each major snake group has distinctive features of envenomation.

The final section was on treatment, and again while reviewing the literature, also established a series of guidelines based on personal experience. The accompanying protocol, though since modified, acted as a firm guideline on the treatment of snakebite in South Australia, where none existed previously.

The publication of this monograph in 1981 drew considerable comment from the toxinology community, receiving good reviews overseas, and resulted in my being invited to join the International Society on Toxinology as a full member, and to present papers at the IST international meeting in Brisbane in 1982.

I presented three papers at this meeting, all subsequently published in the IST journal "Toxicon". These were (1) Patterns of elapid envenomation and treatment in South Australia, (2) Local tissue destruction and Australian elapid envenomation, (3) Haematological problems and Australian elapid envenomation.

The first paper reviewed presentation and treatment of snakebite at three Adelaide hospitals, retrospectively, and clearly demonstrated the inconsistent level of care offered. In particular, some patients were given antivenom inappropriately, and others denied it when it was clearly indicated. The paper ended with a plea to establish formal consultant expertise in clinical toxinology to rectify this problem in snakebite management.

The second paper documented differences in local effects between the major groups of snakes causing bites in South Australia, as well as documenting the few cases of significant tissue injury. In effect this paper

demonstrated that different types of snake caused sufficiently different effects for this to be used to determine the type of snake involved. Such an analysis had not been made before in Australia, and this is one of the areas of significant contribution documented in this thesis.

The third paper, on haematological problems, extended the data and review given in the monograph, and also significantly added to the information base differentiating bites and outcomes for various groups of snakes. It also reviewed snakebite coagulopathy and renal failure, an issue concurrently developed in a paper published in the MJA in 1983, "Acute renal failure and coagulopathy after snakebite".

This latter paper, co-authored with Robert Fassett (at the time a renal unit registrar) documented a case of severe acute renal failure following brown snake envenomation, and associated with a true DIC. The case record was largely written by Dr Fassett, the discussion largely by me.

At the same time a severe case of tiger snake envenomation was reported, ultimately published in 1984, titled "Tiger snake bite". Though a multi-author work, the paper was both conceived and written by me and, like the previous paper, documents some aspects and complications of severe snakebite either poorly reported before, or hitherto unreported. In particular, this case illustrates the classical local and general features of tiger snake envenomation, which are quite distinctive from brown snake envenomation.

During 1983 Dr Derrick Pounder (IMVS, Adelaide) and I commenced discussions on fatal snakebite. Dr Pounder, as a forensic pathologist, was interested from this point of view, while I was keen to research the causes of death in fatal snakebite. As I had not (and still have not) had any cases of snakebite with fatal outcome under my care, such research would have to be

based on a retrospective survey across Australia, to generate sufficient numbers.

In considering what to look for in snakebite fatalities, we generated some basic guidelines, and these formed the basis of our paper, "Fatal snakebite in Australia", subsequently quoted as recommended reading for trainee pathologists in Australia. This paper incorporated my clinical data on differences between bites by various groups of snakes, as a table for the first time.

The general aspects of fatal snakebite and potential autopsy findings were further canvassed in a paper presented at the First Asian-Pacific Congress in Legal Medicine, Singapore, 1983, "Ophidian envenomation in Australia; problems in the autopsy diagnosis".

At this time I was trying to further define clinical and laboratory findings in snakebite, in correlation with the species or species group involved. This resulted in collaboration with Dr T.D. Schwaner at the S.A. Museum. These ideas were first presented as a paper, "Snakebite in South Australia", to the annual meeting of the Australian Society of Herpetologists in 1983, but given much firmer focus in a second paper, "Correlation of phylogeny of Australian elapid snakes, determined by immunologic methods, with venom structure and clinical envenomation". This latter paper, presented at the 5th European Meeting of the International Society on Toxinology, Hanover, 1983, was authored by me, but also drew on experimental work on snake taxonomy by Dr Schwaner and colleagues, hence the multi-authorship. This paper for the first time correlated my own clinical data with emerging knowledge of the phylogeny of Australian snakes, a merging of disciplines which has since developed further, and is part of the focus for my current venom research, as will be mentioned later.

By 1984 some case data on fatal snakebite had been gathered by Dr Pounder, facilitated by the establishment of a research group, "The Australian Snakebite Study Group", consisting of myself and Dr Pounder in Adelaide, and Dr J. Pearn and Mr J. Morrison in Brisbane. The first objective of this group was to collect data on fatal snakebites, and I analysed and presented this data first, as "A perspective on the problems of snakebite in Australia", at the Australasian Herpetological Symposium, Sydney, 1984. The analysis of data, and concept and writing of the paper were mine, but all other members of the ASSG were listed as co-authors, as the paper also promoted the ASSG. This paper also further developed ideas on the principles behind medical management of snakebite, stressing the potential role for a clinical toxinologist expert in both medicine and herpetology. The statistics on snakebite fatalities documented a decline in death rate from snakebite in Australia. This important paper helped stimulate interest in snakebite related issues amongst herpetologists, especially as it was published in a major textbook on herpetology, "The Biology of Australasian Frogs and Reptiles", in 1985.

Shortly after the 1984 Sydney meeting, I presented another paper, "Clinical problems in the management of Australian snakebite - the choice of antivenom" at the 11th International Conference on Tropical Medicine and Malaria, Calgary, 1984. This paper presented new information on the use of venom detection kits in South Australia, and their relationship to the choice of antivenom. The alternatives in choosing antivenom were discussed and the role of a clinical toxinologist developed to a much greater extent than previously. Unfortunately this paper, which was well received by its toxinologist audience, and subsequently refereed for publication as proceedings of the conference, did not ultimately reach publication as the whole project was withdrawn at the end of 1987. It has not yet been revised and updated for publication elsewhere.

However, the data from this and previous papers mentioned were drawn together in textbook form for four chapters on snakebite in the Queensland Museum Publication "Toxic Plants and Animals; a Guide for Australia", ultimately published in 1987. The four chapters were: "Elapid snakes; venom production and bite mechanism", "Elapid snakes; venom toxicity and actions", "Elapid snakes; aspects of envenomation", "Elapid snakes; management of bites". Covering over 100 pages, these four chapters were both an updated and expanded version of my monograph, with attendant literature reviews, distilled into a useable textbook format, and a presentation of new or expanded data based on my own research on clinical aspects of snakebite.

Of particular relevance here is the chapter on envenomation, which amongst other information incorporated a review of 76 cases of snakebite in South Australia, one of the largest such series ever published from Australia. The data on fatal snakebite was greatly expanded, with a detailed review of 21 cases which clearly documented for the first time the importance of snakebite coagulopathy, and associated haemorrhage and renal failure as a cause of death.

The syndromes of envenomation for each snake group were analysed in detail, with previously unpublished case reports used to illustrate the various comments made, and the overall patterns reduced to a single table for ease of use by toxinologists.

The chapter on treatment, while covering areas discussed in the monograph, also fully discussed the concepts behind the role of clinical toxinologists in determining appropriate antivenom therapy. These concepts are the present culmination of the major theme of my research and management of snakebite in South Australia.

Following publication of this textbook, which received very favourable reviews, several other publications on snakebite or snake venom have also been produced.

"A review of 105 cases of suspected snakebite in S.A." was presented at the First Asian-Pacific Congress on Animal, Plant and Microbial Toxins, Singapore, 1987. This has now been published in the proceedings of the Congress. While it is the largest such review yet published from Australia, it will ultimately form the basis of an even larger review, now being undertaken. Nevertheless, as it stands, it is an important addition to the epidemiology of snakebite.

The first reported case of snakebite by the five-ringed brown snake was documented in "The five-ringed brown snake; *Pseudonaja modesta* (Gunther): report of a bite and comments on its venom". This paper was written by me, with clinical data on the case supplied by Dr Passehl, and laboratory data supplied by Mr Williams, the latter work performed under my direction.

A severe case of envenomation by the brown snake was documented in "Severe envenomation following multiple bites by a common brown snake, *Pseudonaja textilis*". Again, Mr Williams performed the laboratory coagulation studies. This paper has been submitted for publication.

My association with primary venom research, initiated by me but bench work being performed in association with Mr Williams, is concentrated on venom effects on coagulation. While our prime focus is the brown snake group, initial venom supplies necessitated work on the black tiger snake group. This will result in a series of papers on the venom of these snakes. One paper has so far been published; "Variation in venom constituents within a single isolated population of peninsula tiger snake (*Notechis ater niger*)". Three more are in various stages of publication. Together, they will document some important results with regard to snake venom evolution. With

this species, we have shown that while isolated populations have homogeneous venom component composition, differences in composition profile occur between isolated populations, and this diversity is not related to prey type or snake morphology, but is most closely explained by time of separation between presently isolated populations. This provides good evidence that the diversity of components found within snake venoms may not completely reflect selective pressures for prey acquisition, but rather random generation of diversity. Two components common to all sampled populations are presumably vital in prey acquisition, and so have been conserved. This work may form a basis for increasing our understanding of the evolution of venoms, and possibly may extend our understanding of more general concepts of evolutionary mechanisms.

These black tiger snake venoms also contain powerful coagulants and platelet aggregation inhibitors, the purification and characterisation of which are the subjects of papers currently in preparation.

While snakebite has been the main theme of these papers, and my work in clinical toxinology, I have also had extensive experience with spiderbite. Some of this experience has been distilled in a recent publication, "Review of clinical and pathological aspects of spiderbite in Australia", first presented as a plenary lecture at the First Asian-Pacific Congress on Animal, Plant and Microbial Toxins, Singapore, 1987, and now published in the proceedings of that Congress. This paper drew favourable comment, and as a result is being used as a basis for two chapters on spiderbite in a forthcoming textbook on spiderbite to be published by the Asian-Pacific Section of the International Society on Toxinology. I am currently preparing these chapters, but in the process I have produced two other papers on specific cases of spiderbite, now submitted for publication, which greatly expand reliable case reports of bites by a variety of species of spiders. These two papers, "Bites by the white-tailed spider, *Lampona*

cylindrata", and "Case reports of spiderbites in South Australia, excluding bites by the red-back spider", are co-authored by Mr Hirst and Ms Hender, although the majority of the cases reported, and the entire text, are mine.

While I have managed many cases of red-back spiderbite, this topic has already been well canvassed in the literature. My contribution, "Latrodectism; an unusual cause of abdominal pain; a cautionary tale", is an unusual report, the details and relevance of which are amply summarised in the title.

Summary of Contribution to Toxinology

The essence of the contribution to the field of toxinology of the works embodied in this thesis will probably already be apparent from the foregoing section, but in brief may be summarised as follows:

1) The provision of a single text providing all required information for the management of snakebite including identification of snakes (1981 monograph). (At that time no such text was available.)

2) The documentation of clinical features of snakebite for major subgroups of Australian snakes, ultimately allowing differentiation of bites for each group, thus assisting use of correct antivenom.

3) The documentation of snakebite coagulopathy and establishment of firm guidelines for its diagnosis and management.

4) The documentation of causes of snakebite fatalities in Australia, which should assist management decisions designed to prevent future fatalities.

5) The documentation of snakebite epidemiology in South Australia, which will assist in developing overall management strategies for snakebite in South Australia.

6) The documentation of patterns of spiderbite with special reference to South Australia, allowing a more rational and factual basis for the provision of medical advice and management of cases of spiderbite.

List of Publications as Main Text of Thesis

1) White J. (1981) Ophidian envenomation; a South Australian perspective.

Records of the Adelaide Children's Hospital, 2(3); 311-421.

2) White J. (1983) Patterns of elapid envenomation and treatment in South Australia.

Toxicon, Suppl. 3; 489-491.

3) White J. (1983) Local tissue destruction and Australian elapid envenomation.

Toxicon, Suppl. 3; 493-496.

4) White J. (1983) Haematological problems and Australian elapid envenomation.

Toxicon, Suppl. 3; 497-500.

5) White J., Fassett R. (1983) Acute renal failure and coagulopathy after snakebite.

Medical Journal of Australia, 2; 142-143.

White J. ~ 60% (including virtually all of discussion).

Fassett R. ~ 40% (essentially the case report section).

6) White J., Tomkins D., Steven I., Williams V. (1984) Tiger Snake bite.

Records of the Adelaide Children's Hospital, 3(2); 169-173.

White J. ~ 95% (whole paper conceived and written).

Williams V. ~ 4% (provided coagulation laboratory results).

Tomkins D.) ~ 1% (involved in managing this case, and

Steven I.) reviewed manuscript).

7) White J., Pounder D.J. (1984) Fatal snakebite in Australia.
The American Journal of Forensic Medicine and Pathology, 5(2); 137-143.

White J. ~ 75% (all information on snakebite including tables, diagrams etc).

Pounder D.J. ~ 25% (sections on general autopsy techniques, heat stroke etc).

8) White J., Pounder D.J. (1983) Ophidian envenomation in Australia; problems in the autopsy diagnosis.
First Asian-Pacific Congress in Legal Medicine, Singapore.

White J. ~ 85% (conception of paper, most of text).

Pounder D.J. ~ 15% (sections on autopsy).

9) White J. (1983) Snakebite in South Australia.
Annual Meeting of the Australian Society of Herpetologists.

10) White J., Schwaner T.D., Dessaur H.C., Baverstock P.R., Mengden G. (1983) Correlation of phylogeny of Australian elapid snakes, determined by immunologic methods, with venom structure and clinical envenomation.
5th European Meeting of the International Society on Toxinology, Hanover, FRG.

White J. ~ 75% (conception and writing of paper, all of clinical and toxinologic correlation).

Schwaner T.D. ~ 20% (immunologic data and analysis).

Dessaur H.D.)
Baverstock P.R.) ~ 5% (advice and comment on immunologic data
Mengden G.) and analysis).

11) White J., Pounder D.J., Pearn J.H., Morrison J.J. (1985) A perspective on the problems of snakebite in Australia.

In Eds. Grigg G., Shine R., Ehmann H.: The Biology of Australasian Frogs and Reptiles; Surrey Beatty & Sons, Sydney.

White J. $\simeq$ 90% (conceived, written, all data analysis and discussion).

Pounder D.J. $\simeq$ 8% (collection of autopsy reports).

Pearn J.H.)

Morrison J.J.) $\simeq$ 2% (advice and comments on text).

12) White J. (1984) Clinical problems in the management of Australian snakebite - the choice of antivenom.

11th International Conference on Tropical Medicine and Malaria, Calgary, Canada.

13) White J. (1987) Elapid snakes; venom production and bite mechanism.

In Eds. Covacevich J., Davie P., Pearn J.: Toxic Plants and Animals; A Guide for Australia, Queensland Museum, Brisbane, pp 356-367.

14) White J. (1987) Elapid snakes; venom toxicity and actions.

In Eds. Covacevich J., Davie P., Pearn J.: Toxic Plants and Animals; A Guide for Australia, Queensland Museum, Brisbane, pp 368-389.

15) White J. (1987) Elapid snakes; aspects of envenomation.

In Eds. Covacevich J., Davie P., Pearn J.: Toxic Plants and Animals; A Guide for Australia, Queensland Museum, Brisbane, pp 390-429.

16) White J. (1987) Elapid snakes; management of bites.

In Eds. Covacevich J., Davie P., Pearn J.: Toxic Plants and Animals; A Guide for Australia, Queensland Museum, Brisbane, pp 430-457.

17) White J. (1987) A review of 105 cases of suspected snakebite in South Australia.

In Eds. Gopalakrishnakone P., Tan C.K.: Progress in Venom and Toxin Research, National University of Singapore, Singapore, pp 15-19.

18) White J., Williams V., Passehl J.H. (1987) The five-ringed brown snake, *Pseudonaja modesta* (Gunther): report of a bite and comments on its venom.

Medical Journal of Australia, 147; 603-605.

White J. $\simeq$ 70% (conceived, written, discussion).

Williams V. $\simeq$ 20% (data on venom).

Passehl J.H. $\simeq$ 10% (some data on case report).

19) White J., Williams V. (1988?) Severe envenomation following multiple bites by a common brown snake, *Pseudonaja textilis*.

Submitted.

White J. $\simeq$ 90% (conceived, written, case report and discussion).

Williams V. $\simeq$ 10% (coagulation data, comments).

20) Williams V., White J. (1987) Variation in venom constituents within a single isolated population of peninsula tiger snake (*Notechis ater niger*).

Toxicon, 25(11); 1240-1243.

White J. ≃ 40% (conceived, partially responsible for discussion etc, review of laboratory aspects at all stages).

Williams V. ≃ 60% (major portion of laboratory benchwork, and significant role in text).

21) Williams V., White J., Schwaner T.D., Sparrow A. (1988?) Variation in venom proteins from isolated populations of tiger snakes (*Notechis* elapidae) in South Australia.

Toxicon (accepted).

White J. ≃ 25% (conception of project, part of text, including discussion).

Williams V. ≃ 50% (most of laboratory benchwork, and major role in text).

Schwaner T.D. ≃ 15% (some statistical analysis and work on text).

Sparrow A. ≃ 10% (statistical analysis).

22) White J. (1987) Review of clinical and pathological aspects of spiderbite in Australia.

In Eds. Gopalakrishnakone P., Tan C.K.: Progress in Venom and Toxin Research, National University of Singapore, Singapore, pp 531-541.

23) White J., Hirst D., Hender E. (1988?) Bites by the white-tailed spider, *Lampona cylindrata*.

Submitted.

White J. ≃ 80% (conception of paper, most case records, all text).

Hirst D. ≃ 10% (some case records).

Hender E. ≃ 10% (some case records, comments on text).

24) White J., Hirst D., Hender E. (1988?) Case reports of spiderbites in South Australia, excluding bites by the red-back spider.

Submitted.

White J. ≃ 80% (conception of paper, most case records, all text).

Hirst D. ≃ 10% (some case records, confirmation of spider ID).

Hender E. ≃ 10% (some case records, comments on text).

25) Harbord M., White J. (1984) Latrodectism: an unusual cause of abdominal pain. A cautionary tale.

Records of the Adelaide Children's Hospital, 3(2); 174-175.

White J. ≃ 50% (some of case record, much of discussion).

Harbord J. ≃ 50% (some of case record, some of discussion).

List of Papers not included in this Thesis

1) White J. (1976) Reptiles of the Corunna Hills.
Herpetofauna, 8(1); 21-23.

2) White J. (1979) Conservation and public education programmes for herpetological societies.
Herpetofauna, 10(2); 21-23.

3) White J. (1979) The Trilling Frog found active in a hot water bore pool.
Herpetofauna, 10(2); 29.

4) White J. (1979) Brief observations on tortoises in Minkie Waterhole, Innaminka.
Herpetofauna, 10(2); 31.

5) White J. (1979) The road to Mokari.
Herpetofauna, 11(1); 13-16.

6) White J. (1979) Courtship display of the Snapping Tortoise, *Elseya latisternum* Gray.
Herpetofauna, 11(1); 27-28.

7) White J., Boldt D.B., David D.J., Sheffield L., Simpson D.A. (1981) Carpenter Syndrome with normal intelligence and precocious growth.
Acta. Neurochirurgica, 57; 43-49.

8) David D.J., White J. (1981) Severe craniofacial deformities and their management.
Annals of the Academy of Medicine, Singapore, 10(2); 180-186.

9) David D.J., White J., Sprod R., Bagnall A. (1982) Nasendoscopy: Significant refinements of a direct viewing technique of the velopharyngeal sphincter.

Plastic and Reconstructive Surgery, 70(4); 423-428.

10) David D.J., Simpson D.A., White J. (1983) Fronto-ethmoidal encephaloceles; morphology and treatment.

Proceedings of the 8th International Congress of Plastic and Reconstructive Surgery.

11) Goss A.N., White J., Townsend G.C. (1983) Craniofacial growth in young marmosets (*Callithrix jacchus*).

Laboratory Animals, 17; 303-306.

12) David D.J., Sheffield L., Simpson D.A., White J. (1984) Fronto-ethmoidal meningo-encephaloceles: morphology and treatment.

British Journal of Plastic Surgery, 37; 271-284.

13) Simpson D.A., David D.J., White J. (1984) Cephaloceles: treatment, outcome and antenatal diagnosis.

Neurosurgery, 15(1); 14-21.

OPHIDIAN ENVENOMATION
A SOUTH AUSTRALIAN PERSPECTIVE

by

JULIAN WHITE

**HONORARY CONSULTANT ON SNAKE BITE
ROYAL ADELAIDE HOSPITAL AND THE ADELAIDE
CHILDREN'S HOSPITAL**

Records of the Adelaide Children's Hospital
Vol. 2, No. 3 1980–81, pp. 311–421

ISSN 0314 612 X

This monograph on snakes and human envenomation is published by the Adelaide Children's Hospital as a continuation of the Board's policy to make available comprehensive studies of interest to both physicians and naturalists. It follows in the tradition already set in previous issues of this journal by Dr. R. V. Southcott with his monographs on lepidopterism, arachnidism, and toxic fungi. Dr. Julian White, the author of the authoritative account presented here, came with his parents from the United Kingdom to live in Adelaide when he was 12 years old, and his interest in the local fauna was stimulated three years later when his father was given two Shingle-back ("stumpy-tailed") lizards as pets. From this start the family menagerie rapidly expanded to over 300 animals, including hopping mice, rats, reptiles, spiders, insects, and even (in 1970) a Johnson's fresh-water crocodile (who still lives with the family in a tank on the front verandah of their suburban home, rather to the discouragement of casual callers).

Julian helped to form the Herpetology Group of the Field Naturalists Society of South Australia, in 1971, and was its President in 1974. In 1975 this became the South Australian Herpetology Group Incorporated, in close association with the S.A. Museum, and now affiliated with the Friends of the S.A. Museum, one of the several specialist bodies officially linked to that very active State Government institution. He was President of the Friends in 1979–80, and is now Honorary Consultant on snake bite to both the Royal Adelaide Hospital and the Adelaide Children's Hospital. Dr. White is currently the Clinical Co-ordinator of the S.A. Cranio-facial Unit which is based at our hospital, and we regard ourselves as very privileged to be able to publish this monograph, which brings together much recent work of medical importance and general interest.

ERIC SIMS

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CONTENTS

INTRODUCTION

Snakes have been a prominent part of many of man's cultures. Some cultures have worshipped the snake, or revered it. Others have cast the snake in the role of a devil or agent of Lucifer. Undoubtedly this has stemmed from the ability of some snakes rapidly to kill animals many times their size, including man. Many groups of animals have independently developed venoms to subdue prey, or discourage predators, but few have done so with such obvious success as the snakes.

Few figures are available on the impact of venomous snakes on man, but it seems likely that many thousands, if not tens of thousands of humans die each year as a result of snakebite or its sequelae.[1] Fortunately, in Australia few humans die from snakebite.[2] This is despite the abundance and diversity of potentially lethal species,[3] which recent studies[4] have shown to be potentially the most deadly ophidians in the world.

Such a low deathrate can be attributed to a variety of causes, amongst which excellence of medical care does not feature strongly. Australian medical practitioners have certainly contributed greatly to basic and clinical research into Australian snake venoms over the years. One of the most important new developments in the first aid treatment of envenomation has originated from Australia.[5] However, until recently there appears to have been no undergraduate teaching on ophidian envenomation and its treatment. Most medical practitioners in Australia only learn about such treatment when faced with treating a case of snakebite, an experience traumatic for both doctor and patient.

Several enlightened doctors have tried to remedy this situation in recent years. Credit must be given to Struan Sutherland, of the Commonwealth Serum Laboratories, for spearheading these moves in the last five years. This article is offered as a further shot in the battle to bring information on snakebite to the mass of Australia's medical practitioners and in particular to those passing through the various training schemes at the Adelaide Children's Hospital.

In this article an attempt has been made at placing snakebite in Australia in the wider perspective of worldwide ophidian envenomation and the biology of snakes. An understanding of the evolution, biology and taxonomy of snakes will make the understanding and treatment of envenomation more meaningful.

SECTION I – SNAKES

1. EVOLUTION

The exact evolutionary origins of snakes is still clouded in uncertainty and an inadequate fossil record. However, most popular theories suggest that snakes derive from burrowing or aquatic lizard-like ancestors. Certainly there are burrowing lizards today, which are often mistaken for snakes to those unfamiliar with reptile taxonomy. There is fossil evidence of snakes or snake-like animals extending back to the lower Cretaceous, about 120 million years ago.[6]

Amongst the earliest true snakes discovered is *Dinilysia* from the upper Cretaceous of Patagonia.[7] Early Typhlopid and Boid snakes are represented at least as far back as the Eocene, about 50 million years ago. The modern families of snakes, including the Colubridae, Elapidae, and Viperidae are known from Miocene fossils, about 15–25 million years ago.[7]

Clearly, the comparatively recent evolution of the Colubrids, Elapids, and Viperids provides some interesting questions for biogeographers and systematists, as these groups would have evolved well after the break-up of Gondwanaland. This provides problems in determining the systematic position of Elapid snakes, the major group in Australia. This family, the Elapidae, is represented on all continents (except Antarctica), with a diverse range of species. However, it is in Australia that this group predominates over all other families of snakes. It is also in Australia that the most potent of all ophidian venoms have evolved.

When considering taxonomy, it is easy to accept a static picture of species unchanging, with a series of groups grading from primitive to advanced in any given set of animals. However, evolution is a continual process, applying to all living organisms. Snakes are undoubtedly still evolving. As each species evolves so its biology will change, including venom structure. Species with a large distribution, containing many isolated populations, may show significant differences between individuals of different populations. Each population may have its own distinctive venom components, as well as components

common to all populations. The rate of increase in change between populations will depend on a large number of variables. In Australia, Black Tiger Snakes are an example of numerous isolated populations with venom diversity between populations. This problem is currently being studied in detail.[8]

Populations need not be isolated to allow evolution of venom diversity within a species. Experience has confirmed that where a successful species is spread continuously over a large range, specimens taken from widely separated parts of the range can show important differences in venom.[9] These differences can be so great that anti-venom prepared to the venom of specimens in one part of the range can be totally ineffective in treating bites by the same species of snake from a different part of its range.

2. TAXONOMY

Despite the continuing evolution of snakes, in nearly all cases clearly defined species can be recognized. The groupings above species are less easy to define, the higher groupings being the subject of continuing debate and change. The most commonly accepted taxonomy has been used in this article.[6]

Snakes are but one sub-order of one of four orders of living reptiles (Table I). There are approximately 6,400 species of living reptiles, of which about 6,000 belong to one Order – Squamata. This Order contains three sub-orders: Sauria, the lizards, with about 2,800 species; Amphisbaenia, worm-like reptiles without legs, with about 100 species; Serpentes, comprising the snakes, about 3,300 species.

The success of snakes as a group is reflected in the diversity and number of living species. They are grouped in 11 families (Table II), six of which occur in Australia. Many taxonomists group the families of snakes into infra-orders. This grouping places the four modern families of snakes, Colubridae, Elapidae, Hydrophiidae, and Viperidae, into one infra-order, Caenophidia.

All these families are widely distributed. The vast majority of species (about 2,500) occur in one family, Colubridae. All species in the other three families are venomous, though not necessarily dangerous to man.

Until recently it was believed that the majority of Colubrid snakes were non-venomous. Recent evidence from a variety of studies suggests this is not the case.[10] It would appear that many Colubrid snakes do have venom glands, but lack adequate venom delivery apparatus. However, very few Colubrid snakes are potentially dangerous to man. Of these few, probably the best known is the Boomslang (*Dispholidus typus*) of Africa.

3. THE SNAKE FAMILIES

(a) Definitions:

Snakes are a diverse group of animals, with diverse life styles and anatomical modifications for these life styles. However, some features are basic to all species and define them from other reptiles.

(i) There is an absence of any functional limbs although some primitive species have vestigial pelvic girdles and hind limbs.

(ii) The body is covered with overlapping scales, which may be smooth, keeled, or granular.

(iii) The eyelids are fused to form a transparent spectacle over the eye, which is shed as part of the slough, periodically.

(iv) There is no external ear, tympanic cavity, or Eustachian tube.

TABLE I: Classification of Class Reptilia

Class	*Order*	*Suborder*
Reptilia	Chelonia (tortoises and turtles)	Pleurodira
		Cryptodira
	Crocodilia (Crocodiles, Alligators, Caimans)	
	Rhynchocephalia (Tuatara)	
	Squamata (Lizards, Snakes, and Amphisbaenians)	Sauria (Lizards)
		Amphisbaenia (Amphisbaenians)
		Serpentes (Snakes)

TABLE II: Classification of snakes

Suborder	*Family*
SERPENTES (OPHIDIA)	Typhlopidae (Blind snakes)
	Leptotyphlopidae (Thread snakes)
	Anilidae (Cylinder snakes)
	Uropeltidae (Shield tail snakes)
	Xenopeltidae (Sunbeam snake)
	Boidae (Pythons and boas)
	Acrochordidae (File snakes)
	Colubridae (Colubrine snakes)
	Elapidae (Front-fanged snakes such as Cobras, mambas, Kraits, and Australian venomous snakes)
	Hydrophiidae (Sea-snakes)
	Viperidae (Vipers, adders and rattlesnakes)

(v) The mandible is highly mobile and is in two parts, joined in the midline by highly elastic tissue, allowing the whole mandible to separate. This enables the snake to swallow prey of greater diameter than the undistended mouth.

(vi) The tongue is bifid, retractile, and can be enclosed in a sheath in the mouth. It is used in conjunction with Jacobson's organ to detect the presence of prey or predators. This is akin to mammalian smell.

(vii) Skull mechanics are altered to allow swallowing of large prey. In particular, palatal bones are mobile, as may be the maxillae, palatine and pterygoid bones, allowing movement of sets of teeth in relation to prey and each other. This assists in ingestion of the prey. The teeth are usually sharp and recurved, and are replaced in perpetual succession. This includes fangs.

(viii) The vertebral column and ribs are modified with accessory articulating facets to allow the hypermobility necessary for the snake's locomotion.

(ix) Internal organs are modified in structure to sit within an elongated body cavity. The left lung is reduced or absent. Other paired organs are usually asymmetrically located.

(b) The Primitives

(i) Family *Typhlopidae* (Blind snakes)

Worldwide about five genera and 200 species. Adapted for a subterranean existence, usually only surfacing at night. Non-venomous. Eyes markedly reduced and vestigial.

(ii) Family *Leptotyphlopidae* (Thread snakes)

Worldwide about two genera and 50 species. Subterranean, vestigial eyes. Non-venomous.

(iii) Family *Aniliidae* (Cylinder snakes)

Worldwide three genera and nine species. Mainly subterranean. Eyes small. Non-venomous.

(iv) Family *Uropeltidae* (Shieldtail snakes)

Worldwide eight genera, about 40 species. Subterranean, with reduced eyes. Non-venomous.

(v) Family *Boidae* (Pythons and boas)

Worldwide about 22 genera and 90 species. Often large snakes, muscular, killing prey by asphyxiation. Non-venomous. May be terrestrial or arboreal.

(vi) Family *Xenopeltinae* (Sunbeam snake)

One species only. Burrowing species, small eyes. Non-venomous.

(vii) Family *Acrochordiae* (File snakes)

Worldwide about two genera, six species. Aquatic snakes, with granular scales. Fish eaters. Probably non-venomous.

(c) Advanced snakes

(1) Family *Colubridae* (Colubrine snakes)

The largest and most diverse family of snakes, with about 2,500 species in 250 genera. A wide variety of morphologies and life styles is extant in this family. Some are terrestrial, some arboreal, some aquatic. Dentition is varied. Some species have evolved fangs in the back of the mouth, others appear to have more anteriorly placed fangs. Although many appear to have venom glands, and produce venom, none appears to have sophisticated venom delivery apparatus. Hence few colubrine snakes are of medical significance.

(ii) Family *Elapidae* (Elapid snakes)

Worldwide about 41 genera and 180 species. This is the most significant group of snakes in Australia. The family includes the Cobras, Kraits, Mambas, and Coral snakes. It is snakebite by elapid snakes that is the largest single cause of ophidian envenomation fatalities in the world.[1]

Elapid snakes have small- to medium-sized fangs, in relation to head size. The fangs are near the front of the mouth, on the maxilla, and usually have an enclosed poison channel (diagram 1). The maxilla has limited mobility.

All elapid snakes are venomous, but only some have the combination of potent venom and effective delivery apparatus to render them potentially dangerous to man. In Australia, the majority of elapid snakes are not dangerous to man. They have adopted many life-styles, including burrowing (fossorial) species, but most are vagrant hunters.

(iii) Family *Hydrophiidae* (Sea snakes)

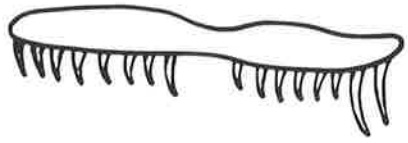
Worldwide about 16 genera and 50 species. All are marine or estuarine aquatic species, spending virtually their entire lives in water, where they hunt for fish and other prey. Their fang structure is similar to elapid snakes, and all species are venomous. Most are probably potentially dangerous to man.

(iv) Family *Viperidae* (Vipers, Adders, Pit-Vipers, Rattlesnakes)

Worldwide about 14 genera and 150 species. Viperid snakes are diverse in form and habit, but all are venomous and have incredibly efficient venom delivery apparatus (diagram 1).

Colubrine aglyphous maxilla

(no fangs)



Colubrine opisthoglyphous maxilla

(back fanged)

Elapid proteroglyphous maxilla

(front fanged)

Viperid proteroglyphous maxilla

(front fanged)

Diagram 1 - The maxilla and fangs in modern snakes

(after Webb, Wallwork, Elgood)

The long fangs, on highly mobile maxillae, can be erected to attack, and folded back when not in use.

Viperid snakes probably account for nearly half of all human ophidian envenomation fatalities. No viperid snakes are found in Australia.

4. SOUTH AUSTRALIAN SNAKES

(a) Identification of Snakes

The basic characteristics of snakes have been defined earlier. Each family of snakes has definite characters that separate its members from members of other families. Similarly, specific characters will define each genus and species. However, while much detailed taxonomy of snakes relates to osteology, particularly of the skull, there are some external characters which can be used in identification of each species.

Commonly used external characters are counts of the mid-body scale rows, the anal and subcaudal scales, and head scales. Colour may be of value in separating some species, especially the small burrowing elapid snakes, but for most dangerous species, colour is highly variable and is not a reliable diagnostic character.

Methods of counting scales, and nomenclature of head scalation are given in diagram 2. Information on identification of each species has been gleaned from Cogger[3] and examination of specimens in the South Australian Muscum. The estimated distribution of each species in South Australia is also based on Museum records, and each specimen of the species registered at the Museum is plotted. However, as our knowledge of reptiles in South Australia is far from complete, these maps should only be taken as an approximation of distribution. Due to habitat variability, there will be many areas included in each range which will not be suitable for the species in question. Similarly, the species may extend beyond the marked range, but we are not aware of this extension. (See appendix II.)

Nevertheless, these distribution maps should provide a helpful guide to the range of each species, and should permit the physician to narrow down the range of culprits in most cases of snakebite. This should allow for the mixing of two or three antivenoms, instead of using polyvalent antivenom in nearly all cases of snakebite by an undetermined species of snake. This will be discussed again in Section IV.

(b) Blind Snakes – family *Typhlopidae*

The blind snakes are widely represented in Australia but only four species are recorded from South Australia. They are probably common but are rarely seen because of their life style. They are burrowing snakes, only coming onto the ground surface occasionally, such as on warm summer nights. They are all small snakes and are completely harmless to man. They appear to feed on termites.

The eyes are reduced to small dark spots seen under the head scales. The mouth is usually situated behind a shovel shaped snout, to prevent soil entering the mouth during burrowing. The head is not distinct in diameter from the neck. There is a virtually constant-diameter, cylindrical body, with a very short tail which ends in a sharp point. This point is used to assist in burrowing, and contrary to some popular opinion, is *not* poisonous. It is merely a leverage device and not a sting.

– *Typhlina australis* (fig. 1)

Snout bluntly rounded from above, sharp and projecting in profile. Nasal cleft joining the second infralabial scale, and ending in front of the nostril. Rostral nearly as broad as long. Scales in 22 rows mid-body. Colour variable, but usually brown with pale flanks and white ventral surface.

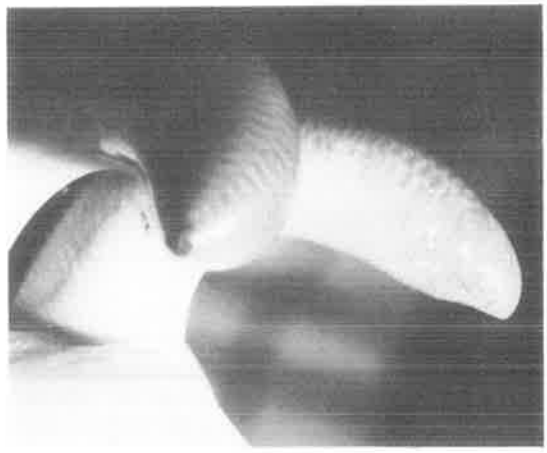
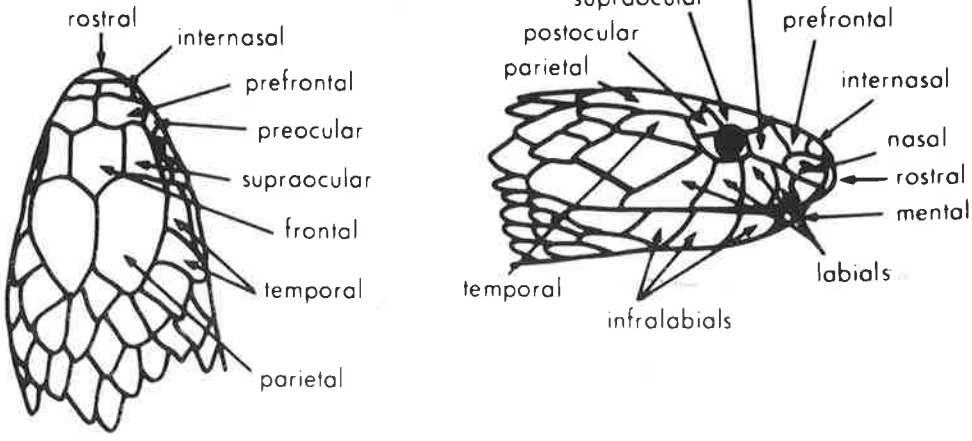
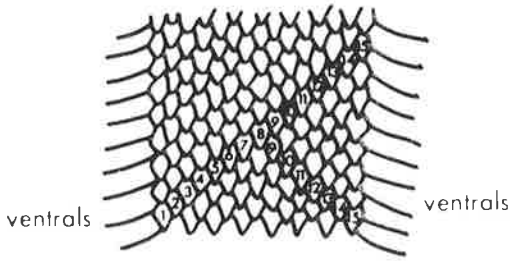


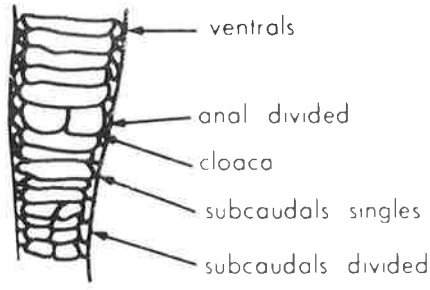
FIG. 1: Blind Snake, *Typhlina australis*. Typical shovel-like burrowing snout, and very short, sharp tipped tail.



Head scalation of an elapid snake



Midbody scale count



Underside of cloaca region
to show ventral, anal and subcaudal scales

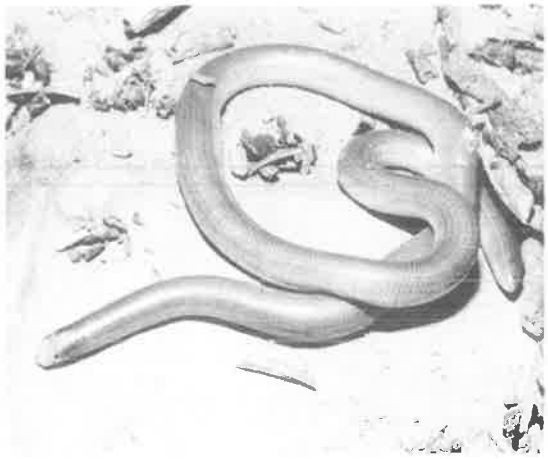


FIG. 2: Blind Snake, *Typhlina bituberculata*. Cylindrical body, reduced eyes, shovel-like rostral scale, trilobular.

– *Typhlina bituberculata* (fig.2)
Snout has distinctive double notched or trilobular appearance from above, and is sharply angular in profile. Nasal cleft does not divide nasal, contacts second labial inferiorly. Rostral longer than broad. Scales in 20 rows mid-body. Colour variable, usually brown to black above, and pale ventrally.

– *Typhlina endotera*
Snout bluntly rounded from above, sometimes slightly trilobular. Bluntly angular in profile. Nasal cleft joins preocular, doesn't extend beyond nostril. Rostral broadly oval, slightly broader than long. Scales in 22 rows mid-body. Colour variable, usually brown above, white ventrally.

– *Typhlina pinguis*
Snout slightly trilobed from above, blunt, bluntly angular in profile. Nasal cleft does not divide nasal, is not visible from above, contacts second labial below. Rostral circular from above. Scales in 20 rows mid-body. Colour variable, usually dark above, pale to white ventrally.

(c) Pythons – family *Boidae*

The pythons are widely distributed in Australia, but there are only three species recorded from South Australia, and all are rarely seen. They are mostly nocturnal in habit, and feed on a variety of prey including mammals, birds, and reptiles. They are non-venomous, and kill prey by asphyxiation. They are occasionally seen in the daytime sunning themselves.

They are often large, slow moving snakes, and have cloacal spurs, which are vestigial hind limbs, adjacent and lateral to the cloaca bilaterally.

– *Aspidites ramsayi* (Woma Python) (Colour plate 1)
Scales smooth, in 50–60 rows mid-body. About 280–315 ventral scales, with single anal, and about 50 subcaudals, mostly single. May reach 2.7 metres in length.
Colour is variable, but usually dark yellow brown above, with regular darker bands which may be indistinct. The head is usually of the same colour as the body in adults, but tinges of black, especially around the eyes, may be seen in juveniles.
Typically found in arid, sandy areas, especially sandy deserts. Common in the north-east of the State, around the Moomba area gas fields where it is sometimes found in earthworks and pipeline trenches in the morning.

– *Liasis childreni* (Children's Python)
Scales smooth, in 37–49 rows mid-body. About 255–300 ventrals, with single anal, and about 30–45 subcaudals, mostly divided. May reach 1.5 metres in length, though more commonly adults are about 0.8 metres.
Colour pattern is distinctive (fig. 3), with a base light brown above, paler

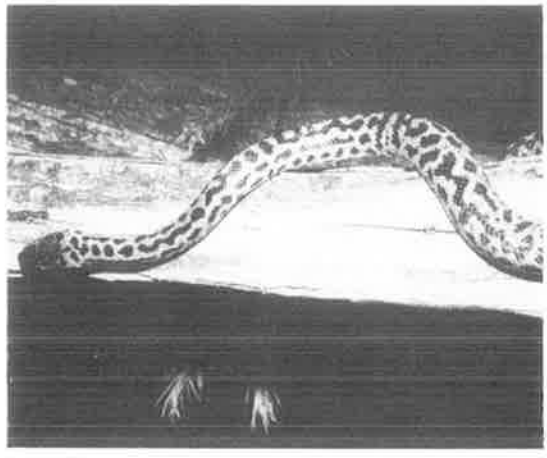


FIG. 3: Children's Python, *Liasis childreni*. Typical dorsal pattern. Head slightly distinct from neck.

on the sides, and pale to white ventrally. The upper surface has a series of irregular dark brown blotches. There are distinct labial heat sensing pits.

Typically found terrestrially, though occasionally arboreal. Usually associated with rock outcrops, such as the mesas and buttes of upper Eyre Peninsula. Also found in the Flinders Ranges and further north. Only found rarely, and it is probably an uncommon to rare snake in South Australia; though very common in northern states.

– *Python spilotes variegata* (Carpet Python)

Scales smooth, in 40–65 rows mid-body. About 240–310 ventrals, with single anal, and about 60–95 subcaudals, usually divided. May reach four metres in length, though two metres is more usual.

Colour pattern is distinctive (fig. 4a), as is head shape, and there are prominent heat sensing pits on the side and front of the head (fig. 4b). The base colour above may be grey, to red to brown, with distinctive dark blotching producing a superbly camouflaged pattern.

Typically found in wetter areas or near water, and is often arboreal. Feeds mainly on birds and mammals. In South Australia is most commonly encountered along the River Murray corridor, but is also found in scrubland on Eyre Peninsula, and in the lower Flinders Ranges.

(d) Elapid Snakes – family *Elapidae*

The bulk of South Australian snakes are elapids, and therefore are venomous, with anterior maxillary fangs. However, most are small, and few are potentially dangerous to man.

The taxonomy of elapid snakes is complex, and is not helped by the variability of important external diagnostic characters. Because of this, identification can be difficult, even for experts. Photographs of head scalation have been included for each species, in addition to the description and map. Photographs of important species will also aid identification.

– Genus *Acanthophis* – Death Adders

– *Acanthophis antarcticus* – Common Death Adder

Scales smooth to keeled, 21–23 rows mid-body. 110–130 ventrals, anal single, 40–55 subcaudals, mostly single. Very distinctive head shape, with small eyes and vertical slit pupil for nocturnal vision. Head shields smooth to strongly rugose. Short, thickset body, thin narrow tail, used as a lure.

Colour variable, from red to brown or grey dorsal ground colour, with irregular cross-banding, sometimes black edged (figs. 5, 6) (Colour plate 7).

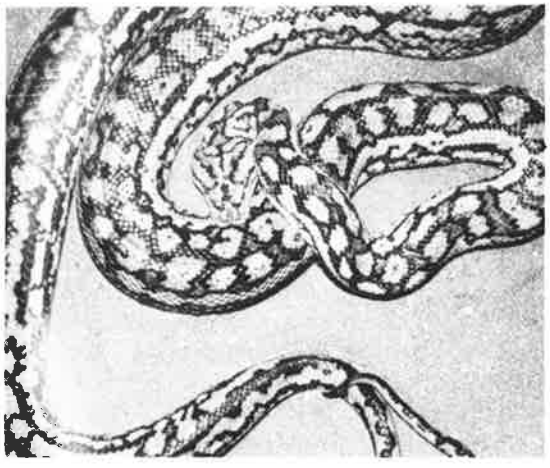


FIG. 4(a): Carpet Python, *Python spilotes variegata.* **Typical River Murray colour pattern. Head distinctively shaped, and very distinct from neck.**

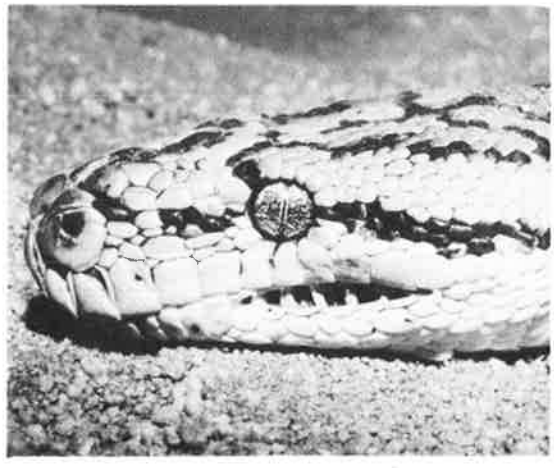


FIG. 4(b): Carpet Python, *Python spilotes variegata.* **Detail of head, showing anterior labial, and posterior infralabial scale infra-red sensing pit organs.**

FIG. 5.

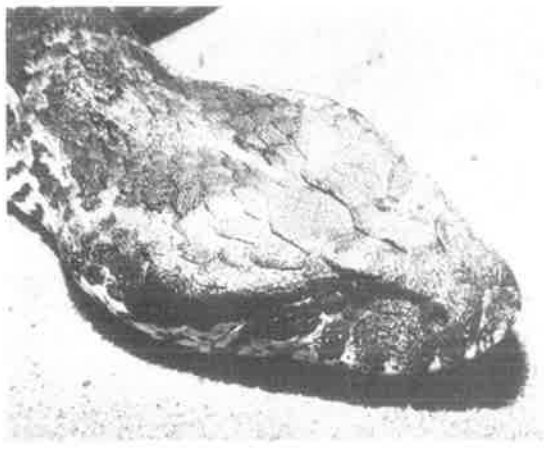


FIG. 6: Death Adder, *Acanthophis antarcticus*. Detail of head, showing distinct triangular shape, nocturnal adapted slit pupil, and keeled neck scales.

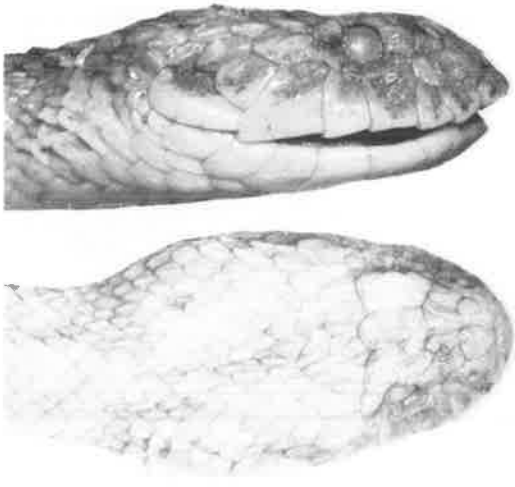


FIG. 7.

Found in a variety of habitats. Strictly terrestrial, and often found hidden in leaf litter or similar debris, from whence it is often reluctant to retreat on the approach of man. Most active at night.

– *Acanthophis pyrrhus* – Desert Death Adder

Very similar to the Common Death Adder, except for its strongly keeled scales, and more rugose head shields. Mid-body 21 rows, 140–160 ventrals, anal single, 45–60 subcaudals.

Colour is usually lighter than Common Death Adder, usually reddish in ground colour (fig. 7).

Habits similar to Common Death Adder, but found in more arid regions.

– Genus *Austrelaps* – Copperheads

– *Austrelaps superbus* – Common Copperhead

Smooth scales, 15 rows mid-body, lateral scales enlarged. 140–165 ventrals, anal single, subcaudals 35–55 single. Head is narrow and only faintly distinct from neck. Head scalation with absent suboculars, internasals present. Maximum length 1.7 metres, average 1.3 metres.

Colour highly variable but in south-east S.A. population, dorsally red-brown, becoming copper-coloured laterally. Labial scales typically with white edging producing a characteristic barred appearance (figs. 8, 9) (Colour plate 8).

Terrestrial, found in association with water and wetlands such as the swamp country of south-east S.A. Both diurnal and nocturnal. Not aggressive. Common, often in large aggregations in the south-east.

– *Austrelaps sp.* – Adelaide Hills Copperhead

Similar to Common Copperhead but considerably smaller, usually only 0.7 – 0.9 metres in length.

Colour usually grey above and pale ventrally, with white edging to labial scales giving distinctive appearance of lip as seen in Common Copperhead (figs. 10, 11) (Colour plate 9).

Terrestrial, found in wetter areas, though not necessarily near swamps. Found in sclerophyl forest in the

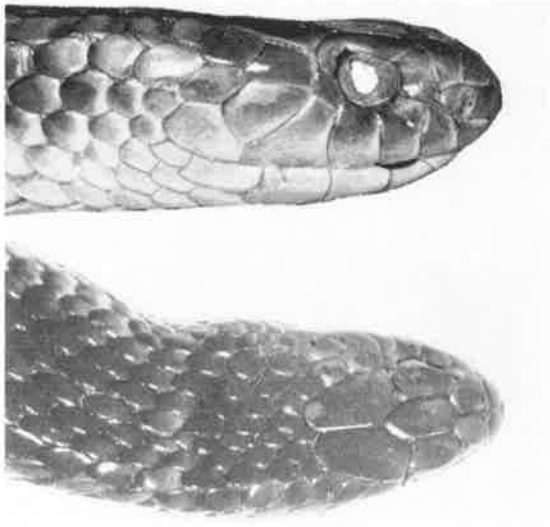


FIG. 8.

FIG. 9: Common Copperhead, *Austrelaps superbus*. Typical south-east colour phase, with white areas on each labial scale.

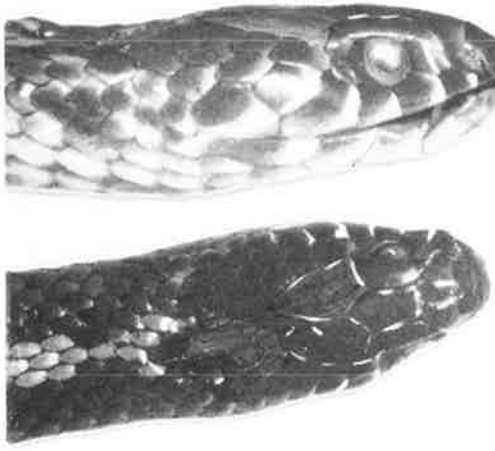


FIG. 10.

Adelaide Hills. Common in the Mount Lofty Ranges, including market garden areas. Also on Kangaroo Island and near mouth of River Murray. Diurnal, may also be nocturnal. Common in much of its range.

– Genus *Demansia* – Whip-snakes

– *Demansia psammophis* – Yellow-faced Whip Snake

Smooth scales, 15 rows mid-body. 165–230 ventrals, anal divided, subcaudals divided 60–105. Characteristic feature is large eyes, diameter of eye being greater than distance from edge of eye to mouth. Nasal and preocular scales in contact. About 0.8 metres long.

Colour distinctive in pattern though variable in colour. Typically the head and anterior body are russet grey green, and the rest of body grey green. The eye is ringed by a pale circle (figs. 12, 13).

Terrestrial, but found in most arid habitats. Very common, and active diurnal snake, rapid moving.

FIG. 11: Adelaide Hills Copperhead, *Austrelaps sp.* Detail of head, with fine white edging to labials and some other head scales.

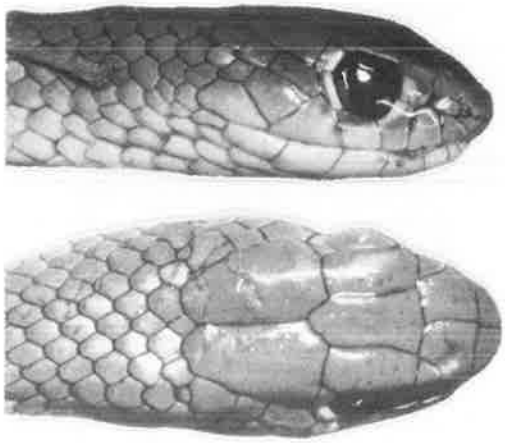


FIG. 12.

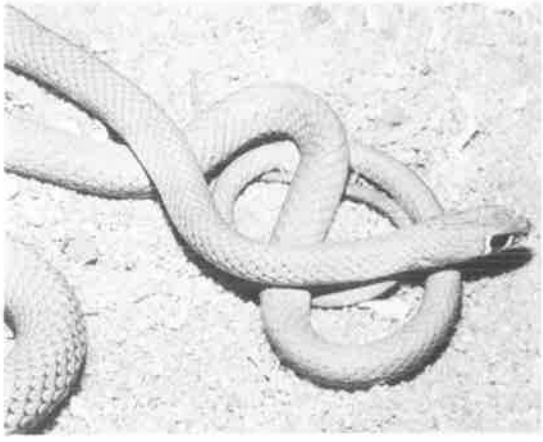


FIG. 13: Yellow-faced Whip Snake, *Demansia psammophis*. Typical colour phase, with very large eye in comparison to head size, and pale ring around eye.

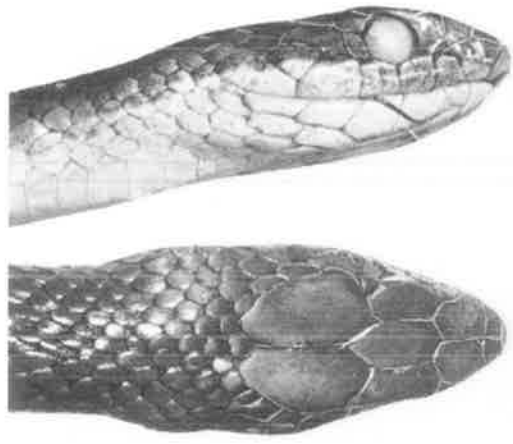


FIG. 14.

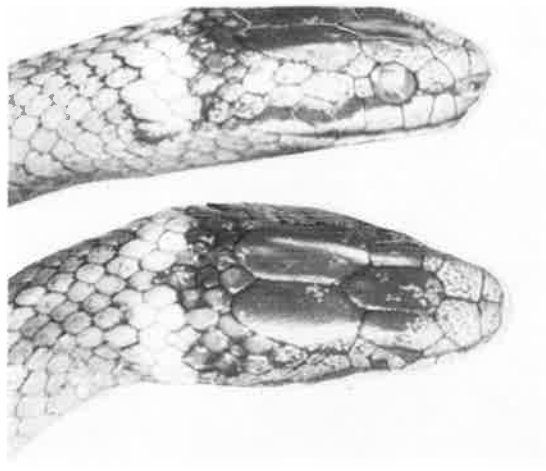


FIG. 15.

– Genus *Drysdalia*

– *Drysdalia coronoides* – White-lipped Snake

Smooth scales, 15 rows mid-body. 130–160 ventrals, anal single, subcaudals single, 35–60. Frontal much longer than broad, usually less than $1\frac{1}{2}$ times as wide as the supraocular. Internasals present. About 0.4 metres long.

Colour variable, usually grey dorsally, with a pale stripe from the snout along the upper lip and fading out along the neck (fig. 14).

Terrestrial, fossorial, found in high rainfall areas. Little known of habits, though may be nocturnal.

– *Drysdalia mastersi* – Masters Snake

Scalation and size as for the White-lipped Snake. However, colouration is distinctive, with usually a grey colour dorsally, dark head, with a pale yellow or orange band or collar immediately behind it. There may be a pale streak from nostril to eye and a darker streak from eye to back of head (fig. 15) (Colour plate 2).

Terrestrial, fossorial, more widely distributed than the White-lipped Snake. Common in low scrub within its range.

– Genus *Echiopsis*

– *Echiopsis curta* – Bardick

Smooth scales, 19 rows mid-body. Ventrals 130–145, anal single, subcaudals single, 30–40. Head is distinctively broad and flat. Suboculars absent, internasals present. Up to 0.6 metres long.

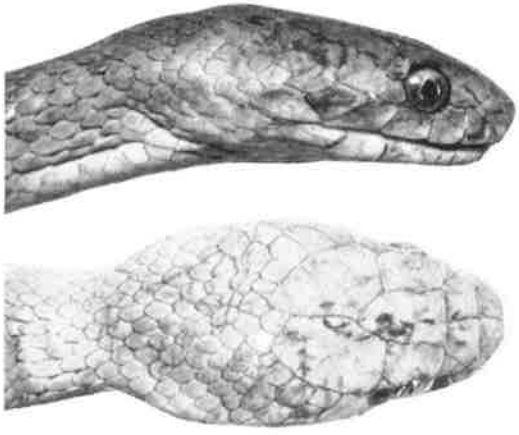


FIG. 16.

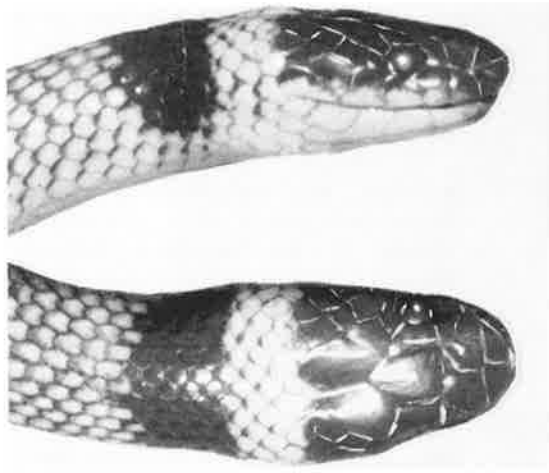


FIG. 17.

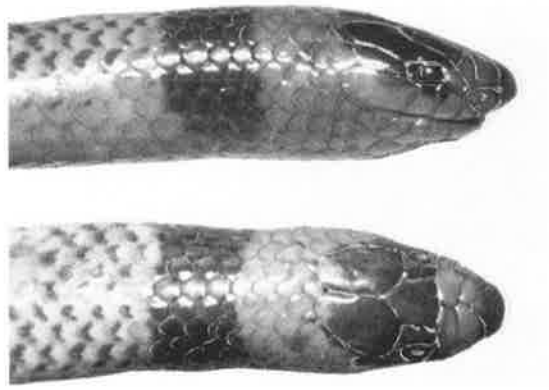


FIG. 18.

Usually brown to russet coloured uniformly, with white specks laterally around the head. Ventrally pale grey-brown (fig. 16) (Colour plate 3).

Terrestrial, fossorial, probably nocturnal. It is only rarely found in S.A., and little is known of its life style.

– Genus *Furina*

– *Furina diadema* – Red-naped Snake

Smooth scales in 15 rows mid-body. Ventrals 160–210, anal divided, subcaudals 35–70, divided. Nasal undivided, widely separated from preocular, suboculars absent. About 0.4 metres long.

Colour pattern distinctive, with dark brown or black head and neck, with a bright orange or red bar on the nape of the neck. Rest of body reddish-brown dorsally, often with dark edging to the scales. Ventrally white or cream coloured (fig. 17).

Terrestrial, probably nocturnal, often found in or near termite or ant colonies. May be aggressive when threatened, striking wildly.

– Genus *Neelaps*

– *Neelaps bimaculatus* – Western Black-naped Snake

Smooth scales in 15 rows mid-body. Ventrals 175–235, anal divided, subcaudals 15–35, divided. Nasal in contact with preocular, 5 upper labials, rostral not sharp-edged or shovel shaped (thus distinguishing it from similar *Simoselaps* species). 0.4 metres long.

Colour usually pale red-brown dorsally, with dark edging to each scale. Dark to black area on head from frontal to parietals, and a black nuchal band, commencing posterior to the parietals. Ventrally pale (fig. 18).

Burrowing species. Little is known of this snake which is very rarely seen in S.A.

– Genus *Notechis* – Tiger Snakes

– *Notechis ater.* – Black Tiger Snake

There are several subspecies of this snake, which will not be dealt with here. Smooth scales in 17 or 19 rows mid-body, ventrals 155–190, anal single, subcaudals 40–60, single. Head characterized by large squarish frontal shield, about as long as broad, lower anterior temporal large, as long or longer than frontal, suboculars absent, internasals present. Up to 1.5 metres long.

Colour consistently black dorsally, and dark to grey or pale ventrally. Juveniles may show some suggestion of banding dorsally (fig. 19) (Colour plate 10).

Terrestrial, found in a variety of habitats. On mainland, is usually found near water, such as along the creeks of the southern Flinders Ranges (e.g. Separation Creek, Spring Creek), and the wetland areas on the tips of Yorke and Eyre Peninsulas, and at the River Murray mouth. Also found on Kangaroo Island, and on many of the islands off Eyre Peninsula, where they are commonly found in association with Mutton Bird nest holes.

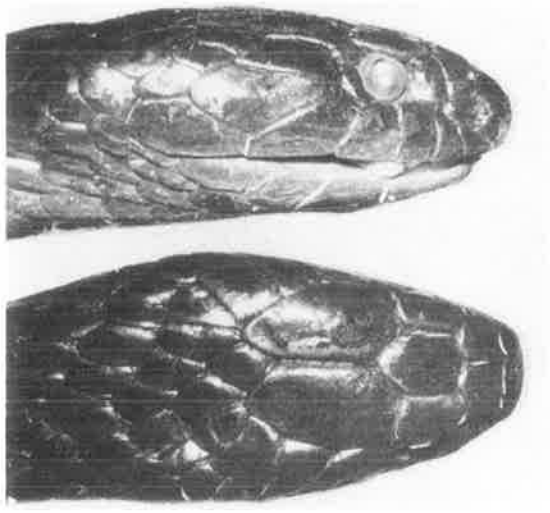


FIG. 19.

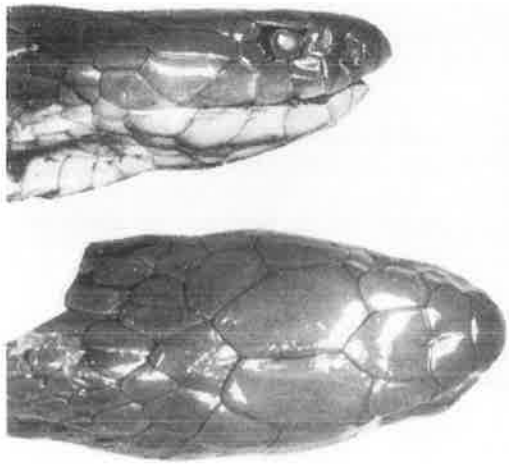


FIG. 20.

– *Notechis scutatus* – Mainland Tiger Snake

Scalation similar to Black Tiger Snake. Scales smooth in 17 rows mid-body, ventrals 140–190, anal single, subcaudals 35–65, single. Up to 1.2 metres long.

Colour is variable. Typical specimens show distinct banding dorsally, with intermittent bands of blue-grey and yellow-orange-brown. However dorsal colour is very variable, and banding may be absent, with a uniform brown to russet colour dorsally, especially in specimens from south-east S.A. Ventrally the colour may vary from pale cream, through yellow to grey (fig. 20) (Colour plates 11, 12).

Terrestrial. Usually found in association with water such as along the River Murray corridor, where it is very common, in the south-east of S.A., where it is common, and also in some sclerophyll forest in the Mount Lofty Ranges, near Woodside.

– Genus *Oxyuranus* – Taipan and Small-Scaled Snakes

– *Oxyuranus microlepidotus* – Small-Scaled Snake

Smooth scales in 23 rows mid-body, ventrals 220–250, anal usually single, subcaudals 55–70, divided. Subocculars absent. Up to 2.5 metres long.

Colour variable, but usually brown dorsally, with dark edging to scales, and occasionally showing faint banding. Head frequently dark to black, deeper colour extending onto neck. Ventrally pale cream, often with dark edging to ventral scales (fig. 21) (Colour plate 19).

Terrestrial. Details of the life style of this species are still being elucidated. Ms. J. Covacevich of the Queensland Museum has suggested that this snake is restricted to black-soil plains type habitat, where it may hunt plague rats in the cracks beneath the surface. However, it has been found in other habitats in South Australia, including canegrass sand-dune country near the Moomba gas fields, and along the Birdsville track. It is only rarely seen, but may be moderately common in its range. Usually

Figure 1

Figure 2

FIG. 1 Woma Python, *Aspidites ramsayi*. Juvenile, with typical black colouration above eyes. Collected near Moomba, S.A.

FIG. 2 Masters Snake, *Drysdalia mastersi*. Adult, with typical dark head, orange yellow nuchal band and grey body. Collected in Lincoln N.P., S.A.

FIG. 3 Bardick, *Echiopsis curta*. Adult with typical colour, and head well distinct from neck. Collected on central Eyre Peninsula, S.A.

FIG. 4 Desert Banded Snake, *Simoselaps bertholdi*. Typical adult with black and orange banding. Collected near Whyalla, S.A.

FIG. 5 Curl Snake, *Suta suta*. Typical sheeny brown body, with dark brown head. Collected at Murnpeowie Station, S.A.

Figure 3

Figure 4

Figure 5

Figure 6

Figure

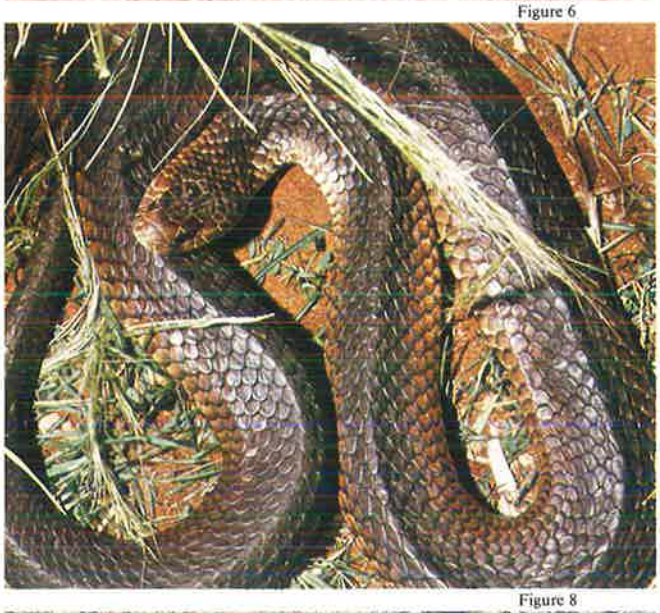


Figure 8

FIG. 6 Mitchell's Short-tail Snake, *Unechis brevicaudus*. **Typical northern colour phase with black head and dark vertebral stripe. Collected at Mount Searle, S.A.**

FIG. 7 Death Adder, *Acanthophis antarcticus*. **Typical colour phase. Collected at Tiddy Widdy Beach, S.A.**

FIG. 8 Common Copperhead, *Austrelaps superba*. **Typical colour phase from south-east S.A. Collected near Penola, S.A.**

FIG. 9 Adelaide Hills Copperhead, *Austrelaps sp*. **Typical colour phase from Mount Lofty Ranges, with grey body, and white edging to labial scales. Collected near Cleland C.P., S.A.**

FIG. 10 Black Tiger Snake, *Notechis ater*. **Typical all black colouration. Collected on Reevesby Island, S.A.**

Figure 9

Figure 1

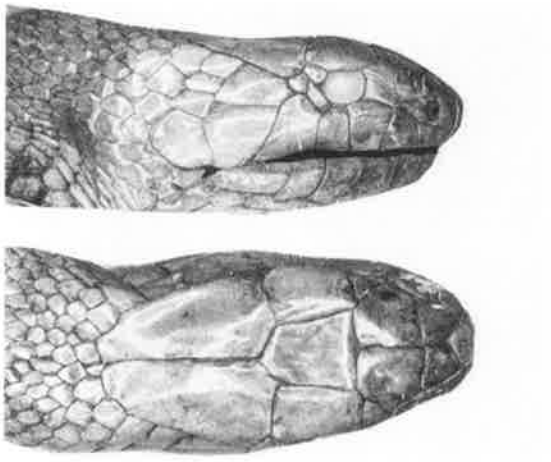


FIG. 21.

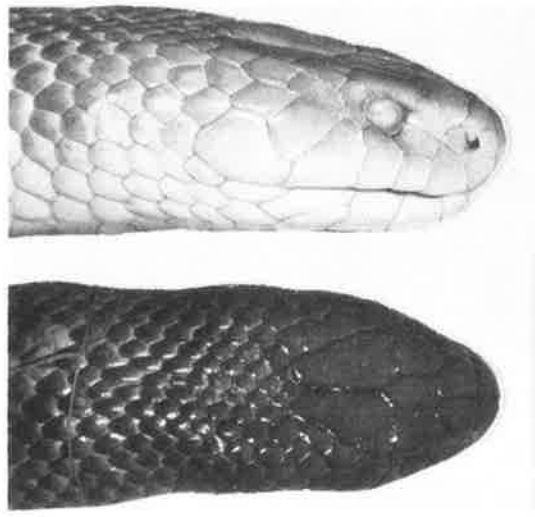


FIG. 22.

placid, but some specimens very aggressive, hence its alternative common name of Fierce Snake.

– Genus *Pseudechis* – Black Snake Group

– *Pseudechis australis* – Mulga Snake or King Brown

Smooth scales in 17 rows mid-body. Ventrals 185–225, anal divided, subcaudals 50–75, anteriorly single, posteriorly divided. Suboculars absent. Up to two metres long.

Colour variable, from pale copper, through russet colour, to brown, or even deep brown black dorsally. Scales often have pale to yellow inner margins, and darker brown outer margins, giving a reticulated appearance. Ventrally pale cream to orange, often with orange to brown blotches (fig. 22) (Colour plate 13).

Terrestrial. Thrives in a wide range of arid and semi-arid habitats. Diurnal predominantly, but nocturnal during hot weather. Often aggressive. A large, powerful snake, common in most of its range.

– *Pseudechis porphyriacus* – Red-bellied Black Snake

Scales smooth in 17 rows mid-body, ventrals 180–210, anal usually divided, subcaudals 40–65, usually single anteriorly and divided posteriorly. Suboculars absent. 1.5 metres long.

Colour black dorsally, merging to bright port-wine red ventral scales ventrally, which usually have black edging. Some specimens may be black ventrally (fig. 23) (Colour plate 14).

Terrestrial. Usually found along creeks, and in wetlands, where it can be very common. Diurnal. Very common along many creeks on both sides of the Mount Lofty Ranges.

– Genus *Pseudonaja* – Brown Snakes

– *Pseudonaja modesta* – Ringed Brown Snake

Scales smooth in 17 rows mid-body. Ventrals 145–175, anal divided, subcaudals 35–55, divided. Nasal and preocular scales in contact, suboculars absent. 0.45 metres long (fig. 24).

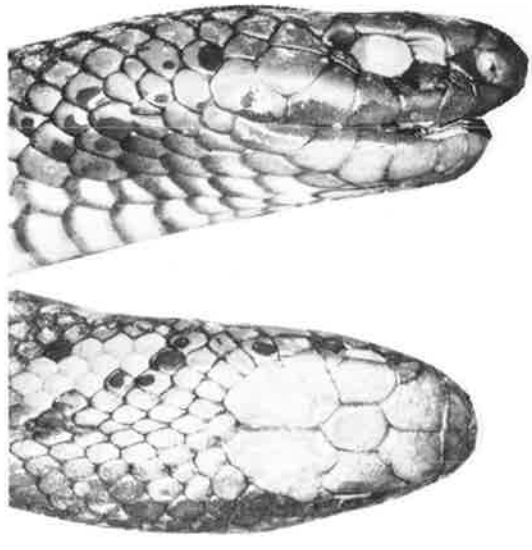


FIG 23.

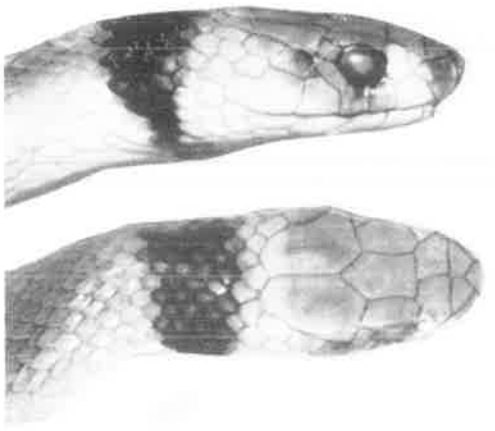


FIG. 24.

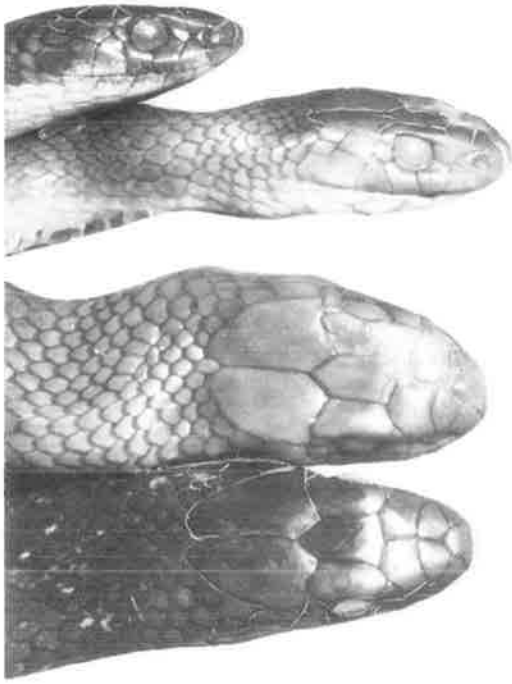


FIG. 25.

Colour variable, usually brown dorsally with grey to black bands, up to five in number, the most prominent being on the neck. The head is usually dark brown to black, and there may be a pale cream bar anterior to the black neck bar. The rest of the body bars are very widely spaced apart, are narrow, and often fade partially or completely in adult specimens. Ventrally variable from cream to dark orange, often with darker blotches.

Terrestrial. Usually diurnal though nocturnal in hot weather. Found in a variety of arid and semi-arid habitats. Moderately common.

– *Pseudonaja nuchalis* – Western Brown Snake or Gwardar

Scales smooth in 17 rows mid-body. Ventrals 180–230, anal divided, subcaudals 50–70, divided. Head scalation similar to the Ringed Brown Snake. Up to 1.5 metres long. Inside of mouth typically dark in colour.

Colour highly variable. Dorsal colour may be uniform, speckled, dark on the head and neck, or even semi-banded. Ground colour can vary from a very pale brown to almost black. Ventrally also variable, from cream to deep orange with or without darker speckling (fig. 25).

Terrestrial. Diurnal usually, but often nocturnal in hot weather. Found in a great variety of arid and semi-arid habitats, and frequently found in association with man, such as in rubbish dumps and around and underneath houses. Rapid moving and often aggressive. Common.

– *Pseudonaja textilis* – Common Brown Snake

Smooth scales in 17 rows mid-body. Ventrals 185–235, anal divided, subcaudals 45–75, paired usually, but occasionally single anteriorly. Head scalation as for the Ringed Brown Snake. Up to 1.5 metres long. Interior of mouth typically pale in colour.

Colour highly variable, as for Western Brown Snake. However most specimens are light to dark brown dorsally, with paler ventrals usually with dark orange speckling. Juveniles very variable, with dark head, followed by orange band, followed by a dark band on the neck, the rest dorsally brown. Some juveniles show continuous banding over the whole length of the body, or even speckling (figs. 26, 27, 28) (Colour plate 15).

Terrestrial. Diurnal and nocturnal in hot weather. Found in a vast array of habitats, from arid to sclerophyll and even wetlands, along creeks. This species is frequently found in association with man and is still prevalent in outer Adelaide suburbs. Very common. Often aggressive.

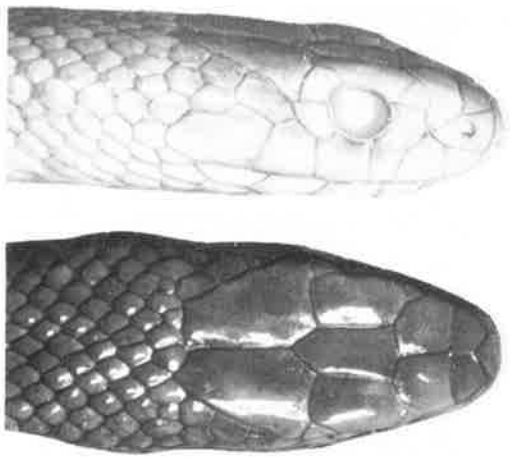


FIG. 26.

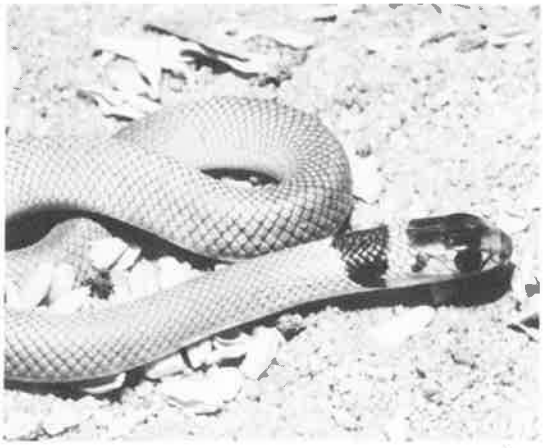


FIG. 27: Common Brown Snake, *Pseudonaja textilis*. Typical juvenile colour phase with black head dorsally, narrow pale anterior nuchal band, followed by broader black nuchal band.

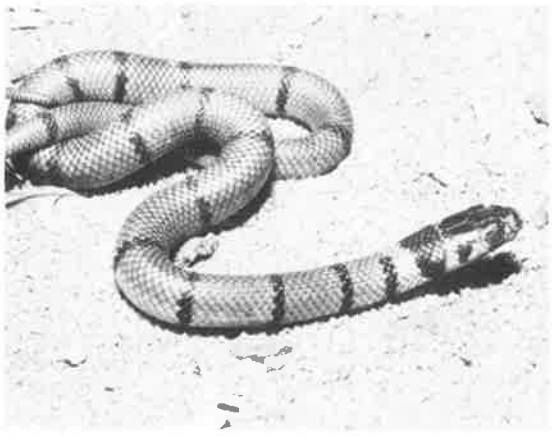


FIG. 28: Common Brown Snake, *Pseudonaja textilis*. A typical juvenile colour phase, with series of bands down whole length of body.

– Genus *Simoselaps* – Burrowing Snakes

– *Simoselaps australis* – Coral Snake

Scales smooth in 17 rows mid-body. Ventrals 140–170, anal divided, subcaudals 15–30, divided. Internasals present, no suboculars between eye and labials, rostral with a sharply upturned, angular, projecting edge. Up to 0.5 metres long.

Colour pale to dark pink to red dorsally, with numerous narrow bands of dark edged, pale centred scales. Usually has a black bar across the head, including eyes, and a second broad black bar on the nape. Ventrally pale (fig. 29).

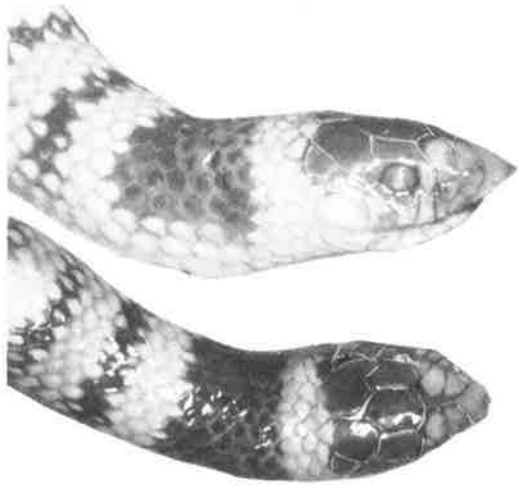


FIG. 29.

Burrowing species, only seen rarely, on the surface at night. Found in a variety of arid and semi-arid habitats.

– *Simoselaps bertholdi* –Desert Banded Snake

Scales smooth in 15 rows mid-body. Ventrals 100–135, anal divided, subcaudals 15–30, divided. Internasals present, no suboculars between the eye and labials. Nasal and preocular scales in contact. 0.3 metres long.

Colour yellow to orange-red dorsally, with distinct black bands along the whole body, extending onto the ventral surface, and as wide as the paler interbands. Head usually dark to black, but with pale snout (fig. 30) (Colour plate 4).

Burrowing species, common in most of its range and frequently found on the surface at night.

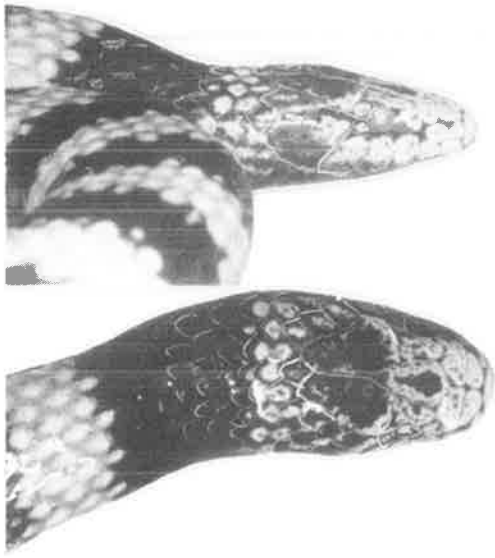


FIG. 30.

– *Simoselaps fasciolatus* – Narrow-banded Snake

Scales smooth in 17 rows mid-body. Ventrals 140–175, anal divided, subcaudals 15–30, divided. Internasals present, no suboculars between the eye and labials, nasal and preocular scales in contact. 0.3 metres long.

Colour light red-brown dorsally. Each scale edged with darker colour, some almost black. These darker tipped scales from numerous irregular cross-bands, sometimes fading caudally. Head black, with pale snout, pale area posterior to parietals, followed by black nuchal band. Ventrally cream to white (figs. 31, 32).

Burrowing species, rarely seen.

– *Simoselaps semifasciatus* – Half-girdled Snake

Scales smooth in 15 or 17 rows mid-body. Ventrals 140–190, and divided, subcaudals 14–30, divided. Rostral has a slightly upturned and angular anterior edge. Internasals present, no suboculars between the eye and labials. 0.3 metres long.

Colour variable, from pale fawn or cream, through brown to red-brown dorsally, with numerous darker bands of variable width. Dark to black bar across head enclosing eyes. May be a dark nuchal bar. Snout pale. Ventrally pale or white (figs. 33, 34).

Burrowing species, rarely seen.

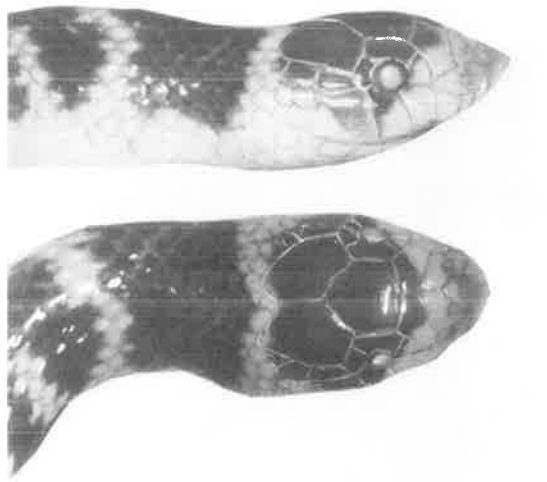


FIG. 31.

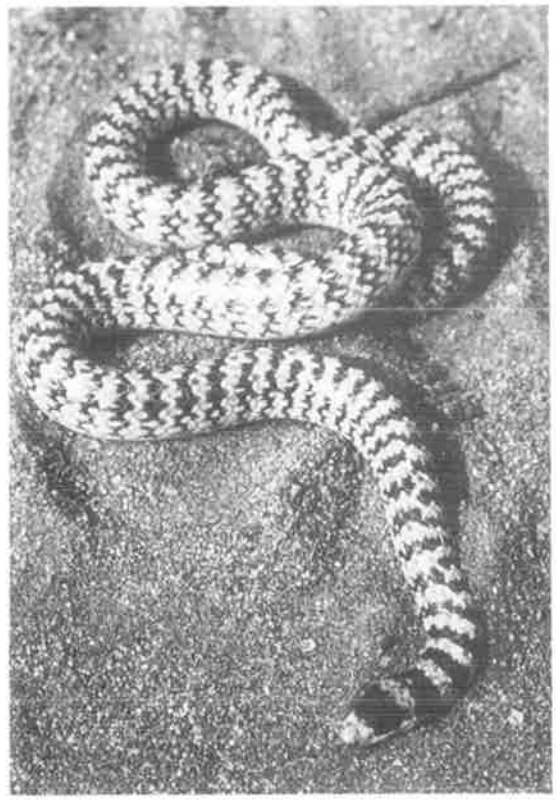


FIG. 32: Narrow-banded Snake, *Simoselaps fasciolatus*. Typical colour phase pattern, with a series of narrow dark to black bands.

– Genus *Suta*

– *Suta suta* – Curl Snake

Scales smooth in 19–21 rows mid-body. Ventrals 140–190, anal single, subcaudals 20–35, single. Suboculars absent, frontal longer than broad, internasals present. Up to 0.6 metres long, but usually 0.35–0.4 metres.

Colour pale brown to rich red-brown dorsally, sometimes with a dark portion on the inner edge of each scale. Head usually distinctively darker in colour, with a pale lateral stripe from canthus through eye, to temporal region, with darker region below, then paler supralabials. Ventrally pale (fig. 35) (Colour plate 5).

FIG 33.

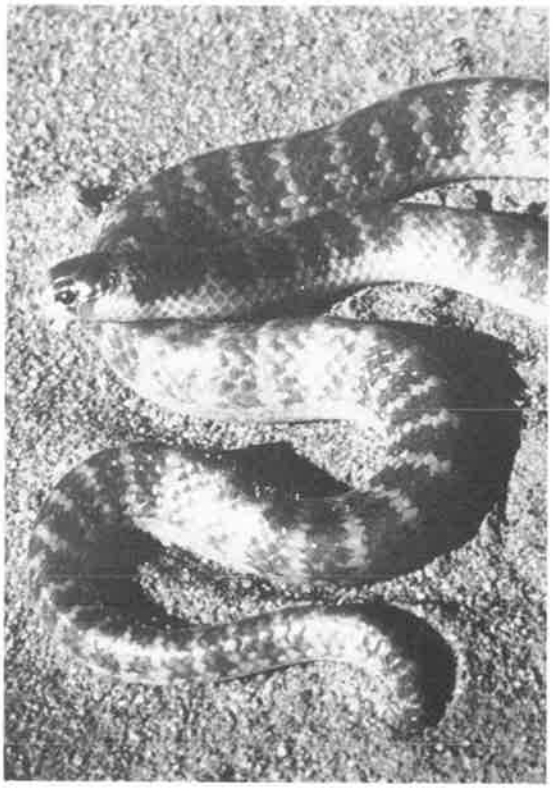


FIG. 34: Half-girdled Snake, *Simoselaps semifasciatus*. Typical colour phase pattern, with a series of broad dark bands.

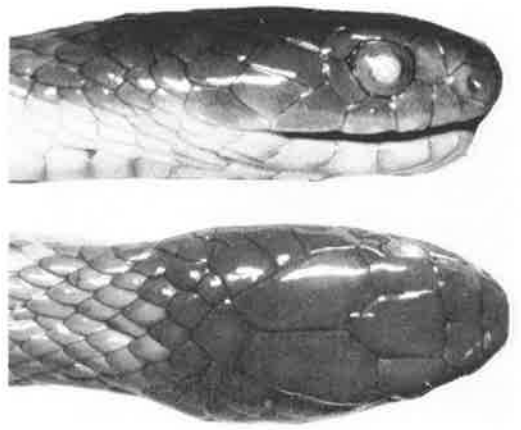


FIG. 35.

Probably fossorial species, usually found under debris such as logs, stones, and rubbish. Thought to be nocturnal, and found active in the open at night. Can be very aggressive if disturbed. Common.

– Genus *Unechis*

– *Unechis brevicaudus* – Mitchell's Short-tail Snake

Smooth scales in 15 rows mid-body. Ventrals 152–164, anal single, subcaudals 23–24 single. Nasal may or may not contact preocular, internasals present, no suboculars. 0.4 metres long.

Colour variable, from cream to dark brown dorsally, often with darker indistinctly edged stripe running from neck along back, vertebrally. Head and nape dark to black. Ventrally pale (fig. 36) (Colour plate 6).

Cryptic species usually found under logs, rocks or rubbish. Probably nocturnal. Probably common in much of its range.

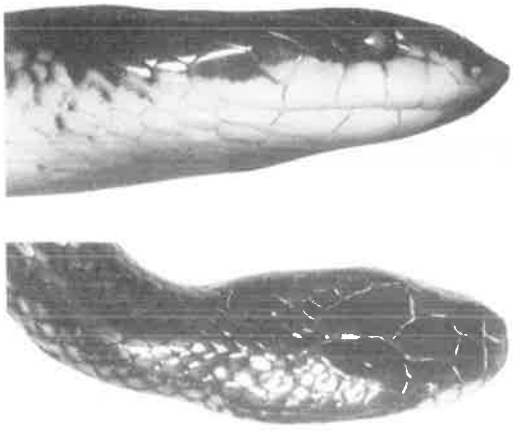


FIG. 36.

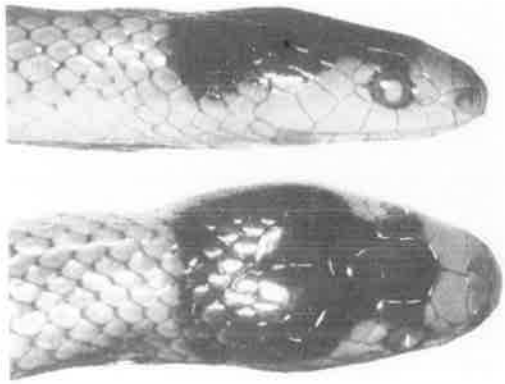


FIG. 37.

– *Unechis flagellum* – Little Whip Snake

Scales smooth in 17 rows mid-body. Ventrals 125–150, anal single, subcaudals 20–40, single. Nasal contacts preocular. No suboculars, internasals present. 0.4 metres long.

Colour usually brown dorsally, with black head extending onto neck, and paler region from snout to prefrontals. Head laterally paler. Ventrally pale, or occasionally dark (fig. 37).

Cryptic but common species, often found under logs, stones, or rubbish. Probably nocturnal.

– *Unechis gouldii* – Black-headed Snake

Scales smooth, in 15 rows mid-body. Ventrals 140–175, anal single, subcaudals 20–35, single. Nasal contacts preocular. Internasals present, no suboculars. 0.4 metres long.

Colour variable from tan to dark brown dorsally, scales often lighter edged, and occasionally with vertebral dark stripe from neck to tail. Head and neck dark to black, pale laterally and on snout, with a dark band nasal to nasal. Ventrally pale (figs. 38, 39).

Cryptic but common species.

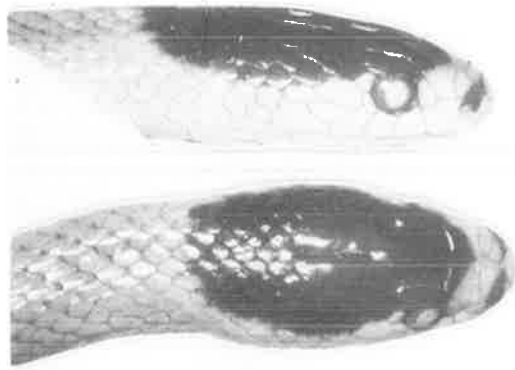


FIG. 38.

– *Unechis monachus* – Hooded Snake

Scales smooth in 15 rows mid-body. Ventrals 140–175, anal single, subcaudals 20–35, single. Single posterior temporal, internasals present, suboculars absent. 0.4 metres long.

Colour variable brown to russet dorsally with black head, and black scales extending to include nape giving black hooded appearance. Snout black. Head may be laterally paler. Ventrally paler (fig. 40).

Cryptic species. Probably similar in habit to the Black-headed snake.

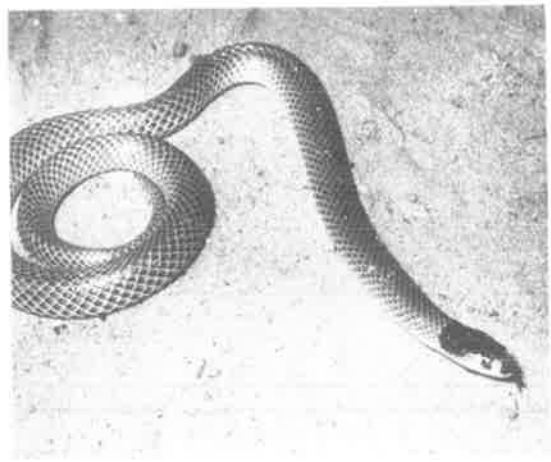


FIG. 39: Black-headed Snake, *Unechis gouldii*. Typical colour phase, with black head, dark russet brown body, and slight reticulation.

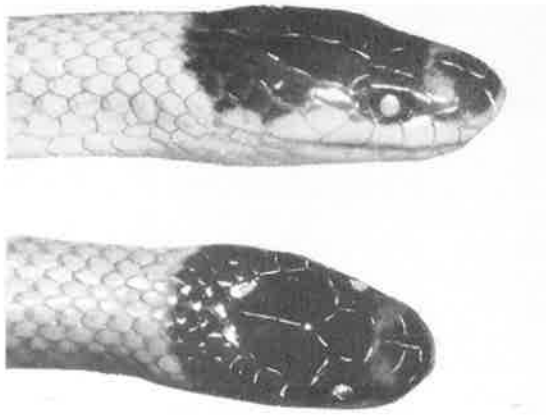


FIG. 40.

– Genus *Vermicella*

– *Vermicella annulata* – Bandy-Bandy

Smooth scales in 15 rows mid-body. Ventrals 180–230, anal divided, subcaudals 12–30, divided. Internasals present, suboculars absent. 0.4 metres long.

Colour very distinctive, with alternate black and white bands, commencing with a black snout. Bands usually extend to the belly (fig. 41).

Burrowing species, nocturnal, found through a great diversity of habitats. Probably common though rarely seen.

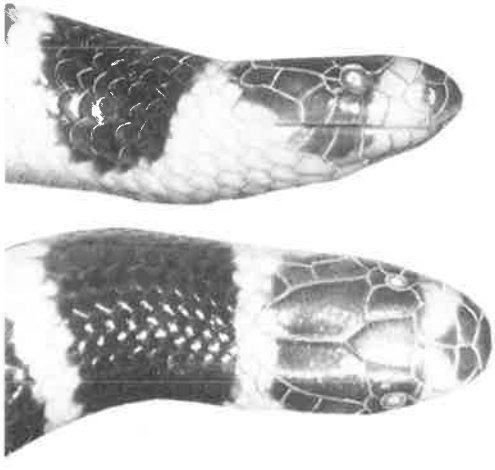


FIG. 41.

SECTION II – VENOMS

1. HISTORY OF VENOM RESEARCH

Snakes and snake venoms have always held a fascination for man. In many cultures snake venoms were used in medicine to produce cures for a variety of ailments. Doubtless much primitive research was involved in this use of venoms, but systematic research on venoms is a relatively recent development, which has followed development of ideas and research concepts in other areas, especially chemistry and physiology.

Pedler first proposed that venoms were protein-like substances in 1878.[11] In 1893 Martin separated Australian Black Snake venom into two components, one haemorrhagic; the other neurotoxic. By this time physiologists were using venoms to study the action of nerves. By the end of the 19th century the existence of a curarizing toxin in the venoms of Elapidae and Hydrophiidae had been shown.

With the further development of physiology and biochemistry, venoms were used extensively, both to study their intrinsic properties, and as powerful research tools in neuropharmacology, neurophysiology, and haematology. Undoubtedly snake venoms played an important part in our understanding of blood clotting mechanisms, and neuromuscular transmission, and in the latter field, continue to be of great importance. Elapid postsynaptic neurotoxins such as α-bungarotoxin (from the Krait, *Bungarus multicinctus*) or α-neurotoxin (from Cobras, *Naja naja*) have been of major importance in defining nicotinic acetylcholine receptors of skeletal muscle[12] and α-bungarotoxin has been instrumental in developing theories and assays for myasthenia gravis.[13]

2. VENOM RESEARCH IN AUSTRALIA

Early settlers to Australia soon discovered the lethality of Australian elapid snakes. Inevitably this prompted many people to find cures for snakebite. These were many and varied and few have validity. Campbell[14] has reported on some of the most controversial ones. Nineteenth century Australian medical literature abounds with treatments for snakebite, and discussion about these. This was especially so in the last two decades of the century, and at this time notable work was commenced on Australian elapid venom research.

C. J. Martin and F. Tidswell both contributed greatly in this area. Martin[15] investigated the venom of the Black Snake, *Pseudechis porphyriacus*, separating it into two components, via high-pressure gelatine filtration. The demonstration of more than one venom component, each component with different properties, helped to open the way to a better understanding of the complex Australian elapid venoms. Tidswell[16] investigated lethalities of various Australian snake venoms, and correlated this with epidemiological studies. As a result it was apparent that Australia had some of the most dangerous snakes in the world. In particular he noted a fatality rate of 50% for Death Adder bites, and 45% for Tiger Snake bites.

The next major contributors were active in the 1920's and 1930's. Amongst these the names of Sir Hamilton Fairley and C. H. Kellaway stand out. Both worked for a time at the Walter and Eliza Hall Institute, Melbourne, and over the course of a decade or so the workers at this Institute published a large number of papers on Australian elapid venoms.[17]

In March 1929, the first part of a *Symposium on Snake Bite* was published in the *Medical Journal of Australia*. Fairley here reviewed previous literature on the venoms and epidemiology of snake bite in Australia, and commenced research with Kellaway and others to expand on earlier research, particularly that of Tidswell. He also initiated research on production of antivenom to Tiger Snake and Death Adder venom. This work was done at the Commonwealth Serum Laboratories, under the direction of Dr. Morgan, then Director of C.S.L..Fairley examined the anatomy and physiology of venom production and delivery in detail[17,18], noting the relative efficiency of the Death Adder and Tiger Snake delivery apparatus compared to that of the Brown Snake. He concluded that the Death Adder was the most dangerous Australian snake possessing a "potent neurotoxic venom".

Fairley and Kellaway, using sheep, tested several Australian venoms, finding potent neurotoxins; a "haemorrhagin", causing multiple haemorrhages in viscera; a "thrombose"; an anticoagulant; and haemolysins.[17] Fairley and Splatt[19] investigated venom yields of Australian snakes, using the rubber diaphragm collection method still widely used. They successfully collected venom from Tiger Snakes, Death Adders, Copperheads, and Black Snakes, but were unable to milk venom from Brown Snakes.

Kellaway further advanced these studies, with detailed studies of each snake venom on a variety of experimental animals. In a series of papers from 1929 to 1937 he reviewed the venoms of the Tiger Snake *Notechis scutatus*,[21,37,39] the Copperhead *Austrelaps superbus*,[22,25] the Spotted Black Snake, *Pseudechis guttatus*,[23] the Death Adder, *Acanthophis antarcticus*,[24,25,27] the Taipan, *Oxyuranus scutellatus*,[26] the Black Snake, *Pseudechis porphyriacus*,[28,39] the Mulga Snake, *Pseudechis australis*,[29] the Brown Snake, *Pseudonaja textilis*[30] and the Black Tiger Snake, *Notechis ater*.[31,39]

However, while determination of venom yields, toxicities of whole venom, and clinical effects, was possible, fractionation of specific venom components was more difficult for these workers. In fractionation of Death Adder venom Kellaway *et al.*[27] used precipitation by heat and subsequent fractionation of the filtrate by half- and full-strength saturation with ammonium sulphate, and by extraction with 45% ethyl alcohol. They described a soluble fraction containing a "neurotoxic principle", and an insoluble fraction with feeble coagulant activity. Holden *et al.*[39] fractionated Tiger Snake and Black Snake venoms using ultra-filtration through pyroxylin membranes, and absorption.

Inability to further fractionate venom appears to have occasioned a halt in research in the late 1930's. Many years later, from the late '50s onwards, new techniques for fractionation allowed more comprehensive venom studies. In Europe, America, Japan and Taiwan detailed studies were performed and specific neurotoxins isolated, such as α-bungarotoxin.

In 1958, Doery,[40] working at C.S.L. successfully fractionated Tiger Snake venom, using resin chromatography and paper electrophoresis. She reported at least two different neurotoxins, as well as a coagulating fraction, haemolysin, hyaluronidase, phosphatases, and cholinesterase.

In 1972, Karlsson *et al.*[41] further fractionated Tiger Snake venom, using ion-exchange chromatography. They reported a very potent pre-synaptic neurotoxin, which inhibited the release of acteylcholine from motor nerve end

plates. They also reported four other potent post-synaptic neurotoxins.

In 1973, Datyner and Gage[42,43] confirmed the pre- and post-synaptic effects of Tiger Snake neurotoxins, concluding that the neurotoxins selectively disrupt the process linking membrane depolarization to the secretion of transmitter. They also noted that Tiger Snake antivenom did not counteract the pre-synaptic effects of the venom, once they had developed. Lane and Gage,[44] showed alterations in synaptic vesicle population and morphology following administration of Tiger Snake venom to toad neuromuscular junctions.

In 1976, Fohlman *et al.*[45] reported the isolation of Taipoxin, a potent pre-synaptic neurotoxin, from the venom of the Taipan. Komenskaya and Thesleff[46] showed that the pre-synaptic block was characterized by gradual reduction and finally cessation of transmitter release. Cull-Candy *et al.*[47] showed concomitant ultrastructural changes, with depletion of vesicles.

In 1979 Coulter *et al.*[48] isolated a pre-synaptic neurotoxin from the Brown Snake, which was more potent than any previous neurotoxin isolated from snake venoms.

Research into other venom components and into different venoms continues. Current research in Australia centres around studies of venoms not previously examined in detail and it seems likely that many more species of Australian elapid snake will prove to have potent venoms.[49] Sutherland *et al.* have initiated new research into the treatment of envenomation in Australia, resulting in major departures from old first-aid treatments which promise more effective treatment of snakebite, and other forms of envenomation.[50,51]

3. VENOM COMPONENTS

The snakes in the design of their venom are as cunning and evil as man has imagined. (Thesleff[61])

Elapid snake venoms contain a complex mixture of components which act in a variety of ways, only a small portion of which are presently understood. Furthermore, some components may act on a variety of different sites within the victim, thus showing a wide variety of actions which may have totally separate clinical significance.

In this paper, the actions of venom components will be subdivided on a clinical basis.

(a) Overall toxicities

The relative toxicities of the venoms of snakes from different species, genera, and families have been the subject of constant revision. New techniques have allowed more accurate assessment of toxicities, with revised LD_{50} and LD_{100} figures. Thus recent work[4] has shown that the venom of the Brown Snake, *Pseudonaja textilis*, had previously been under-estimated in toxicity because of absorption to glassware using a standard diluent. The change of diluent from saline to 0.1% bovine serum albumin allowed consistent results and a sixfold increase in measured lethality.

In addition, as new snake venoms are studied, so the list must be further revised. The re-recognition of a distinct species of Taipan-like snake, the Small-scaled Snake, *Oxyuranus microlepidotus* from south-east Queensland, north-west New South Wales, and north-east South Australia,[52] and subsequent research on its venom,[53] revealed a snake whose venom was more toxic than any other snake known.

Differences in techniques and type and size of laboratory animals used have led to a wide disparity in LD_{50} and LD_{100} figures from various studies. The most recent review of relative toxicities by Broad *et al.*[4] has avoided some of these problems by testing a wide selection of venoms in one laboratory under rigid control conditions. Broad's figures (Tables III, IV) shows that Australian elapid snake venoms are the most potent of all snake venoms, but that previous estimations of relative toxicity of Australian venoms were significantly inaccurate.

(b) Venom production and delivery

Just as there is a diversity of venom components, so there is a diversity of sizes and position of venom glands. However, in Australian Elapid snakes, the venom gland resides behind the eye, with a duct passing anteriorly beneath the eye to open next to the fang base. From here the venom travels in an enclosed groove from the base of the fang to an opening or papilla near the tip of the fang, on the anterior wall of the vagina dentis (diagram 3; fig. 42) (Colour plate 16).

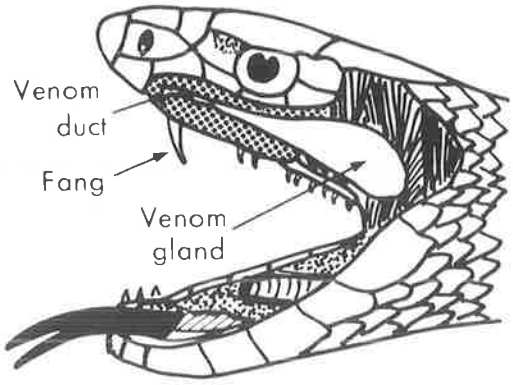


Diagram 3 - Position of venom gland, duct and fang in the Taipan

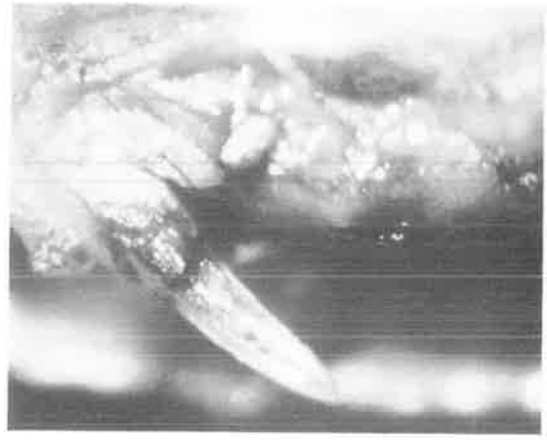


FIG. 42: Fang of Brown Snake, with duct ending at base of fang, and enclosed venom channel to opening near tip of fang.

TABLE III: Relative toxicity of snake venoms in mice
(after Broad *et al.*[4])

(S = sea-snake; E = elapid snake; EA = Australian elapid snake; V = viperid snake)

Snake		LD_{50} (Saline) in mg/kg	LD_{50} (bovine serum albumin)
Small-scaled Snake (*Oxyuranus microlepidotus*)	EA	0.025	0.010
Common Brown Snake (*Pseudonaja textilis*)	EA	0.053	0.041
Taipan (*Oxyuranus scutellatus*)	EA	0.099	0.064
Tiger Snake (*Notechis scutatus*)	EA	0.118	0.118
Reevesby Island Tiger Snake (*Notechis ater niger*)	EA	0.131	0.099
Beaked Sea Snake (*Enhydrina schistosa*)	S	0.164	0.173
W.A. Tiger Snake (*Notechis ater occidentalis*)	EA	0.194	0.124
Chappell Island Tiger Snake (*Noetchis ater serventyi*)	EA	0.338	0.271
Death Adder (*Acanthophis antarcticus*)	EA	0.400	0.338
Western Brown Snake (*Pseudonaja nuchalis*)	EA	0.473	0.338
Copperhead (*Austrelaps superbus*)	EA	0.560	0.500
Indian Cobra (*Naja naja*)	E	0.565	0.500
Dugite (*Pseudonaja affinis*)	EA	0.660	0.560
Papuan Black Snake (*Pseudechis papuanus*)	E	1.09	1.36
Stephens Banded Snake (*Hoplocephalus stephensii*)	EA	1.36	1.44
Rough-scaled Snake (*Tropedechis carinatus*)	EA	1.36	1.09
King Cobra (*Ophiaphagus hannah*)	E	1.80	1.91
Blue-bellied Black Snake (*Pseudechis guttatus*)	EA	2.13	1.53
Collett's Snake (*Pseudechis colletti*)	EA	2.38	–
Mulga Snake (*Pseudechis australis*)	EA	2.38	1.91
Red-bellied Black Snake (*Pseudechis porphyriacus*)	EA	2.52	2.53
Small-eyed Snake (*Cryptophis nigrescens*)	EA	2.67	–
Eastern Diamond Back Rattlesnake (*Crotalus adamanteus*)	V	11.4	7.70

TABLE IV: Average venom yields and various species of snake (LD_{50} for mice)
(after Broad *et al.*[4])

Snake	*Average Venom Yield* mg	Total LD_{50} doses	*Maximum Venom Yield* mg	Total LD_{50} doses
Small-scaled Snake	44	217,821	110	544,554
Taipan	120	94,488	400	314.961
Brown Snake	2	2,469	67	80,426
Chappell Island Tiger Snake	75	13,838	388	71,587
Indian Cobra	169	16,900	610	61,000
Death Adder	78	11,538	236	34,911
King Cobra	421	11,050	(? 500)	13,123
Eastern Diamond Back Rattlesnake	410	2,662	848	5,505

The venom duct and gland are closely related, being divided into a posterior main venom gland, and an anterior secretory duct with an accessory mucous gland, all of which are enclosed in a tough connective tissue capsule.[54] The posterior or main venom gland is situated superficially, directly beneath the supralabial and lower temporal scales, and appears to be a derivation of the parotid gland.

The gland capsule is attached to facial processes suspending the gland in position and into which fibres of the anterior temporal muscle are inserted.[18]

The gland is organized into many continuous tubules, which usually run in a postero-anterior direction converging on the centre of the gland. There is a serous secretory epithelium, the height of the cells probably changing with the stage of secretion.

The total production of venom, as well as the average and the maximum venom yields on milking, vary from species to species (Table V). The Mulga Snake, *Pseudechis australis*, produce the most venom per milking of any Australian snake, and the Brown Snake, *Pseudonaja textilis*, the least (of those tested). It is likely that some of the smaller elapids whose venoms are now being studied produce even smaller quantities of venom.

The anatomy and physiology of venom injection were extensively studied by Fairley.[18] The complex osteology of the elapid skull (diagrams 4, 5) allows independent movement of each side of the mandible, and of each side of the upper jaw and of the palate. The mobile anteriorly-positioned trachea allows breathing to continue during ingestion of prey. The complex dentition assists in the ingestion of prey, separate movements of each segment being used to advance different sets of teeth along the prey, and to pull it into the mouth, and thence to the oesophagus. Typical elapid dentition can be seen in diagram 6, and colour plate 17. The fangs, anteriorly placed on the maxil-

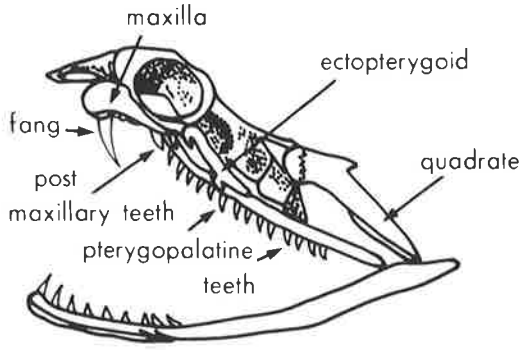


Diagram 4 - Lateral view of the skull of a Death Adder

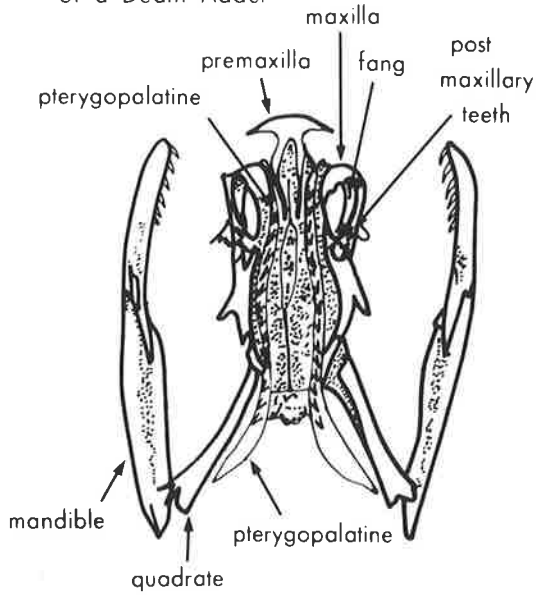


Diagram 5 - Ventral view of skull of a Death Adder. Note independent maxillae, mandibles and pterygopalatine bones and attached teeth

TABLE V: Average venom yields of several species Australian elapid snake[4,114]

	Average Venom Yield mg	*Maximum Venom Yield* mg
Mulga Snake	180	–
Taipan	120	400
Death Adder	78	236
Chappell Island Tiger Snake	75	388
Small-scaled Snake	44	110
Red-bellied Black Snake	40	–
Tiger Snake	35	189
Copperhead	20	–
Rough-scaled Snake	6	–
Brown Snake	2	67

lae, can be elevated in elapid snakes, though they cannot achieve the rotation used by viperid snakes (diagram 7).

The fang elevation in elapid snakes is achieved by maxillary elevation, but the angle of fang entry to the victim, and therefore depth of penetration, depends on both maxillary elevation and the extent of mouth opening, combined with the obliquity of the upper jaw relative to the bitten surface. Of Australian elapid snakes studied by Fairley, the Death Adder, *Acanthophis antarcticus*, showed the greatest capacity to elevate the fangs, and the Brown Snake, *Pseudonaja textilis*, the least (diagram 8, table VI).

TABLE VI: Elevation angle of the fangs of some Australian elapid snakes[18]

Snake	*Elevation Angle*
Death Adder	40 – 50°
Tiger Snake	30 – 35°
Copperhead	25 – 30°
Red-bellied Black Snake	25 – 30°
Brown Snake	10 – 15°

TABLE VII: Length of fangs of some Australian elapid snakes[18]

Snake	*Length of fang* (mm) (average)
Taipan	13
Mulga Snake	6.5
Death Adder	6.2
Red-bellied Black Snake	4.0
Tiger Snake	3.5
Copperhead	3.3
Brown Snake	2.8

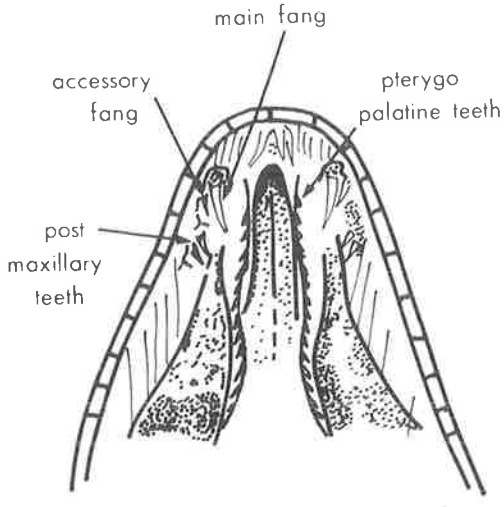
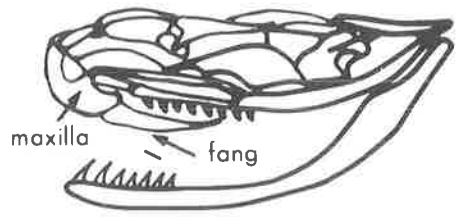


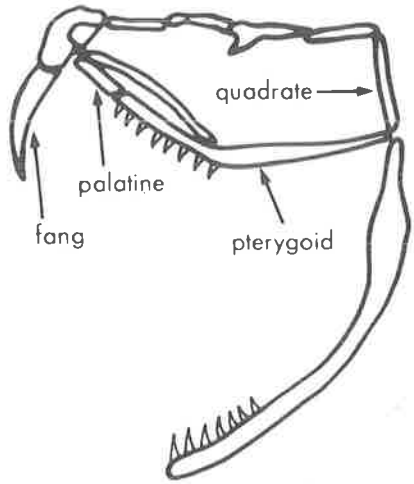
Diagram 6 - Ventral view of mouth of Death Adder showing fangs, post maxillary and pterygopalatine teeth

The fangs of Australian elapids are proteroglyphous (diagram 9), with a distinct closed-groove venom passage, and an opening at the tip of the fang (fig. 4). There are usually several successional or back-up fangs, which can rapidly become functional following loss of the primary fang. Fang length varies with species, the Brown Snake having the smallest fangs (Table VII). Fairley[18] noted that on reviewing the Australian elapids, the average venom yield increases with widening of the distance between fangs.

Fairley, in studying the biting mechanisms, concluded that there were four phases of biting, and that the local lesion produced may show only one fang mark, or up to four marks for each bite. Clearly the local lesion becomes even more variable if multiple bites occur. He concluded



Mouth closed with fang parallel to palatine and pterygoid bones



Mouth open with fang erection as maxilla rotates as quadrate pulls prefrontal, frontal, parietal and supratemporal backwards

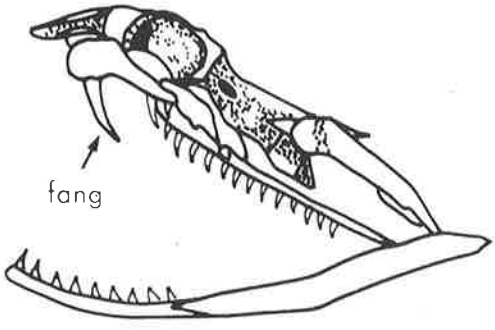
Diagram 7 - Dynamics of fang erection in the Rattlesnake
(after Webb, Wallwork, Elgood)

that the Death Adder had the fastest strike of Australian elapids, and that all species except the Brown Snake strike with the jaws closed until time of impact. The strike is the first phase of the biting mechanism.

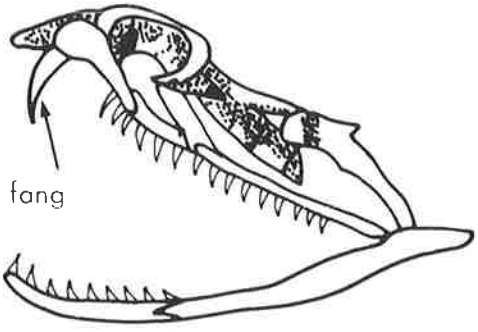
The second phase of the biting mechanism occurs as the snake's head reaches the victim. The mandibles are depressed by rapid contraction of the digastric, cervico-mandibular and vertebro-mandibular muscles, and simultaneously the fangs are rotated forward by the forward swing of the pterygo-palatine transverse arch. This is brought about by the simultaneous contraction of the spheno-pterygoid and parieto-pterygoid muscles.

The third phase is closure of the mouth, brought about by the simultaneous contraction of the anterior, middle, and posterior temporal muscles which strongly elevate the mandibles. With closure of the jaws the fangs simultaneously penetrate the victim and immediate inoculation of venom occurs. This is brought about mainly by contraction of the superior and inferior portions of the anterior temporal muscle which compresses the gland by producing torsion on its capsule with the expulsion of venom into the duct. The venom passes through the dental papilla into the vagina dentis which, by the tense approximation of its edges to each other, and to the surface of the fang, prevents escape of the venom except into the fang channel, whence it is conveyed under pressure into the victim.

The fourth phase occurs immediately following the entry of the rotated and elevated fangs. Accompanying the discharge of venom there occurs another set of movements due to the contraction of the retractor muscles operating on the pterygo-palatine-transverse arch which results in the distribution of venom along an oblique, posteriorly-directed, fang track. The fangs enter the tissue of the victim in a position of maximal elevation, and continue their subsequent course in a downward and backward direction, producing the scratch mark typically seen in cases of Australian snake bite. Small animals can be drawn into the mouth by this movement, while on large animals the snake's head is drawn forward over its victim. Also occurring during this move-



Fang position at rest



Fang position at maximum elevation

Diagram 8 - Rotation of maxilla and attached fang and post maxillary teeth in the Death Adder

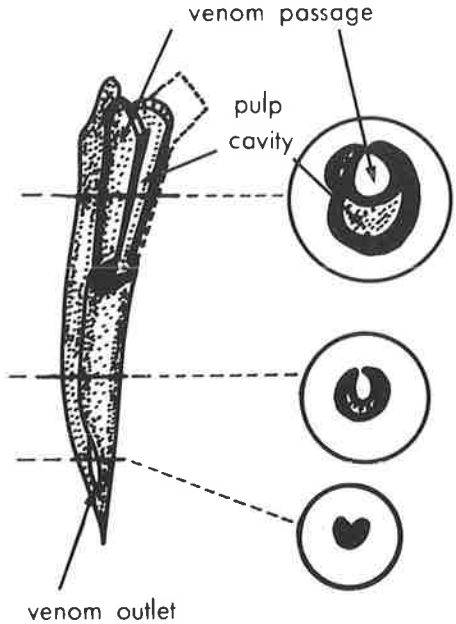


Diagram 9 - Typical proteroglyphous fang of Australian elapid snake, with enclosed venom groove

ment is the contraction of the superior bundle of the parieto-palatine muscle, which tenses the vagina dentis, and contraction of the external pterygoid muscle which thus compresses the inferior surface of the venom gland. This, acting synchronously with contraction of the anterior temporal muscle, facilitates injection of venom under pressure.

Both the forward rotation and elevation of the fangs, and venom injection, are under voluntary control, so that the snake can inject no venom, some venom or all its venom, unilaterally, or bilaterally. This degree of control explains why experiments using live snakes to bite test animals are unreliable, and why the range of effects and degree of envenomation in man is so variable.

(c) Neurotoxins

The most important and most intensively studied components of Australian snake venoms are the neurotoxins. Neurotoxin components of a venom were first demonstrated by Martin[1] who separated Black Snake venom into haemorrhagic and neurotoxic components in 1893. Fairley, Kellaway and their co-workers in the 1920s and 30s were also well aware of the neurotoxins, characterizing them as curare-like. More recently, it has been possible to separate different neurotoxins from within individual venoms, and gain some understanding of their action.

These neurotoxins have a definite peripheral action, affecting transmission at the neuro-muscular junction, and so causing paralysis. This is caused in two main ways. Firstly, some of the most potent neurotoxins have a pre-synaptic effect. Secondly, the more numerous and less potent toxins work post-synaptically. Both may also have a central effect as well, although it is likely the role of central neurotoxic effects in lethality of the venom is a minor one. Kellaway *et al.*[33] demonstrated that direct application of venom to the fourth ventricle could not produce respiratory paralysis and death at venom concentrations in excess of those required to cause paralysis of respiratory muscles and death following intravenous or intraperitoneal administration of venom.

There are several reports of human envenomation in Australia, where the victim has had signs suggestive of central nervous system involvement.[55–60] These are usually drowsiness or lethargy, although in children, the victim may be unconscious for a period.[59] I have observed drowsiness and lethargy, with slowness or absence of response to commands in all severe cases of envenomation I have encountered. Sutherland[60] reported a grand mal epileptic attack in a previously well middle-aged woman following envenomation by a Red-bellied Black Snake, *Pseudechis porphyriacus*, and I know of a similar case, where a $2\frac{1}{2}$-year-old boy, previously well, suffered a "grand mal epileptic attack" following massive envenomation by a Brown Snake, *Pseudonaja textilis*. This "epileptic attack" was related to me by an attending casualty officer, but the details are unfortunately very vague.

However, the most important role of snake neurotoxins is at the neuromuscular junction.

(i) Pre-synaptic neurotoxins

The pre-synaptic neurotoxins of Australian elapid venoms are the most toxic substances isolated from any snake venom at this time. To date, four pre-synaptic neurotoxins have been isolated from Australian venoms, and two from the venoms of exotic snakes.[61,48] All have in common a basic phospholipase A_2, which is typically a single peptide chain of about 120 amino-acids, with disulphide bridges. This primary unit may be complexed with acidic, neutral, or basic protein units. It appears that the neurotoxicity resides in the phospholipase A_2 unit, with enhancement of potency by sub-units.[61] Thus the largest such toxin, *Textilon*, is the most potent.[48] Provisional schematics of these toxins have been suggested by Fohlman[62] (diagram 10). The sub-units are non-toxic, and may enhance toxicity either by providing ligands for specific binding to pre-synaptic sites, or by preventing binding of the principal phospholipase A_2 unit to non-specific sites.[61]

The first Australian, pre-synaptic

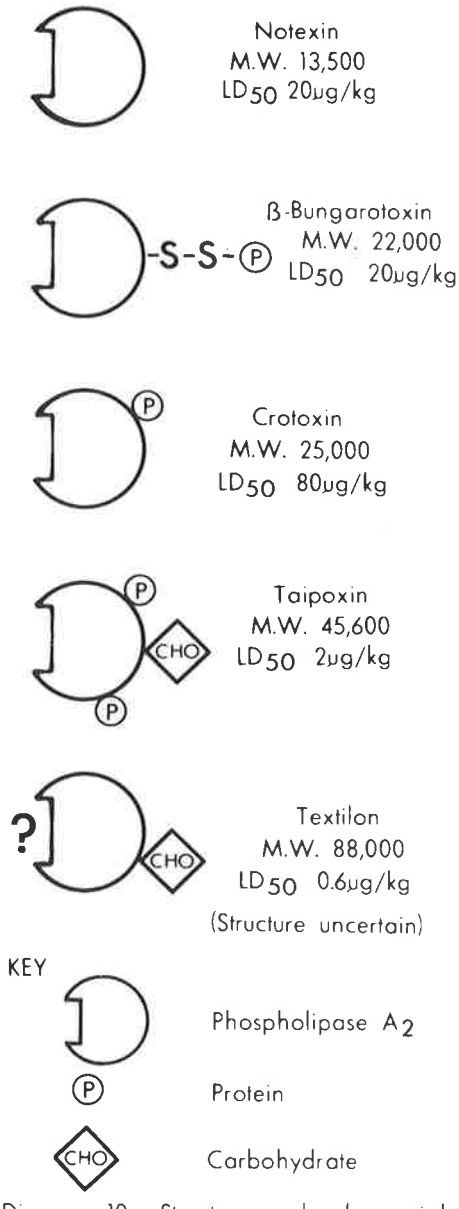


Diagram 10 - Structure, molecular weight and toxicity (mice) of presynaptic neurotoxins

neurotoxin isolated was *Notexin*, from the Tiger Snake.[41]

Notexin constituted 5% of the crude venom, and is a basic phospholipase of 119 amino acids in a single peptide chain, with seven disulphide bridges. The molecular weight is 13,500.

Taipoxin, from the Taipan, is a moderately acidic sialo-glycoprotein with a 1:1:1 ternary complex of subunits (α, β, γ). The α and β subunits are each of 120 amino acids, with seven disulphide bridges. The γ subunit has 135 amino acids with eight disulphide bridges. The α subunit is the only subunit with lethal neurotoxicity, but is much less toxic than the whole *Taipoxin* (LD_{50} = 300 μg/kg for α subunit, and 2 μg/kg for whole *Taipoxin* (mouse)). The molecular weight of *Taipoxin* is 45,600.[45]

A *Taipoxin* analog, *Paradoxin*, has been isolated from the venom of the Small-scaled Snake.[129] It accounts for 12% of venom protein, and has similar γ, α, and β components, and an I.V. LD_{50} (mice) of 2 μg/kg.

Textilon, from the Common Brown Snake, constitutes 4–5% of the crude venom. It possesses approximately 70% of the total venom toxicity. The molecular weight is 88,000.[48]

The LD_{50} (mouse) of these neurotoxins shows an increase in toxicity with increasing size. Thus *Notexin* has an LD_{50} of 20 μg/kg, *Taipoxin* 2 μg/kg, and *Textilon* 0.6 μg/kg, which is therefore the most toxic component so far identified from any snake venom.

These pre-synaptic neurotoxins typically cause a progressive neuromuscular paralysis, which may take 2–3 days to kill the test animal. Paralysis is preceded by a latency period of 30–60 minutes, which is reduced by nerve activity, but not by increased toxin concentration.[61] The toxin is rapidly and irreversibly bound to the nerve terminal during the latency period, and binding may take as little as 3–5 minutes. They cause immediate inhibition of the high-affinity choline transport system in synaptosomes.

During the latency period, spontaneous transmitter release is increased, evidenced by an increase in miniature end-plate potentials (mepps). Nerve impulse evoked release (epp) is also altered, with a rapid fall in amplitude of epps.

At the time of paralysis, there is a marked reduction in the number of vesicles, with a wide variation in use and shape of remaining vesicles, and

swelling and disruption of mitochondria.

There is an associated myotoxic effect, with an acute necrotizing myopathy of surrounding muscle fibres.

Thesleff[61] has suggested that these "pre-synaptic neurotoxins rapidly bind with a high degree of specificity and irreversibility to the axolemma of cholinergic nerve terminals, possibly to the membrane protein involved in the high-affinity choline transport system. Upon binding, the neurotoxins enter the nerve terminal axoplasm by the endocytic mechanism. Once inside the nerve terminals the phospholipase A_2 activity of the toxin exerts its hydrolytic action on the vesicle membrane and on other intracellular membraneous constitutents. This causes a reduction in synaptic vesicle number, a fusion of vesicles to form large synaptic vesicles, and damage of mitochondria and other intracellular organelles storing calcium thereby increasing the level of free calcium inside the nerve terminal. Paralysis results when the number of synaptic vesicles were sufficiently reduced by the hydrolytic phospholipase A_2 activity of the toxin molecule."

(ii) Post-synaptic neurotoxins

In addition to the potent presynaptic neurotoxins, Australian elapid venoms contain post-synaptic neurotoxins. Kellaway *et al.*[32,33,34,35] showed a curare-like neuromuscular block in both frog neuromuscular and rabbit diaphragm preparations, following administration of the venoms of the Death Adder, Copperhead, Common Brown Snake, Tiger Snake, Taipan, and Red-bellied Black Snake, but this effect was not observed after use of venom of the Mulga Snake, *Pseudechis australis*. The post-synaptic block is rapidly established, but less severe than that of presynaptic neurotoxins.

In delineating *Notexin*, Karlsson *et al.*[41] also reported the presence of four post-synaptic neurotoxins in Tiger Snake venom. The first of these, had an LD_{100} (mouse) of 100 μg/kg (compared to an LD_{100} of 25 μg/kg for *Notexin*) and comprised 60 amino acids.

The second had an LD_{100} of 150 μg/kg, with about 70 amino acids, and the third an LD_{100} of 600 μg/kg, and 120 amino acids. The fourth toxin was not elucidated. All caused respiratory distress, and the first two appeared similar to cobratoxin from the Cobra, *Naja naja*. The third in sublethal doses caused respiratory distress for up to 48 hours.

In delineating *Taipoxin*,[45] post-synaptic neurotoxins were again present, in addition to the pre-synaptic neurotoxin. Fraction III showed paralytic effects, with an LD_{100} of 100 μg/kg, and Fraction IV caused respiratory paralysis in mice, with an LD_{100} of 100 μg/kg.

Fractionation of Death Adder venom has revealed five separate lethal fractions, including a postsynaptic neurotoxin, *acanthophin a*. It is a single chain of 63 amino acids, with four disulphide bridges, an approximate molecular weight of 7,700, and an intraperitoneal LD_{50} (mice) of 0.16 mg/kg.[128]

These elapid post-synaptic neurotoxins bind to and inhibit the function of nicotinic acetylcholine receptors.[12] The mechanism of these "α-neurotoxins" has been used to delineate the acetylcholine receptor on muscle.[12,63] Though curare-like at least some of these post-synaptic neurotoxins show irreversible block (e.g. α-bungarotoxin on rat diaphragms[64]).

(d) Myotoxins

The myolytic action of some snake venoms is well documented, especially for Sea Snakes.[64] However, the myolytic action of Australian elapid venoms has not been extensively investigated, and is only now gaining the attention it deserves.

Kellaway[29] alluded to the effect of Mulga Snake venom on heart muscle but did not describe post-mortem findings. Several case reports of rhabdomyolysis following envenomation by Australian elapids exist. The Tiger Snake,[66] Mulga

Figure 11

Figure 12

FIG. 11 Common Tiger Snake, *Notechis scutatus*. **Typical River Murray colour phase, with distinct banding. Collected near Morgan, S.A.**

FIG. 12 Common Tiger Snake, *Notechis scutatus*. **Almost unbanded, brown colour phase found in south-east S.A. Collected near Penola, S.A.**

FIG. 13 Mulga Snake, *Pseudechis australis*. **Typical brown colour phase showing yellow inner edging to each scale giving a semi-reticulate pattern. Collected near Innaminka, S.A.**

FIG. 14 Red-bellied Black Snake, *Pseudechis porphyriacus*. **Typical colour phase, black dorsally, crimson edged scales ventrally. Collected near Tungkillo, S.A.**

FIG. 15 Common Brown Snake, *Pseudonaja textilis*. **Peninsula colour phase, with some dark speckling dorsolaterally. Collected on Yorke Peninsula, S.A.**

Figure 13

Figure 14

Figure 15

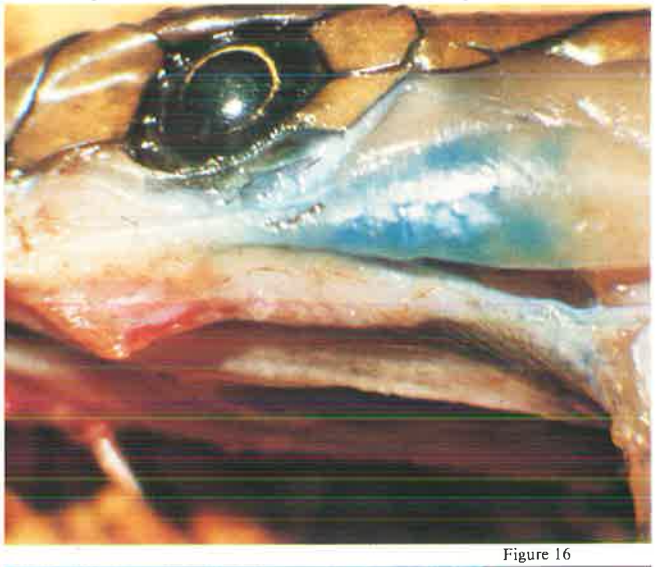


Figure 16

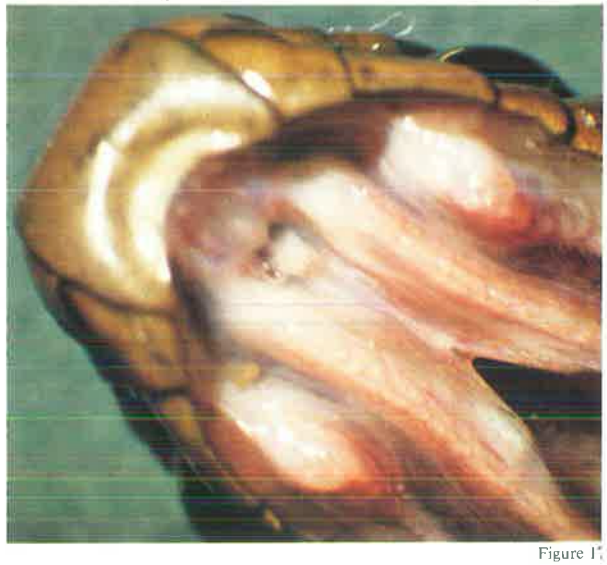


Figure 17

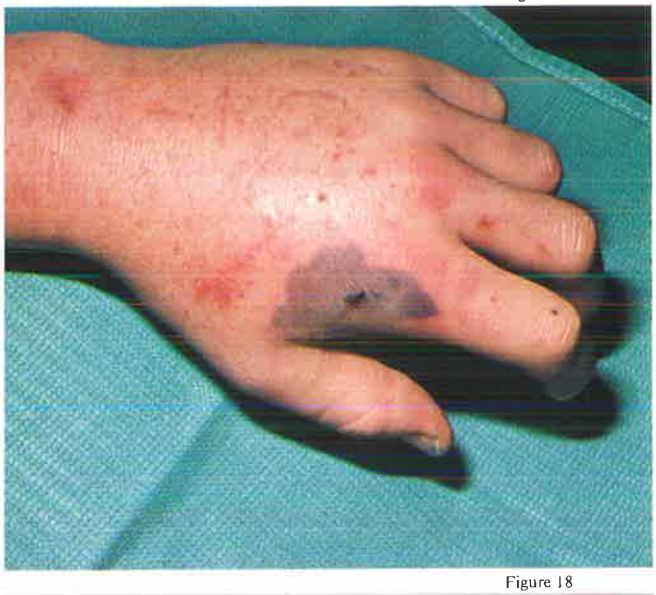


Figure 18

Figure 19

FIG. 16 Dissection of side of head of Brown Snake, to show venom gland, duct, and fang in sheath. Anterior portion of venom gland and adjacent duct has been stained blue.

FIG. 17 Underside of mouth of Brown Snake, showing sheathed fangs anterolaterally, and the more medially placed rows of pterygopalatine teeth.

FIG. 18 Hand of victim of Tiger Snake bite, showing oedema of whole hand, bruising around bite with early skin necrosis, and clot of blood on the skin surface at the site of fang puncture.

FIG. 19 Small-scaled Snake, *Oxyuranus microlepidotus*. Typical colour phase of adult specimen, with dark coloured head. Collected near Birdsville Track, S.A.

Snake,[55] and Small-eyed Snake, *Cryptophis nigrescens*[65] have caused myolysis and myoglobinuria in human victims. In the latter two cases, the victims died.

Recent work[67] shows that the venoms of the Tiger Snake, Mulga Snake, Red-bellied Black Snake and Copperhead, all have potent myolytic activity. The venoms of the Death Adder, Western Brown Snake (Gwardar) *Pseudonaja nuchalis*, and Common Brown Snake, *Pseudonaja textilis*, do not exhibit myolytic activity. The venoms of the Dugite, *Pseudonaja affinis*, and Small-scaled Snake, *Oxyuranus microlepidotus* have given equivocal results.

The nature of these myotoxins is not completely understood, but *Notexin* causes local reversible muscle damage, and some other snake toxins also with a phospholipase structural unit consistently causes myonecrosis.[68] A potent myotoxin and neurotoxin with a basic phospholipase A_2 unit, 120 amino acids, and a molecular weight of 13,500, has been isolated from the venom of the Sea Snake, *Enhydrina schistosa*.[68]

A lethal myotoxin has been isolated from Mulga Snake venom.[130] One of four lethal fractions identified from this venom, and labelled *Mulgatoxin a*, it is a basic single polypeptide chain of 122 amino acids, with seven disulphide bridges. It causes myoglobinuria in mice, with an LD_{50} (mouse) of 200 μg/kg, and its activity is specific for skeletal muscle, causing massive cell damage both *in vitro* and *in vivo*. Molecular weight is 13,710.

(e) Cardiotoxins

No specific cardiotoxins have been identified from Australian elapid venoms, but some features suggestive of cardiac actions have been reported. However, the role of cardiotoxins in cases of envenomation by Australian elapids remains unresolved, though it seems probable that if cardiotoxins are present, they play only a minor part.

Kellaway[35] noted that Mulga Snake venom has a direct effect on cardiac muscle, which he concluded was important in the lethality of this venom. He also noted a weak cardiotoxic action in the venoms of the Copperhead and Red-bellied Black Snake. He found Mulga Snake venom caused cessation of the beat in isolated rabbit auricle in about one hour, at concentrations of 1:10,000.[29] He considered this venom to be similar in cardiac action to Cobra venom.

A pure cardiotoxin has been isolated from Cobra venom,[64] and causes systolic arrest in the frog heart. It irreversibly reduces the resting membrane potentials of the cardiac muscle. There is an increase in the Q-T interval of the electrocardiogram. Purified neurotoxin and phospholipase A from the same venom do not produce these cardiac changes. However, there does appear to be a synergistic action between phospholipase A and cardiotoxin.

In addition to a direct action on the heart, cardiotoxins may also act on the vascular system. The venoms of the Red-bellied Black Snake and the Copperhead can cause severe progressive hypotension if administered intravenously.[60] This may be due to a direct effect of a cardiotoxin, or to the release of pharmacologically active substances such as histamine or SRS, both of which can be liberated by Red-bellied Black Snake venom. It is likely that a phospholipase A in the venom is responsible for the release of these substances indirectly, by forming lysolecithin, which in turn damages cells causing the release of these substances.[69]

(f) Haematologically-active components

Nearly all snake venoms studied have some effect on the blood. Two major areas of activity can be delineated. These are interference with the coagulation system to produce either excess coagulation, or incoagulability; and haemolytic activity.

(i) Coagulation disturbance

Snake venoms contain enzymes and toxins concerned with all three basic reactions of blood coagulation. These three basic reactions are:–

(1) formation of auto prothrombin C, (factor Xa)
(2) formation of thrombin
(3) formation of fibrin[70]

Australian elapid venoms appear to possess powerful prothrombin converters, thus affecting coagulation at step two. This can cause either coagu-

TABLE VIII: Coagulant activity of some Australian elapid venoms (updated from Kellaway's original work and partly based on clinical cases)

Snake	*Relative coagulant activity* (this may manifest as defibrination syndrome and resultant hypo-coagulability)	*Defibrination Syndrome Recorded*
Brown Snake	+	+
Western Brown Snake	+	+
Dugite	+	+
Tiger Snake	+	+
Black Tiger Snake	+	(no cases described but probable)
Taipan	+	+
Small-scaled Snake	+ (uncertain)	(probable)
Rough-scaled Snake	+	+ (probable)
Papuan Black Snake	+	+
Red-bellied Black Snake	+	–
Mulga Snake	?	? (one possible case)
Death Adder	–	–
Copperhead	–	–

TABLE IX: Clotting times of Factor V – deficient plasma, with four Australian elapid venoms, showing complete and incomplete prothrombin activation[71]

Snake	*Clotting time of normal plasma* (sec.)	*Clotting time of Factor V deficient plasma* (sec.)	*Type of Prothrombin Activator*
Tiger Snake	10	67	Incomplete
Death Adder	12	170	Incomplete
Taipan	8	8	Complete
Brown Snake	6	6	Complete

lation of blood, or a defibrination syndrome with consequent hypocoagulability.

Martin[15] first demonstrated the thrombotic action of Red-bellied Black Snake venom, and Tiger Snake Venom. Subsequent work by Kellaway *et al.*[17,21,22,25,26,29,39] showed that only some Australian elapids have significant effects on coagulation (Table VIII). Later, Denson[71] studied some Australian elapid venoms with thrombotic effects, finding that Tiger Snake venom was an incomplete thrombin activator and that the Taipan and Common Brown Snake venoms were complete thrombin converters (Table IX).

The exact nature of the thrombin activators has not been established, but it is conceivable that the neurotoxins may also have some prothrombin conversion activity.

Arvin, a coagulant fraction isolated from the Malayan Pit Viper, *Agkistrodon rhodostoma*, can cause rapid and effective hypocoagulability by defibrination. The lethal dose of the whole venom is 100–500 times the defibrinating dose.[71]

Phospholipases may also exert a direct anticoagulant action by action on platelet or plasma phospholipid.[71] Kaire[72] reported anticoagulant activity in all Australian elapid venoms studied, and linked this activity with phospholipase A (Table X).

TABLE X: Anticoagulant activity of Australian elapid venoms according to Kaire[72]

Snakes – in descending order of anticoagulant activity	
1	Mulga Snake (highest anticoagulant activity)
2	Papuan Black Snake
3	Red-bellied Black Snake
4	Copperhead
5	Death Adder
6	Tiger Snake
7	Taipan
8	Brown Snake (lowest anticoagulant activity)

(ii) Haemolysins occur in many snake venoms, and have been noted to exist in Australian elapid venoms for many years. Kellaway and others made extensive examinations of the haemolytic properties of these venoms,[73,74,75] and their work was extended by Doery and Pearson.[76] Using the venom of the

Red-bellied Black Snake as a control, the haemolytic activity of most major Australian elapid venoms was documented (Table XI). It is clear that the *Pseudechis* group have the most haemolytic activity, and that the venoms of the Brown Snakes are only very weakly haemolytic.

TABLE XI: Haemolytic activity of Australian elapid venoms (after Doery and Pearson[76]), as a percentage of activity of red-bellied black snake venom

Snake	*Phospholipase A*	*Direct Haemolysis*
Red-bellied Black Snake	100	100
Mulga Snake	150	50
Papuan Black Snake	150	100
Copperhead	50	50
Tiger Snake	50	2–3
Taipan	100	<1
Death Adder	50	<1
Brown Snake	<20	<12

Two forms of haemolytic activity are evident. There is direct haemolysis by venom components, and indirect haemolysis by phospholipase A venom components which appear to work by converting lecithin to lysolecithin, which then cause haemolysis.[76]

The exact relationship of these haemolytic components to other venom components has yet to be established. However, clinically, haemolysis is not a significant contributor to mortality following envenomation by Australian elapids.

(g) Enzymes

All snake venoms contain a multiplicity of enzymes, the composition of which vary from species to species, and at higher taxonomic levels. They may act in three major ways:

(a) By causing local capillary damage and tissue necrosis (e.g. proteinases, phospholipases, arginine ester hydrolases, hyaluronidase).

(b) By causing coagulation or anticoagulation (e.g. proteinases, phospholipase A).

(c) By causing acute hypotension and pain secondary to the release of vasoactive peptides by kinin-releasing enzymes (e.g. Kininogenase).

Of about 26 enzymes detected in snake venoms, most are hydrolases, and 12 enzymes appear common to all venoms.[77] Those common enzymes are phospholipase A_2, L-amino-acid oxidase, phosphodiesterase, 5-nucleotidase, phosphomono-esterase, deoxyribonuclease, ribonuclease, adenosine triphosphatase, hyaluronidase, NAD-nucleosidase, arylamidase, and peptidase. Enzymes found in some Elapid venoms also include acetylcholinesterase, phospholipase B, and glycerophosphatase. It appears that acetylcholinesterase is exclusive to elapid venoms, being absent from Viperid and Crotalid venoms. The enzyme composition of Australian elapid venoms is not fully elucidated, and the above listing can be a guide only, based mainly on non-Australian elapid venoms. These studies suggest that elapid venoms in general are poor in enzymes compared to Viperid and Crotalid venoms. These last two families of snakes have venoms which are rich in locally-acting enzymes which cause tissue damage and necrosis at the site of the bite, a feature which is fortunately absent from most Australian snake bites.

The role of enzymes in envenomation by Australian elapids is not well-defined, but it seems likely their action is overshadowed by the more specific activity of other toxins such as the neurotoxins.

4. SUMMARY

Australian elapid venoms contain a diverse array of toxic substances which are only now being elucidated, and a complete understanding of these venoms is not yet possible. They are characterized by potent specific toxins which act on the victim at sites distant from the bite site, and have relatively little local action at the bite site. Potent neurotoxins, both pre-synaptic and post-synaptic, are evident in many of these venoms, and where present are usually the chief lethal toxins.

Potent myotoxins exist in some of these venoms, and some contain haematologically-active substances which can cause coagulation, anticoagulation via defibrination; anti-coagulation, and haemolysis.

The known or suspected activities of each major Australian elapid snake are listed in Table XII.

TABLE XII: Relative activities of major Australian elapid snakes (after Sutherland[114] and others)

Snake	*Local tissue oedema and damage*	*Neurotoxic effects*	*Haemolytic effects*	*Defibrination Syndrome*	*Renal Failure*	*Myotoxicity*
Brown Snake Western Brown Snake Dugite	–	+++	+	+++	+	–
Death Adder	–	+++	+	–	–	–
Taipan	+	+++	+	+	?	?
Small-scaled Snake	–?	+++	+	?+	?	?
Tiger Snake Black Tiger Snake	+	+++	+	+++	+	++
Copperhead	–	++	++	–	–	++
Mulga Snake	+	?++	+++	?+	+	+++
Red-bellied Black Snake	+	+	+++	–	–	++
Rough-scaled Snake	–	++	++	?+	+	++

SECTION III – ENVENOMATION

1. EPIDEMIOLOGY

(a) Whole World

Information and statistics on envenomation for all the regions of the world are scattered and cover different periods, and encompass different standards of health care. There is one World Health Organization study,[1] published in 1954 which documented statistics on snakebite mortality worldwide. Problems in compiling such statistics include inaccurate diagnoses and reporting of envenomation, and problems with statistical recording of envenomation. For instance, the International Statistical Classification of Diseases, Injuries, and Causes of Death does not have a specific category exclusively for snakebite, so that statistics on snakebite are sandwiched in with statistics on spiderbite, insect bites and stings, including bee stings, and attacks from other venomous or possibly venomous animals.

However, allowing for these problems, the W.H.O. study concluded that between 30,000 and 40,000 people die annually from snakebite. The breakdown of estimated deaths for each world region (Table XIII) shows that the region containing Australia has the lowest average mortality totals. These figures, however, do not include the population and health care differences between these regions. It is unknown what the effects of increased population versus improved medical care have had on this appalling death rate.

The same study estimated that about 500,000 people were bitten by snakes annually. Thus less than 10% of people bitten by snakes die as a result. Analysis of the W.H.O. statistics, with correlation to total death rates, provide a better perspective on the importance of snakebite as a cause of mortality in various regions of the world. Thus Burma has a snakebite mortality of 36.8 per 100,000 population in Sagaing province, and an overall rate of 15.4 per 100,000 population. In contrast, the Netherlands has a rate of 0.004 per 100,000 population. The U.K. has a rate

TABLE XIII: Total snakebite mortality worldwide (based on W.H.O. 1954 statistics[1])

Region	*Estimated Mortality*
Asia	25,000 – 35,000
South America	3,000 – 4,000
Africa	400 – 1,000
North America	300 – 500
Europe	50
Oceania (includes Australia)	10
TOTAL	30,000 – 40,000 deaths per year

of 0.02 per 100,000 population, and France a rate of 0.07. During the same period, Australia had a rate of 0.07 per 100,000 population. This puts the danger of snakebite mortality in Australia in perspective. Advances in treatment of snakebite in Australia, and improved public awareness of snakes and snakebite, have undoubtedly decreased this rate further. Thus the chance of death from snakebite in Australia is small compared to some other parts of the world, and is now probably similar to parts of Europe whence many migrants have come.

(b) Australia

Patterns of death from snakebite in Australia inevitably vary with time and place. The W.H.O.[1] noted that recorded snakebite deaths in Australia in the period 1945–9 varied greatly between States, Queensland having the most deaths, and South Australia no recorded deaths (Table XIV). Trinca[78] reported a similar breakdown of snakebite deaths in 1963, which again showed Queensland with the highest number of deaths, and with only one death in South Australia in the 10-year period (Table XV). These figures show an average death rate of 4.5 per year. Both the W.H.O. figures, and those of Trinca cover periods after the introduction of antivenom in Australia.

TABLE XIV: Geographic Distribution of snakebite mortality in Australia, for the period 1945–9[1]

State	*Recorded deaths from snakebite*
Queensland	18
New South Wales	6
Western Australia	3
Tasmania	1
South Australia	–
Victoria	–
Northern Territory	–
TOTAL	28

Fairley in 1929,[17] estimated that 80% of deaths from bites and stings in Australia from 1910 to 1926 were due to snakebite. Again, the highest incidence was in Queensland, and South Australia had one of the lowest mortality rates (Table XVI).

TABLE XVI: Geographic distribution of mortality from venomous bites and stings in Australia for the period 1910–26[17]

State	*Recorded deaths* (approx. 80% due to snakebite)
Queensland	92
New South Wales	71
Victoria	54
Tasmania	10
South Australia	10
Western Australia	7
Australian Capital Territory	1
Northern Territory	1
TOTAL	244

During this period, 244 deaths from envenomation were recorded, giving an average annual death rate from snakebite of 11.5 and 14.4 for all bites and stings.

Queensland's predominance in mortality from snakebite is probably due to the abundance of potentially lethal snakes in areas commonly frequented by men, especially the abundance of Taipans in cane fields. The low mortality in South Australia reflects the relative rarity of venomous snakes in areas frequented by significant numbers of people. The exception to this is the Common Brown Snake, which often frequents areas inhabited by man, both on the fringes of metropolitan areas and around farming properties. Fortunately bites from the Brown Snake have a low mortality rate, despite its potent venom (Table XVII). Even without specific treatment, less than 10% of Brown Snake victims died, and with specific antivenom, the mortality rate has almost certainly been reduced further.

TABLE XV: Geographic distribution of snakebite mortality in Australia for the period 1952–61[78]

State	*Recorded deaths from snakebite*	*Male*	*Female*
Queensland	18	11	7
New South Wales	9	6	3
Victoria	9	5	4
Western Australia	6	6	1
South Australia	1	1	–
Tasmania	–	–	–
Australian Capital Territory	1	1	–
Northern Territory	1	1	–
TOTAL	45	30	15

TABLE XVII: Mortality rates for common lethal snakes in Australia, prior to the advent of antivenom treatment[17]

Species	*Number of Persons Bitten*	*Deaths*	
		Number	*Percentage*
Death Adder	10	5	50%
Tiger Snake	45	18	40%
Brown Snake	70	6	8.6%
Red-bellied Black Snake	125	1	0.8%
TOTAL	250	30	12%

There are several recent series on snakebite in Australia, and New Guinea. Campbell has reported experience with snakebite in New Guinea,[79,80,81] where two of the three main species involved are subspecific varieties of Australian species. (Taipan, *Oxyuranus scutellatus canni*; Death Adder, *Acanthophis antarcticus*). Of 73 patients admitted with snakebite, five died (7%).[81] Sutherland[82] reported that there were 45 deaths from snakebite reported in the 10-year period 1960–1970. Munro and Pearn[83] reported experience with snakebite in children in south-east Queensland, for the 5-year period 1971–1975. There were 71 cases in the series, and in only 27% did any symptoms or signs of snakebite or other illness develop. Only 17% showed definite signs of envenomation and there was only one fatality. Sutherland and Lovering[84] reported experience Australia-wide with C.S.L. antivenoms, and noted that 203 cases of snakebite requiring antivenom therapy were reported to C.S.L. in the 12 months July 1978 to June 1979. Three cases ended fatally.

In Pearn's series[83] only two of the 71 cases received antivenom. Even allowing for the fact that this may represent an abnormally low use of antivenom, it seems likely that between 500 and 1,000 people are bitten by snakes annually in Australia.

The figures may be much higher than this. Despite this few people die from snakebite in Australia. Indeed, a recent report[51] suggests that only one person, a child bitten by a Taipan, died in the 18 months to September 1980.

(c) South Australia

South Australia's lack of prominence in snakebite statistics has already been mentioned. There are no studies reporting on snakebite in South Australia specifically and there are very few case reports of snakebite from this State. Fotheringham[58] reported a case of neurotoxic envenomation in a child, and Sutherland *et al.*[85] reported two cases of envenomation of adults from Balaklava, a town about 70 miles north of Adelaide. In the latter two cases, the Common Brown Snake was implicated. In my experience, nearly all snakebites in South Australia are caused by the Common Brown Snake or the Western Brown Snake. Bites from other species are most often seen in snake handlers, who unfortunately have a propensity for being bitten by their pets with monotonous regularity. I am aware of one snake handler who was bitten 11 times in 18 months by dangerously venomous snakes, covering a variety of species. I know of another who has been bitten three times in two years in exactly the same manner, whilst trying to feed his pets.

(d) Records from the Adelaide Children's Hospital

A study of admissions to the Adelaide Children's Hospital over the 10-year period 1968 to 1977 showed 23 cases admitted with suspected or definite snakebite. The average age of these patients was 8.2 years, and seven cases (30%) showed evidence of envenomation. There were no fatalities. In 12 cases (52%) a snake was seen, and "identified", although in only four cases (17%) was the snake brought in and positively identified. Of these 12 cases, 10 were due, or probably due, to a Brown Snake, and two probably due to a Red-bellied Black Snake. Eleven of the victims were bitten on the foot or leg, and 11 on the hand or arm. One was bitten on the lower lip. In this latter case the snake was seen by the mother, and described as a four-feet-long Brown Snake, although the mother did not actually see the child bitten. The only symptoms or signs were two puncture marks on the lower lip, with

associated oedema. No treatment was instituted. Of the 23 cases, one exhibited severe systemic involvement, with neurotoxicity. This case has been reported elsewhere.[58]

In the 18 months from July 1979, there have been four admissions to the Adelaide Children's Hospital with snakebite. Three had definite signs of envenomation, and two had serious systemic involvement, with classical defibrination syndrome. All survived.

(e) Risk groups

Although anyone can be bitten by a snake, certain groups in the community are more likely to be bitten.

(i) Young children

Munro and Pearn[83] identified the toddler age-group (1–3 years old), as the most common group of children involved in snakebite. This age group will readily play with any snakes they encounter in the garden, especially small snakes such as juvenile Brown Snakes, which are common in outer metropolitan Adelaide gardens in late summer and autumn.

(ii) Late primary school children

Munro and Pearn[83] found a second peak incidence in the 10–12-year-old group. These children are inquisitive about wild animals, and have relatively little fear of animals such as snakes. I have noted several seven- and eight-year-olds bitten by snakes in South Australia recently, who also were inquisitive about animals, and were trying to catch the snakes which bit them.

(iii) Farm Workers

Persons working in areas frequented by snakes, such as around pastoral station homesteads, haystacks and swamps and drainage channels, have a greater exposure to snakes and are more likely to be bitten. In the area around Adelaide horticultural workers are at particular risk from bites by the Common Brown Snake, *Pseudonaja textilis*, and the Adelaide Hills Copperhead, *Austrelaps* sp.

(iv) Reptile keepers

Despite the protection of all reptiles, including venomous snakes, in South Australia and the consequent bureaucratic impediments to obtaining snakes as pets, there are a considerable number of amateur reptile keepers in South Australia. Based on membership of local herpetological societies, there are at least 150–200 people in South Australia who keep or study reptiles on an amateur basis. There are at least 20 of these people who keep one or more dangerously venomous snakes. Clearly this group, who range in age from about 14 years to retired people over 65 years, have a greatly increased likelihood of snakebite. While many of this group are very responsible about keeping and handling venomous snakes, inevitably there are some who are either less careful than they should be, or who are simply irresponsible. The mixing of alcohol consumption with handling of venomous snakes is a disturbingly frequent association with snakebite in this latter group.

(v) Professional reptile keepers

There are an unknown number of people in Australia who make some or all of their income from catching, keeping, and exhibiting dangerously venomous snakes. In South Australia there are two major reptile parks, at Renmark, and at Whyalla. In addition there are smaller parks, such as that at Naracoorte, and other wildlife parks which have venomous snakes amongst their displays. There is at least one large amateur reptile collection open for public display in the Adelaide metropolitan area.

These professional snake keepers handle large numbers of venomous snakes, and although their safety consciousness is high, they inevitably are bitten occasionally. The director of one major park has been bitten many times by a variety of venomous snakes. This group provides special problems for the treating physician, for they may easily develop hypersensitivity to both the venom and the antivenom, after a series of bites.

(f) Other risk factors

Munro and Pearn[83] identified risk fac-

tors. They found 36% of victims were bitten while in paddocks, fields or open country, and a further 29% were bitten in gardens or yards. Only two cases were bitten in creeks, and two whilst in a house. In 75% of cases with reliable data there was no intentional provocation of the snake. They found a peak seasonal incidence in Summer (42%), and in the afternoon (51%). There was a random distribution throughout the week, with no weekend peak.

The Adelaide Children's Hospital figures also show that the majority of victims are bitten in fields, paddocks, and especially long grass (65%), the remainder being bitten in gardens or yards. Data on provocation were incomplete, but at least 60% showed no history of provocation. The majority of bites were in Spring, later Winter (August), or early Summer. The majority of bites were in the afternoon. Males were involved more than three times as commonly as females.

2. SYMPTOMS AND SIGNS

The constellation of symptoms and signs in definite and suspected cases of snakebite in Australia is highly variable, depending on such factors as species of snake, size of snake, success of attack, record of snake, mood of victim, age and size of victim, attitude of victim to snakebite, concurrent medical problems of victim, and past exposure to snakebite. To define a precise set of symptoms and signs which define snakebite is clearly impossible. However, certain symptoms and signs are suggestive of snakebite. The treating physician must have a high index of suspicion, and careful history, examination, and investigation are essential to correct diagnosis and treatment of snakebite.

(a) History of bite and symptoms of envenomation

(1) Circumstances of bite

The sophisticated venom delivery apparatus of snakes has been discussed earlier, in detail, but in resumé, the snake may deliver as much or as little venom to the victim as it desires; thus the fact that someone has been bitten by a snake does not mean that venom has been inoculated. This is a fundamental point in understanding the investigation and management of snakebite in Australia.

Secondly, it can be seen from the composition of Australian elapid venoms, that for some species at least, local reaction to a bite may be negligible. Thus we can have the situation of a person with a very minor scratch on a leg, without local pain or other symptoms, yet who may be inoculated with a lethal dose of venom.

The above two factors inject the major amount of uncertainty into determining whether a serious snakebite has occurred. Careful questioning of the victim and witnesses is important for the way a snake strikes, and how long it hangs on for, can give some indication of the amount of venom inoculated. If the snake strikes rapidly with only a glancing blow, then it is unlikely that much if any venom will have been inoculated. If a snake hangs on to the victim for even a matter of seconds, then considerable venom may be inoculated. If the snake strikes repeatedly, and especially if it hangs on for several seconds at each bite, then it is probable that a large quantity of venom will be inoculated. However, some snakes will strike and hit the victim repeatedly without opening the mouth. Clearly no venom will be inoculated in this circumstance, yet to most observers it will appear that the snake has bitten the victim. Only careful examination of the wound can determine the true story.

Unfortunately, in many cases the victim will not actually see the snake or its strike. They may just feel something brush up against their leg, or may feel a quick stinging sensation, as if a thorn has scratched them. Only later may the snake be seen. This story is particularly commonly told by people bitten while walking through long grass, in areas frequented by Brown Snakes, such as the Adelaide Hills. In many of these cases no immediate first aid is applied, as it is not apparent that the person has been bitten by a snake. Realization that a snake is responsible may only occur

when the snake is seen by a companion, or when the victim becomes physically ill and the fang marks are noticed.

(ii) General symptoms of elapid envenomation

Early symptoms following snakebite are obviously variable, and will depend in part on the victim's attitude to snakes and snakebite. Some may become extremely agitated, or hysterical, and may develop tachycardia, hyperventilation and dizziness as a result of this rather than the venom.

Early symptoms of envenomation are dizziness, nausea and vomiting, sweating, and headache. Tachycardia may also be present. These symptoms may then progress to include pain in regional lymph nodes, abdominal pain, which may be very severe, and occasionally diarrhoea. There may be an impairment of conscious state, the victim being slow to answer or ignoring questions. The victim may be irritable, and this is often associated with tossing and turning on the bed.

The patient may become unconscious, and it is common in significant cases of snakebite for loss of consciousness to occur before any other symptoms are reported to observers. This is particularly so in children, who may be bitten by a snake, ignore the bite, and then suddenly collapse unconscious from 15 minutes to an hour after the unsuspected bite.

(iii) Symptoms of neurotoxic envenomation

Symptoms of neurotoxic involvement are usually progressive. The patient may first notice blurring of vision and double vision. Speech will start to change, often subtly at first, as the tongue and soft palate, velopharyngeal sphincter, and vocal cords are all progressively paralyzed. The voice may become nasal as the velopharyngeal sphincter ceases to close completely for vowel sounds. Speech is distorted as the tongue becomes paralyzed.

Without treatment, as neurotoxic paralysis develops, the victim may note a progressive flaccid paresis of all voluntary muscles. This may first be noted when the victim attempts to move, or write. It has been noted that the first muscles to show paralysis are the extrinsic ocular muscles, and the last, the diaphragm.[81] As paralysis of facial muscles progresses, the patient finds he cannot open his mouth or protrude his tongue. The jaw then relaxes, and falls backward along with the tongue. This can cause respiratory obstruction and rapid death if not treated. Weakness of respiratory muscles will impair the ability to cough up secretions, which may be more profuse due to envenomation. Eventually if untreated, the neuromuscular paralysis will become complete, the patient being unable to move, or, eventually, breathe.

(iv) Symptoms of haematological disturbance

There are no symptoms specific to coagulopathy, and defibrination syndrome, but careful questioning of the victim may reveal that the scratches at the bite site oozed continuously. This is a good indication of significant envenomation. In some cases, especially those reported from Papua New Guinea by Campbell,[79,80,81] the patient may vomit or expectorate blood, or pass blood-stained urine. This is clearly a symptom of severe envenomation.

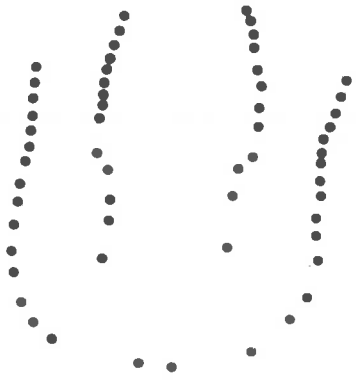
(v) Symptoms of myopathy

Generalized muscle pain and especially muscle movement pain usually indicate a developing rhabdomyolysis, and should be especially looked for in victims of the Mulga Snake, Tiger Snake, Taipan, and Small-scaled Snake.

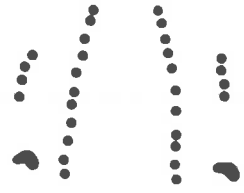
(b) Signs of envenomation

(i) Bite site

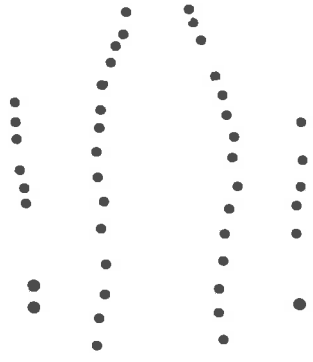
The site of the bite can have a very variable appearance, and may give a clue to the species of snake involved. Fairley[18] carried out studies on bite patterns of Australian elapid snakes, and found distinctive patterns for each species group (diagram 11). However, these impressions were made in laboratory conditions, and are not seen in



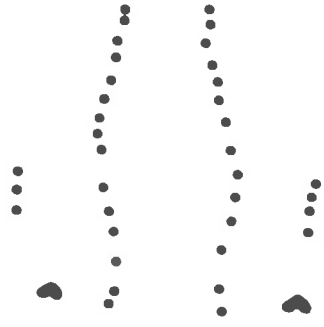
Carpet python



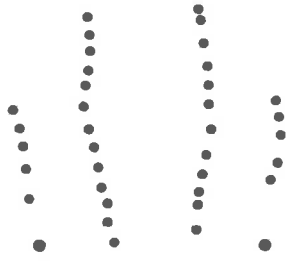
Red bellied Black snake



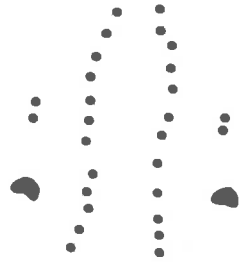
Brown snake



Tiger snake



Copperhead



Death adder

Diagram 11 - Ideal upper jaw bite patterns of a non-venomous python and five dangerous Australian elapid snakes

(after Fairley (17))

human cases of envenomation as a rule. The snake may strike the victim from an angle, only one fang penetrating, and the strike may be only a glancing blow, or a firm bite with chewing and movement of the fangs through the skin. Either can leave a shallow laceration rather than a definite puncture mark. Multiple bites leave an even more complicated pattern of tissue injury. As stated before, the presence of fang marks is not necessarily indicative of significant venom inoculation, but multiple bites are nearly always associated with massive inoculation of venom.

The clinical appearance of fang marks can be seen in figures 43–49. The Brown Snakes typically leave little local evidence of a bite. There may be a single short laceration (fig. 44) which looks like a scratch mark. There is no associated local pain or oedema, erythema, or necrosis. The shallow scratches left by the tiny Brown Snake fang may not even be apparent until some hours later (fig. 45). At the time of initial examination there may only be slight surface irregularities visible on the skin, which will not be seen unless very carefully searched for. They are more easily seen with a magnifying glass.

Copperhead bites may similarly show little or no local reaction (fig. 46). Death Adder bites may show little

FIG. 44: Brown Snake Bite. Close up of single laceration caused by fang.

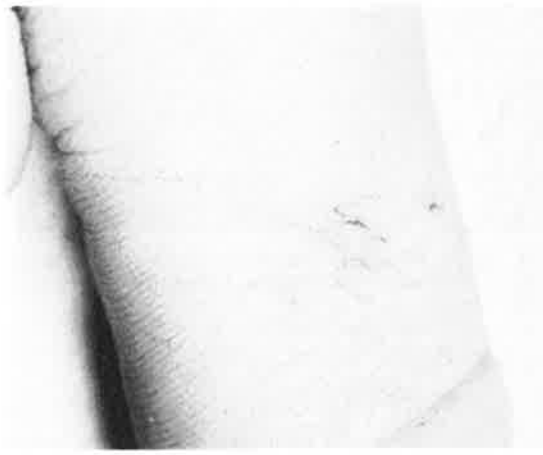


FIG. 45: Brown Snake Bite. Multiple fang marks on finger without oedema or erythema.

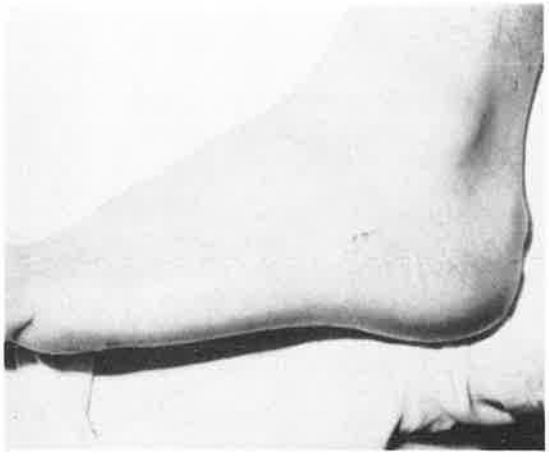


FIG. 43: Brown Snake Bite. Fang mark on side of foot. No local pain, oedema, or erythema.

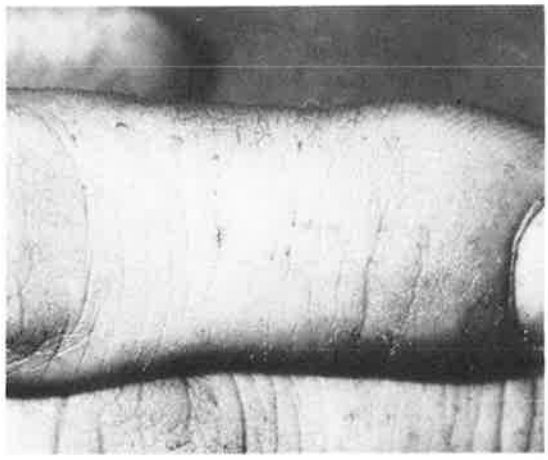


FIG. 46: Adelaide Hills Copperhead Bite. Dual fine fang marks without oedema or erythema.

erythema, but can show slight oedema, associated with local pain. I have encountered two cases of Death Adder bites to a finger, where there has been only mild local oedema, associated with stiffness and marked limitation of movement in the interphalangeal and metacarpophalangeal joints (fig. 47). Attempts to move these joints cause the victim considerable pain, and this problem can last for weeks after the bite, despite an otherwise complete recovery.

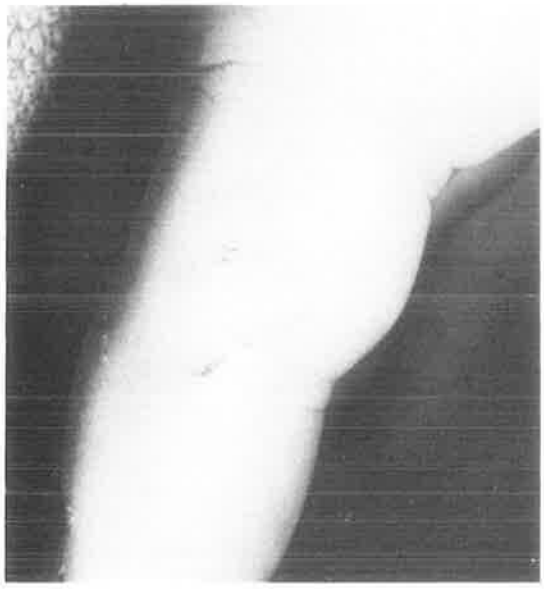


FIG. 47: Death Adder Bite. Dual fang marks on finger with no erythema, mild oedema, and limitation of movement of finger.

Red-bellied Black Snakes, Tiger Snake, and Mulga Snake bites are all associated with local pain, oedema, and occasionally necrosis (figs. 48, 49) (Colour plate 18) though the extent of local reaction is variable. There are few bites of the Taipan documented, and only one definite bite from a Small-scaled Snake documented. In the latter there was some local bruising around the bite site.

Continued bleeding from the bite site is naturally a good indication of a coagulation disorder, and therefore of effective systemic envenomation.

(ii) Lymph node involvement

Involvement of regional lymph nodes should theoretically be a universal feature of all significant cases of envenomation as the venom moves principally via the lymphatic system from the bite site to the general circulation.[5,50,51] However, lymph node

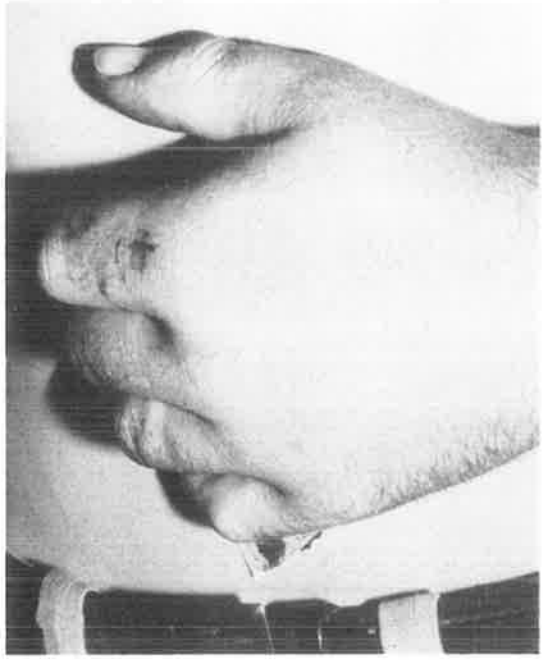


FIG. 48: Red-bellied Black Snake Bite. Marked oedema of whole hand, with marked local pain.

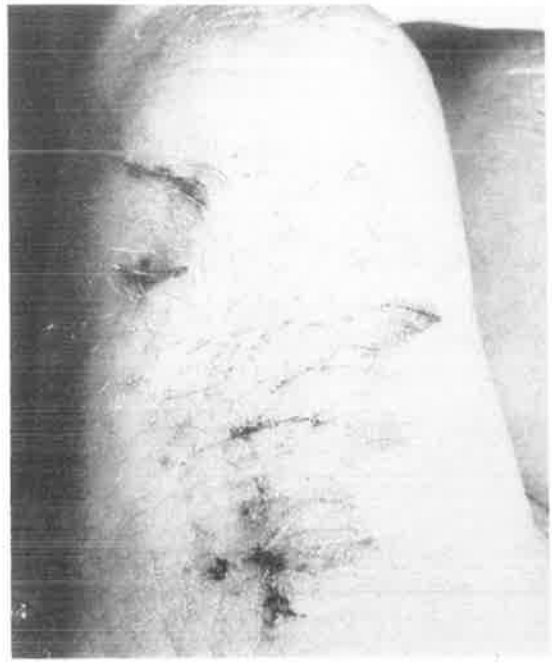


FIG. 49: Red-bellied Black Snake Bite. Detail of multiple bite lacerations to finger, after multiple bite.

tenderness and/or enlargement is not universally found in serious cases of snakebite. Nevertheless, it should be looked for and is usually a harbinger of systemic envenomation.

Campbell[79] reported that the earliest sign of snakebite in patients envenomated in Papua New Guinea, was tender, enlarged regional lymph nodes, which were involved about 1–2 hours after the bite.

(iii) Systemic involvement

The pattern of systemic signs, like all other signs of snakebite, is highly variable. General systemic signs observed in snakebite include tachycardia, hypotension, hypertension, vomiting, restlessness, and impairment of conscious state. Haematemesis, haemoptysis, and haematuria may occur. Hyperthermia may be observed.

However, of great significance in observing a patient with systemic envenomation, are signs of specific toxin activity, and especially neurotoxicity.

Neurotoxic signs usually develop and progress over several hours, and the early signs may be subtle and easily missed. The most highly innervated muscles are those first to show neurotoxic paralytic effects. Thus extrinsic ocular muscles and the ciliary muscles controlling the lens usually provide first evidence of paralysis. Vision will be blurred, and careful testing of ocular movement may show abnormalities. There may be diplopia on lateral or upward gaze. The muscles associated with speech are often affected early, and as mentioned earlier, the voice may acquire distortions from nasal escape, then slurring as the tongue becomes progressively paralyzed. All other voluntary muscle groups will usually be involved in the untreated or inadequately treated case, and tendon reflexes will be decreased or abolished. As paralysis advances, the physician should carefully observe the upper respiratory pathways, as paralysis of the tongue and facial muscles can lead to respiratory obstruction. Signs of respiratory embarrassment as respiratory muscles are progressively paralyzed should be sought. There may be an initial tachypnoea, and use of accessory muscles of respiration, but as these are paralyzed and the diaphragm has an increasing percentage of the respiratory workload, so chest movement with respiration will fall and abdominal movement with breathing may be more apparent.

Signs of myopathy and rhabdomyolysis should be sought, and these typically are muscle movement pain and, in later stages, wasting of skeletal musculature.

The conscious state is frequently impaired in cases of significant systemic envenomation, although it is not a universal finding. As noted earlier, the relation of conscious state impairment to neurotoxins is uncertain. Cerebral haemorrhage has been described in association with snakebite[108,86] and evidence of such a catastrophe should be sought in any envenomed patient who is unconscious. However, it should be stressed that this complication has only been reported twice from Australia.

Convulsions have been reported in several cases, especially with Taipan bite, and are sometimes a very early manifestation of envenomation.[87,88,51]

(c) Time scale

Like other clinical features discussed in relation to snakebite, the timing of progression of symptoms and signs is highly variable.

In children, where a bite may occur without an adult's knowledge, and no first-aid may be applied, symptoms and signs of systemic envenomation may be seen within 10–15 minutes of the bite, and often within 30 minutes. The first sign of trouble may be the collapse of the child, who may rapidly become semi-conscious or unconscious. At least in the case of Brown Snake bites, and probably for some other species too, the defibrination syndrome, with hypocoagulable blood, may be detectable at this stage.

However, peripheral neurotoxic problems usually take longer to become established. Certainly experimentally, there is a

latent period of between 30–60 minutes between application of venom to the neuromuscular junction, and established block of transmission.[61] This latent period is decreased by muscle activity. Clearly, in a human victim, unless venom is injected intravenously, there will be considerable delay between the bite, and the passage of the venom through the lymphatic network, via the thoracic duct, to the general circulation, and hence to neuro-muscular junctions. The speed of passage will depend on first-aid undertaken by the victim. A child bitten on a leg, who continues to play and run around after the bite will sustain much faster venom movement than an adult who immediately has a lymphatic bandage and splint applied, and is then motionless until medical care is reached.

In a series of 52 cases of snakebite in Papua New Guinea Campbell[79] reported that 33% developed symptoms within one hour or less of the bite, 15% developed symptoms from 1–2 hours after the bite, and the remaining 52% developed symptoms in 2–12 hours after the bite. The earliest signs – lymph node tenderness – appeared about 1–2 hours after the bite.

3. COMPLICATIONS OF ENVENOMATION

(a) Neurotoxicity

The neurotoxins are probably the most distinctive, important, and lethal components of Australian elapid snake venoms. They act post-synaptically, and in some species, pre-synaptically as well. The occasional catastrophic paralysis of voluntary muscle that they can cause, if untreated, will almost inevitably lead to the lingering death of the victim. Details of their pharmacology have been dealt with earlier in this paper.

Surprisingly, detailed analyses of neurotoxic envenomation in man are not legion in the Australian medical literature. The most detailed accounts of series of cases are from Papua New Guinea, where the three snakes most involved were the Taipan, Death Adder, and Papuan Black Snake.

Campbell[79] noted that the earliest signs of neurotoxic envenomation were slight ptosis usually associated with slight impairment of upward and lateral gaze. Following this, a nasal voice developed, with difficulty in speaking, in swallowing, and in opening the mouth. This progressed, untreated, to a general muscle weakness. A more detailed description of these neurotoxic symptoms and signs appears earlier in this paper.

In severe cases of paralysis, Campbell notes complete ocular muscle paralysis, with fixed gaze, bilateral ptosis, but pupils still reacting to light. In the most severe cases, the victims "lay as if dead, and the only movement detectable was an ineffectual twist of the pelvis".

Paralysis was symmetrical. The diaphragm appeared most resistant to paralysis, and with maintenance of life (e.g. tracheostomy, etc.), it took from 24–30 hours for complete diaphragmatic paralysis. This paralysis remained complete for about six hours, after which weak diaphragmatic movements recommenced. It took a further one – four days for sufficient recovery to enable the patient to breathe unaided.

Next to recover were ocular muscles, which showed signs of recovery about 48 hours after the bite. Within two – five days of the first sign of ocular movement, most muscle function was recovered. A further week or more was required for recovery of muscle power.

Fotheringham[58] reported a case of neurotoxic envenomation in an $8\frac{1}{2}$-year-old male with partial respiratory paralysis. The snake involved was not identified but is likely to have been either a Tiger Snake or a Brown Snake. The victim saw the snake brush his right leg but felt no pain (suggestive of Brown Snake). He later developed a severe frontal headache, and became tired and lethargic. He was seen by a doctor, but the diagnosis of snakebite was not made. The following day he had respiratory distress, and a hoarse voice with slurred speech. He was drowsy and slow to obey commands. Pupils were dilated and unreactive, and there was a bilateral facial palsy and ophthalmoplegia. However, no limb palsies were evident, although reflexes were sluggish. Despite antivenom (polyvalent), there were significant signs of neurotoxic para-

lysis for at least five days. This illustrates the effect noted by Campbell, that when once established, neurotoxic block is not reversed by antivenom, except for the Death Adder venom. This also accords with experimental work on pre-synaptic toxins, which appear unaffected by antivenom once in the nerve terminal. This case also illustrates the natural history of neurotoxic paralysis, with slow recovery over several days, as noted by Campbell (fig. 50).

Other accounts of progressive neurotoxic paralysis, some fatal, have been reported following envenomation by the Taipan,[87,88,91,102] Small-scaled Snake,[56] Tiger Snake[59] and Rough-scaled Snake.[92,106] This is not an exhaustive list of all such reports.

(b) Central nervous system involvement

The action of Australian elapid venoms on the C.N.S. is not well-understood, although it has been suggested that C.N.S. toxicity is of little significance compared with other effects of these venoms.[35]

However, the early onset of unconsciousness, without apparent anoxia, has been recorded by a number of authors, for a variety of snake species.[56,57,59,87,88,89] In some cases, the first noted symptom or sign of systemic involvement is a sudden lapse into unconsciousness, sometimes associated with convulsions.[88] I have seen two boys envenomated by Common Brown Snakes, who have had C.N.S. involvement. One, age $2\frac{1}{2}$ years, was bitten just below the buttocks, (fig. 51) and appeared well for about 15 minutes. The child then became unconscious and had what appears to have been a grand mal convulsion (not observed by me). This child remained drowsy and irritable for about six hours. The second child, age seven years, was bitten on the hand, and the first problem noted, about 20 minutes after the bite, was drowsiness and lethargy, quickly followed by peripheral shutdown.

Convulsions have been reported following snakebite, as mentioned earlier and above.[57,60,87,88] From the scanty information available they appear to be of the grand mal type, and there may be a single fit or multiple fits.

There are no reports of long-term C.N.S. problems following Australian snakebite. There is one report of dementia following Tiger Snake bite,[90] but this appears to be the result of cerebral anoxia

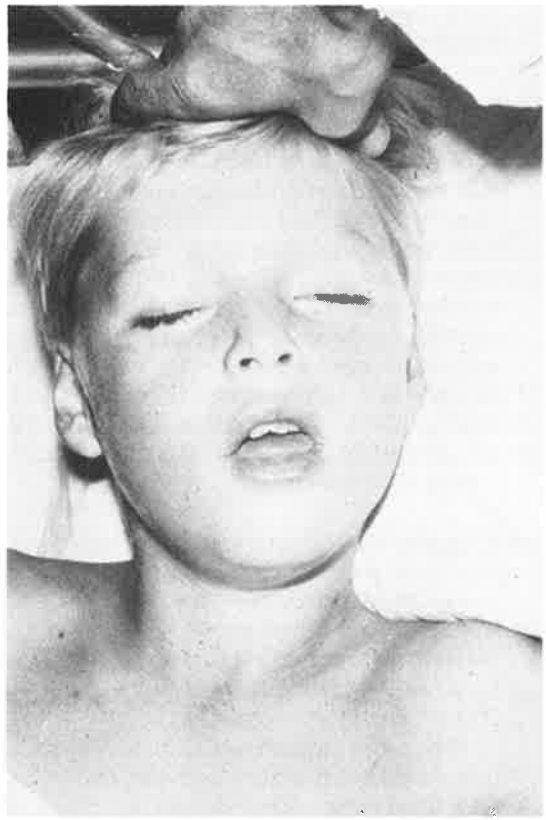


FIG. 50: Neurotoxic paralysis following snakebite with bilateral ptosis, slack jaw, and slightly protruding tongue.

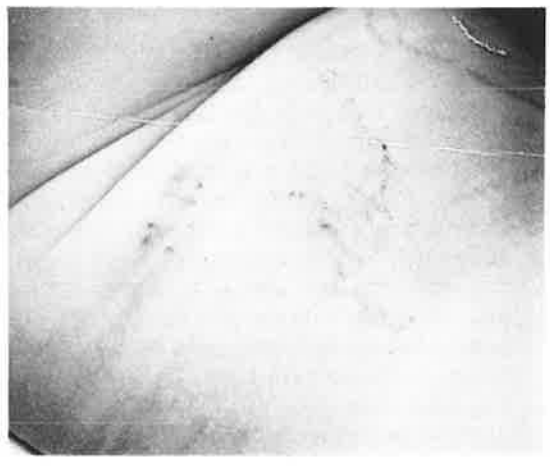


FIG. 51: Brown Snake Bite. Multiple bites on posterior thigh, just below buttocks. No local oedema. Patient developed defibrination syndrome.

following a severe anaphylactic shock on administration of antivenom. However, there are reported cases of cranial nerve malfunction persisting after recovery from snakebite. These usually relate to loss of smell and disturbance of taste, and have been reported following Taipan bite.[88] In one case there was complete loss of taste and smell, but two months later there was partial return of smell, which was altered, everything smelling the same, and disagreeable.[88] Taste likewise returned, but was altered. Salt and sugar tastes returned to normal but "sauces and the like [which perhaps really depended more on the sense of smell] all tasted the same." In another case of Taipan bite, there was short-term disturbance of smell and taste, with later complete recovery.[91] I am aware of one case of envenomation by a Red-bellied Black Snake, with loss of smell, which the victim claims is still completely absent five years later.

(c) Coagulation disturbance – the defibrination syndrome

As mentioned in the section on Venoms, Australian elapid venoms have a variety of effects on human blood, amongst which disturbances of coagulation are the most prominent.

The most important clinical coagulation problem in snakebite is the defibrination syndrome. This occurs in cases of systemic envenomation, probably as a result of prothrombin converters in the venom, which in turn cause formation of thrombin, then conversion of fibrinogen to fibrin, which is then destroyed causing elevated titres of fibrin degradation products, and very low fibrinogen titres, with consequent hypocoagulability.

The defibrination syndrome is usually detected when laboratory investigation of coagulation function is performed. Sometimes it is evidenced by prolonged bleeding from the bite site, or a venepuncture site. Rarely cutaneous ecchymoses, or subcutaneous haematomata may be seen. Scalp haematomata have been reported[59] in a four-year-old child. Bleeding from the gastro-intestinal tract may occur, with haematemeses.[59,93] Haemoglobinuria is a common finding.[59,56,92,93,94,95] Haemoptysis has been reported in cases from Papua New Guinea.[93] Continued ooze from tracheostomy wounds in those patients with co-existent neurotoxic paralysis has also been reported.[93,92] There is one case of abnormally increased menstrual flow in a mild case of defibrination syndrome.[96]

Laboratory findings vary with the species of snake involved. Several interesting series have been published. In 1966 Champness[93] reported experience with the defibrination syndrome in snakebites in Papua. The snakes implicated were the Taipan and Papuan Black Snake. In a series of 22 cases of alleged snakebite, 11 had clinical signs of envenomation, and six of these had defibrination syndrome. Five of these six also had severe neurotoxic envenomation. In three cases there was haematuria. One patient died (of neurotoxic problems). In five of the cases the blood would not clot at initial testing. In only one case was there definite haemolysis. Platelets, where checked, were normal. Only one case had sufficient blood loss to be a threat to circulation. Fibrinogen titres were reduced in all cases. Indeed in four cases fibrinogen was not detectable.

In Australia, the defibrination syndrome is most commonly reported after envenomation by members of the Brown Snake genus. Herrmann *et al.*[96] described the defibrination syndrome following envenomation by the Dugite, *Pseudonaja affinis*. They reported three non-fatal cases, all in adults. Two of these cases had initially incoagulable blood, with very low or undetectable fibrinogen, and marked elevation of fibrin degradation products. These cases also showed depletion of Factor II (25%, 12%), Factor VIII (55%, 2%), Factor V (19%), Factor IX (78%), and Factor VII plus X (55%). Assay of Factor X was normal in both cases. Their third case had definite symptoms of envenomation, but was apparently not seen until 18 hours after the bite, by which time symptoms were receding. Here clotting time was normal with good clot retraction, but the fibrinogen titre was reduced, and Factor VIII was only 60%

Schapel *et al.*[94] reported a case of envenomation by the Common Brown Snake with defibrination syndrome in addition to neurotoxic problems. The

blood was initially incoagulable, with no detectable fibrinogen. Factor assays were not reported.

Crawford[95] reported a case of envenomation by a juvenile Western Brown Snake, or Gwardar (*Pseudonaja nuchalis*). Though the length of the snake was not given, the photograph depicts a colour phase usually only seen in juveniles of less than 40 cm length. The patient was a 57-year-old man. No neurotoxic problems developed but he had a definite defibrination syndrome, with afibrinogenaemia. Factor assays showed depletion of several factors; Factor II (28%), Factor V (18.5%), Factor VII (76%), Factor VIII (6.5%), Factor IX (44%), Factor XI (85%), and Factor XII (14%). The platelet count was normal. Fibrin degradation products were markedly elevated. Intravascular haemolysis was also noted.

I have seen two cases of defibrination syndrome in children following envenomation by snakes. In one case the snake was seen by parents and described as grey or brown and about four feet long. This was in an Adelaide suburb where the Brown Snake and Adelaide Hills Copperhead are the only known species. This child developed typical defibrination syndrome, with afibrinogenaemia, and depletion of several clotting factors, most notably Factors V and VIII. Serial results for this child are given in Table XVIII. This child suffered multiple bites to the upper thigh, and no first-aid was instituted until after the child collapsed in hospital (fig. 51). The response to therapy in this case will be discussed later (page 379).

The second case was a $7\frac{1}{2}$-year-old boy bitten by a brown-coloured snake about two feet long. The C.S.L. ELISA later confirmed that serum for this case contained Brown Snake venom. Serial results on this case are shown in Table XIX. He was playing with reptiles and claimed he had been bitten by a legless lizard, which was caught and brought home. He continued to play after the bite, then went home where he collapsed about 15 minutes after the bite. A girl who was with him when he was bitten claimed he was bitten by a Brown Snake, so an ambulance was called. However, the legless lizard was produced, and so the parents decided his collapse was false and no first-aid was given. On arrival of the retrieval party and ambulance, about 35 minutes after the bite, he was still pallid, sweaty, irritable and drowsy, and blood taken then failed to clot over the following half-hour. On this basis he was given Brown Snake antivenom. This case is very instructive, for on available history, the child had not been envenomated, but simple testing of whole blood clotting time showed clear evidence of envenomation. This initial blood sample contained Brown Snake venom on ELISA testing. As with the previous case, the defibrination syndrome was established within 30 minutes of the bite.

From this small group of cases it is clear that defibrination syndrome may occur following envenomation by any of the three major species of Brown Snake. A comparative summary of initial laboratory findings in such cases of defibrination syndrome is given in Table XX. These cases confirm that the defibrination syndrome can be established in less than three hours after the bite, and may be apparent within 30 minutes after the bite. Typical findings in the untreated state are incoagulable blood, afibrinogenaemia, elevated fibrin degradation products and significant deficiencies of Factors II, V and VIII, with variable deficiency of other factors. Platelet counts are normal.

The defibrination syndrome has also been described following Tiger Snake envenomation.[59] In one case, a four-year-old boy, there was marked prolongation of Prothrombin Time (P.T.) and Activated partial thromboplastin time (A.P.T.T.) at four hours after the bite, with no detectable fibrinogen and bleeding from the venepuncture site. Following further antivenom, the P.T. and A.P.T.T. were normal at 18 hours, and the fibrinogen titre normal at 48 hours. In a second case, a 28-year-old male, defibrination syndrome with fibrinogen depletion and oozing from injection sites was confirmed at two hours and following treatment, normal at 24 hours.

Campbell[97] describes a case of Tiger Snake bite in a 42-year-old male with defibrination syndrome. About one hour after the bite his blood was unclottable,

TABLE XVIII: Defibrination syndrome in a 2½-year-old male following envenomation by an unidentified snake, presumed to be a common brown snake

	TIME AFTER BITE						
	35 mins.	3 hours	4 hours	4 hours 40 mins.	6 hours	19 hours	43 hours
Antivenom – Tiger Snake	3000u.	–	3000u.	–	–	–	–
Antivenom – Brown Snake	1000u.	–	2000u.	–	–	–	–
Clotting time	Unclottable	Unclottable	–	Very weak clot with lysis	6½ mins. weak clot	7¾ mins.	7¾ mins.
Prothrombin time	>150 secs.	>15 mins.	–	>15 mins.	120 secs.	14.5 secs.	12.5 secs.
A.P.T.T.	> 90 secs.	>15 mins.	–	>15 mins.	55 secs.	34 secs.	34 secs.
Fibrinogen (mg/ml)	–	Not detected	–	Not detected	Not detected	74	174
Fibrin degradation products (mg/ml)	–	>5000	–	>5000	2500	800	30
Platelet Count	–	250000	–	–	–	250000	224000
Factor II	–	54%	–	58%	55%	–	–
Factor V	–	15%	–	34%	72%	–	–
Factor VII	–	44%	–	49%	55%	–	–
Factor VIII	–	8%	–	23%	50%	–	–
Factor IX	–	100%	–	100%	100%	–	–
Factor X	–	50%	–	80%	80%	–	–
Clinical state	Irritable drowsy	Irritable drowsy	Irritable drowsy	Irritable drowsy	Fully conscious, playing with parents	Normal	Normal

TABLE XIX: Defibrination syndrome in a $7\frac{1}{2}$-year-old male following envenomation by a brown snake

	TIME AFTER BITE						
	25 mins.	1 hour	5 hours	7 hours	approx 18 hrs.	approx 40 hrs.	approx 64 hrs.
Antivenom – Brown Snake	–	1000u.	–	–	–	–	–
Clotting time	Unclottable	–	20 mins.	$5\frac{1}{2}$ mins.	5 mins.	$5\frac{1}{2}$ mins.	$5\frac{1}{2}$ mins.
Prothrombin time	–	–	$3\frac{1}{2}$ mins.	30 secs.	18 secs.	13 secs.	13 secs.
A.P.T.T.	–	–	74 secs.	51 secs.	46 secs.	34 secs.	32 secs.
Fibrinogen (mg/ml)	–	–	14	19	–	99	156
Fibrin degradation products (mg/ml)	–	–	2000	1000	500	10	6
Platelet count	–	–	265000	–	–	187000	253000
Clinical state	Irritable drowsy	Irritable drowsy	Irritable – less drowsy	Awake, not irritable	Normal	Normal	Normal

TABLE XX: Comparison of initial coagulation studies in cases of defibrination syndrome following envenomation by members of the brown snake genus

	Common Brown Snake[94] (*Pseudonaja textilis*)			Dugite[96] (*Pseudonaja affinis*)			Gwardar or Western Brown Snake[95] (*Pseudonaja nuchalis*)
	Case 1	Case 2	Case 3	Case 1	Case 2	Case 3	Case 1
Time after bite	3 hrs.	5 hrs.	?	2 hrs.	18 hrs.	2 hrs.	?
Whole blood clotting time	Unclottable	20 mins.	Unclottable	Unclottable	6 mins.	Unclottable	Unclottable
Prothrombin time	>15 mins.	$3\frac{1}{2}$ mins.	>20 mins.	>120 secs.	16 secs.	>120 secs.	Grossly prolonged
A.P.T.T.	>15 mins.	74 secs.	–	–	–	–	Grossly prolonged
Fibrinogen assay (Mg/100 ml)	Not detected	14	Not detected	<10 mg	<10 mg	Not detected	Not detected
Fibrin degradation products (μg/ml)	>5000	2000	–	Increased	Slightly increased	Increased	Markedly increased
Factor II	54%	–	–	25%	–	12%	28%
Factor V	15%	–	–	–	–	19%	18.5%
Factor VII	44%	–	–	–	–	55%	76%
Factor VIII	8%	–	–	55%	60%	2%	6.5%
Factor IX	100%	–	–	–	–	78%	44%
Factor X	50%	–	–	100%	–	100%	–
Factor XI	–	–	–	–	–	–	85%
Platelet count	250000	265000	nil	190000	185000	250000	Normal

with gross disturbance of clotting factors. Virtually no fibrinogen was detected. There was a return to virtually normal values within 12 hours of the bite and early administration of specific antivenom.

Catastrophic haemorrhage related to defibrination syndrome in these cases appears to be rare. Though persistent oozing from the bite site or venepuncture sites may be a problem, this will not exsanguinate the patient. In series from Papua New Guinea where tracheostomy was required, persistent bleeding from the tracheostomy wound was a problem, but in only one case was transfusion required for this.[93]

Sullivan[86] quotes Sutherland as reporting a case of death from sub-arachnoid haemorrhage following a snakebite in Queensland. Foxton[108] describes a fatal case of Brown Snake bite, with haemorrhages in the floor of the lateral ventricles, the pons and medulla at autopsy. Death occurred two hours after convulsions and subsequent coma, and was ascribed to the brainstem haemorrhages.

(d) Rhabdomyolysis

As noted earlier, rhabdomyolysis is a feature of envenomation by several Australian elapid snakes. Experimentally, rhabdomyolysis can be caused by the venoms of the Tiger Snake, Taipan (and presumably the Small-scaled Snake), Mulga Snake, Red-bellied Black Snake, Copperhead and Rough-scaled Snake. No myolytic activity has been detected in the venoms of the Death Adder, Common Brown Snake, or Western Brown Snake (Gwardar).[67]

Clinically, severe rhabdomyolysis following Australian elapid envenomation has only been reported infrequently. In a fatal case of Mulga Snake (*Pseudechis australis*) envenomation, Rowlands *et al.*[55] noted all muscles examined at autopsy showed swelling and acute coagulative necrosis of numbers of muscle fibres. The most severely affected muscles were those of the bitten limb (right arm), the respiratory muscles, and the extraocular muscles. An inflammatory reaction was only observed in the muscles of the right arm. The myocardium showed many small foci in which muscle fibres were swollen with indistinct markings, vacuolation of the sarcoplasm and absent nuclei. The victim, a 20-year-old male farm labourer, died 40 hours after the bite. Early symptoms included nausea and vomiting. Some hours later he developed lethargy, and limb movements were weak. Presumed myoglobinuria was present at this stage, with red discolouration of the urine. There were progressively developing signs of neurotoxic problems with ptosis, poor chest expansion, limited jaw opening, and partial paralysis of the tongue. He was unable to maintain any voluntary muscle contraction in the limbs against more than slight resistance. All deep tendon reflexes were decreased. His condition progressively deteriorated and he died following a cardiac arrest. It is not recorded if he had any pain on muscle movement, which is typical of rhabdomyolysis.

Hood and Johnson[66] reported a non-fatal case of severe renal failure associated with rhabdomyolysis and myoglobinuria following a Tiger Snake bite. The victim, a 47-year-old male, who had been bitten by snakes many times previously, was bitten on the left hand. He promptly received Tiger Snake antivenom. The following day he developed aching tender muscles and dark discolouration of his urine. The next day he was oliguric and delirious, with diminished deep tendon reflexes. Creatinine phosphokinase was grossly elevated (4,640 U.; N < 50 U.). He was oliguric, requiring peritoneal dialysis and haemodialysis for about 10 days, and muscle wasting was gross. Muscle biopsies showed a focal necrotizing myopathy. Recovery in this case was complete, with return to normal muscle bulk and power.

Furtado and Lester[65] reported a fatal case of acute renal failure and myoglobinuria in a 20-year-old male bitten by a Small-eyed Snake (*Cryptophis nigrescens*) in north Queensland. As this snake was not considered dangerous, no treatment for the bite was sought. Three days later he developed jaw muscle weakness and pain, followed two days later by intense muscular pain in both lower limbs, with decreased power. His urine was dark in colour. The association with snakebite was not made, but by the eighth day he was oliguric and he had severe muscle weakness

with poor respiratory excursion, requiring a tracheostomy. Soon after this he died following a cardiac arrest. Autopsy confirmed myoglobinuria, and areas of muscle necrosis.

(e) Renal problems

Renal failure has been reported following envenomation by a variety of Australian elapid snakes, and one case of nephrotic syndrome secondary to envenomation has also been described. The mechanisms of renal failure in snake bite are varied, and renal lesions reported worldwide include glomerulitis, glomerulonephritis, arteritis, interstitial nephritis, tubular necrosis, cortical necrosis, and renal infarct.[98] No specific nephrotoxins have been isolated from Australian venoms.

In those cases of renal failure reported from Australia, acute tubular necrosis is the lesion most often seen. Harris *et al.*[99] reported three cases of acute renal failure with recovery after envenomation by the Gwardar and Dugite in Western Australia. In two of their three cases no antivenom was given. All three cases had blood pictures consistent with microangiopathic haemolytic anaemia, and were oliguric for 14–21 days. No renal biopsies were done but acute tubular necrosis was thought to be the renal lesion. None of these cases developed documented defibrination syndrome or rhabdomyolysis. There was no significant neurotoxic envenomation. All cases required dialysis. Renal recovery was complete in two cases, and in the third case the blood urea was still elevated at 12 months.

Three cases of myoglobinuria reported[55,66,65] (see section on Rhabdomyolysis) following snakebite all showed renal damage. In a case of Tiger Snake envenomation with myoglobinuria and acute renal failure[66] the victim developed oliguria about two days after the bite, which was treated promptly with specific antivenom. He developed hyperkalaemia (to 8.3 mEq/1), hyperphosphataemia (to 12.8 mg/100 ml) and hypocalcaemia (4.6 mg/100 ml), requiring peritoneal dialysis, haemodialysis, Resonium A, insulin and glucose, calcium gluconate and calcium chloride. About two weeks after the bite he entered the diuretic phase and a renal biopsy at this time showed the recovery phase of acute tubular necrosis. His renal function subsequently returned to normal.

A fatal case of snakebite by the Small-eyed Snake (*Cryptophis nigrescens*)[65] had myoglobinuria associated with renal failure which developed several days after the bite. There was some hyperkalaemia (6.7 mEq/1). At autopsy the kidneys were macroscopically tense with dark medullae, and microscopically the distal tubules were congested with eosinophilic material.

A fatal case of Mulga Snake envenomation[55] died before renal failure was definitely established, but at autopsy, the kidneys were congested and swollen, with swollen glomeruli filling almost completely Bowman's capsule. The renal vessels were normal.

A fatal case of envenomation by the Rough-scaled Snake (*Tropedechis carinatus*) developed oliguria and gross elevation of blood urea[92]. Autopsy showed pallor of the cortex, and congestion of the medulla. Histologically there was fragmentation of the cytoplasm of the proximal convoluted tubules, and the epithelium of the distal tubules was flattened, with reduction of the number of nuclei. The glomeruli and renal vessels were normal.

Steinbeck[100] reported a case of nephrotic syndrome thought to have been secondary to envenomation by a snake, presumed to be a Brown Snake or Taipan. The victim, a 23-year-old female was uncertain about being bitten, but later that day became lethargic, with oedema of the bitten foot. She later developed oedema of the feet, legs, abdomen and around the eyes. On the third day she developed polydipsia and oliguria, and this and the oedema became worse. She sought medical aid on the fifth day, and the oedema progressed. There was definite proteinuria and low blood albumin and protein levels. She developed bilateral pleural effusions and ascites, and an elevated blood urea. The oliguria persisted for seven weeks, but by the fifteenth week the urine was consistently free of protein.

(f) Respiratory problems

As noted earlier, neurotoxins can cause

progressive and fatal respiratory paralysis, and tracheostomy and I.P.P.V. may be required. However, in addition to these problems, pulmonary oedema may also occur, and be severe enough to require treatment.

In one fatal case,[92] autopsy showed a fibrinous and diffuse polymorphonuclear cell exudate in grossly congested and oedematous lungs. Both lower lobes were consolidated. The ante-mortem profuse pulmonary oedema undoubtedly contributed to difficulties with ventilation despite tracheostomy and I.P.P.V., and death was considered to be due to the acute pulmonary oedema.

In a fatal Mulga Snake bite[55] there was basal congestion of the lungs and pulmonary oedema at autopsy.

One non-fatal case of Tiger Snake bite[66] with renal failure developed extensive bilateral bronchopneumonia, requiring I.P.P.V. for 10 days.

(g) Local tissue injury

Though common after viperid bite, local tissue destruction around the site of a bite is unusual in Australian elapid envenomation. As discussed earlier, local reaction to envenomation varies with the species of snake. In my experience Brown Snakes usually cause little or no local pain, oedema or erythema, and the same applies to Copperheads. I have seen two Death Adder bites, both on fingers. In both cases, although there was little oedema, the fingers remained painful and stiff for several weeks.

Tiger Snake bites and Red-bellied Black Snake bites are often associated with local pain and oedema.

Two cases of Mulga Snake bite with local damage have been reported. In a fatal case there was gross oedema and discolouration of the bitten hand and arm, with severe subcutaneous oedema, haemorrhage and infiltration with polymorphonuclear cells at autopsy.[55] In a non-fatal case, the victim was bitten at the base of the thumb, and subsequently developed gangrene of the thumb requiring amputation and plastic surgical reconstruction (fig. 52).[101]

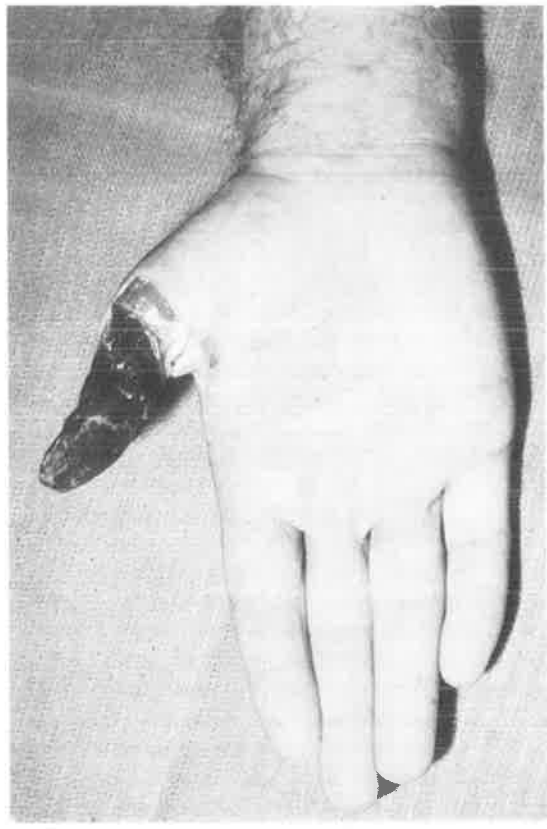


FIG. 52: Mulga Snake Bite. Gangrene of thumb requiring amputation, following bite at base of thumb.

I have seen local gangrene of skin requiring skin grafting, following envenomation by a Tiger Snake (Colour plate 18). In this case an excessively tight tourniquet was applied for a prolonged period. Local skin necrosis has also been reported following a Taipan bite.[102]

In Fotheringham's report of a case of neurotoxic paralysis in a $8\frac{1}{2}$-year-old boy bitten by an unidentified snake, he also noted some necrosis of skin between the supposed puncture wounds on the leg.[58] This wound later became infected and surrounded with a cellulitis, which responded well to antibiotic therapy.

(h) Death

In Australia's early history death was probably a frequent outcome of snakebite. However, as discussed under epidemiology, even in the pre-antivenom era, most people bitten by snakes survived. The pre-antivenom mortality rates for several snake species have already been given (Table XVII).

Of those people who died in those days nearly half took more than 24 hours to die, and only 15% died in the first six hours. (Table XXI).[17] With the availability of specific antivenom, use of correct first-aid, and good medical care, death should become almost unknown as a consequence of snakebite in Australia.

TABLE XXI: Death time in fatal cases of elapid snakebite in Australia, prior to the advent of antivenom[17]

Death time after bite	*Number of cases*	*Percentage*
0–3 hours	8	8.8%
3–6 hours	7	7.7%
7–12 hours	14	15.4%
13–24 hours	24	26.4%
24 hours and over	38	41.8%
TOTAL	91	

4. SUMMARY OF EFFECTS OF ENVENOMATION FOR EACH SOUTH AUSTRALIAN ELAPID SNAKE

(a) Death Adder and Desert Death Adder (*Acanthophis antarcticus* and *A. pyrrhus*)

There is no direct information available on the Desert Death Adder, but it probably has venom similar to the Common Death Adder, for which there is a considerable amount of information.

The largest series of Death Adder bites reported are from New Guinea. Campbell[103] has reviewed reports of Death Adder envenomation.

The Death Adder usually conceals itself in leaf litter or other debris, and will not move on the approach of humans. Thus it may be trodden on more easily than other Australian snakes. It is often active at night. It strikes low and so bites above the ankle in an adult are unlikely to be due to a Death Adder. The obvious exception to this is in snake handlers.

The fangs are quite long and mobile and a moderate amount of venom can be injected.

The site of the bite may be painless or mildly painful. Oedema is not common, and slight when present. However, when bitten on a finger, there may be considerable limitation of movement of the digit. Bleeding from the bite site does not occur.

Symptoms are usually mild, until severe neurotoxic paralysis occurs. Early mild headache and occasional vomiting are seen in some cases. There may be some local lymph node pain, which can be severe. Preparalytic neurotoxic symptoms are vague, and there may only be a slight blurring of vision.

The earliest signs of envenomation may develop within one hour of the bite, and include tenderness of local lymph nodes. Ptosis is the earliest sign of paralysis.

In severe cases, paralysis may be total for all voluntary muscles. Cardiac muscle appears unaffected.

The neurotoxic paralysis is reversed readily by antivenom. Coagulation disorders do not appear to occur, and defibrination syndrome has not been reported. Renal failure also has not been reported, nor has local tissue destruction. The venom has no myolytic activity.

(b) Copperhead and Adelaide Hills Copperhead (*Austrelaps superbus* and *A.* sp.)

Information on envenomation by the Copperhead and Adelaide Hills Copperhead is very limited. Both have small fangs, and deliver a small amount of venom. However, venom studies suggest the Copperhead venom is highly toxic. There are no clinical studies but it is apparently strongly neurotoxic, although no pre-synaptic neurotoxins have yet been isolated. It does not appear to cause coagulation disturbances or defibrination syndrome, although it has been reported to have anticoagulant properties *in vitro*. The venom has moderate to strong haemolytic activity. No cases of renal failure or local tissue damage have been reported.

I am aware of two cases of snakebite by the Adelaide Hills Copperhead, both in healthy adult males, the bites being to a finger in both cases. No symptoms or signs suggestive of envenomation occurred in either case, and there was no local oedema or pain. In both cases the snakes were positively identified, and were about 60 cm long. However, this does not imply that the Adelaide Hills Copperhead is harmless. In both cases the bite was glancing and it is possible that little or no venom was injected.

(c) Yellow-faced Whip Snake (*Demansia psammophis*)

This species, which is very common in South Australia, does not appear to be lethal. I am unaware of any reports of envenomation by this species but I know of two amateur herpetologists bitten by adults of this species, both on the hand, who developed severe local oedema which lasted several days and was associated with some malaise and lethargy (fig. 53).

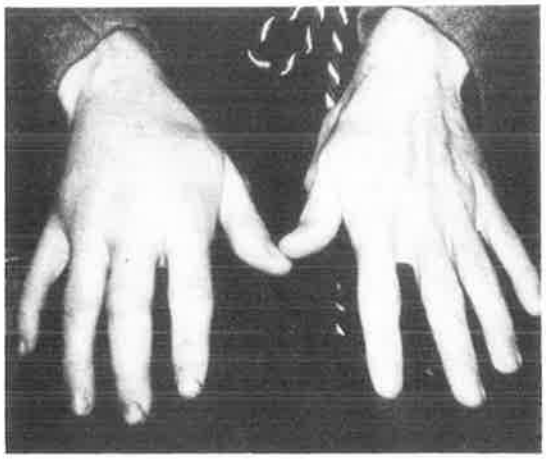


FIG. 53: Yellow-faced Whip Snake Bite. Marked oedema of bitten hand.

(d) Masters Snake and White-lipped Snake (*Drysdalia masteri* and *D. coronoides*)

No information is available on these species, but both are small, unlikely to bite except when deliberately caught, and it is very unlikely that either is dangerous to man.

(e) Bardick (*Echiopsis curta*)

No information is available on envenomation by the Bardick, which is very rarely found, even by herpetologists. It is probably harmless to man, but a recent verbal report to me from Western Australia indicated that a closely related species can cause coagulation disturbances in man. This report is unconfirmed.

(f) Red-naped Snake (*Furina diadema*)

No information available. Almost certainly harmless to man because of its small size and small fangs, and its tendency to attack without opening its mouth.

(g) Western Black-naped Snake (*Neelaps bimaculatus*)

No information is available. A small burrowing species, almost certainly harmless to man.

(h) Tiger Snake and Black Tiger Snake (*Notechis scutatus* and *N. ater*)

The Tiger Snake is one of the best studied of Australian snakes and there are many case reports of human envenomation. Campbell has summarized this information.[97]

Tiger Snake venom is one of the most toxic snake venoms known, and has potent pre- and post-synaptic neurotoxins. The Black Tiger Snake is found in a series of isolated populations, and there appears to be a corresponding diversity of venom potencies. However, those venoms studied from South Australia appear to be highly potent. The fangs are efficient and a moderate amount of venom can be delivered.

Tiger Snakes are usually associated with swamps, rivers or creeks, and prefer moist environments. They are usually active diurnally, but may be active nocturnally in hot weather. As a result bites may occur in a variety of circumstances, and may involve many different regions of the body. It is not inconceivable that a water-skier in the Murray River could be bitten on the trunk.

Local pain and oedema around the bite site are commonly found. In significant bites, pre-paralytic symptoms appear universally, and include nausea, vomiting, headache and sudden loss of consciousness. The onset of these symptoms may be within minutes of the bite or delayed several hours. There may be profound shock with hypotension. Persistent oozing from the bite site is also commonly encountered.

Without treatment neurotoxic manifestations will often ensue, following the typical pattern, with early visual disturbance, dysarthria, dysphagia and then progressive paralysis. Death may occur from respiratory paralysis before significant limb paralysis is evident.

Rhabdomyolysis can occur with Tiger Snake envenomation, and may be associated with renal failure. The defibrination

syndrome can occur. Haemolysis is not prominent but local tissue injury can occur, with skin necrosis.

(i) Small-scaled or Fierce Snake and Taipan (*Oxyuranus microlepidotus* and *O. scutellatus*)

The Small-scaled or Fierce Snake has only been resurrected in herpetological circles in the last decade,[52] and until then was considered synonymous with the Taipan. With its recognition as a distinct species has come work on its venom which reveals it as the world's most venomous land snake.[53,4]

However, there is only one confirmed bite from this species[104] and this victim survived. The case was initially reported as a Taipan bite.[56]

In this single case, the victim, an adult male, was bitten on the right thumb. He applied a tourniquet. Within one hour he was unconscious and incontinent of faeces and urine. However, he regained consciousness about 15 minutes later, and complained of muscle pain. He next developed nausea and vomiting. His arm was swollen, with bruising around the bite site. He experienced difficulty in phonating and swallowing about six hours after the bite, and became progressively agitated, and restless; then disoriented and confused. He then suffered a cardiac arrest, but was successfully resuscitated. At this stage he reached a hospital and was given Brown Snake antivenom, as he thought he had been bitten by a Western Brown Snake. He was on a Bird respirator at this stage, and had frequent ventricular extrasystoles, fixed pupils, bilateral ptosis and ophthalmoplegia. He could voluntarily move his limbs and reflexes were present. He was unable to pass urine and required catheterization.

His next problem was an episode of hypotension, with haematuria and bloody diarrhoea. His blood was now found to be unclottable. However, over the next few hours his condition improved and continued to do so gradually over several days, although he still required assisted ventilation, and a tracheostomy was performed. He was discharged nearly a month after the bite.

From this single case it is clear that envenomation by the Small-scaled Snake can cause rapid development of symptoms including collapse and loss of consciousness. Progressive neurotoxic paralysis may then ensue. There may be some rhabdomyolysis. There may be cardiac arrhythmias and vasomotor signs. A coagulation disturbance may be seen, and this is probably a classic defibrination syndrome. The rapidity of onset and severity of symptoms and signs is indicative of the urgency with which a bite from this species should be treated. The fangs are quite large, and a considerable quantity of venom can be injected.

The closely related Taipan has equally effective fangs and can inject large quantities of venom. In consequence its bite is particularly dangerous, and the death rate from Taipan bite is probably still high. A child in Queensland recently died in less than an hour after a Taipan bite.[89]

Although the Taipan does not normally occur in South Australia, there is an unknown quantity of live Taipans being kept in Reptile Parks, and private homes in the State. Hence it is quite possible that Taipan bites may occur here.

There is a number of individual reports of Taipan bites from Queensland,[87,88,91] and several series from Papua New Guinea.[79,80,81] Campbell has summarized some of the typical findings.[105]

Though headache, nausea and vomiting may occur as early symptoms, in several of the reported cases first manifestations were sudden collapse and convulsions within 4 – 90 minutes of the bite. The patient may then rapidly progress to respiratory paralysis, failure and death, or may regain consciousness. There may be haematemesis, haematuria, and oozing from the bite site. Progressive signs of neurotoxic envenomation will usually ensue, with ophthalmoplegia, dysarthria and dysphagia, progressing to general muscle weakness and respiratory paralysis and failure. Coagulation disturbances can occur, although they are not well documented. It seems likely that the defibrination syndrome can occur. Similarly, in spite of the fact that rhabdomyolysis has not been reported, it probably can occur, and with it, the possibility of renal failure. Although in those cases reported, the

neurotoxic problems have dominated the clinical picture, local tissue damage may occur, with localized necrosis.

(j) Mulga Snake or King Brown (*Pseudechis australis*)

The Mulga Snake is a large aggressive arid land snake with large efficient fangs, and capable of injecting very large quantities of venom. Fortunately the venom is not as toxic as some of its cousins. There are very few reports of Mulga Snake envenomation but one fatal case has been recorded.[55]

As it is a large snake, the Mulga Snake may strike quite high. The local bite site is probably painful and oedematous in all cases. There may then be a delay of several hours before symptoms develop. Early symptoms include nausea, vomiting and headache. In the fatal case these progressed to lethargy and weakness of limbs. The victim then became drowsy, restless, with abdominal pain, respiratory paralysis (partial), and ptosis. Jaw and tongue movement was impaired suggesting progressive neurotoxic envenomation. There was also myoglobinuria indicative of rhabdomyolysis which was extreme in this case – the venom of the Mulga Snake is known to have very powerful myolytic activity.[67] There was associated renal damage. Progressive hypotension followed and the terminal event was a cardiac arrest.

In a non-fatal case there was evidence of coagulation disturbance, with prolongation of clotting time, and severe local tissue necrosis.[101]

(k) Red-bellied Black Snake (*Pseudechis porphyriacus*)

Unlike its close relative, the Mulga Snake, the Red-bellied Black Snake is usually found near water, such as along creeks. It has moderately-sized fangs, but injects only a modest amount of relatively weak venom. It can be a large snake, and may strike high.

There is usually local pain and oedema at the bite site and oedema may involve much of the bitten limb. Symptoms vary and include headache, nausea, and vomiting. There may be oozing from the bite site and a coagulation disorder. Defibrination syndrome has not been recorded but is conceivable and severe intravascular haemolysis may occur. Renal failure and local tissue necrosis have not been reported, nor do neurotoxic problems appear to be significant in this species.

(l) Common Brown Snake, Western Brown Snake (or Gwardar), *Pseudonaja textilis* and *P. nuchalis*

The Brown Snake and Western Brown Snake are very widely distributed in South Australia and undoubtedly are responsible for most cases of snakebite in the State. There are a number of reports of Brown Snake and Western Brown Snake envenomation. Recent studies[4,48] have shown that the Common Brown Snake has the second most toxic land snake venom known, eclipsed only by the Small-scaled Snake. The venom of the Western Brown Snake is considerably less toxic, though still potentially lethal.

However, both species have very small fangs and can deliver only very small quantities of venom. This, plus the tendency of Brown Snakes to strike with the mouth closed, is probably responsible for the low mortality rate.

As the fangs are small, the bite marks may be exceptionally difficult to find. There is no local pain, erythema, or oedema. Systemic symptoms may develop rapidly, and headache, nausea, and vomiting, rapidly followed by collapse and sometimes unconsciousness may occur within 15 minutes of the bite, at least in children. Convulsions may also occur. In adults, events may be less sudden or severe. Intense abdominal pain is often present. Neurotoxic problems may ensue over several hours with ptosis, dysarthria and dysphagia, which can progress to general muscular paralysis, respiratory paralysis, and death.

Oozing from bite sites is uncommon, because of the small size of the puncture wounds, but persistent oozing from venepuncture wounds is common. The defibrination syndrome is very commonly associated with significant cases of envenomation, even when no muscular paralysis is present, and may dominate the clinical picture. Renal failure has been reported, but not local tissue damage, which is unlikely to occur.

(m) Ringed Brown Snake (*Pseudonaja modesta*)

This snake is small with small fangs, and though a member of the Brown Snake group, is not regarded as dangerous. There are no data on its venom, and no cases of envenomation reported. In view of the lack of information, and its close relationship to the Brown Snake, bites from this species should be treated with caution.

(n) Burrowing Snakes:
Coral Snake (*Simoselaps australis*)
Desert Banded Snake (*S. bertholdi*)
Narrow Banded Snake (*S. fasciolatus*)
Half-girdled Snake (*S. semifasciatus*)

All these snakes are primarily burrowers, all are small, with small fangs. There is no information on their venom but they are almost certainly harmless to man. It is very difficult to induce any of these species to bite.

(o) Curl Snake (*Suta suta*)

The Curl Snake or Myall Snake is not reported as causing injury in man, but work now in progress indicates that its venom is probably quite potent, and bites from this species should be treated with care and caution. The fangs are small, and it is unlikely that more than small amounts of venom could be injected. Bites are reputed to be very painful.

As it is often found under debris, it may be encountered by searching children or herpetologists, these being the main risk groups. It is, in fact, an aggressive snake, readily disposed to bite.

(p) *Unechis* group
Mitchell's Short-tail Snake (*Unechis brevicauda*)
Little Whip Snake (*U. flagellum*)
Hooded Snake (*U. monachus*)
Black-headed Snake (*U. gouldii*)

There are no reports of human envenomation by this group of snakes which are cryptic. All are small with small fangs. I am aware of two bites by the Little Whip Snake. In one there was no symptoms or signs of envenomation. In a second case, an eight-year-old boy was bitten by a snake he claimed was a Little Whip Snake, although the snake was not captured or positively identified. He developed marked oedema of the bitten hand and forearm which took several days to subside. He was an asthmatic, with a history of allergies to insect bites.

(q) Bandy Bandy (*Vermicella annulata*)

This small burrowing snake has very small fangs, and is very reluctant to bite. There is no information on its venom or envenomation in man. It is almost certainly harmless to man.

(r) Rough-scaled Snake (*Tropedechis carinatus*) (also called the Clarence River Snake)

This species does not naturally occur in South Australia, being limited to the east coast of Australia, in the Cairns-Innisfail-Mossman area, and in a larger pocket in the Brisbane-to-Taree area, but it is occasionally kept by herpetologists; hence its inclusion in this list. It is a small snake, with small fangs and a low venom yield. Its venom is potentially lethal to man, but it is one of the less dangerous of Australia's venomous elapid snakes (see table III).

Envenomation in man by this species has been well discussed in a review by Trinca *et al.*[106] More recent studies suggest that the venom may contain a presynaptic neurotoxin.[107] In Trinca's series there were 12 cases with one fatality, and that in a case of multiple bites to a man intoxicated by alcohol. However, nine cases developed systemic signs of envenomation, some quite rapidly, and in only one case was there significant local reaction (local oedema and urticaria). Symptoms and signs recorded include initial headache, nausea and blurred vision. Vomiting and abdominal pain occurred in several cases. One case had a haematemesis. Two non-fatal cases collapsed unconscious. In no cases was renal failure or local necrosis reported, nor the defibrination syndrome manifested.

(s) Broad-headed Snakes
Broad Headed Snake (*Hoplocephalus bungaroides*)
Pale Headed Snake (*H. bitorquatus*)
Stephen's Banded Snake (*H. stephensi*)

All three species are confined to the eastern seaboard of Australia. At least one species is classified rare in some of its range. For a variety of reasons, all three species have been popular with reptile keepers, and there may be some in captivity in South Australia.

I am unaware of any fatalities ascribed to envenomation by any of this group, but recent venom studies suggest that at least one species (*H. stephensi*) has a potentially lethal venom, listed as more toxic than the Rough-scaled Snake, Mulga Snake, or Red-bellied Black Snake[4] (table III). In view of this, bites from members of this genus should be treated with great care and caution, and because of the lack of data, attending physicians should make sure cases of envenomation are meticulously recorded, investigated, and reported in the medical literature.

Cogger[3] comments that bites from *H. bitorquatus* are painful, and that bites from *H. bungaroides* may produce acute symptoms.

(t) Small-eyed Snake (*Cryptophis nigresens*)

Another inhabitant of the eastern seaboard of Australia, the Small-eyed Snake is occasionally kept by reptile keepers, and so such people may present with bites from this species.

There is one recorded fatality ascribed to envenomation by this species.[65] The victim was a 20-year-old male reptile keeper who had been bitten by this species on several previous occasions without harm. Details of the case have been described earlier, under Renal Problems. He developed myoglobinuria and renal failure several days after the bite, and died some days later of respiratory failure. There was an apparent coagulation disorder as well, with persistent ooze from a tracheostomy wound.

Venom toxicity studies[4] place the Small-eyed Snake well down the list of potentially lethal snakes (table III).

SECTION IV – TREATMENT

1. INTRODUCTION

Controversy and the treatment of snakebite seem inextricably linked. There have been a vast array of suggested treatments, and much argument in the medical literature about the merits of each. This applies both internationally and in Australia. Some authors lately have reviewed these controversies, such as that surrounding Professor Halford's Ammonia treatment for snakebite, promulgated in Australia at the end of the last century.[14]

Fortunately, treatment of snakebite in Australia has been rationalized in recent years, following careful experimental work involving staff of the Commonwealth Serum Laboratories, Melbourne. (C.S.L.)

In discussing treatment, the aims of such treatment should be remembered. In the case of snakebite they may be summarized, in order of application as:

(1) Prevention of venom reaching the systemic circulation.
(2) Neutralization of any circulating venom.
(3) Correction of venom-induced abnormalities.
(4) Maintenance of vital functions and life.

2. FIRST AID

The principal aim of first-aid for snakebite is the retardation of venom movement from the site of the bite to the systemic circulation.

Over the years a variety of measures to secure this aim have been advocated, including incision of the wound, excision of the wound, the use of a variety of topical chemicals, such as potassium permanganate, the use of arterial tourniquets, and venous tourniquets. These treatments still remain in vogue in some areas, despite recent research which has established a standard treatment for envenomation in Australia.[5]

Sutherland *et al.*[5,109] have clearly demonstrated in experimental work with monkeys, that most venom is transported from the bite site to the systemic circulation via the lymphatics, and not via direct capillary absorption into the venous system. This means that retardation of lymphatic return will immobilize most of the venom, and especially the principal toxic fractions, which are of large molecular weight.

In a classic paper in 1941, Barnes and Trueta[110] demonstrated that Black Tiger Snake venom movement from the bite site was largely via the lymphatics, and that immobilization of the limb greatly retarded lymphatic flow. Sutherland and colleagues were able to study this process in more detail, using monkeys (*Macaca fascicularis*) which closely simulate human victims of envenomation. It was possible to assay circulating concentrations of venom, and even venom components, using a radioimmunoassay technique.

Initial studies using this technique and envenomation with Tiger Snake venom, using a variety of first-aid measures, showed that a

firm crepe bandage at about 55 mm Hg pressure, combined with immobilization of the limb, prevented virtually all venom reaching the systemic circulation.[5] Later studies using the same techniques and studying all major Australian snake venoms, revealed that this method of first-aid was universally effective.

Fairley in 1929[20] had already demonstrated the difficulties of local incision or excision in Australian snakebite, showing that unless these measures were carried out expertly within a few minutes of the bite, they had no value at all.

In view of these studies a definite course of first-aid for snakebite in Australia can be stated:

(1) Bind the bitten limb firmly with crepe bandage or something similar, commencing at the bitten area, and extending to involve the whole limb. The bandage should be at a pressure of about 55 mm Hg, which is about the pressure used to bind a sprained ankle.[5] The arterial inflow and venous outflow of the limb should not be obstructed by such a bandage, and it should be left on until medical facilities, including antivenom and resuscitation equipment, are reached (figs. 54, 55).
(2) The bitten limb should be immobilized with a splint and kept immobilized as long as the bandage is on.
(3) The patient should be encouraged to be as inactive as possible, and should be reassured.
(4) Transport should be brought to the victim whenever possible.
(5) The bite wound should be left untouched. It should *not* be washed, sucked, incised, excised, or treated with chemicals or other agents.

3. MEDICAL TREATMENT

The physician presented with a case of definite, suspected, or possible snakebite must proceed along standard lines of assessment, albeit with some urgency (see Appendix I).

A history should be sought, including time and place of bite or possible bite, circumstances of bite, first-aid applied, details of offending reptile if available, type and progression of symptoms, and type and progression of signs noted by the victim's companions (see Section III).

Enquiries should also be made about previous illnesses, previous snakebite if any, and allergies, particularly in regard to horse serum.

While this is proceeding, examination of the victim may also be commenced, looking for signs of a bite and signs of systemic envenomation, especially signs of neurotoxic envenomation (see Section III).

If the patient has a lymphatic bandage in place, it should be removed over the area of the bite only to inspect the bite, as this may give clues to the extent and type of envenomation. Swabs should be taken from the bite site. The bandage should then be replaced.

If the patient does not have a lymphatic bandage present then one should be applied immediately after inspection of the bite site, unless the victim has been bitten in excess of

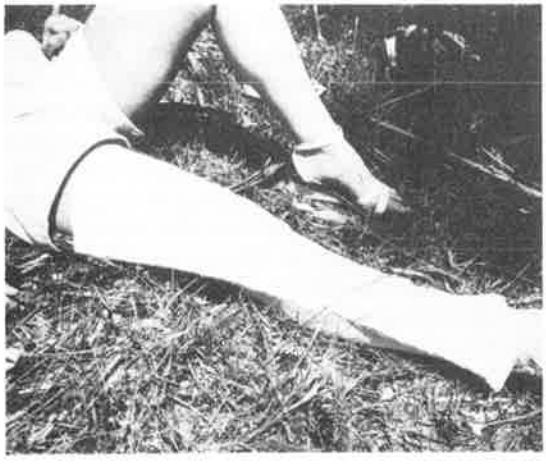


FIG. 54: First aid to lower limb following snakebite. Lymphatic occlusal bandage to whole limb, and splinting of limb.

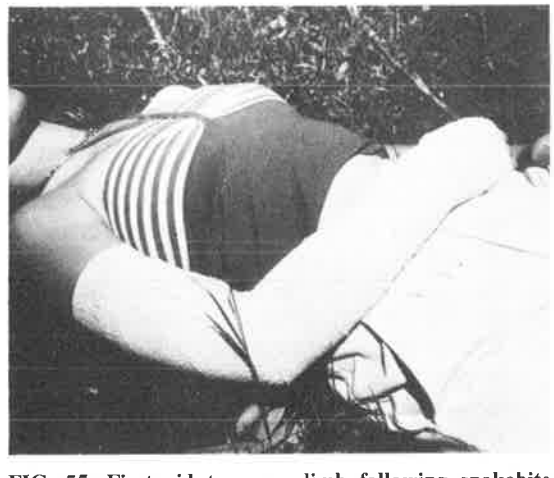


FIG. 55: First aid to upper limb following snakebite. Lymphatic occlusal bandage to most of limb. Limb should now be immobilized.

six hours previously, without subsequent development of signs or symptoms of envenomation.

If a tourniquet is in place, rather than a lymphatic bandage, it should be removed and replaced with a lymphatic bandage as soon as possible, but not until measures are taken to cope with sudden envenomation problems on release of the tourniquet.

In all cases where there is a definite snakebite or a reasonable possibility of snakebite, an intravenous line should be promptly inserted while other assessment procedures are going on. During insertion of the line, before actual attachment of I.V. fluid lines, or as a separate procedure, venous blood should be taken for investigation.

(a) *Laboratory investigations*

Some blood should be frozen for future analysis for venom, if that should prove necessary. Some blood should be tested for whole-blood clotting time, as a screen for coagulation disorders such as the defibrination syndrome.

If a suitable laboratory is available, detailed coagulation studies should be performed, including fibrinogen levels, fibrin degradation products, and factor assays in addition to usual coagulation investigations.

Routine electrolyte studies should also be performed, including assays of serum calcium and phosphate levels. Renal and liver function should be checked, and serum enzyme studies performed, for evidence of rhabdomyolysis.

A routine blood picture should be done as well as platelet count. In cases with evidence of major envenomation or isolated defibrination syndrome, blood should be grouped and matched, or at least grouped and held.

Urine should be collected and examined by naked eye for discolouration or other evidence of haemolysis, haemoglobinuria, or myoglobinuria. The volume also should be noted, and a sample kept for future venom assay, if this should prove necessary. Samples should also be investigated for proteinuria, microscopic haematuria, haemoglobinuria and myoglobinuria.

These tasks, including history and examination, application of lymphatic bandage, insertion of IVT, and collection of blood and urine can be done virtually concurrently.

At this early stage of management, the physician must decide whether the patient has evidence of systemic envenomation or not. If there is definite evidence of *systemic envenomation* then specific treatment should be instituted. If there is *no* definite evidence of systemic envenomation then the patient should be carefully observed in a high-intensity nursing area such as an Intensive Care Unit for a minimum of 24 hours from the time of the bite. If no symptoms or signs have developed in that time then the patient may be discharged.

(b) *Antivenom*

In those cases where there is definite evidence of systemic envenomation, either symptoms and signs, or positive laboratory results, then specific treatment must be instituted without delay. The most important specific treatment is the administration of *antivenom* intravenously.[78,82,86,111,112,113] The aim of antivenom is to neutralize all circulating venom and all venom which will reach the circulation.

Snake antivenom in Australia is manufactured by the Commonwealth Serum Laboratories, Melbourne, and is produced in the standard method, using horses. Thus the antivenom is horse-serum-based, with the inherent antigenic problems.

The first snake antivenom in Australia was specific for Tiger Snake venom and was released in 1930. Antivenoms are now available to cover all major dangerous snakes in Australia (table XXII). Each vial contains enough antivenom to neutralize, *in vitro*, the average yield of venom milked from the snake species in question. Polyvalent antivenom contains equivalent quantities as for each specific antivenom.

The quantity of antivenom per vial can only be taken as an approximate guide to appropriate dosage, as the size of the snake, how many times it bit, and other factors may greatly vary the amount of venom injected. The size of the patient is irrelevant, and children should receive the same dose of antivenom as adults. For a single uncomplicated bite, one vial of antivenom is sufficient in most cases. For

multiple bites at least two vials should be used. For severe cases 10 or more vials may be needed. The correct dose of antivenom is that required to neutralize the venom, and must be titrated for each patient. If after administration of antivenom, there are indications of progressive envenomation, then more antivenom should be given. Campbell reports cases requiring 10 vials, or 10 times the normal dose of antivenom.[112] I know of one child requiring five times the normal dose, and

TABLE XXII: Antivenoms available to Australian elapid venoms, produced by C.S.L.[114]

Antivenom (AV)	*Species effective for*	*Units per vial*
Brown Snake AV	Brown Snake Western Brown Snake Dugite	1000u.
Tiger Snake AV	Tiger Snake Black Tiger Snake Copperhead Rough-scaled Snake	3000u.
Death Adder AV	Death Adder Desert Death Adder	6000u.
Taipan AV	Taipan Small-scaled Snake	12000u.
Black Snake AV	Red-bellied Black Snake Mulga Snake Collets Snake Blue-bellied Black Snake	18000u.
Sea Snake AV	Covers all species of sea snake	1000u.
Polyvalent AV	Covers all of above except sea snakes	
	Contains: Brown Snake AV	1000u.
	Tiger Snake AV	3000u.
	Death Adder AV	6000u.
	Taipan AV	12000u.
	Black Snake AV	18000u.

TABLE XXIII: Recommended *minimum* doses of antivenom (intravenous)[114]. Note that many times this dose may be needed. Child's dose the same as for adults.

Snake	*Appropriate antivenom*	*Minimum dose*
Brown Snake Western Brown Snake (Gwardar) Dugite	Brown Snake AV	1000u.
Tiger Snake Copperhead Rough-scaled Snake	Tiger Snake AV	3000u.
Black Tiger Snake	Tiger Snake AV	6000u.
Red-bellied Black Snake	Tiger Snake AV	3000u.
	OR Black Snake AV	6000u.
Mulga Snake	Black Snake AV	18000u.
Death Adder Desert Death Adder	Death Adder AV	6000u.
Taipan Small-scaled Snake	Taipan AV	12000u.

one adult who received 11 times the normal dose. Suggested minimum doses are given in table XXIII.

Antivenom must be given intravenously. Except in cases of severe or catastrophic envenomation where very rapid neutralization of venom is required, it should not be given as a rapid bolus. It should be given over 10–30 minutes, diluted in a paediatric IVT measure (100 mls), via the IVT line. Dextrose solution 5% is a suitable diluent,[86] as is normal saline or Hartmann's Solution.[114]

Before antivenom is given, resuscitation equipment should be prepared for the treatment of anaphylactic reaction should this occur. Fortunately this is a rare complication.[84,112,81] Sutherland has recommended the administration of adrenaline and antihistamine prior to the administration of antivenom.[114,115] Steroids have also been suggested, in an attempt to reduce adverse reactions to the antivenom. In cases receiving single doses of antivenom, without previous history of allergic problems, and provided that adequate resuscitation equipment is available, I have not used prophylactic adrenaline. I have not seen any cases of anaphylactic reaction to antivenom but reaction to antivenom will be discussed later.

Whenever possible specific antivenom should be used as incidence of adverse reactions is greatly increased with the use of polyvalent antivenom.[84] However, when there is real doubt about the identity of the offending snake, polyvalent antivenom should be used. A significant cause of "failed" antivenom treatment is the use of an incorrect specific antivenom.

As the offending snake rarely accompanies the victim, determination of the species involved can be difficult. Verbal descriptions of the snake are unreliable, and even expert herpetologists can misidentify a snake seen only briefly during attack. The information in Section I may help to narrow the range of possible species so that a mixture of two or three specific antivenoms may be used, rather than the full polyvalent product.

(c) *Venom identification*

However, to further assist the physician C.S.L. have recently developed an ELISA (Enzyme-linked immunosorbent assay) for detection and identification of snake venoms. These are now becoming more readily available, and can be obtained from C.S.L. State branches (for South Australia; C.S.L., 282 Gilbert Street, Adelaide, 5000).

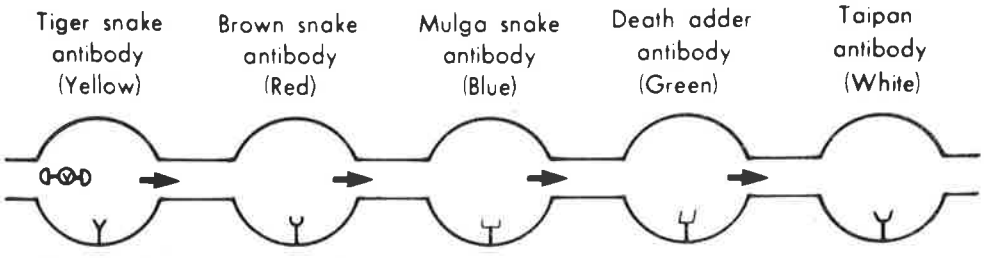
Venom can be identified best from swabs taken from the bite site,[116] but blood and urine may also be tested. Recent studies in monkeys show that a large percentage of venom may be excreted in the urine.[109] The current C.S.L. ELISA kits can detect venom concentrations of 10–25 ng/ml with standard 10-minute incubations (about 50 minutes for completion of test), and 5–10 ng/ml with 30 minute incubations (about 90 minutes for completion of test). The sensitivities to various species are shown in table XXIV.

ELISA tests were first reported in 1971,[117] and were used for quantitative assay of IgG. In 1977 they were first used for snake venom detection.[118] The first prototype C.S.L. ELISA kits for identifying Australian elapid venoms were released in 1979[119] and have subsequently proved very effective.[120] A new revised and simplified kit was released in late 1980.

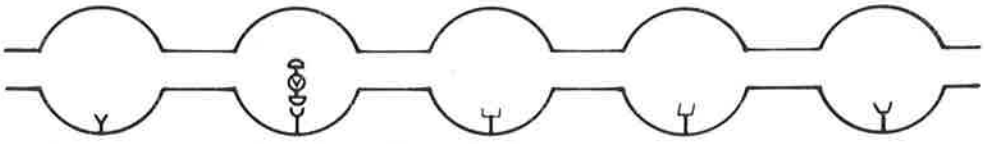
The principles of the kit are relatively simple. Each of the five colour-coded tubes has a specific antibody linked to it (see table XXIV). The sample containing venom is washed through all the tubes and the venom links with the specific antibody. Further incubation with an enzyme conjugate allows this to link to the venom.

TABLE XXIV: Specificities of C.S.L. ELISA snake venom detection kits

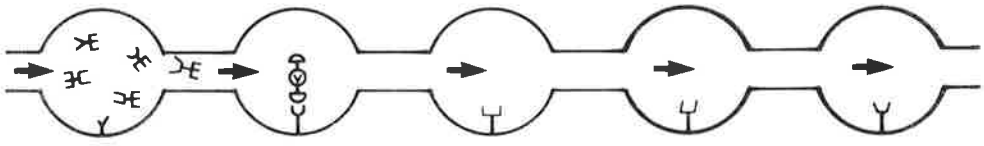
Tube (specific antibody)	*Species giving positive reaction*
1) Yellow	Tiger Snake Red-bellied Black Snake Copperhead
2) Red	Brown Snake Western Brown Snake Dugite
3) Blue	Mulga Snake Papuan Black Snake Red-bellied Black Snake Copperhead
4) Green	Death Adder
5) White	Taipan Small-scaled Snake



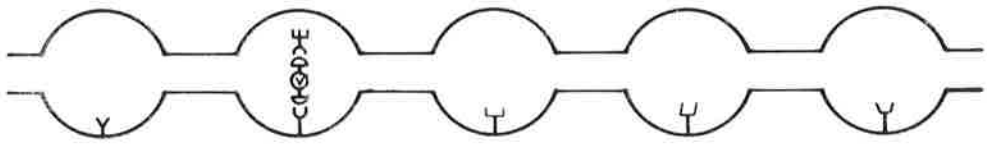
Step 1 : Add venom sample to system.



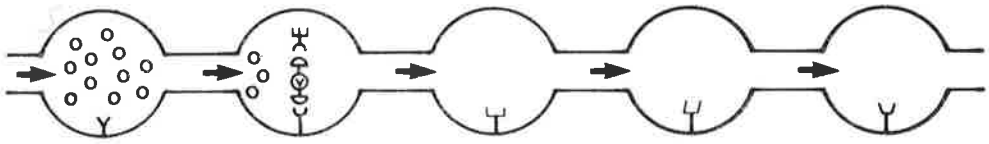
Step 2 : Venom links with specific antibody only.



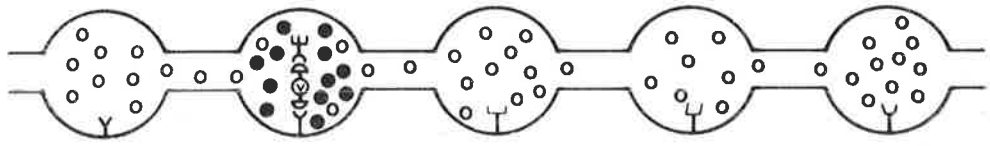
Step 3 : Incubate with enzyme linked to specific antibodies.



Step 4 : Enzyme links only to venom and is washed out of other tubes.



Step 5 : Incubate with substrate



Step 6 : Substrate is oxidised by the enzyme, and changes colour. This will happen only in the specific tube initially, thus indicating the venom involved in this case Brown snake venom.

KEY

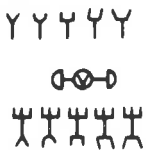


Diagram 12 - Schematic of operation of C.S.L. ELISA snake venom identification kit

Incubation with a substrate then allows the enzyme conjugate to oxidize the substrate, causing a change in colour from yellow to purple. As this will only occur in the tube with linked enzyme-conjugate-venom-antibody, it gives a direct indication of the venom group, and therefore the species involved (diagram 12).

In most cases of significant envenomation, the ELISA can be expected to give a positive result in about one hour. This will allow use of specific rather than polyvalent antivenom. It should be stressed, however, that in a serious case of envenomation, with established and progressing signs of neurotoxic envenomation, it is better to give polyvalent antivenom initially while awaiting ELISA results. Negative ELISA results in the presence of definite signs of envenomation is not an indication to withhold antivenom. Positive ELISA results on bite-site swabs is not an indication to give antivenom, if there are no definite symptoms or signs of systemic envenomation. The ELISA should be considered a valuable tool for identifying the species of snake involved in envenomation, and not as an arbiter of the administration of antivenom.

C.S.L. have also developed a sensitive radio-immunoassay for detecting and quantifying snake venoms.[121] However, it is a lengthy and expensive procedure, and is best used as a research tool. In this function it has proved very effective.[5,109]

(d) *Non-specific treatment*

Local pain at the bite site is only occasionally encountered, but generalized pain and especially abdominal pain may be a problem. Pain relief in this situation is difficult. Morphine is contraindicated.[114] I have seen one case where pethidine was used, without great relief of pain, but with development of nausea and dry-retching. Sutherland[114] suggests diazepam and paraldehyde.

In cases with pulmonary oedema, standard treatment is advocated, using diuretics such as frusemide. Because of the potential for vomiting and aspiration pneumonia, oral fluid should be withheld, and fluid requirements provided via the I.V.T. line. Unconscious, semiconscious or semi-paralyzed patients, should be nursed on their side and secretions sucked out, as necessary.

As snakebite wounds are penetrating, the usual precautions about tetanus should be followed.

The need for meticulous observation should be impressed on all attending staff, and documentation should be complete. As discussed earlier, neurotoxic envenomation may take several hours to develop, and initial signs may be easily missed. A standard 15-minute neurosurgical observation routine is usually known and understood by nursing staff, and should pick up early signs of neurotoxic envenomation. However, the physician should ensure that attending nursing staff are alert to subtle changes such as changes in voice, articulation, and minimal ocular problems such as early ophthalmoplegia with minimal diplopia on lateral gaze only. Because of these frequent and detailed observations, the Intensive Care Unit is the ward of choice for observation and management of envenomation, at least in the first 24 hours. Resuscitation facilities and the expertise to use them will be most readily available in such a place.

Fluid input and output should be carefully monitored, and the patient's weight should be recorded on admission to the Intensive Care Unit. Apart from the initial extensive laboratory investigations, consideration should be given to further repeat tests at regular intervals. In particular, routine coagulation tests (e.g. whole-blood clotting time, P.T., A.P.T.T.) should be repeated even if initially normal. A patient who has had correct first-aid applied promptly may not develop the defibrination syndrome as rapidly as in the untreated case. It is therefore conceivable that initial coagulation studies may be normal, but subsequent studies may show hypocoagulability, as an early sign of venom entry to the systemic circulation, before other signs are apparent. I would recommend repeating the coagulation studies one hour after removal of the lymphatic bandage, even in the absence of symptoms or signs of envenomation.

4. TREATMENT OF SPECIFIC ENVENOMATION PROBLEMS

A number of distinct clinical problems of envenomation were discussed in Section III. Some of these are worthy of separate discussion of the treatment problems they involve.

(a) Neuromuscular paralysis

As discussed earlier, neuromuscular paralysis due to potent neurotoxins is a prominent feature of Australian elapid envenomation. Symptoms and signs have already been discussed (Section III). The only specific treatment for neuromuscular paralysis is appropriate antivenom, which should be given as soon as there is evidence of systemic envenomation. If there is a continuing progression of envenomation after antivenom, then the physician must decide if the correct antivenom, and enough of it, has been given. If an ELISA is available then determination of the correct antivenom is possible. If it is not available, and specific antivenom has been given without the expected improvement, then the antivenom should be reassessed. Firstly, the antivenom should be checked for viability. The average shelf life for C.S.L. antivenoms is three years, if kept refrigerated. However, in the majority of cases, failure of a specific antivenom will be because it is the wrong antivenom. Therefore, in this situation, polyvalent antivenom should be used.

In severe cases of envenomation, as discussed earlier, huge doses of antivenom may be required. In a case of established neurotoxic paralysis, several times the minimum dose of antivenom should be used.

However, it has been shown, both clinically and experimentally, that for some species of Australian elapid snakes, established neuromuscular paralysis will not be reversed by antivenom. This appears to be linked to pre-synaptic neurotoxin activity. Thus Campbell[80,81,112] found that neuromuscular paralysis following Death Adder envenomation was reversed by antivenom, but similar paralysis following Taipan envenomation was not reversed by antivenom. As noted in Section II, Taipain venom has a pre-synaptic neurotoxin, and Death Adder venom does not (or rather, none has been detected at this stage).

In a case of severe neuromuscular paralysis, with ventilatory embarrassment, antivenom should always be tried, in high doses. However, at least in cases of envenomation by the Taipan, Small-scaled Snake, Tiger Snake and Black Tiger Snake, Brown Snake, Western Brown Snake, Dugite, and Rough-scaled Snake, all of whose venoms definitely or probably contain a pre-synaptic neurotoxin, the physician should be prepared for little amelioration of the paralysis.

In this situation, ventilation must be maintained by artificial means, and tracheostomy is occasionally needed. In cases with concomitant defibrination syndrome, persistent oozing from the tracheostomy wound may be a problem. The best treatment for this is correction of blood hypocoagulability (discussed later).

The duration of neuromuscular paralysis and the sequence of recovery have already been discussed in Section III.

(b) Defibrination syndrome

The problems and laboratory findings in defibrination syndrome have been discussed in Section III. The primary treatment of defibrination syndrome is complete neutralization of all circulating venom with specific antivenom. This may require high doses of antivenom. After the circulating venom is neutralized, homeostatic mechanisms usually restore adequate clotting function in 1–3 hours, although complete return to normal may take several days.

The reader is referred back to tables XVIII and XIX (Section III) where progressive results from two children with defibrination syndrome are given. In the first case (table XVIII) with gross defibrination syndrome, initial antivenom did not prevent progression of defibrination. However, within two hours of further antivenom, the blood was again clottable. In less than 48 hours all parameters measured were virtually normal. In the second case (table XIX), defibrination was still evident four hours after antivenom was given, but the coagulation was near normal at six hours post-antivenom. It could be argued that this case received

insufficient antivenom. In both cases, fibrinogen levels took more than 40 hours to return to normal.

Champness,[93] reporting Papuan experience with defibrination syndrome following envenomation, also noted that fibrinogen levels took 24–48 hours to return to normal after antivenom administration. He concluded that administration of fibrinogen was helpful. However, Reid[122] found fibrinogen unhelpful in defibrination following Malayan Pit Viper envenomation. Hermann *et al*[96] successfully treated defibrination syndrome following Dugite envenomation with antivenom only.

Both Sutherland[114,82] and the *Medical Journal of Australia*[123] have suggested that antivenom is the treatment of choice for defibrination syndrome following envenomation, but that after all venom has been neutralized, administration of fibrinogen and possibly other factor concentrates may be helpful in severe cases.

A dose of 10 g of fibrinogen in adults, and 0.1 to 0.2 g per kilogram in children is recommended.[123] It should be emphasized that use of fibrinogen be restricted to a small minority of cases of defibrination syndrome with severe envenomation and persistent hypo-coagulability, despite neutralization of all venom. Antivenom remains the primary and most important treatment of defibrination syndrome.

Rarely, a true disseminated intravascular coagulation (D.I.C.) may be present. In this event there will be a thrombocytopenia, which is not seen in defibrination syndrome. If a D.I.C. is present, then in addition to antivenom, fresh platelets and fresh blood may be required. Heparin may be useful in this situation.[123]

(c) Rhabdomyolysis

The only documented treatment for rhabdomyolysis is the rapid neutralization of circulating venom with sufficient appropriate antivenom. In the recovery phase a high-protein diet would seem advisable.

(d) Renal failure

It is beyond the scope of this article to detail the management of renal failure. In those cases of renal failure following envenomation haemodialysis and peritoneal dialysis were required, as discussed in Section III. Hyperkalaemia, hyperphosphataemia, and hypocalcaemia were also encountered, requiring specific treatment. The majority of cases responded to treatment, with complete recovery, over a period of weeks or months.

(e) Local Tissue Damage

As discussed earlier, this is a rare problem in Australian envenomation. The avoidance of tourniquets should reduce the chance of ischaemic damage on top of venom damage. In both the patients with local tissue necrosis whom I have seen, an arterial tourniquet was used by the victim.

Where there is gross oedema of a digit, hand, foot, or limb, in association with the bite wound, and this oedema is sufficient to potentially impair circulation, then an escharotomy should be considered, as for gross oedema following burns. As an extreme measure in extreme circumstances where it may prevent necrosis it would seem worthwhile. However, I have not managed a case requiring this treatment. Most cases of oedema should respond to elevation of the limb.

Superimposed infection should always be considered in these cases and vigorously treated.

5. ADVERSE REACTIONS TO ANTIVENOMS

There is a widely-held belief, especially amongst some amateur herpetologists and reptile keepers, that antivenoms are more dangerous than venoms. This belief is groundless. There is no doubt that antivenoms are almost solely responsible for the decrease in mortality following snakebite in Australia and there are no reported deaths ascribed to antivenom in Australia.

However, considering the horse serum base of antivenoms, it is not surprising that their use is associated with significant complications. The physician using antivenom should be aware of these problems, and should prepare for them. Antivenom should not be used indiscriminately because of potential hazards, nor should it be used by untrained people. However, for all cases of systemic envenomation, antivenom is the treatment of choice.

Sutherland[124] has investigated the anti-complementary activity of antivenoms, which

is high in all tested samples, including C.S.L. snake antivenoms. There is therefore a potential for these antivenoms to cause untoward reactions which would be unpredictable. These reactions, being unrelated to previous exposure to equine protein, would not be predicted by prior skin sensitivity testing, or subcutaneous test doses. This is one reason why the use of subcutaneous test doses before administration is now considered undesirable. Campbell[81,112] has reported complications following use of antivenom in Papua New Guinea. In a series of 61 patients, 28 (46%) had some form of adverse reaction. Only two (3%) developed anaphylactic shock. The complications are summarized in table XXV.

More recently, Sutherland and Lovering[84] have analysed 181 cases of snakebite. Their findings are summarized in table XXVI. Polyvalent antivenom had the highest incidence of complications, at 20%, or 79% of all recorded complications from snake antivenom. Overall incidence of complications for all antivenoms was 13%, which is much less than in Campbell's series. Immediate reactions to antivenoms included sudden hypotension, rash and bronchospasm, urticaria, collapse, hyperthermia, sweating, headache, colicky abdominal pain and vomiting. Delayed reactions included urticaria, arthralgia and polylymphadenopathy. Immediate reactions only occurred when the combination of premedication and slow infusion of diluted antivenom was not used.

The high incidence of complications following polyvalent antivenom administration further strengthens the case for specific antivenoms or specific antivenom combinations to be used whenever possible.

Serum sickness may occur as a delayed reaction to antivenom administration. It usually occurs 4–10 days after inoculation with foreign serum, though it may occur sooner, or be delayed as much as three weeks. It is characterized by skin eruptions, lymphadenopathy, and arthralgia.

Usually, a pruritis develops first followed by a rash, usually an urticaria. Later lymphadenopathy develops, and there may be oedema of the face, or rarely glottis.

TABLE XXV: Complications of antivenom therapy in Papua New Guinea[81]

Complication	*Number with complication (n=61)*	*Percentage*
– Reaction involving skin – urticaria, itching or oedema	15	25%
– Febrile reaction alone – rigor, shivering, fever	8	13%
– General reaction	3	5%
– Anaphylaxis	2	3%
TOTAL	28	46%

TABLE XXVI: Adverse Reactions to snake antivenoms[84] (n = 181)

Antivenom type	*Immediate reaction*		*Delayed reaction*		*Overall total*
	Severe	*Mild*	*Severe*	*Mild*	
Tiger Snake AV (n = 16)	1	1	–	–	3 (13%)
Brown Snake AV (n = 20)	–	1	–	–	1 (5%)
Tiger Snake AV (n = 36) + Brown Snake AV	2	–	1	–	2 (6%)
Black Snake AV (n = 12)	–	–	–	–	– –
Polyvalent AV (n = 97)	4	10	5	4	19 (20%)
Total – Specific AVs (n = 84)	3 (3%)	2 (2%)	1 (1%)	0	– –
Polyvalent AV (n = 97)	4 (4%)	10 (10%)	5 (5%)	4 (4%)	– –
TOTAL (n = 181)	7 (4%)	12 (7%)	6 (3%)	4 (2%)	24 (13%)

There is usually a fever often accompanied by an arthralgia or even arthritis, with effusion into some joints. Other symptoms include headache, nausea, vomiting, abdominal pain, diarrhoea, cardiac arrhythmias, and pericarditis. Occasionally there may be neurological disorders such as a unilateral mononeuritis, with weakness and sensory deficit in the area affected. The blood picture may show a mild neutrophilic leucocytosis.

Serum sickness is usually a benign self-limiting disease, and subsides within one–three weeks. Where neuropathy is present, a longer recovery period may occur.

There is one report of dementia following anaphylactic reaction to administration of Tiger Snake antivenom.[90] The victim, a 33-year-old man, collapsed five minutes after the antivenom was given. He remained deeply shocked and pulseless at the wrist for about 30 minutes. After recovery he was completely aphasic and incontinent of urine and faeces. There was some recovery over the following weeks.

6. IMMUNIZATION AGAINST SNAKE VENOM

Mass immunization against snake venom has been used overseas, with some success.[125] However, there is only one report of immunization against Australian elapid venom, that of Wiener in 1961.[126] He actively immunized a 47-year-old man with Tiger Snake venom, because the patient, a snake handler who had been bitten many times, had developed reactions to antivenom on two previous occasions. He injected him 20 times in 13 months, with an initial dose of 0.002 mg of venom, and a final dose of 25 mg. The maximum level of circulating antivenom recorded was 5.2 units/ml, and at four months the level was 2 units/ml. The patient was subsequently bitten by a Tiger Snake and although he developed local oedema, no systemic problems ensued. As there was a subsequent rise in antibody titre, Wiener felt that there had been effective envenomation, and that the immunization was successful.

While this case is encouraging, it should be remembered that the venom is now very expensive and such a course of treatment would be costly indeed. The low incidence of snakebite in Australia makes the mass immunization against snakebite unnecessary, and totally unjustifiable on a cost-benefit basis.

7. AUTOPSY AFTER FATAL SNAKEBITE

Hopefully very few cases of snakebite in Australia will come to autopsy. However, there is a paucity of autopsy material published on fatal cases of snakebite. Any physician involved in such a fatal case should ensure that a full autopsy is performed, and the report published.

In addition, there are occasional cases where death occurs from an unknown cause, and snakebite should be included as a possible diagnosis in some of these cases. Sutherland *et al.* have reported two such cases, where radioimmunoassay proved the diagnosis of snakebite.[85,127]

At autopsy, samples of blood and urine should be secured for positive venom identification and assay. Swabs from the bite site should also be taken for the same purpose, and the vitreous humour of the eye may also be useful in this regard, especially if the autopsy is delayed.

Samples of tissue from the bite site should be taken, after prior photography of the site. Local and regional lymph nodes should also be taken. All may be assayed for venom content. A complete autopsy should be performed in the usual fashion, and particular attention should be paid to the kidneys, heart, brain, and lungs. Evidence of haemorrhage should be sought, especially in the brain, gut, and kidneys. Muscle biopsies should be taken, and examined for evidence of rhabdomyolysis.

ACKNOWLEDGEMENTS

The production of this paper has been a major undertaking, and has drawn on over twelve years' experience as a herpetologist in South Australia, and nearly eight years' experience in treating snakebite in this State. Many people have helped me during this time, and it would be impractical to personally thank them all. However, I would like to thank all my fellow doctors who have sought my advice about snakebite during those years.

I was encouraged to write this paper by several people in the Adelaide Children's Hospital, and to those people, Neil Smyth, Richard Cockington, and Brian Fothering-

ham, I am very grateful. I owe a special debt to Eric Sims, who as editor of this journal, and a fellow naturalist, has given me such magnificent support and advice on producing the paper. Donald Simpson, as deputy editor, has also been most helpful.

The Adelaide Children's Hospital Haematology Department has assisted with laboratory investigations on several snakebite victims, and their Coagulation Laboratory kindly provided the details of studies on two cases of Defibrination Syndrome listed in Section III. The A.C.H. Microbiology Department kindly assisted with the ELISA assays.

Terry Schwanner, Curator of Reptiles at the South Australian Museum, has given much helpful advice and information on the paper, and especially taxonomy, and has greatly helped me with preparation of some of the illustrations. Several other members of the Museum staff have also assisted in various ways; Jenny Thurmer, Adrienne Edwards, Beryl Morris. Terry Schwanner, and Adrienne Edwards have kindly made their distribution maps available to me, and these are the basis of the maps in the text.

Ron Southcott, Ivan de la Lande, and Struan Sutherland have provided stimulus to my interest in envenomation, and have provided valuable advice and perspective.

Chris Sprod and Colleen Lloyd of the A.C.H. Photography Department have helped prepare my illustrations, and so increased the professionalism of the presentation.

Lastly, Bernadette Madigan has typed the long and difficult manuscript with consummate ability, and I am most grateful to her.

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APPENDIX I

PROTOCOL FOR TREATMENT OF SNAKEBITE

(Guideline only, refer to main text for details)

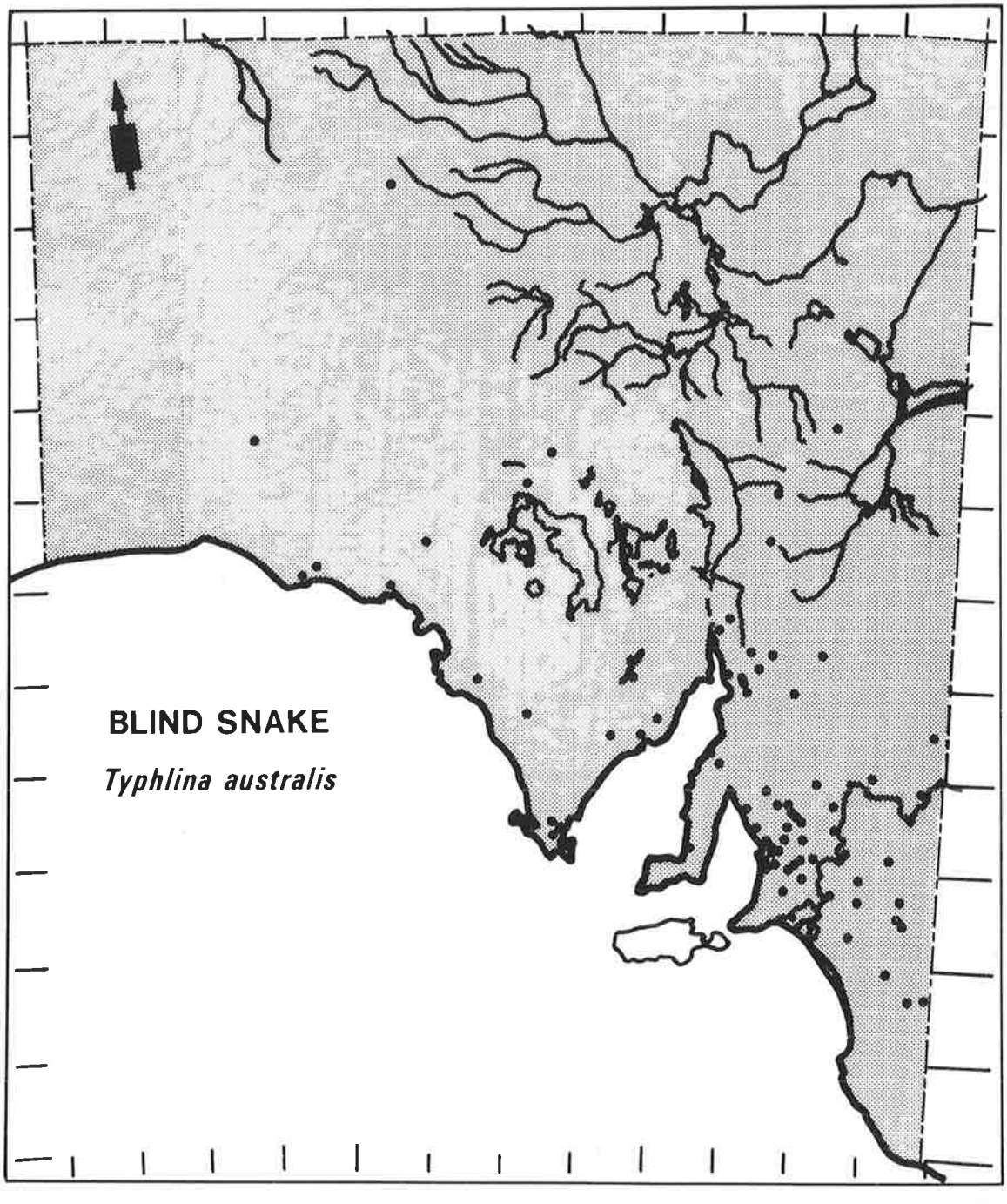
1. *First Aid*
   (a) Apply a lymphatic occlusive bandage over the bitten area and as much of the rest of the limb as possible, at the same pressure as for a sprained ankle.
   (b) Immobilize the limb.
   (c) Whenever possible bring transport to the victim.
   (d) Transport to nearest hospital.
2. *Hospital*
   (a) In treating snakebite remember that –
      (i) Adequate specific Antivenom intravenously is *the* treatment of choice in any case of snakebite with systemic symptoms.
      (ii) Majority of people bitten will not develop systemic envenomation, and so will not require Antivenom.
   (b) Simple step outline of treatment plan for various presentations of snakebite *de novo* to hospital.
      A – Patient has symptoms or signs of envenomation.
      ..... go to *B*.
      – Patient has no symptoms or signs of envenomation.
      ..... go to *C*.
      B – Symptoms and/or signs of systemic envenomation present.
      – If no first aid applied and less than six hours after bite, apply first aid.
      – Insert intravenous line.
      – Collect blood for – bleeding and clotting studies
      – electrolytes, renal function
      – serum enzymes, especially CPK
      – freeze portion for later venom analysis if this should prove necessary.
      – Collect urine for – venom assay
      – myoglobinuria
      – haemoglobinuria.
      – Give intravenous Antivenom, slowly over 15–20 minutes, diluted in 5% dextrose or similar, with everything ready to treat anaphylactic reaction should this occur. If any history of allergy, reactions to horse serum, or asthma, give adrenaline, antihistamine, and corticosteroids prior to Antivenom. Use specific Antivenom where possible, or mixture of specific Antivenoms, in preference to polyvalent Antivenom. If available, use ELISA kit to determine appropriate specific Antivenom. In severe cases use several times the normal dose of Antivenom. In multiple bites use at least twice the normal dose of Antivenom.
      – Observe in high intensity nursing area (e.g. I.C.U.), and monitor for progression of signs, development of complications, and treat appropriately (see main text).
      – Specifically watch for –
         (i) Progressive neuromuscular paralysis
         (ii) Rhabdomyolysis
         (iii) Defibrination Syndrome
         (iv) Renal Failure.
      C – Less than six hours after bite
      ..... go to *D*.
      – More than six hours after bite
      ..... go to *G*.
      D – Definite snakebite
      ..... go to *E*.
      – Doubtful snakebite
      ..... go to *F*.
      E – Apply first aid if not already applied and observe for at least 24 hours, test blood and urine (see *B*) and insert intravenous line.
      – If signs or symptoms, or laboratory tests show systemic envenomation developing then treat as in *B*.
      – If no signs or symptoms or positive laboratory tests of systemic envenomation then discharge after minimum 24 hours.
      F – Observe for a minimum 24 hours, and test blood coagulation.
      – If symptoms or signs of systemic envenomation develop, treat as in *B*.
      – If no symptoms or signs of systemic envenomation, after 24 hours, discharge.
      G – If a definite snakebite
      ..... go to *H*.
      – If a doubtful snakebite
      ..... go to *I*.

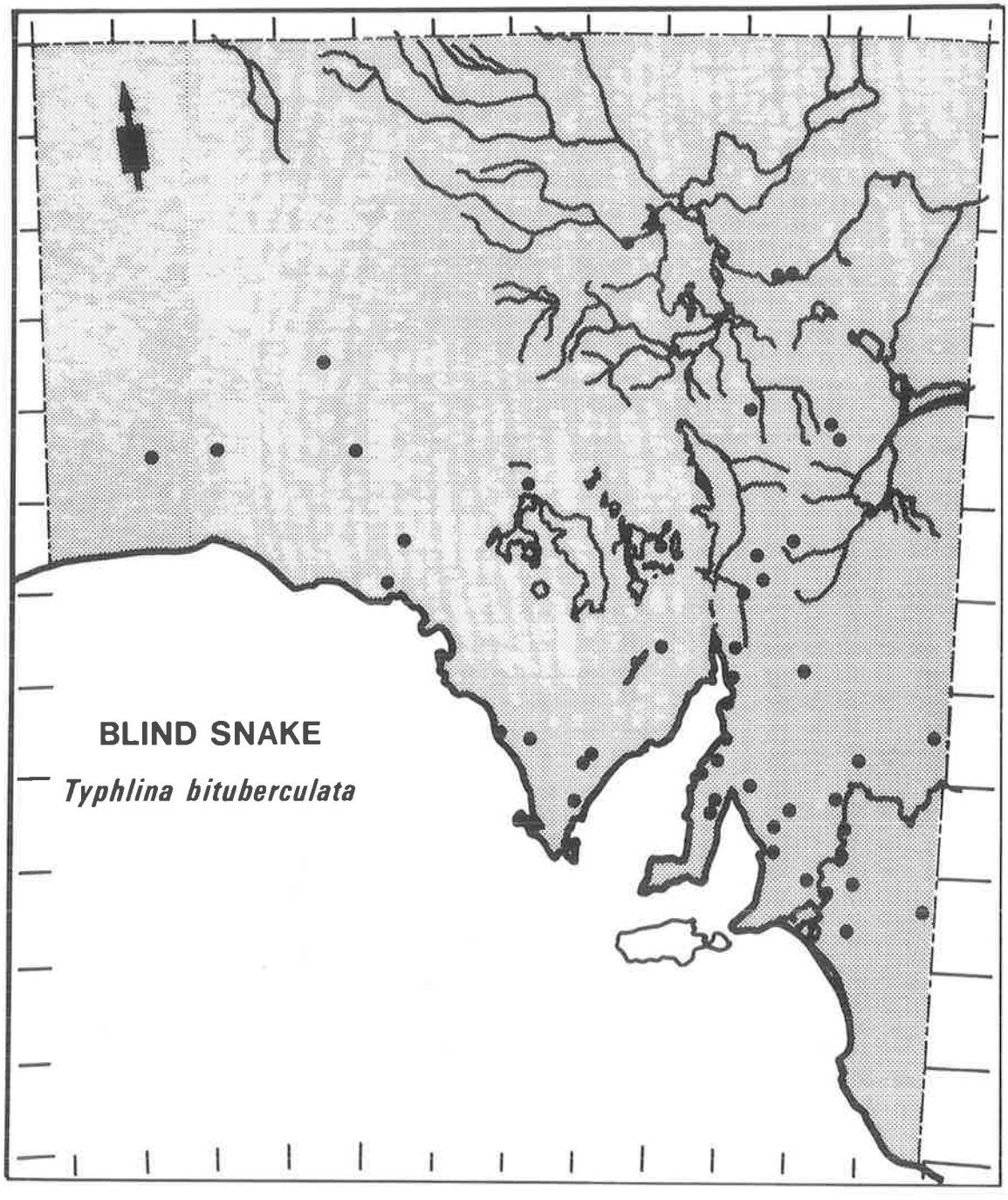
H – If first aid applied, then remove cautiously after insertion of intravenous line. Test for coagulation disturbance. If systemic envenomation develops treat as in *B*. If no systemic envenomation develops discharge after 24 hours.
– If no first aid applied, observe for 24 hours. If systemic envenomation develops treat as for *B*. If no systemic envenomation, discharge after 24 hours.

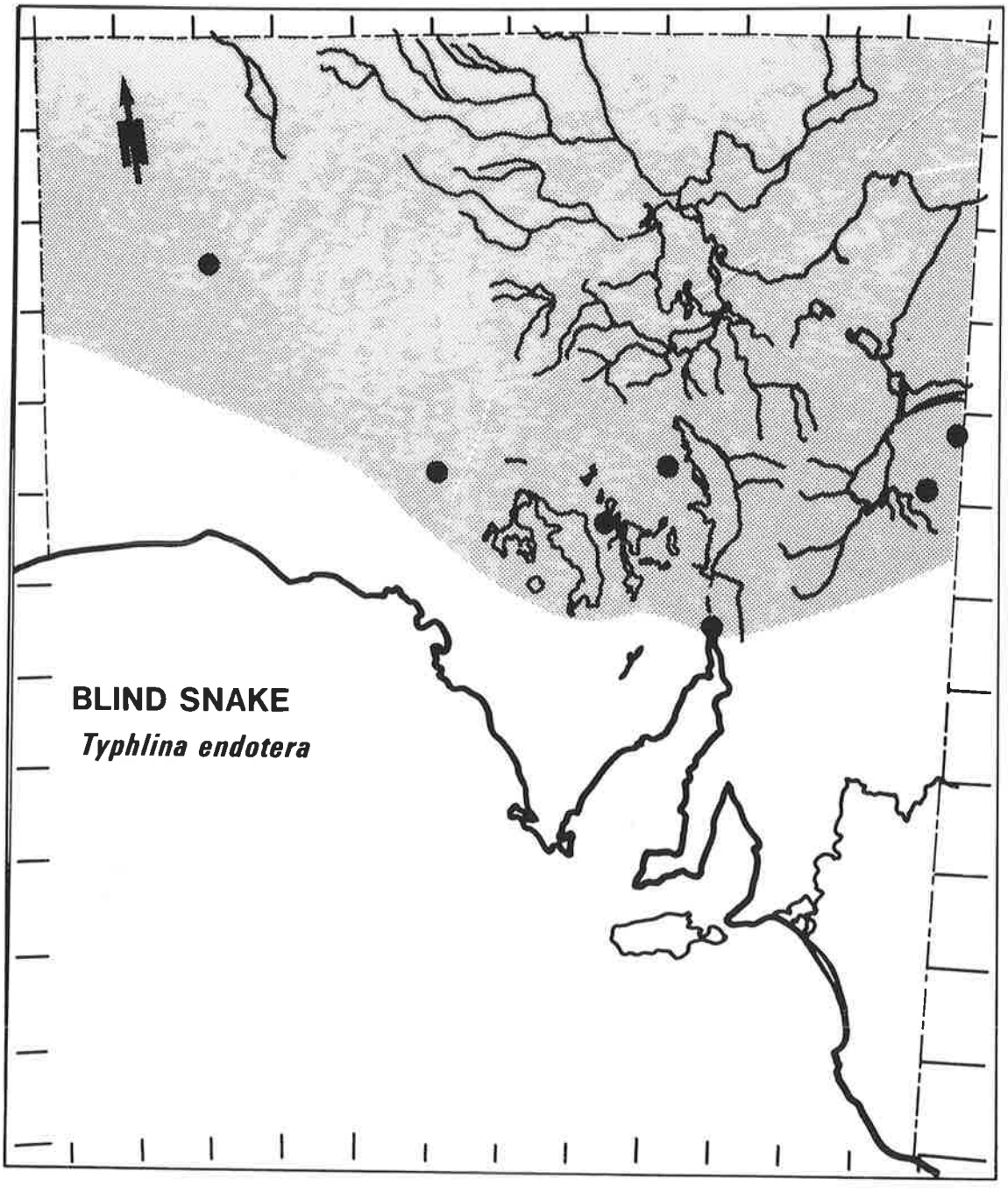
I – Observe for 24 hours, test blood coagulation.
– If systemic envenomation develops treat as for *B*.
– If no systemic envenomation, discharge after 24 hours.

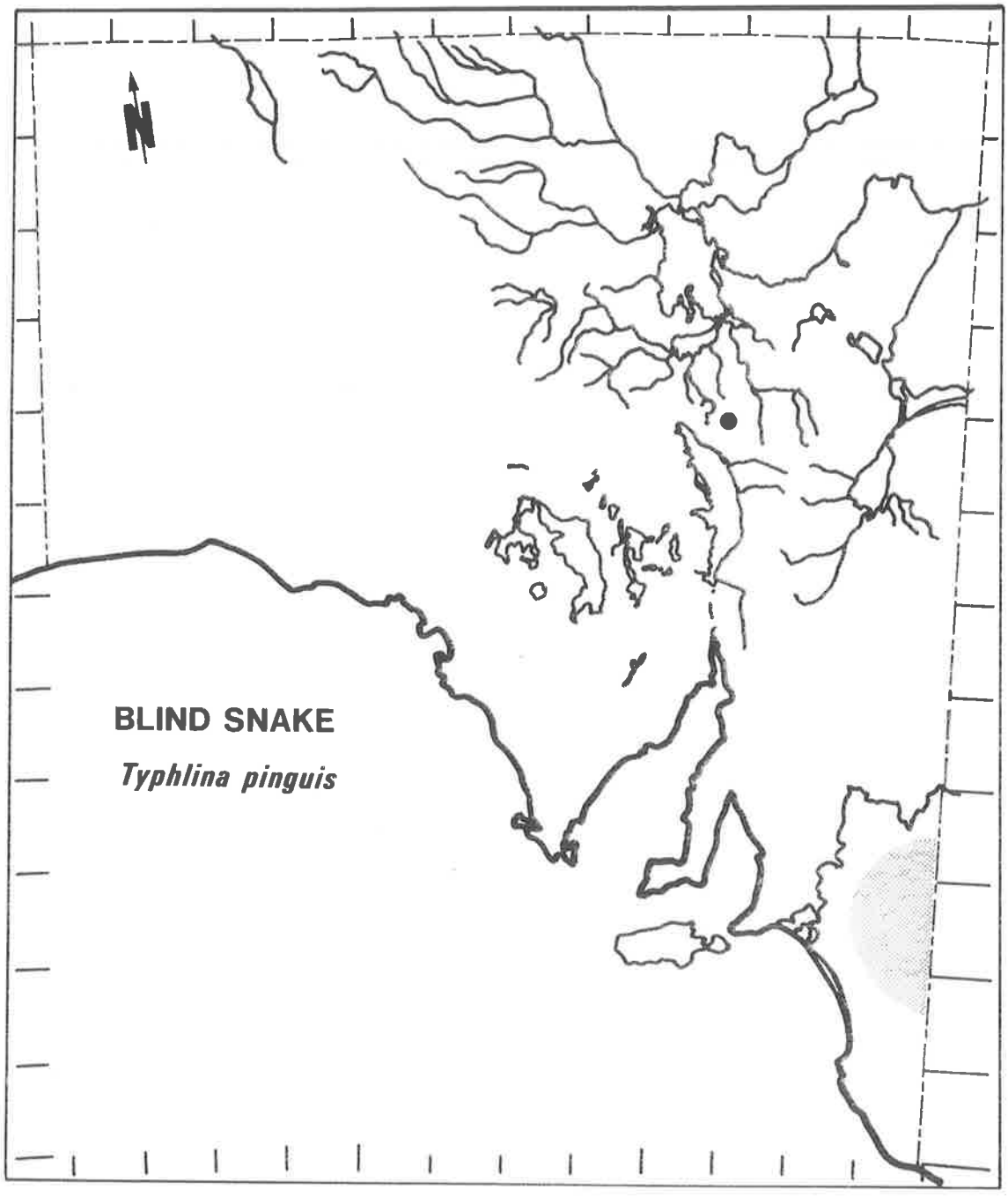
APPENDIX II

These distribution maps have been prepared from personal experiences and records of specimens at the South Australian Museum. They represent a guide to distribution but it is possible that some species have a wider range than shown. They are arranged in the order that the species are described in the text.




BLIND SNAKE
Typhlina bituberculata


BLIND SNAKE
Typhlina endotera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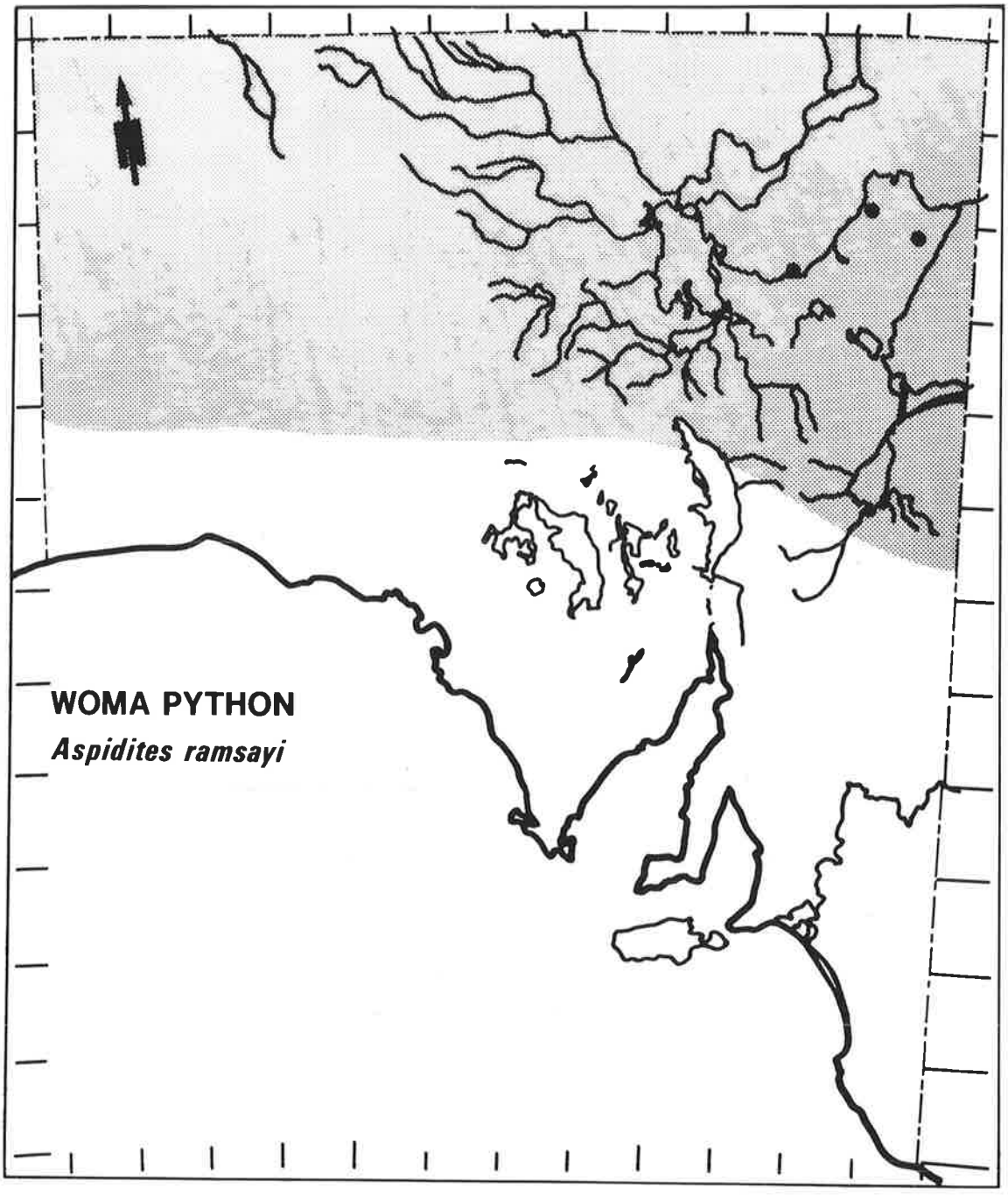
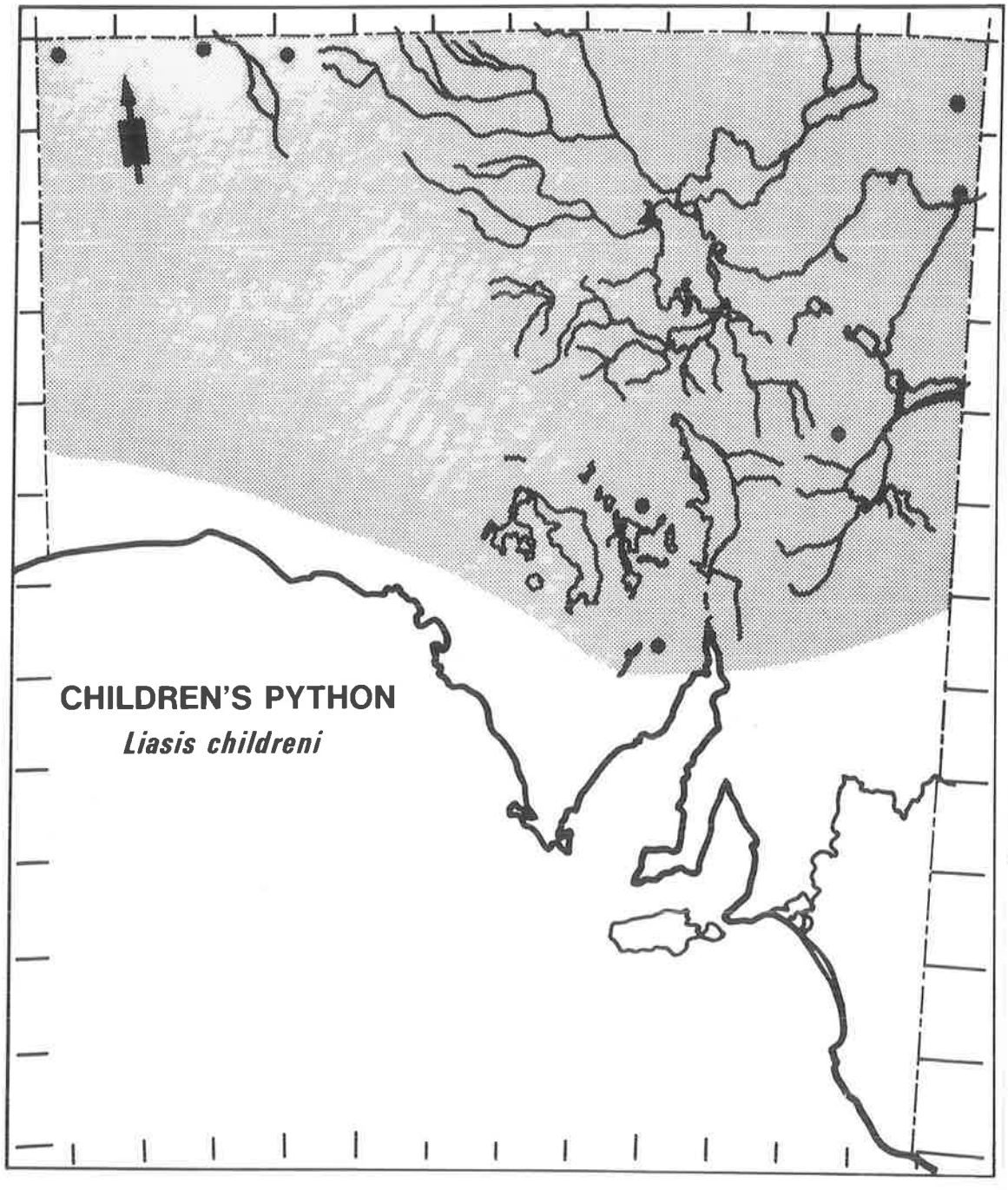
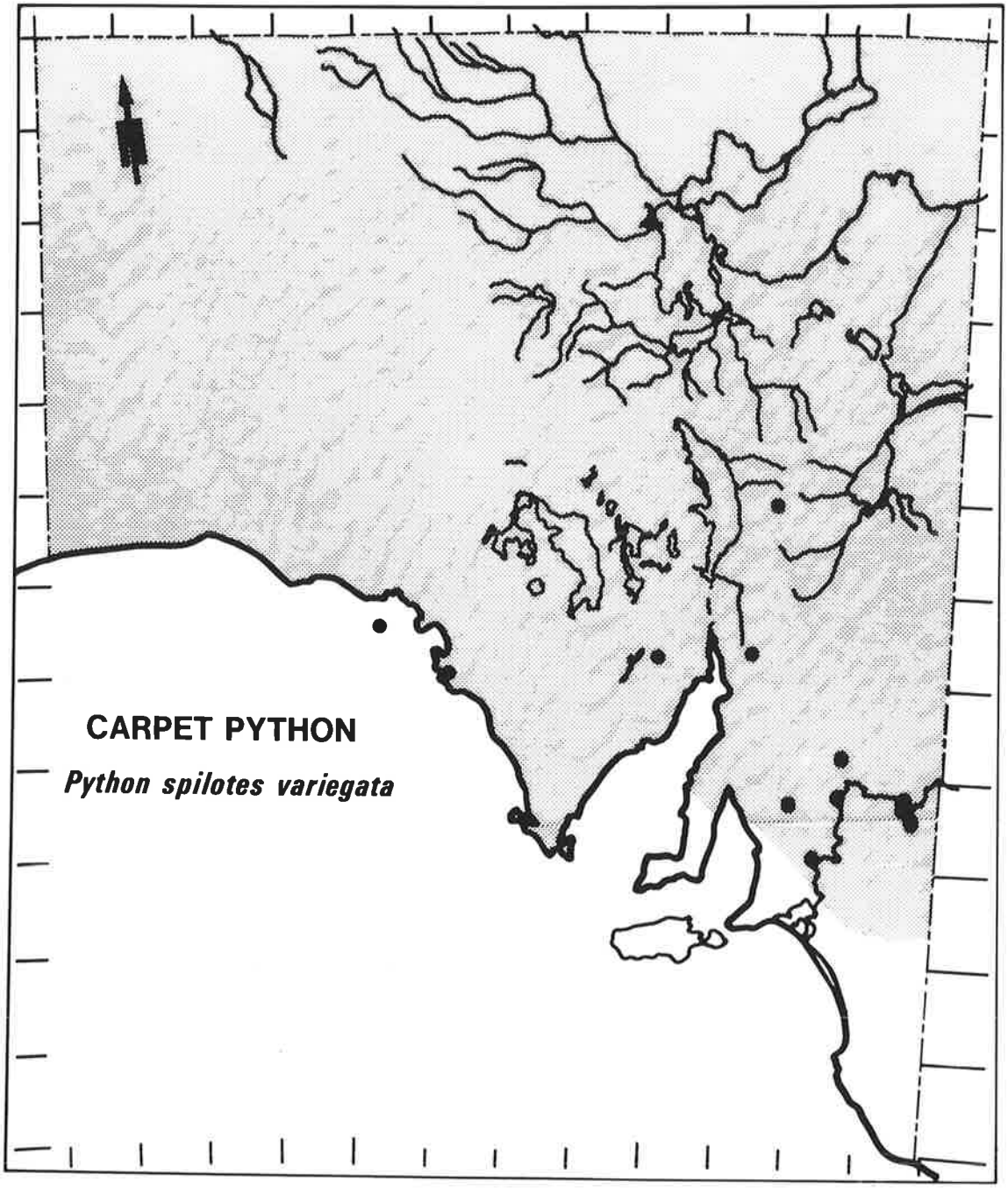
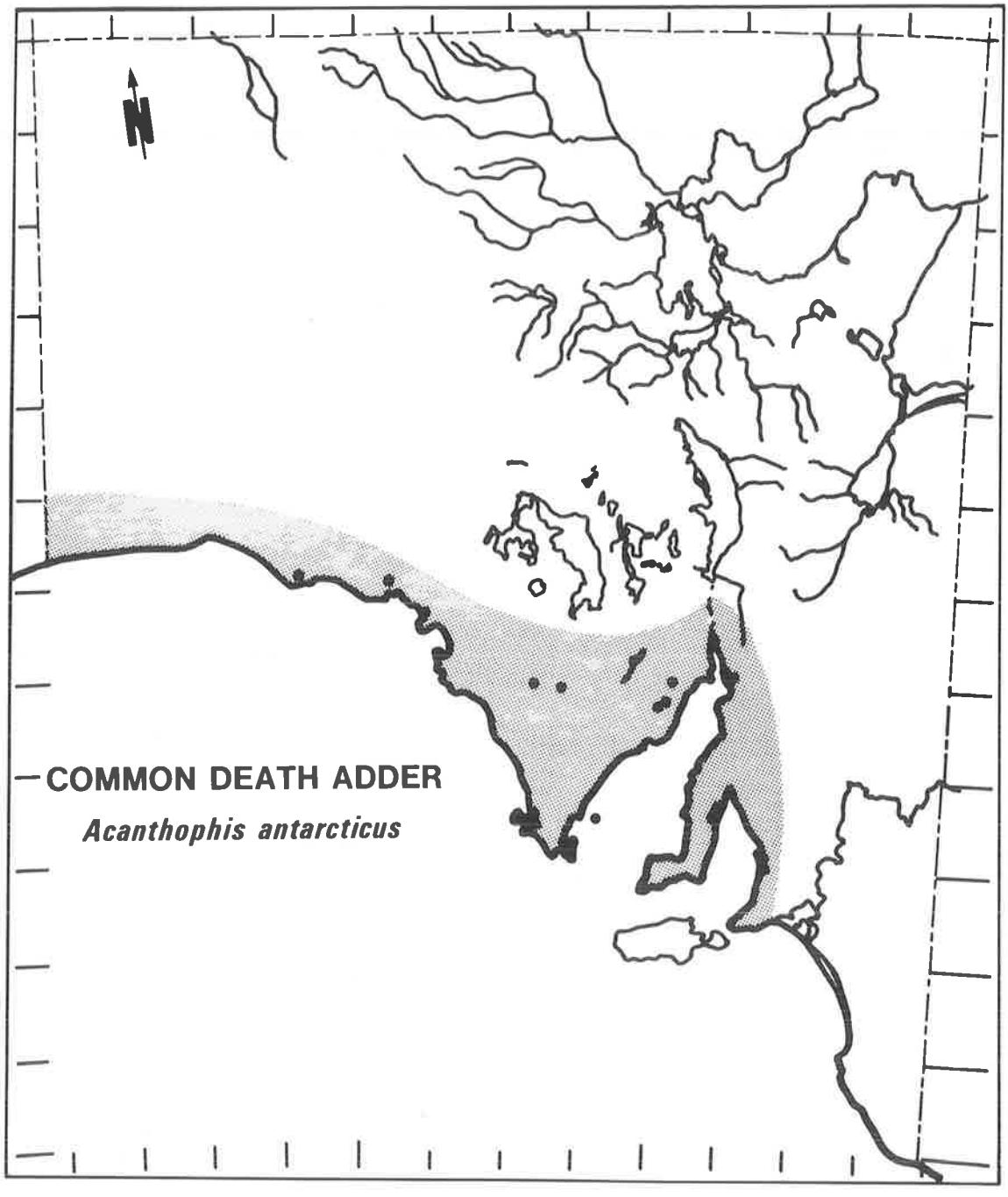
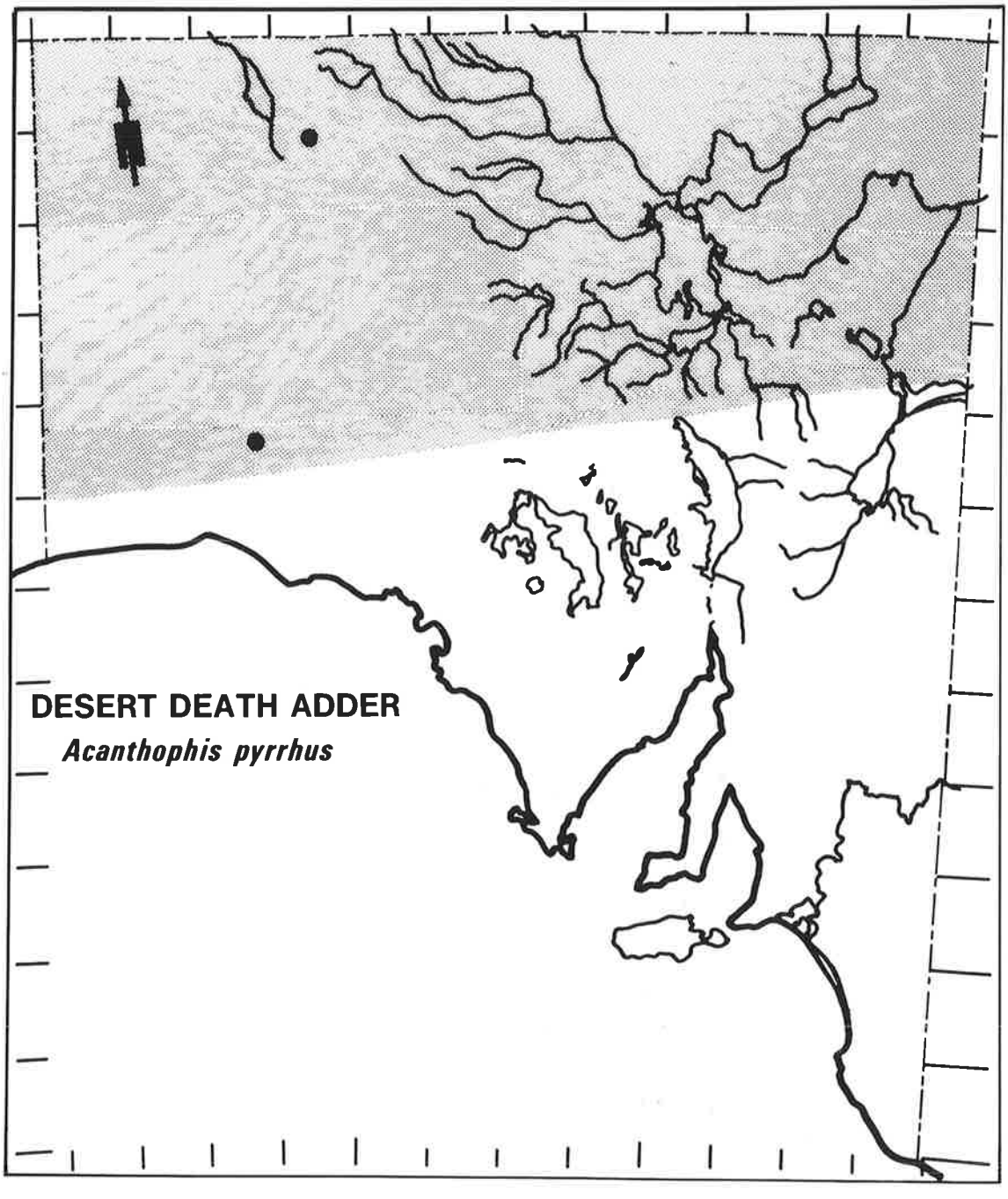
BLIND SNAKE
Typhlina pinguis

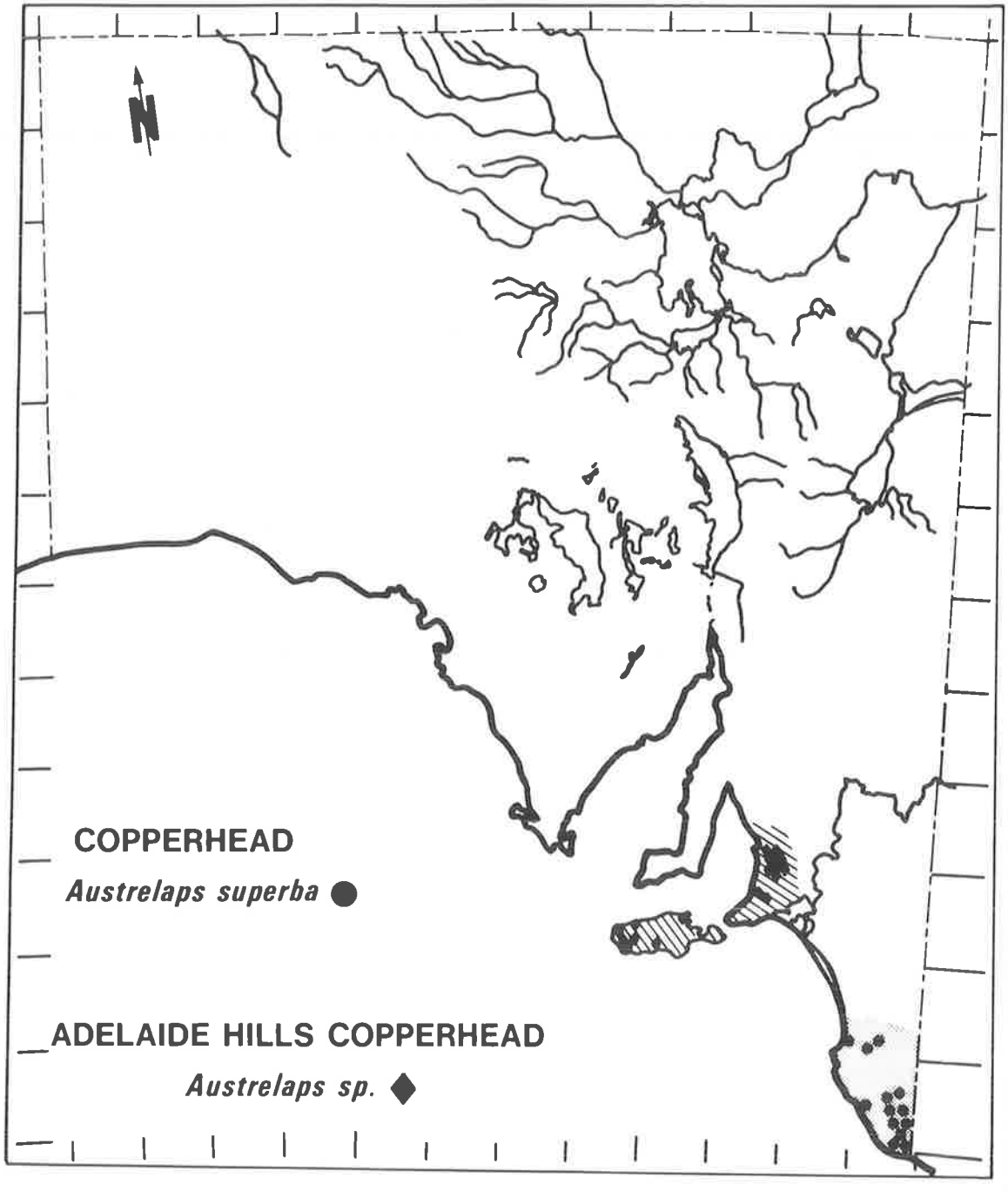

WOMA PYTHON
Aspidites ramsayi

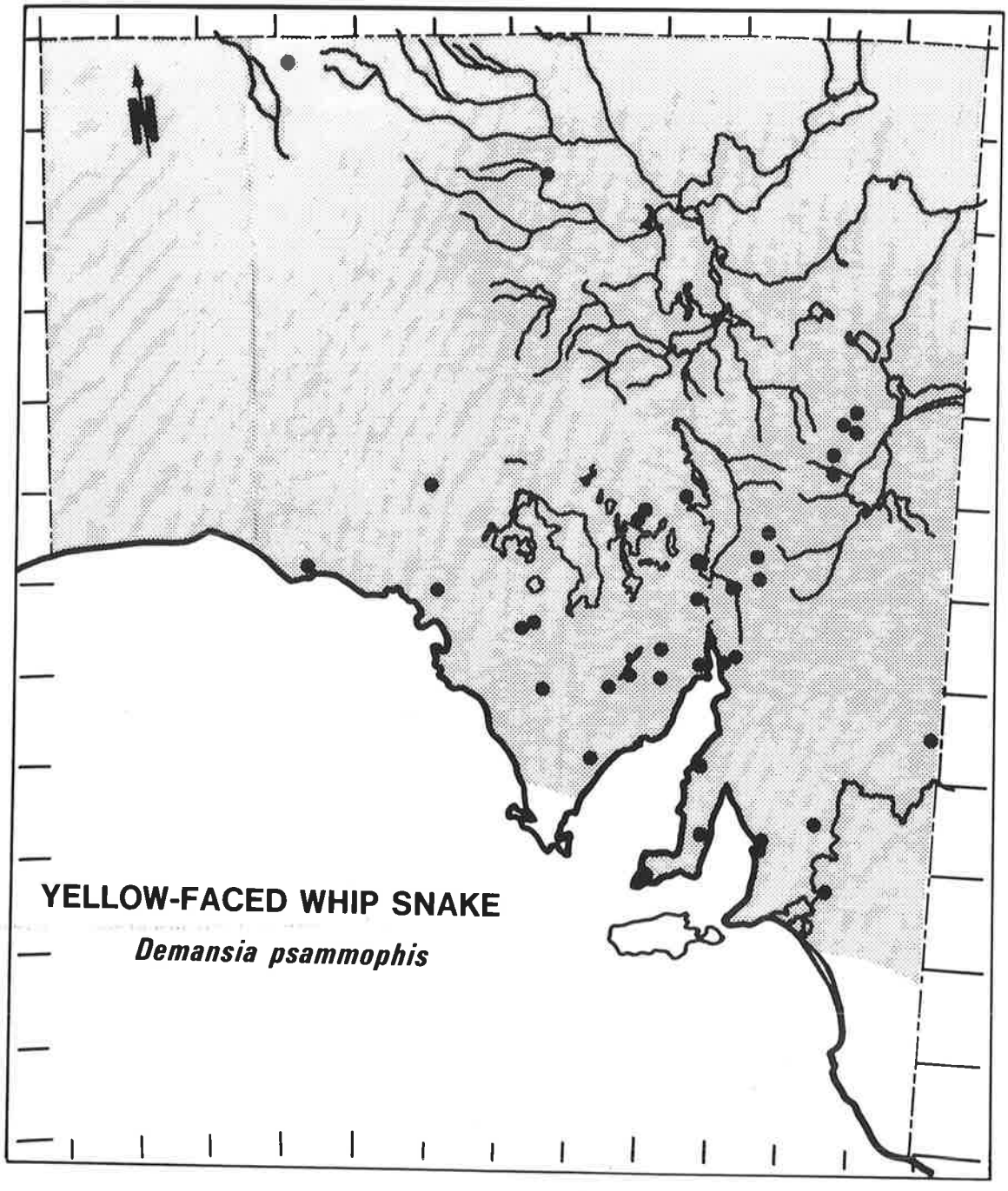

CHILDREN'S PYTHON
Liasis childreni

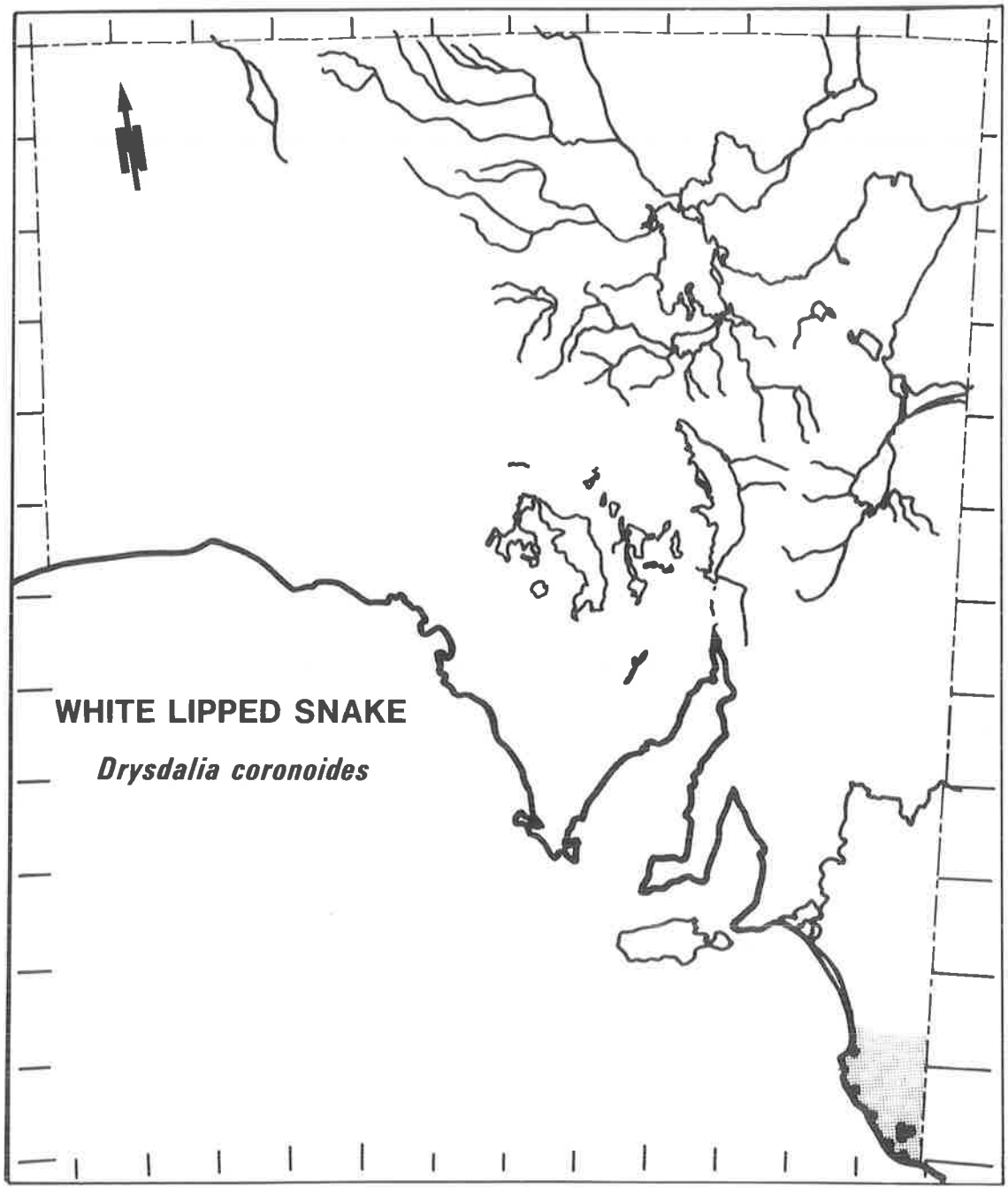

CARPET PYTHON
Python spilotes variegata


N
COMMON DEATH ADDER
Acanthophis antarcticus


DESERT DEATH ADDER
Acanthophis pyrrhus


N
COPPERHEAD
Austrelaps superba
ADELAIDE HILLS COPPERHEAD
Austrelaps sp.


YELLOW-FACED WHIP SNAKE
Demansia psammophis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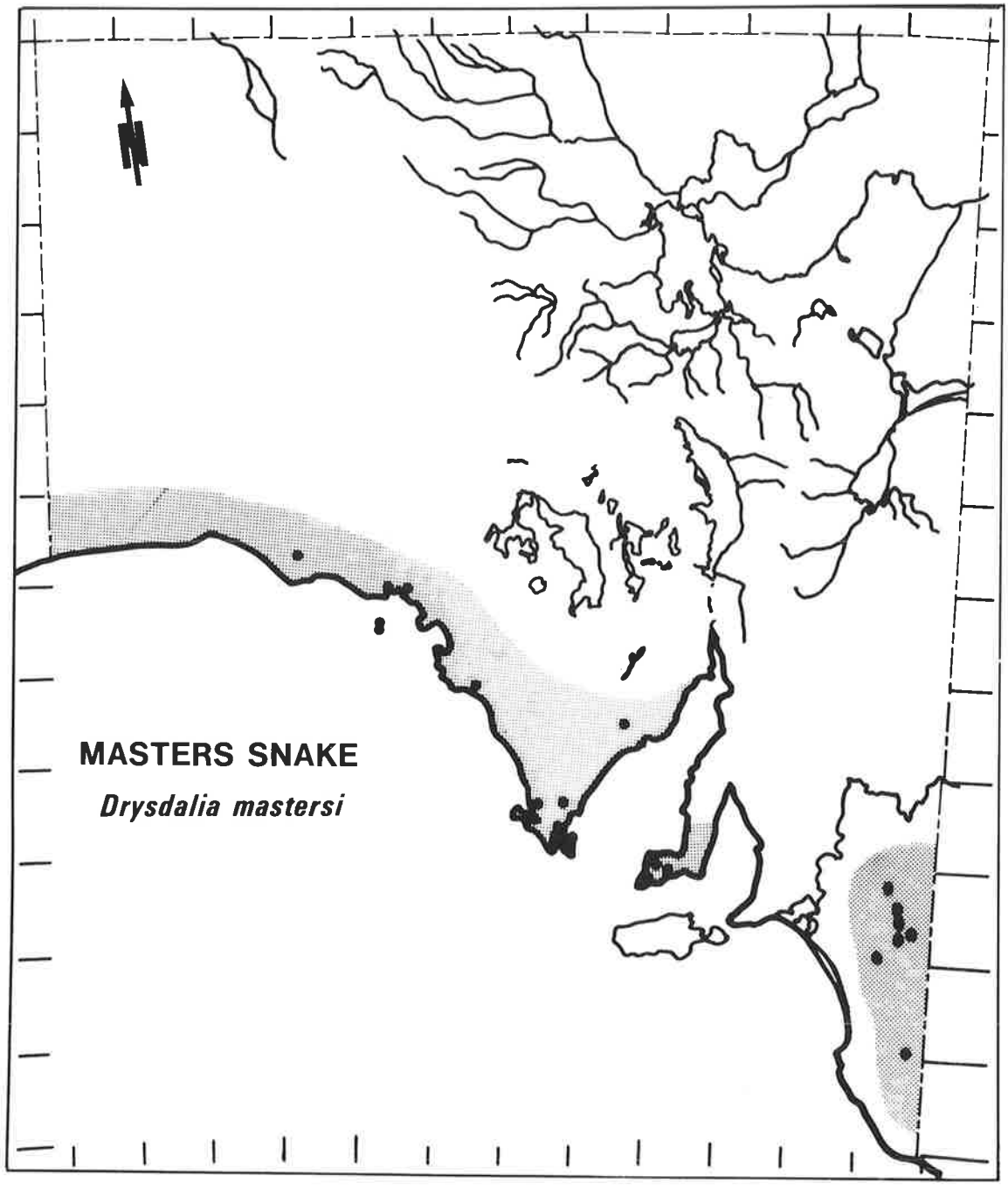
Drysdalia coronoides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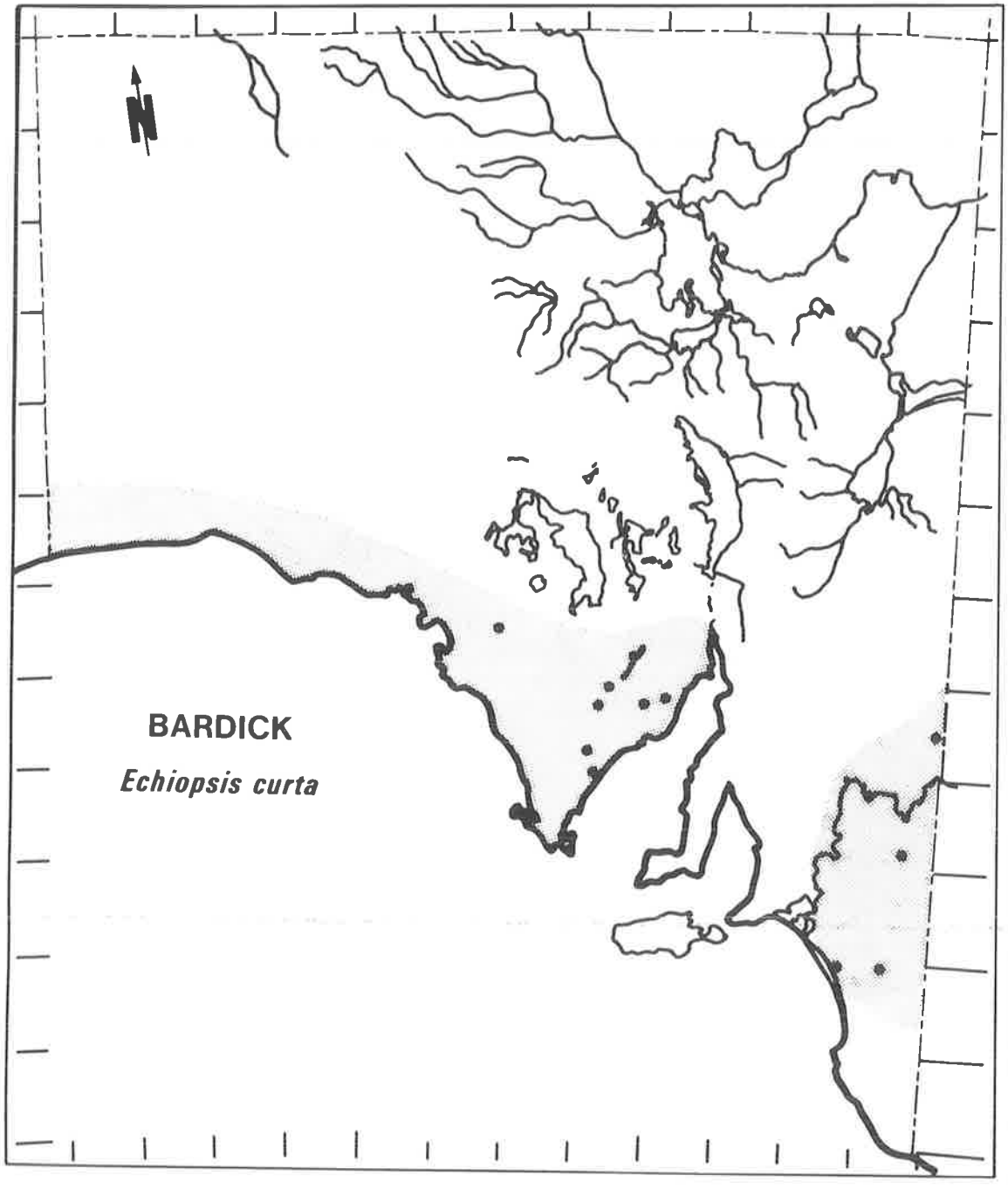
Drysdalia mastersi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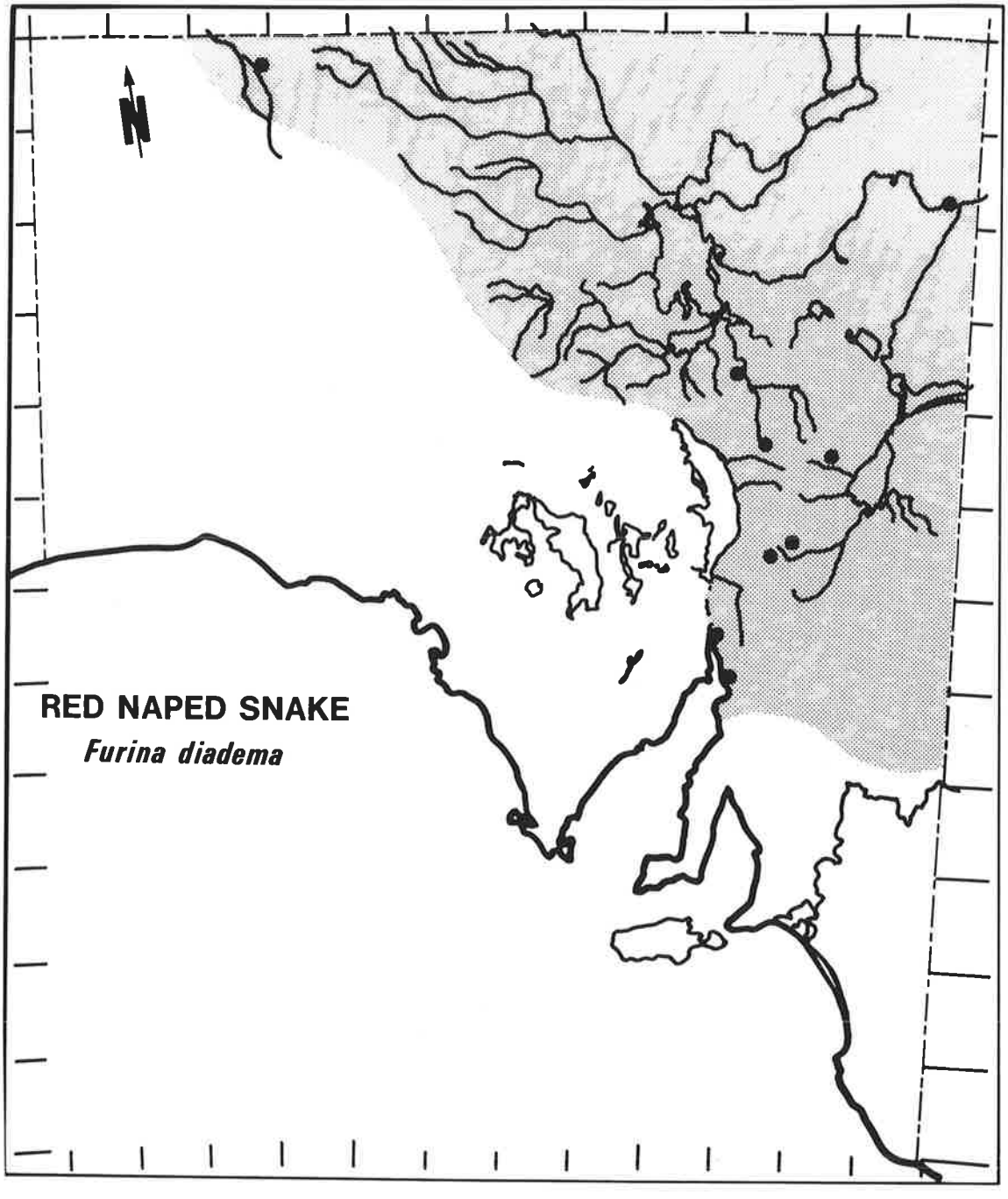
Echiopsis curta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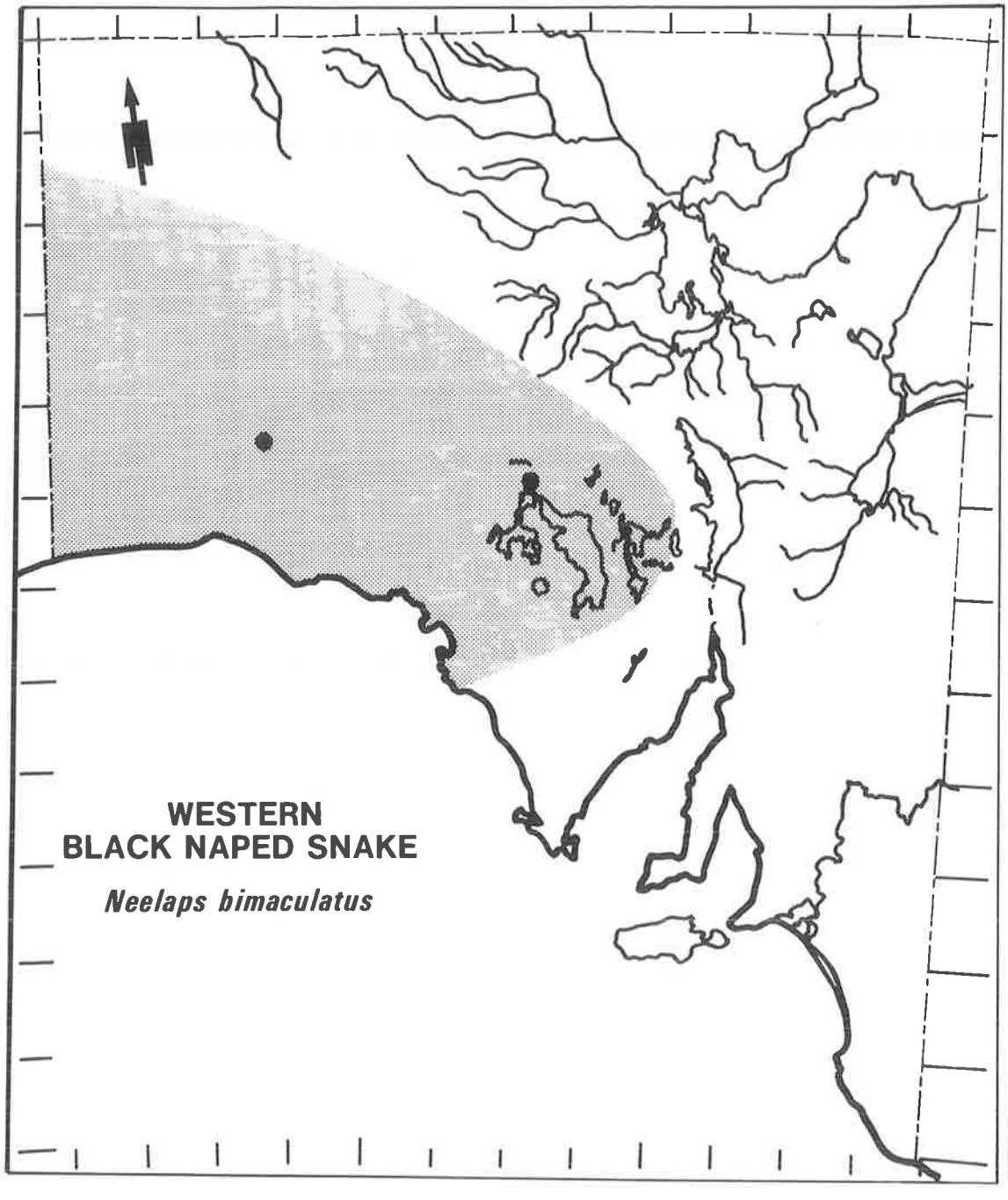
Furina diadema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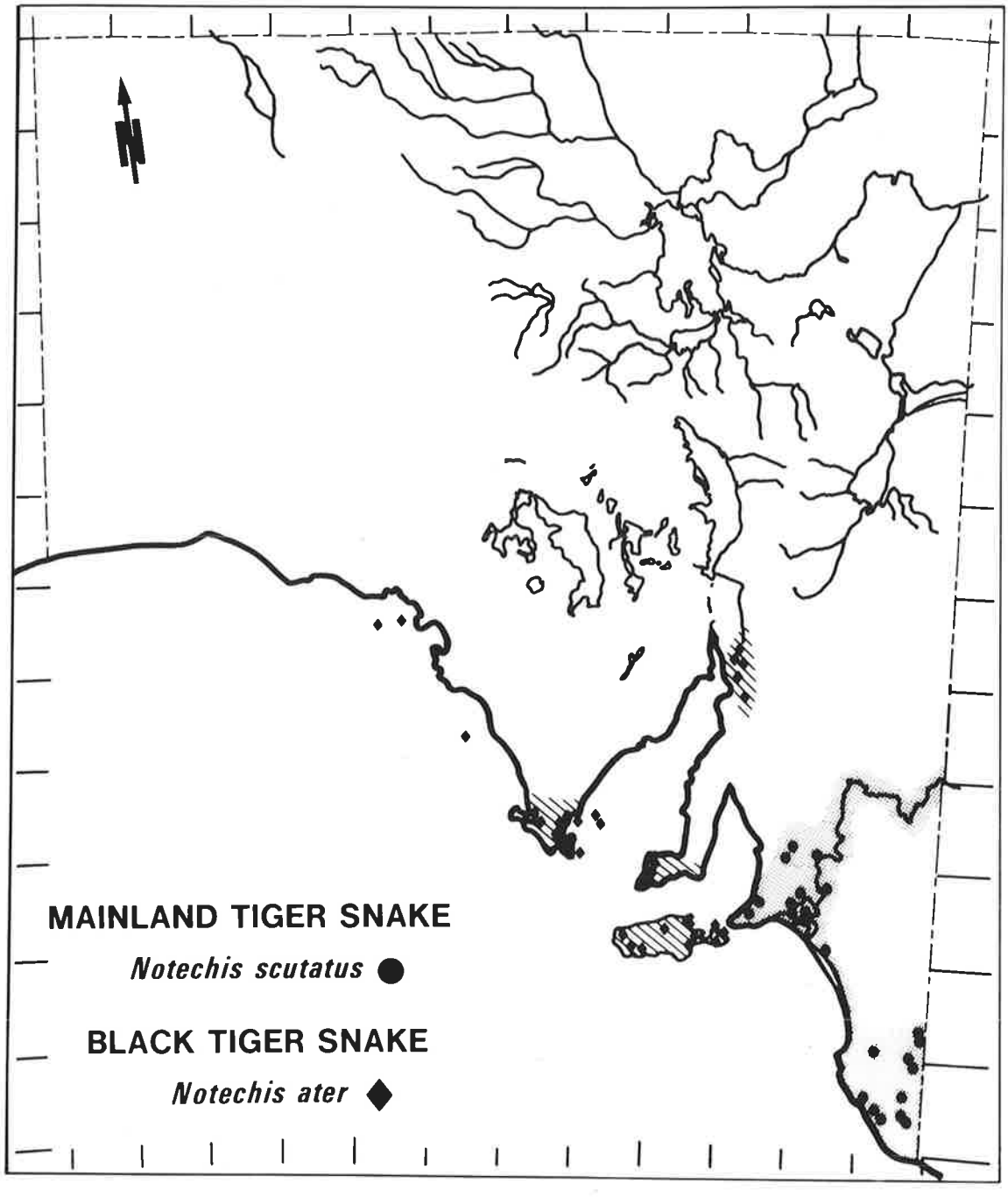
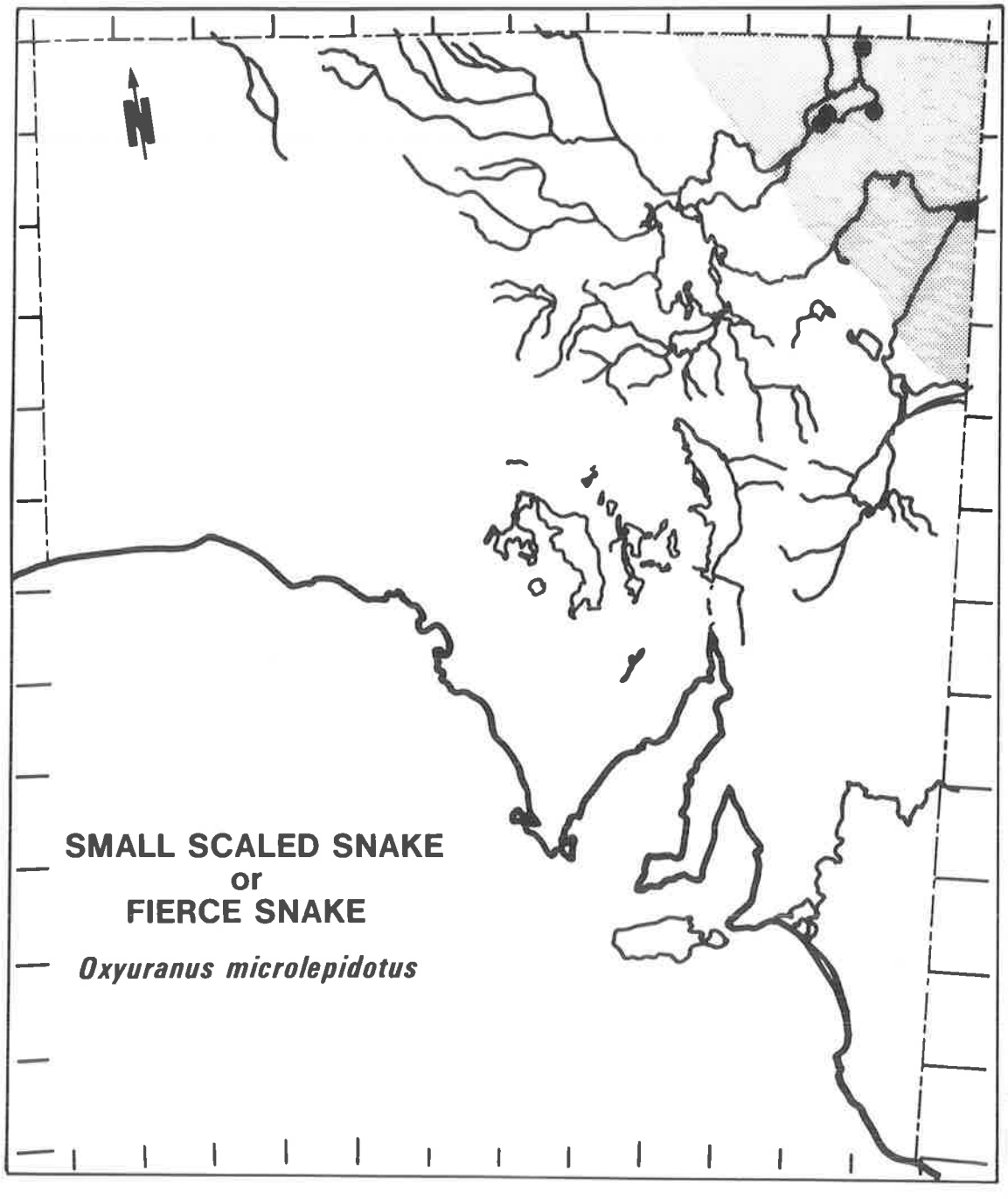
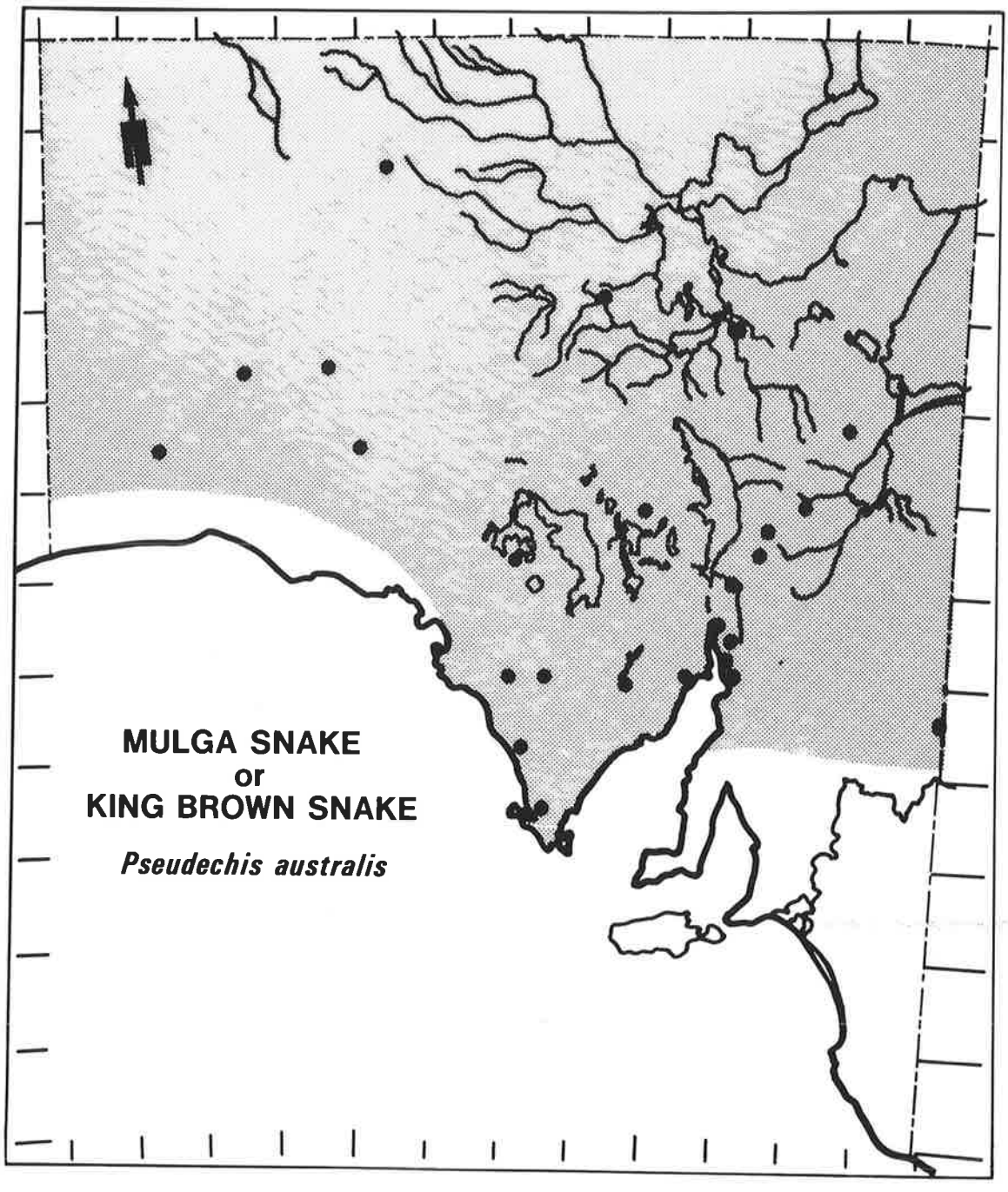
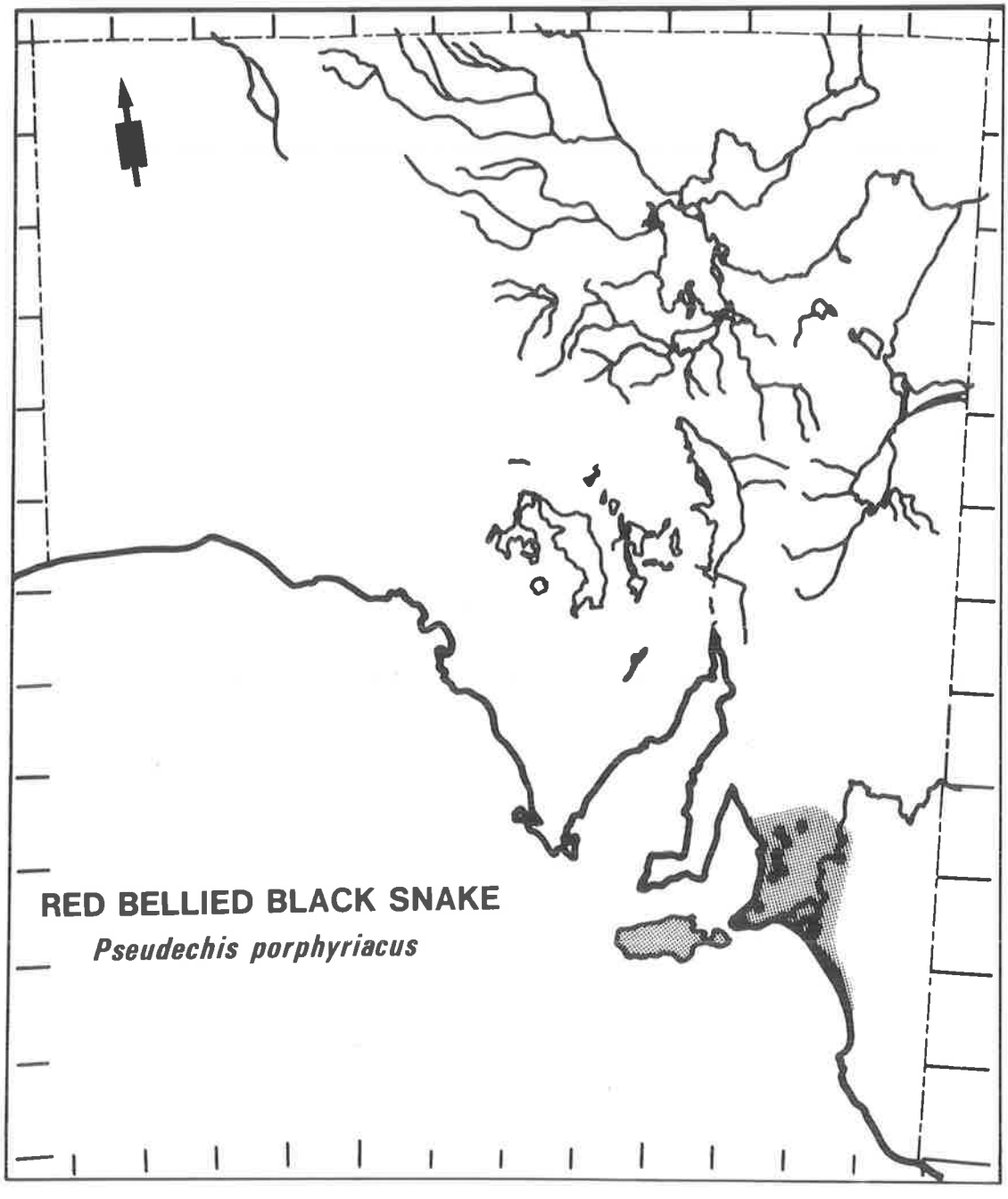
BLACK NAPED SNAKE
Neelaps bimaculatus


MAINLAND TIGER SNAKE
Notechis scutatus
BLACK TIGER SNAKE
Notechis ater


SMALL SCALED SNAKE
or
FIERCE SNAKE
Oxyuranus microlepidotus


MULGA SNAKE
or
KING BROWN SNAKE
Pseudechis australis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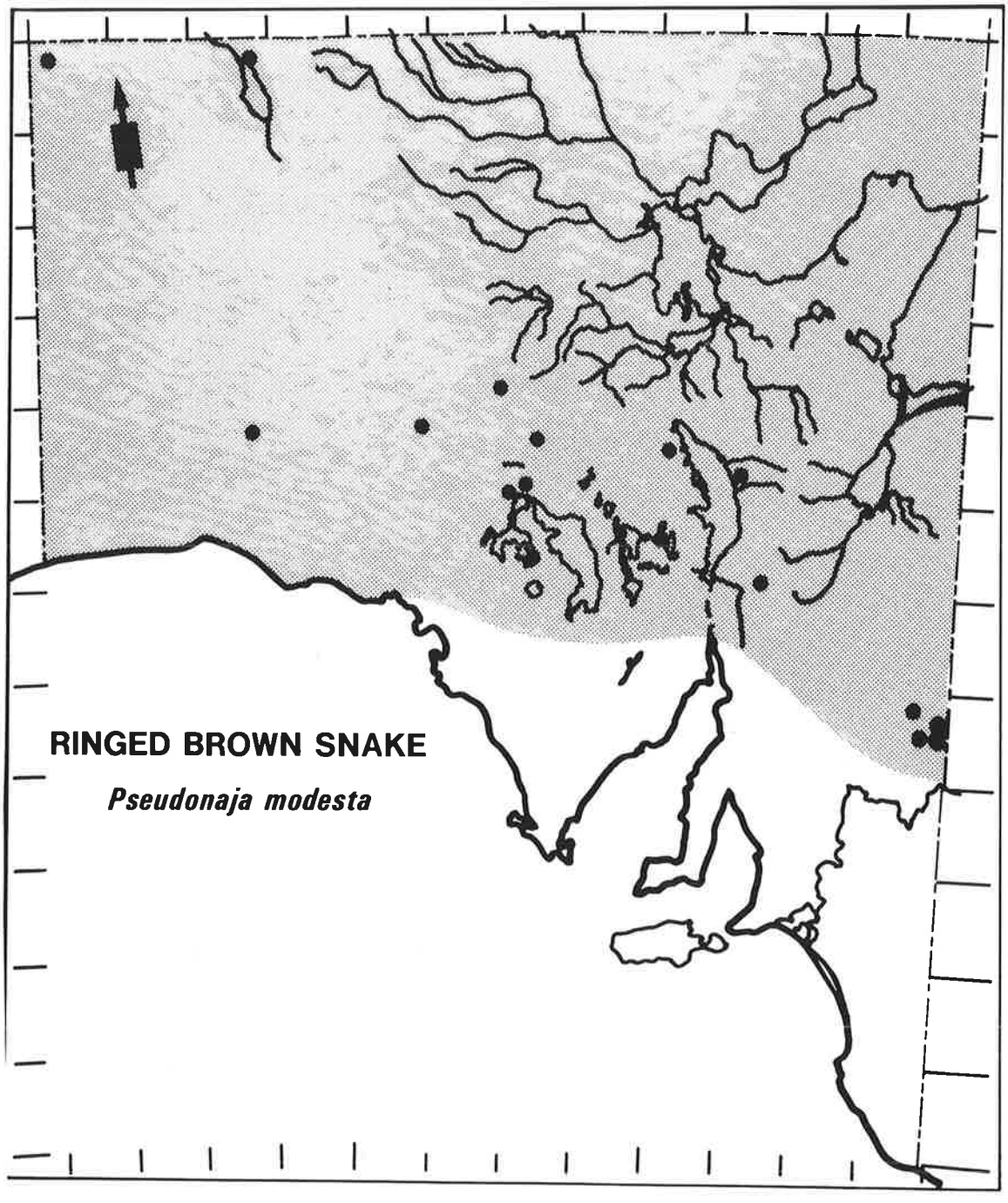
Pseudechis porphyriacus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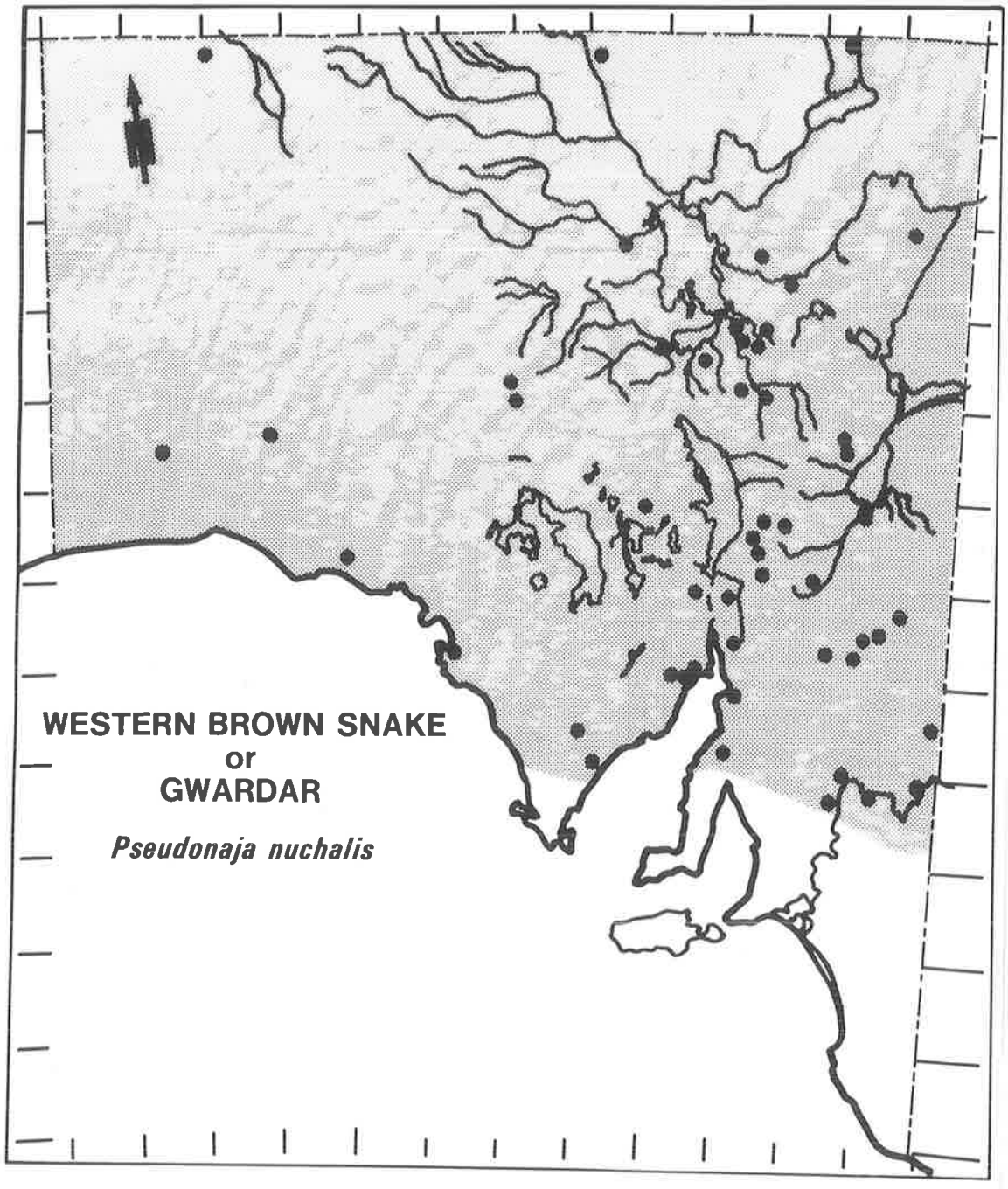
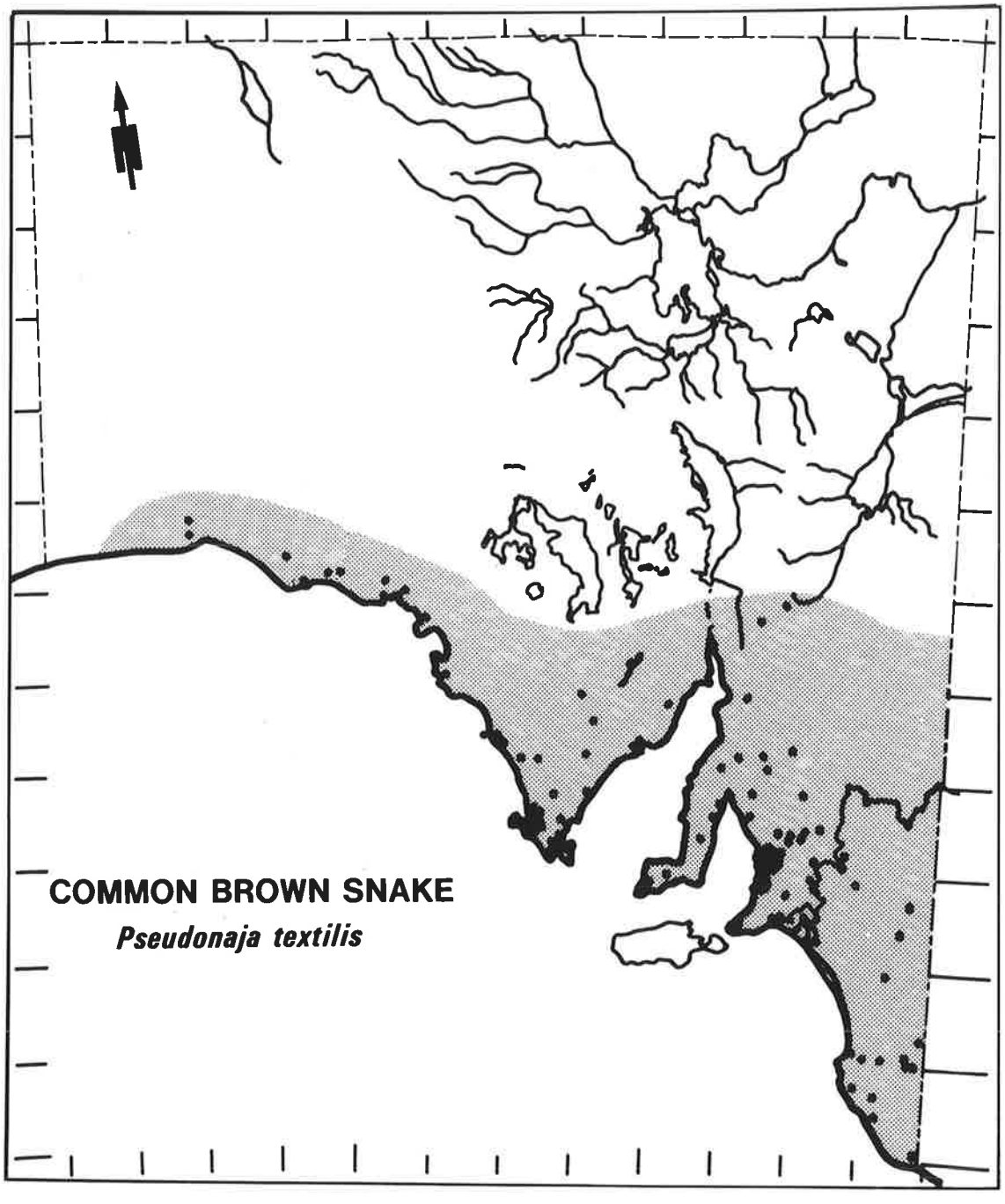
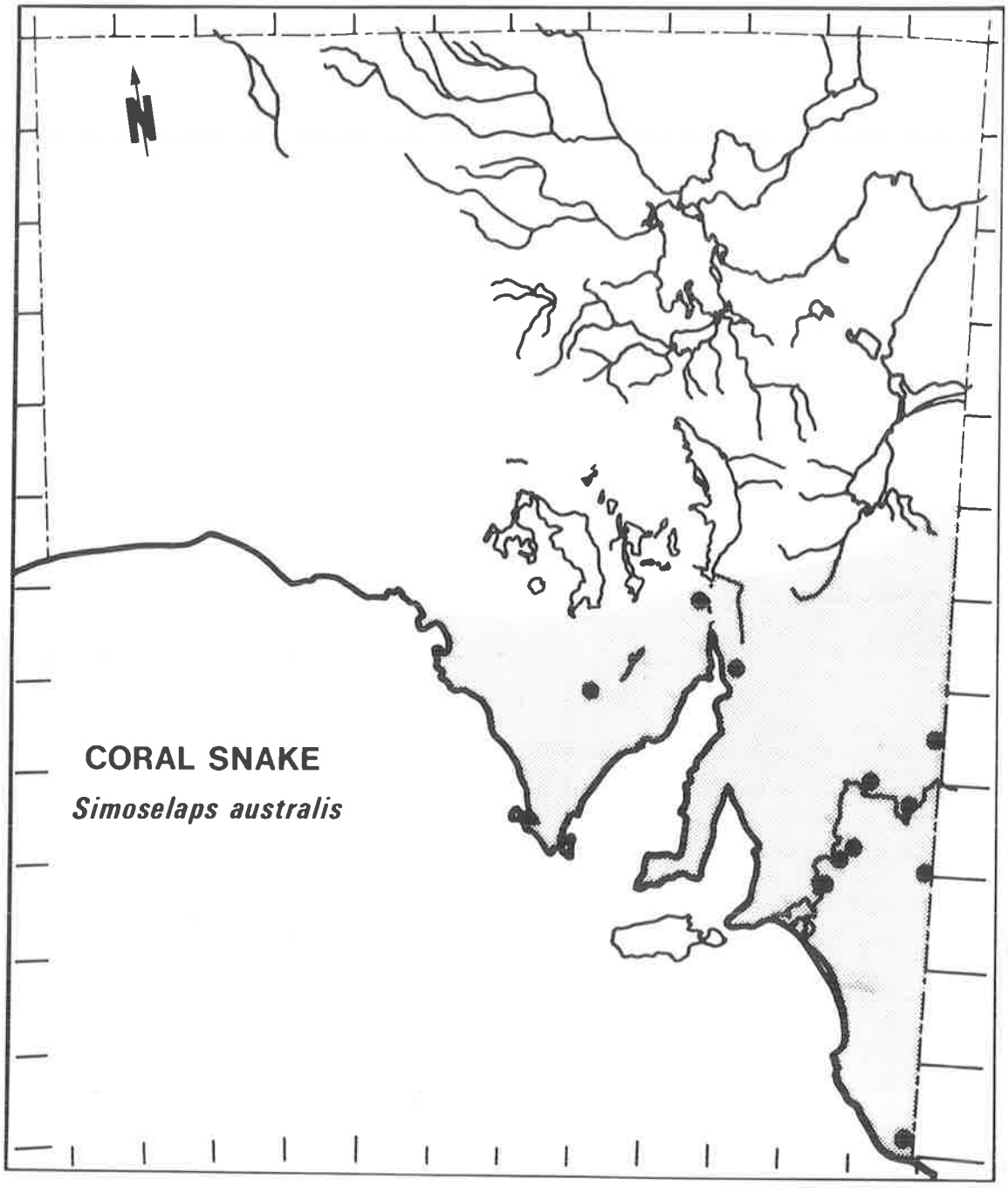
Pseudonaja modesta


WESTERN BROWN SNAKE
or
GWARDAR
Pseudonaja nuchalis


COMMON BROWN SNAKE
Pseudonaja textilis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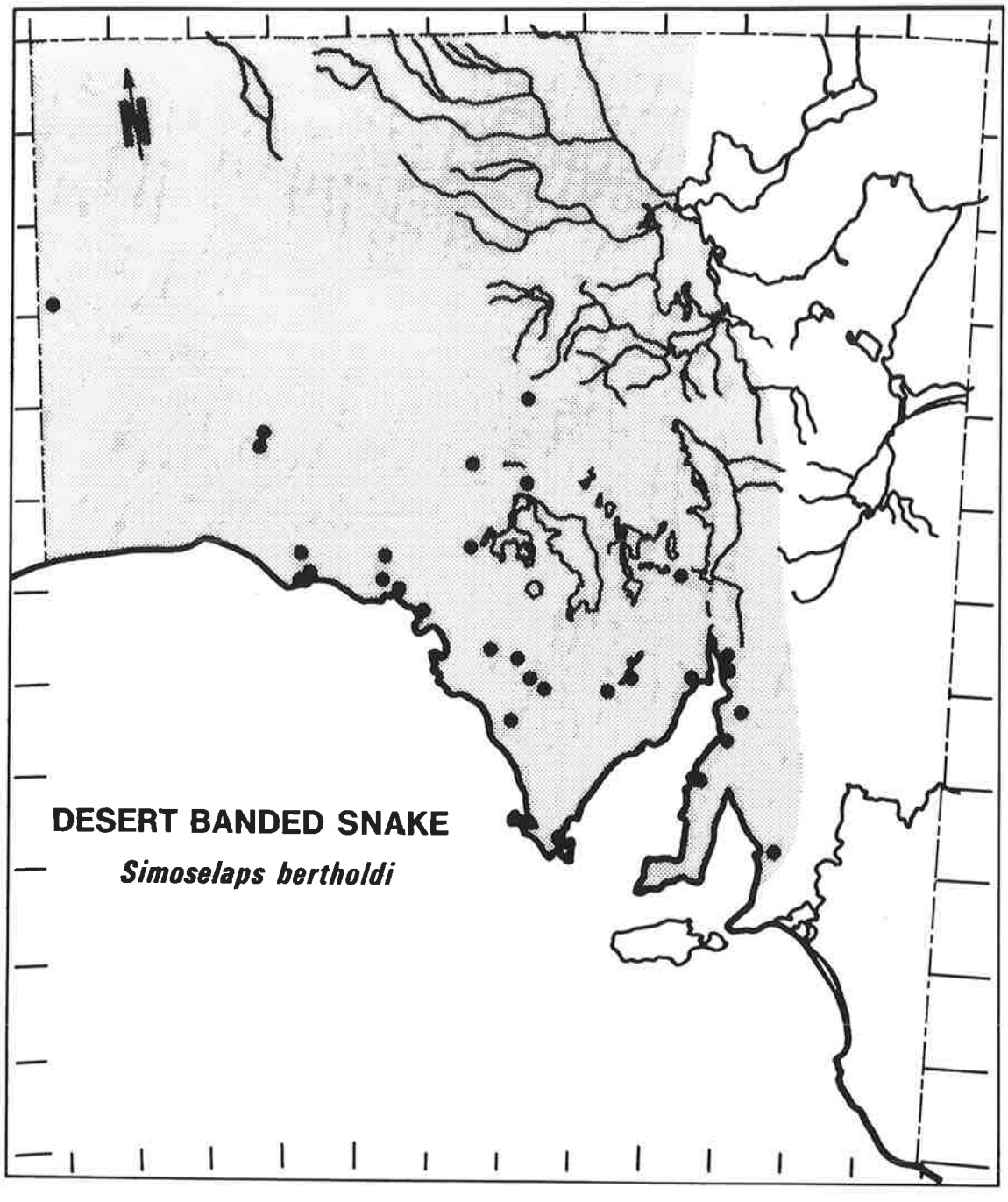
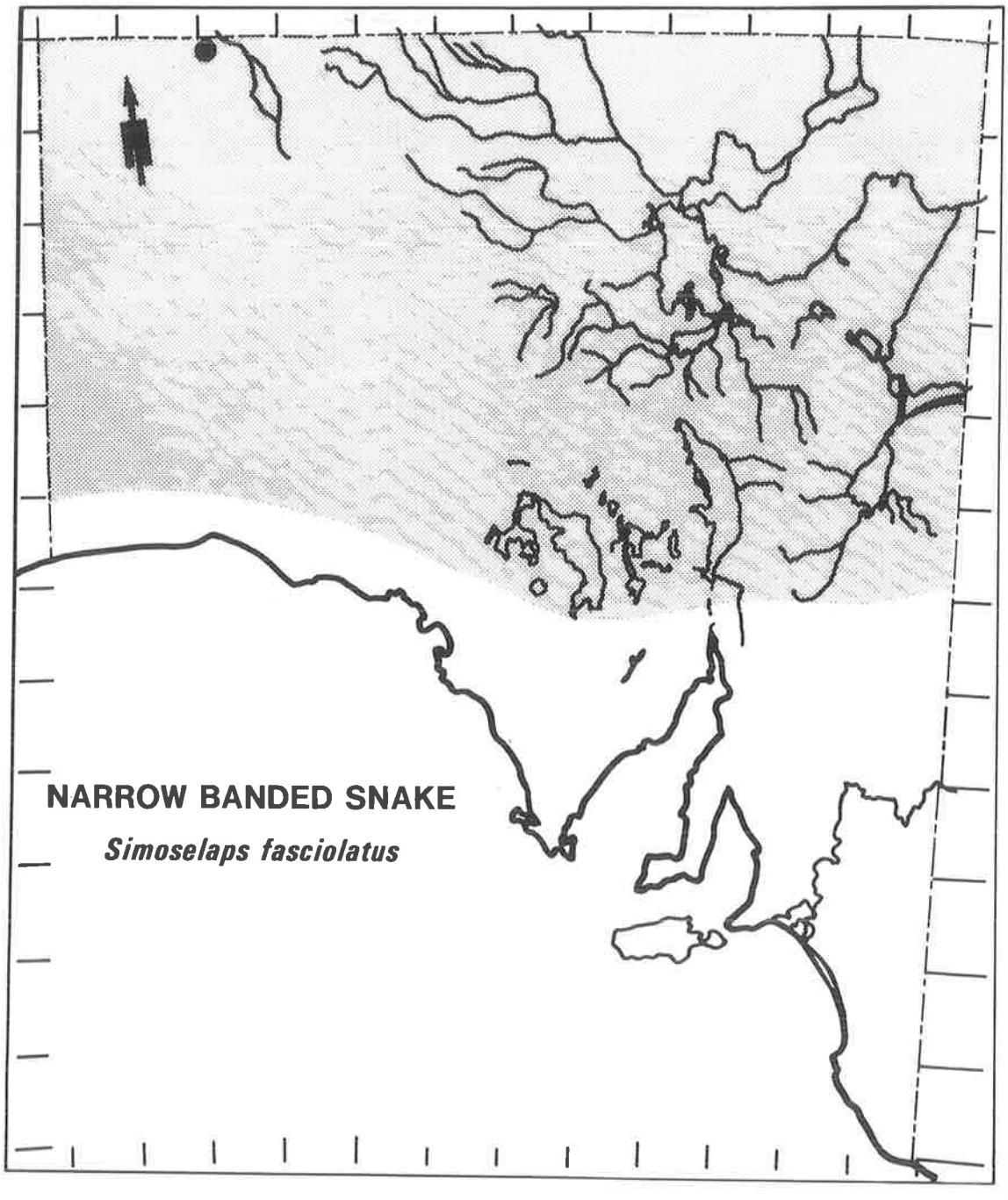
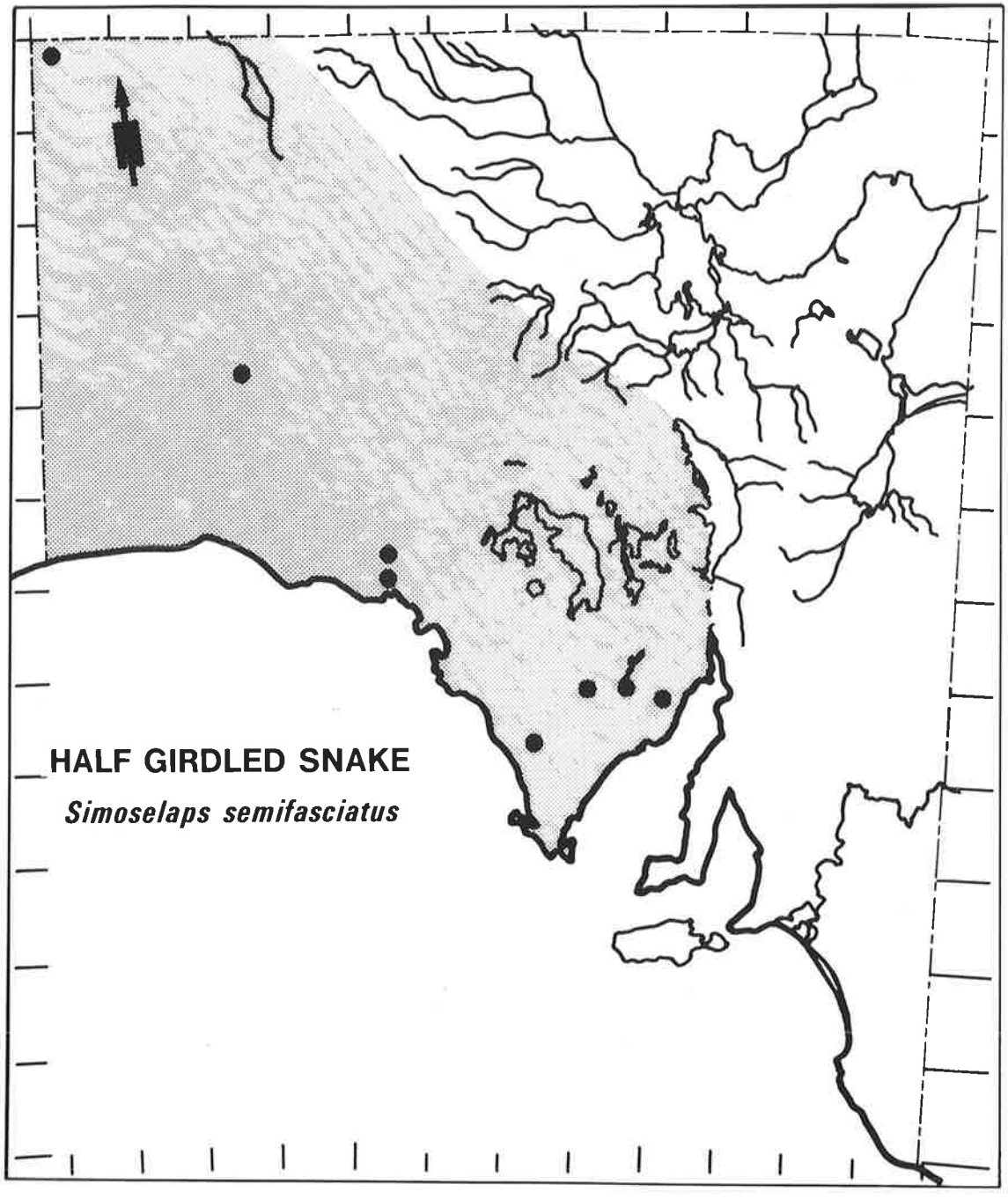
CORAL SNAKE
Simoselaps australis


DESERT BANDED SNAKE
Simoselaps bertholdi


NARROW BANDED SNAKE
Simoselaps fasciolatus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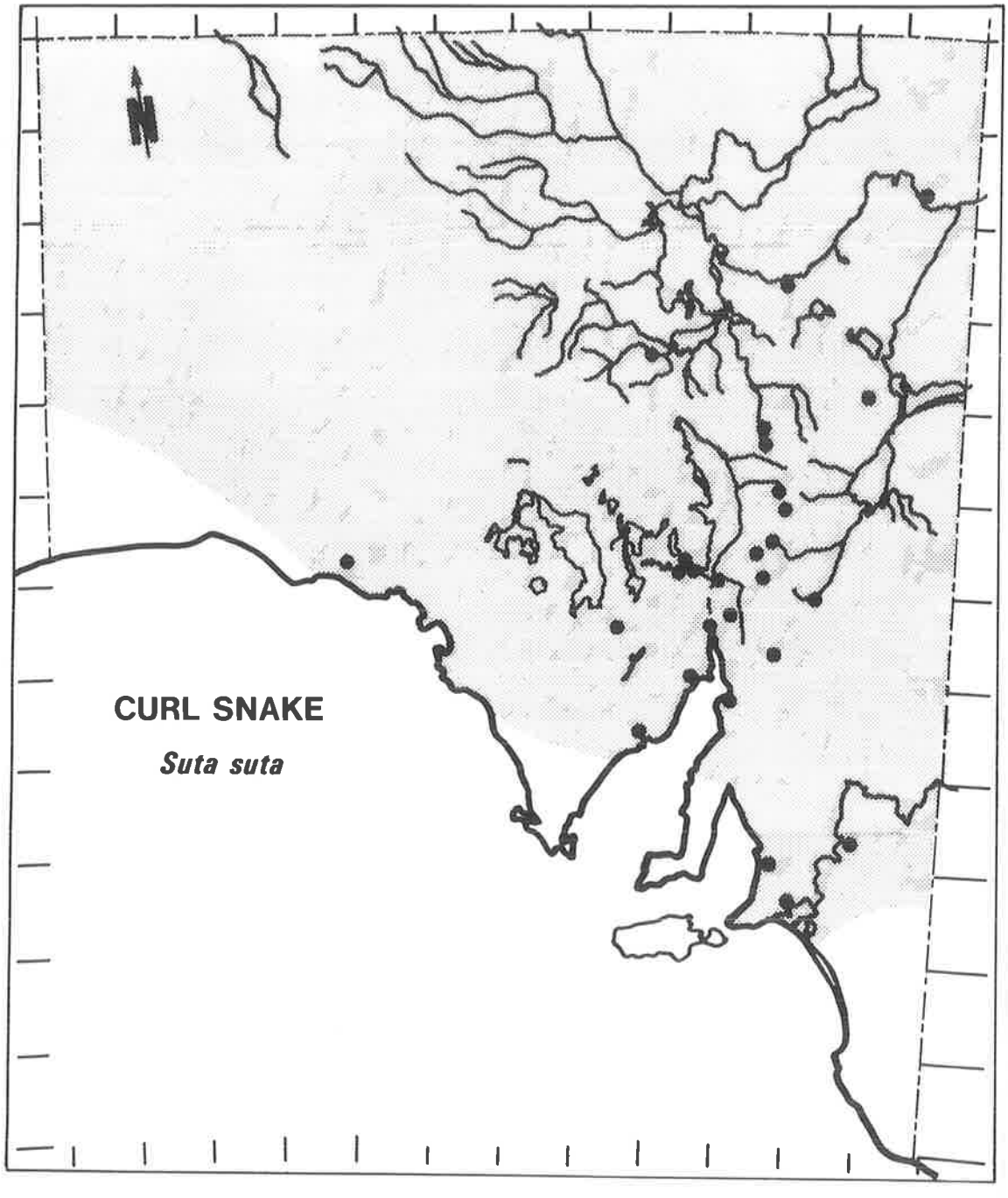
Simoselaps semifasciatus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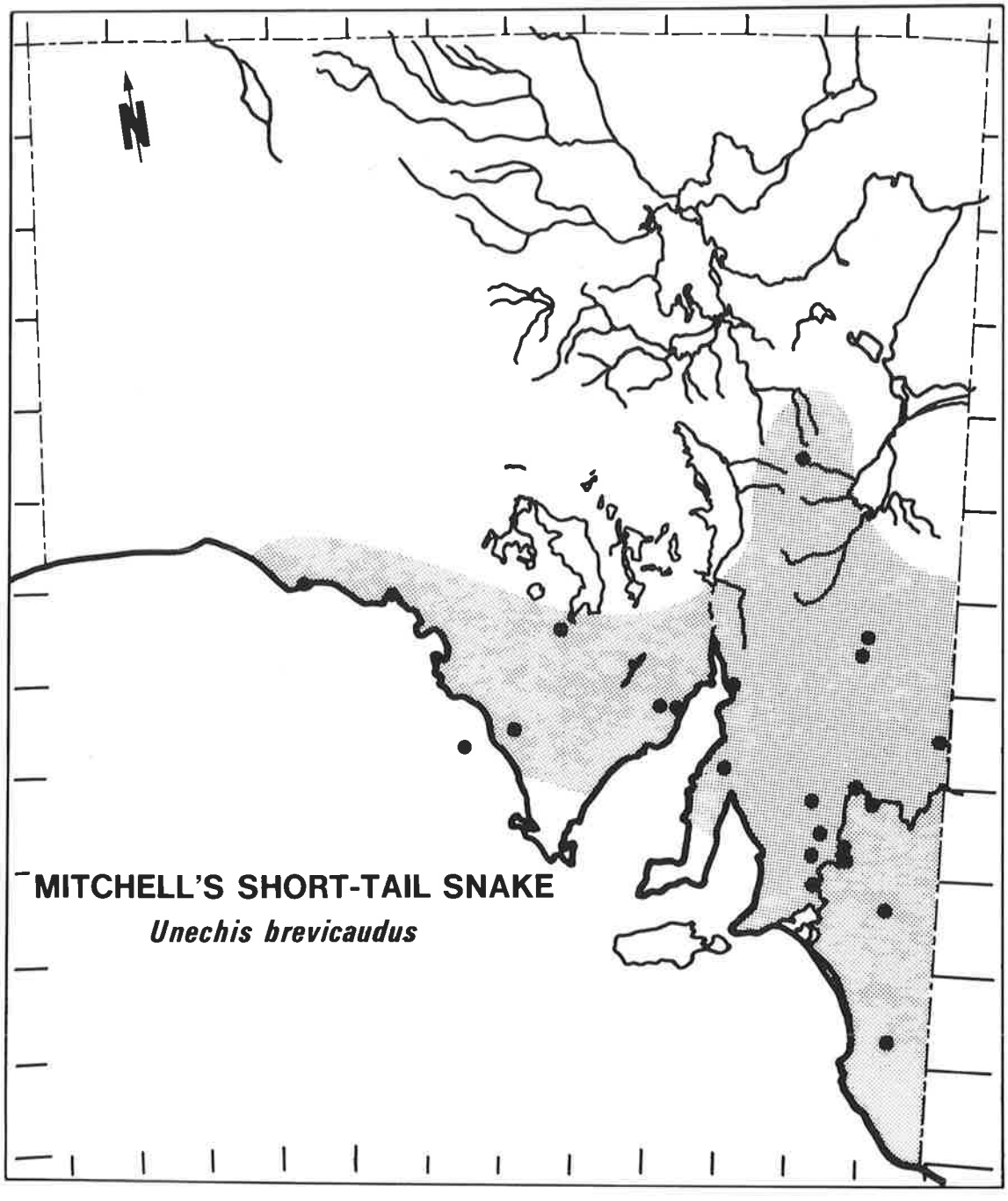
Suta suta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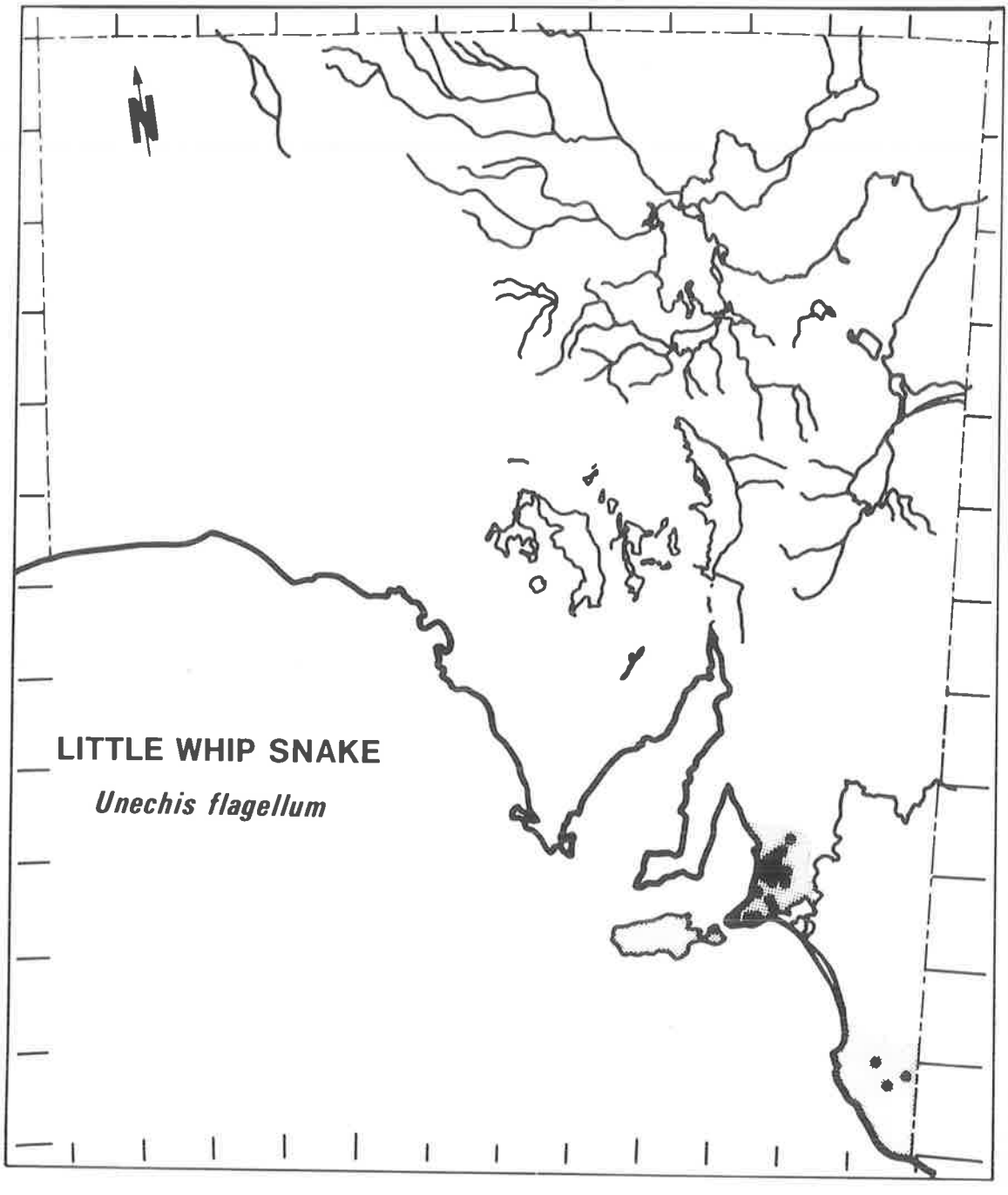
MITCHELL'S SHORT-TAIL SNAKE
Unechis brevicaudus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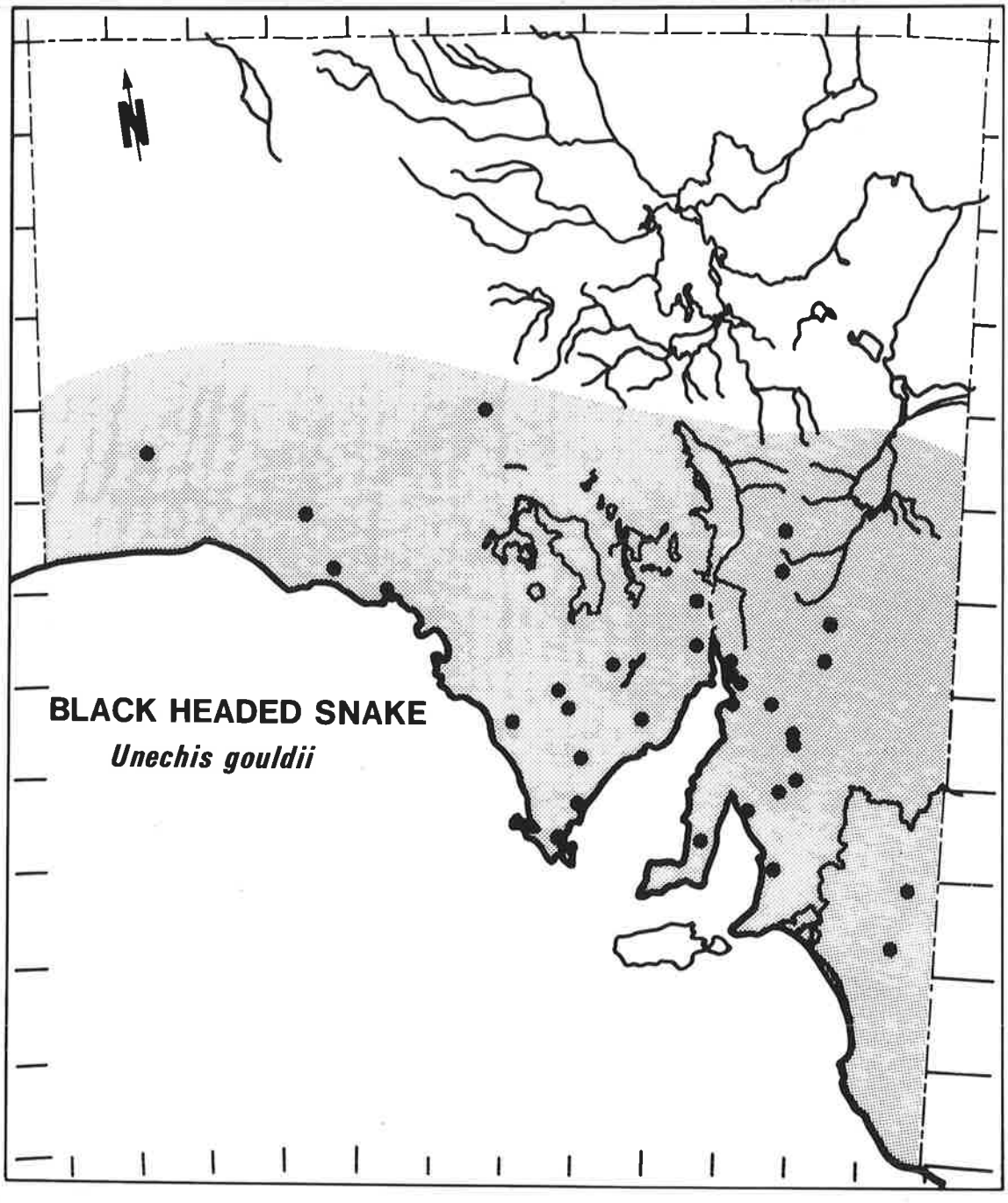
Unechis flagellum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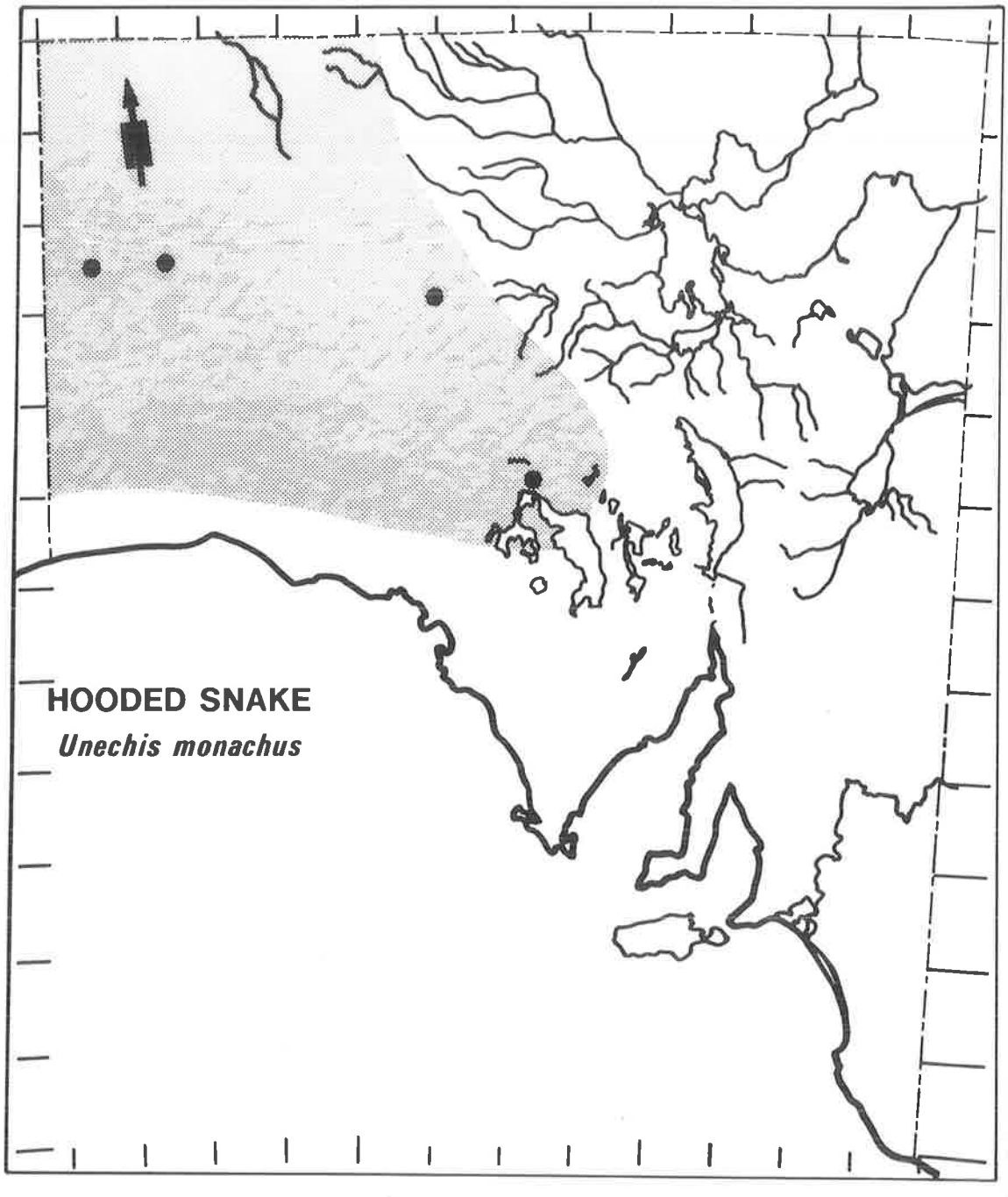
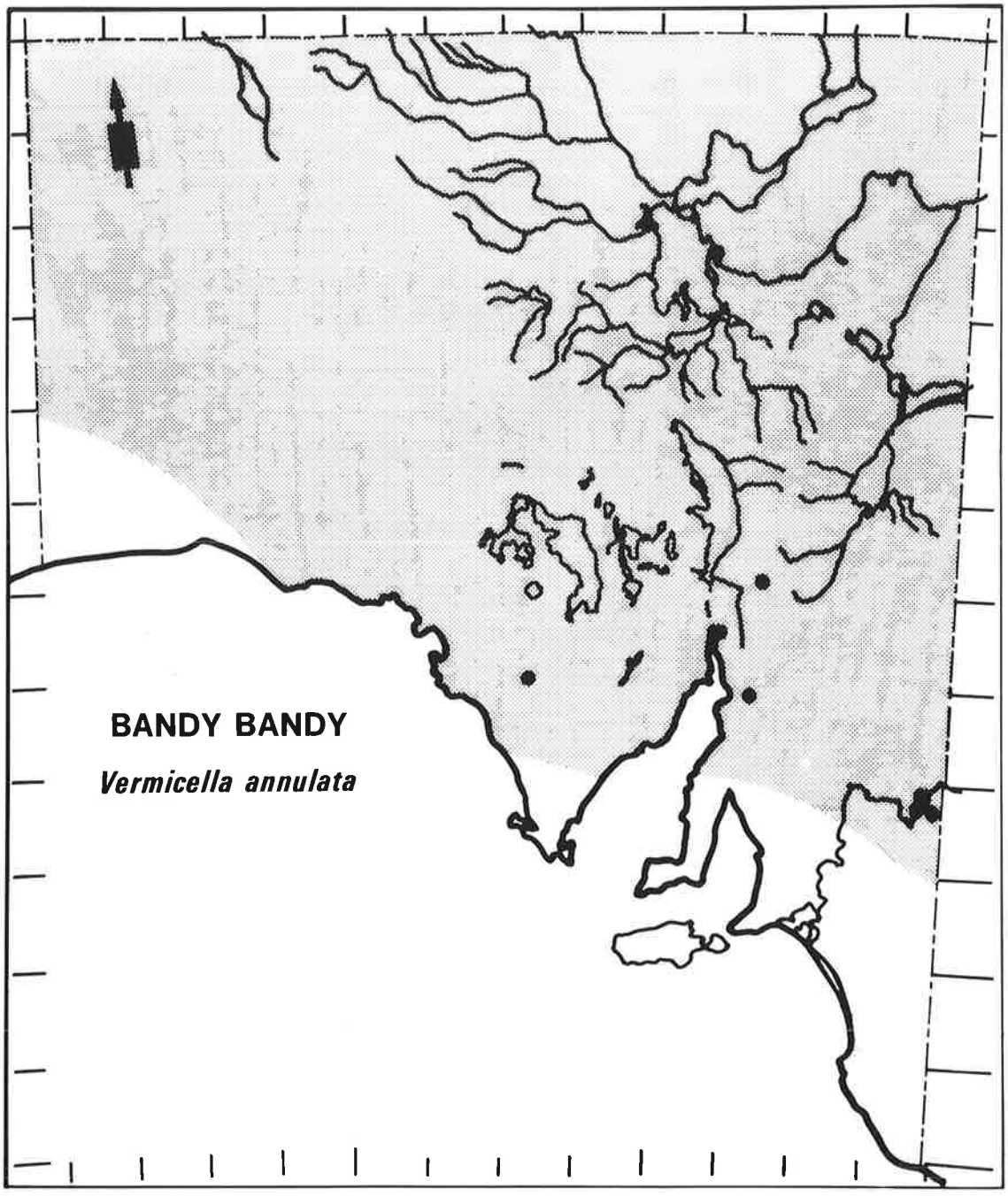
BLACK HEADED SNAKE
Unechis gouldii


HOODED SNAKE
Unechis monachus


BANDY BANDY
Vermicella annulata

AN ENGLISHMAN'S FOOD

An unimportant but interesting change which occurred about 1850 was that oysters quite suddenly became scarce and expensive. They had for centuries been so cheap that they were within the reach of all but the very poorest. At the beginning of the nineteenth century they were even associated with poverty, for as Sam Weller put it:

> . . . poverty and oysters always seem to go together . . . the poorer a place is, the greater call there seems to be for oysters . . . here's an oyster stall to every half-dozen houses [in Whitechapel]. The street's lines with 'em. Blessed if I don't think that ven a man's wery poor, he rushes out of his lodgings, and eats oysters in reg'lar desperation.

As late as 1840 they cost only 4d [4 pence] a dozen but within a very few years they were so dear as to be a luxury only the wealthy could afford. There is little doubt that the cause of this rise in price was reckless dredging of the natural beds as a result of increasing demand from the rapidly-expanding towns. Had it not been that something was known by this time of the breeding habits of oysters, and of methods of cultivating them artificially the mollusc might have become almost extinct in English waters.

J. C. Drummond and Anne Wilbraham (1939):
The Englishman's Food,
Revised, with a new chapter by Dorothy Hollingsworth, 1957.
Jonathan Cape, London.

* * *

A singular occasion for tracheotomy is related by Antoine Louis. A Londoner named Gordon had for many years carried on the double life of a butcher and a highwayman, and had amassed considerable wealth. At last, however, he was detected, tried and condemned to the gallows. A young surgeon named Chovell, who had performed tracheotomy successfully on dogs, persuaded the condemned man to allow him, for a large pecuniary reward, to perform tracheotomy on him the night before the morning fixed for his execution. The prisoner consented and the operation was successfully carried out, and a small tube left in the trachea. The gaoler observed some blood about the man's neck, but was persuaded that he had tried to commit suicide. The next morning the convict was hung in due course. When cut down after the usual period and handed over to his friends for burial he was found to be still living, but he died after a few minutes. The disappointed surgeon attributed death to the excessive pressure of the rope on the patient's neck, for the man was very heavy.

E. W. Goodall (1934):
The story of tracheotomy,
The British Journal of Children's Diseases,
Volume 31, page 256.

* * *

The now popular *Alstroemeria* commemorates the very modest journeys through Spain and other European countries, beween 1760 and 1764, of Baron Clas Alströmer, son of the more famous Jonas Alström whom Linnaeus had visited at Alingsås . . . *Alstroemeria* is familiarly known as "the Lily of the Incas" or "the Peruvian Lily". Since it is not a lily and does not come from Peru, the names are unfortunate, "Chilean daffodil" would be more appropriate. As Miss Alice Coats has pointed out, "the *Alstroemeria* has the distinction of being the only garden plant to wear its leaves upside-down, every leaf-stalk having a twist in it that brings what should have been the underside of the leaf uppermost". Linnaeus used often to associate some peculiarity of a plant he named, with some quality or characteristic of the man it honoured. "It is commonly believed", he wrote in his *Critica Botanica*, "that the name of a plant which is derived from that of a botanist shows no connection between the two. But anyone who has but the slightest knowledge of the history of letters will easily discover a link by which to connect the name with the plant . . ." He gives a few examples, such as: "*Commelina* has flowers with three petals, two of which are showy, while the third is not conspicuous; from the two botanists called Commelin, for the third died before accomplishing anything in botany". What, one may wonder, did he see in Alströmer to associate him with a plant whose peculiarity seems to be a desire to stand on its head?

Wilfred Blunt (1971):
The Compleat Naturalist:
A Life of Linnaeus,
Collins, London, pages 188–189.

Toxicon, Suppl.3, pp.489-491, 1983.
Printed in Great Britain.

Pergamon Press Ltd.

PATTERNS OF ELAPID ENVENOMATION AND TREATMENT IN SOUTH AUSTRALIA

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INTRODUCTION

Snakebite is a medical problem encountered in most countries, yet in many areas, Australia included, is poorly understood by most physicians. There are few studies of patterns of snakebite in Australia, a situation not helped by the grouping of snakebite with all other bites and stings in a single category in the International Statistical Classification of Diseases, Injuries and Causes of Death. In 1929 FAIRLEY noted that of 244 such deaths reported in the years 1910-1926 from Australia, approximately 80% were due to snakebite. Of these deaths, only 10 were from South Australia, despite the diversity and abundance of snakes in the State. In the five year period 1945-9, the W.H.O. (SWAROOP and GRAB, 1954) reported 28 cases of death from snakebite in Australia, and none from South Australia. TRINCA'S figures for the ten years, 1952-61 showed 45 deaths, with only one from South Australia (TRINCA, 1963). However, snakebite is not especially uncommon in S.A. 207 cases were reported to the Commonwealth Serum Laboratories (CSL) in the 24 years 1957 to 1982, and this does not include those cases either not requiring antivenom, which would be the case in the majority of snakebites, or those given antivenom without reporting to C.S.L. (COWLING and SUTHERLAND, 1982).

Most of Australias potentially dangerous elapid snakes are represented in S.A. (WHITE, 1981). The most commonly encountered are the Brown Snakes. The Common Brown Snake *(Pseudonaja textilis)* isabundant in the southern half of the State, including the outer metropolitan areas of Adelaide, where about 900,000 of the State's 1.2 million people live. The closely related Western Brown Snake *(P. nuchalis)* is equally abundant in the northern part of the State. The Mainland Tiger Snake *(Notechis scutatus)* is common along the River Murray Corridor and in the south east of the State. The Black Tiger Snake *(N. ater)* is found in a series of isolated populations on islands, in the Lower Flinders Ranges, and on the tips of Eyre and Yorke Peninsula. The Black Snake group is represented by the Red Bellied Black Snake *(Pseudechis porphyriacus)* in the Adelaide Hills region, and the King Brown or Mulga Snake *(P. australis)* throughout the northern part of the State. The Copperhead group is represented by the Common Copperhead *(Austrelaps superbus)* in the south east, and the Adelaide Hills Copperhead *(A. sp)* in the Adelaide region and Kangaroo Island. The two closely related Death Adders *(Acanthophis sp.)* are found throughout much of sandy and arid S.A., though their ranges are contracting under encroachment by man. The Inland Taipan or Fierce Snake *(Oxyuranus microlepidotus)* is now thought to be quite well represented in the north east of the State, where there is active exploration for and development of oil and gas resources. The potential lethality of these snakes has been defined by BROAD *et al*, (1979). The Common Brown Snake, the commonest snake in areas of human habitation, has the second most potent venom described, eclipsed only by the Inland Taipan. In view of the range of snakes and paucity of information available on snakebite patterns in South Australia, cases of snakebite admitted to selected hospitals in S.A. were reviewed.

MATERIAL AND METHODS

This retrospective study was based on cases of snakebite admitted to three of Adelaide's major teaching hospitals over an approximate ten year period. Cases were located by their disease coding. Thus cases of snakebite incorrectly coded would have been excluded, and all cases presenting to casualty, but not admitted, would likewise be excluded. One other teaching hospital and two large peripheral hospitals were not included for logistic reasons. Similarly, country hospitals were excluded. Thus there is considerable bias in the statistics. To give a more complete picture for the whole State, cases reported to C.S.L. have been reviewed (COWLING and SUTHERLAND 1982). The hospital records examined were from the Adelaide Children's Hospital, 1968-1977; the Royal Adelaide Hospital, 1970-1978; and the Flinders Medical Centre, 1976-1980, these being the periods for each hospital which were completely coded at the time of review.

RESULTS

At the Adelaide Children's Hospital, in a 10 year period 23 cases of definite or suspected snakebite were admitted, and there were no fatalities. The average age of patients was 8.2 years, with a range of 1.6 years to 13.8 years, and a sex ratio of 19 male to 4 female. There was a definite increased incidence in the toddler age group, and in the early teen years. In only 12 of the 23 cases was any reasonable identification of the snake made, and in these, 10 were Common Brown Snakes and 2 were Red Bellied Black Snakes. The area bitten was evenly divided, with 11 to the upper limb, 11 to the lower limb, and 1 to the lower lip. 12 of the bites occurred during spring and 8 in the summer. Of the 23 cases, only 7 developed any signs or symptoms of envenomation. Of these, only 2 were significantly envenomated, and both received polyvalent antivenom as the species of snake involved was in doubt. One had initial collapse, followed by nausea, vomiting and restlessness. The other had definite neurotoxic envenomation, with impaired conscious state, diplopia, dysarthria, ptosis and general facial weakness, all of which were well established when antivenom was given over 24 hours after the bite. There were no haematological problems documented. Of the 7 patients with possible evidence of envenomation, 4 received antivenom. More puzzling is the fact that 5 patients were given antivenom in the absence of either signs or symptoms of envenomation, and without a snake even having been seen.

At the Royal Adelaide Hospital in a 9 year period, 16 cases of snakebite or possible snakebite were admitted, and there were no fatalities. The average age of patients was 26 years, with a range of 14 years to 46 years, and a sex ratio of 15 male to one female. 4 of the cases were in amateur reptile keepers. Of the remaining 12 bites, 7 were due to Brown Snakes, 2 to Tiger Snakes and 2 to Red Bellied Black Snakes, with 1 uncertain. The area bitten was the upper limb in 6 cases and the lower limb in 5. Surprisingly, the area bitten was not stated in 5 cases. Of the 16 cases, 8 developed some symptoms or signs suggestive of envenomation, but none were severe, although one case developed renal failure requiring haemodialysis for several weeks. This case is interesting for several reasons. The man concerned was apparently well known in his country town for his frequent and excessive inbibing of alcohol. While thus intoxicated one night, he was bitten by a snake later positively identified as a Brown Snake. In retaliation he replied in kind by biting the snake, and succeeded in biting its head off, which grizzly momento he pocketed. He then presented to the local hospital for treatment, but was not taken seriously until the snake's head was produced. However he developed no signs or symptoms of snakebite and was transported to another country hospital where he was given Tiger Snake antivenom. This certainly was followed by problems, culminating in acute renal failure, from which he eventually recovered. Other complications seen following snakebite were minor. Of the 16 cases, 10 received antivenom, but in 4 of these, there were very doubtful grounds for use of antivenom. Also, 3 patients who did not receive antivenom, did have symptoms or signs suggestive of envenomation. Of the four bites in reptile keepers, making 25% of all snakebite admissions, 2 were the same keeper. This young man also accounted for 5 of the 14 admissions at the Flinders Medical Centre, and I believe he also has had multiple admissions to other hospitals not surveyed by me.

At the Flinders Medical Centre in a 5 year period 14 cases of snakebite or possible snakebite were admitted, and there were no fatalities. The average age of patients was 18.5 years, with a range from 1 year to 30 years old, and a sex ratio of 12 males to 2 females. 6 of the cases, or 43%, were in reptile keepers, although 5 of the 6 were just one individual, as mentioned earlier. Of the remaining 8 bites, all were due to Brown Snakes or unidentified snakes which in retrospect, were probably Brown Snakes. The area bitten was the upper limb in 7 cases, and the lower limb in 6. 8 of the 14 bites were at night, but 6 of these were in reptile keepers. Significant problems following snakebite only occurred in the reptile keepers, and these accounted for the only 2 cases given antivenom.

DISCUSSION

Several interesting points arise from this limited study. Firstly, though snakebite is not a major problem for health authorities in S.A., there is a consistent influx of snakebite victims to teaching hospitals in Adelaide, and to country hospitals, and

therefore snakebite and its management should be an integral part of undergraduate training in the State's medical schools. Secondly, though the range of species found in S.A. is wide, members of the Brown Snake group account for most of the bites, most of which are minor. Figures from C.S.L. confirm this for all of S.A., with 63% of all reported snakebites where the snake was identified were due to Brown Snakes. Tiger Snakes were the only other common culprit, with 21% of snakebites. Thirdly, at least in Adelaide, reptile keepers do form a significant percentage of admissions for snakebite. Except at the Adelaide Children's Hospital, many of the serious snakebites are in reptile keepers bitten while handling their pets. I suspect that in the vast majority of cases this is in association with a heavy intake of alcoholic beverages, and a desire to display their potentially lethal pets to friends. To my knowledge, almost every potentially lethal species of Australian snake is represented in amateur collections in Adelaide. This includes Taipans and Inland Taipans, and numerous Death Adders.

All reptiles are protected in S.A., and a permit from the National Parks and Wildlife Service is required to keep them, but no special provision is made for dangerous snakes. There are some amateur reptile keepers who have been catching and keeping dangerous snakes for years, without ever sustaining a bite, and their expertise is often used by professional herpetologists. However, many keepers have been bitten repeatedly, and this group require some further regulation. It is proposed, therefore, that legislation be amended in S.A., to allow special restrictions on keeping of dangerous snakes. Persons wishing to keep these snakes would have to be vetted by a committee. The problems inherant in such a system are obvious, and suitable legislation has yet to be framed.

The last point in relation to this study is the variable and sometimes poor quality and consistency of medical care of snakebite victims. Better medical education will help. Concentration of cases in as few centres as possible will allow greater attainment of skills in management by consultant staff. The extensive facilities of teaching hospitals should be mobilized to allow detailed assessment of cases of snakebite, to extend our understanding of envenomation in man. This will be most practical when all cases of snakebite are managed directly or indirectly by a small cadre of consultants with a commitment to furthering our understanding of snakebite. Individual doctors at every hospital taking sole management of cases because of their novelty interest, rather than referring on to a centre of expertise, is not in the interest of patient, medicine or research.

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ACKNOWLEDGEMENTS

The author gratefully acknowledges the administrators and medical staff of the Adelaide Children's Hospital, Royal Adelaide Hospital and Flinders Medical Centre for permission to examine case records; Wendy Cowling and Struan Sutherland of C.S.L. for preparing figures on snakebites reported to C.S.L. from South Australia; Brian Fotheringham and Les Sheffield for helpful comments on drafts of the paper; the Department of Clinical Photography, A.C.H., for helping prepare illustrations; and Maureen Booth for typing the manuscript.

Toxicon, Suppl.3, pp.493-496, 1983.
Printed in Great Britain.

Pergamon Press Ltd.

LOCAL TISSUE DESTRUCTION AND AUSTRALIAN ELAPID ENVENOMATION

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INTRODUCTION

Local tissue injury is a common feature of snakebite in many regions of the world and is assocaited with bites by both Viperid and Elapid snakes. Some elapid snakes, particularly the Cobras, frequently cause severe local tissue injury, in addition to problems caused by systemically active venom components such as neurotoxins. However, snakebite in Australia is apparently rarely associated with significant local tissue injury, for local problems are virtually never mentioned in published cases of snakebite from Australia. (WHITE, 1981). Australian elapid venoms are more potent than venoms of elapid snakes elsewhere, and contain a variety of systemically active toxins including presynaptic neurotoxins, postsynaptic neurotoxins, myotoxins, haemolysins, and prothrombin activators. They also contain a variety of enzymes which may potentially cause local tissue injury, although the precise enzyme composition of these venoms is not known. However, the lethal activity of these venoms lies in the systemically active components.

The venom delivery apparatus of elapid snakes is not as sophisticated as that of viperid snakes. While the modified maxilla can rotate slightly, it cannot move sufficiently to fold the fang, so fang length is restricted. In some Australian species, fang length is very short, with an average of only 2.8mm for the Brown Snake, which is one of the most important species implicated in snakebites. Another important species, the Tiger Snake, has an average fang length of 3.5mm. Those Australian species with larger fangs are less commonly implicated in snakebite. The quantity of venom delivered matches imperfectly to fang length, but those species with large fangs do produce more venom on average than those with smaller fangs. FAIRLEY (1929) studied the bite mechanism of Australian elapid snakes, and delineated four phases of the bite. The first is the strike. The fangs are rotated forward at contact, to maximum elevation. The mouth then closes with fang entry and venom inoculation. The maxillae and fangs then rotate backwards leaving a track of venom beneath the skin. This latter movement may be responsible for linear marks on the skin rather than a single puncture wound. It would also assist in drawing small prey items into the snakes mouth.

The range of similarly coloured potentially dangerous snakes in most regions of Mainland Australia, has made the treatment of snakebite difficult, and necessitated the frequent use of Polyvalent Antivenom, which has a significantly higher incidence of complications than monovalent antivenoms. (SUTHERLAND and LOVERING, 1979). The Commonwealth Serum Laboratories have recently produced an ELISA (Enzyme Linked Immuno Sorbent Assay) Kit to allow identification of species from venom swabbed from the victim. This will substantially improve treatment of snakebite in Australia, if it becomes widely utilized. Nevertheless, if clinical differences in the signs and symptoms between species exist, this may be very useful in allowing the use of monovalent antivenom when the ELISA is not available. However, little attempt has been made to delineate such differences in the past. PEARN and COVACEVICH (1981) published a brief "Atlas of Skin Lesions in Snake Bites", and WHITE (1981) reviewed a large personal series of snakebites from South Australia which included comments on local tissue appearances. As the range of systemic problems is similar in most Australian elapids, differences in local appearances may be more useful as differentiators.

MATERIALS AND METHODS

Cases of snakebite admitted to the Adelaide Children's Hospital (1968-1982), Royal Adelaide Hospital (1970-1982) and the Flinders Medical Centre (1976-1980) were reviewed. Other snakebites in the author's personal series were also reviewed. Photographs of the bite site were reviewed where available.

RESULTS AND DISCUSSION

Bites by members of the Brown Snake group, and in particular the Common Brown Snake *(Pseudonaja textilis)* and the Western Brown Snake *(Pseudonaja nuchalis)* are the most common in South Australia. (WHITE, 1982) They are usually associated with minimal or even no local pain, oedema, or erythema. The only local sign is usually one or more faint scratch marks which are often visible only under a magnifying glass at the time of the bite. They become more readily visible over the following hour. Even in the presence of severe defibrination syndrome bleeding from the bite is not seen, although venepuncture sites may ooze persistently. Multiple bites may occur, often in close proximity to each other. Rarely, the site of the bite may be tender to palpation. There are no reports of local tissue destruction.

Tiger Snakes*(Notechis scutatus)* are probably responsible for more snakebites than any other species in Australia (SUTHERLAND, 1982). They have larger fangs and inject significantly more venom than Brown Snakes. Local scratch marks from the fang are often seen and may be multiple and patchy; even in a single bite. There may be prolonged ooze from the bite, even in the absence of a documented defibrination syndrome. Oedema and erythema are usually seen, and an area of superficial tissue necrosis may be seen. This will initially manifest as an area of darkly discoloured skin which will usually contract in size over the subsequent 24 hours or more before final delineation of the actual area of necrosis. This area of necrosis may be quite small, and often heals spontaneously over the following weeks without need for surgical intervention. The bite is usually acutely painful and an area of associated hypo and hyperaesthesia is frequently found if looked for.

The Red Bellied Black Snake *(Pseudechis porphyriacus)*and the congeneric Mulga Snake *(Pseudechis australis)* are both relatively large snakes, especially the Mulga Snake, which delivers more venom on average milking than any other Australian species. However there are relatively few reports of bites from these species, especially the Mulga Snake. Bites by the Red Bellied Black Snake usually are painful, sometimes intensely so, and are associated with extensive oedema and some erythema. Fang marks are usually scratches, and oozing may occur. Oedema may involve the whole bitten limb and may be slow to subside. Small areas of local necrosis or subcuticular haemorrhage are seen, and these may become secondarily infected. One case had a deep vein thrombosis in the bitten arm.

Bites by the Mulga Snake cause similar problems which may be more severe. In a fatal case there was gross oedema and discolouration of the bitten hand and arm, with severe subcutaneous oedema, haemorrhage and infiltration with polymorphs at autopsy (ROWLANDS *et al*, 1969). Another non-fatal case had necrosis of the thumb distal to the bite and this will be discussed further later. Local pain is a prominent feature of Mulga Snake bite, as is extensive local oedema which may involve the whole limb. Paraesthesia associated with the bite site may occur. Some days after the bite the surface layers of skin around the bite peel off.

Death Adders *(Acanthophis antarcticus)* are notresponsible for many bites in Australia, but have a reputation for severe and often fatal envenomation. Death Adder bites are usually associated with very little local oedema or erythema, but considerable local pain. Where significant bites have occurred on fingers, the finger is usually tight rather than grossly oedematous, and there is severe local tenderness and almost total limitation of joint movement due to pain and tissue tightness. This local tenderness, pain, and limitation of joint movement may persist for days or even several months, without evidence of infection. Ooze from the bite site is not seen, and the fang marks are usually scratches.

The Taipan *(Oxyuranus scutellatus)* and Inland Taipan or Small Scaled Snake *(O. microlepidotus)* are potentially the most lethal snakes in the world. They have long fangs and produce large quantities of a very potent venom. The Taipan has longer fangs and produces more venom than the more toxic Inland Taipan. Local reactions to bites by these species are not well documented. There may be local pain with extensive oedema and local subcuticular haemorrhages, although some significant bites are apparently painless and without local tissue reaction. At the other extreme, local tissue necrosis following

Taipan bite has been described (BENN, 1951).

There is little clinical information reported about Copperhead bites, although SUTHERLAND (1982) suggests that at least in Victoria, bites by the Common Copperhead *(Austrelaps superbus)* are often associated with significant local reactions including tissue necrosis, and presumably pain and oedema. Bites by the as yet undescribed species of Copperhead from the Adelaide Hills region in South Australia do not cause significant clinical problems. All confirmed cases so far have had no local pain or oedema, and fang marks are usually slight scratches on even classic punctures.

Bites by the Rough Scaled Snake *(Tropedechis carinatus)* are usually not associated with local problems, although one reported case developed oedema and urticaria (TRINCA *et al*, 1971). There are no reports of local tissue necrosis.

Though not thought to be dangerous to man, the Yellow-faced Whip Snake *(Demansia psammophis)* is responsible for a number of snakebites. Systemic problems are not significant, but extensive local oedema with some pain, usually not severe, is described, and appears to be the most common reaction to bites by this species. The oedema may take a week or more to subside.

It can be seen there are real differences in local reactions to the bites of various Australian elapid species, and when an ELISA is unavailable, this could be used to determine the monovalent antivenom required. Except in those areas where the Taipan and Inland Taipan occur, a bite by a large brown coloured snake will most probably be due to a Brown Snake or Western Brown if no local oedema, or a Mulga Snake if extensive local oedema. The Death Adder is sufficiently distinct in appearance to be readily identified from a description by the victim. Of the other species, most will produce some local reaction, and will respond to monovalent Tiger Snake antivenom which is effective for Tiger Snake, Copperhead, Rough-scaled Snake and Red Bellied Black Snake bites.

Severe local tissue injury in non-fatal cases of snakebite has only been described following Mulga Snake bite and Tiger Snake bite. The worst such injury was in an amateur snake keeper bitten by a large Mulga Snake while putting the snake in a bag. He was bitten just proximal to the base of the right thumb, and applied a tight tourniquet. On arrival at hospital the hand was tense, swollen and blue-white in colour. The tourniquet was released and he was given one ampoule of polyvalent antivenom i.v. The oedema increased, spreading to the chest wall, and 24 hours later the distal phalanx of the thumb was gangrenous. 24 hours later, though the rest of the oedema was subsiding, the rest of the thumb became gangrenous. The thumb was later amputated and attempts at reconstruction were not wholly successful. Another amateur snake keeper bitten by a pet Tiger Snake developed an area of skin necrosis surrounding the bite on his left hand, in the first web space. He had also used a tourniquet which was both far too tight, and left on too long. The area of necrosis subsequently required skin grafting. FROST (1981) has described another case of Tiger Snake bite, to the calf, which resulted in an area of skin necrosis requiring susequent skin grafting. The lymphatic pressure bandage recommended by SUTHERLAND (1979) was used in this case, but was not applied until 45 minutes after the bite and was left on for 12 hours. This last case has prompted discussion in the Australian Medical press about the length of time the lymphatic pressure bandage is left in place.

In all three cases there was immobilization of venom in the bitten limb for a significant period by the first aid measures. This may have allowed more local effect to occur that would have been the case if the venom was able to move normally from the bite site. These local effects could be due to cytotoxic enzymes, and also due to local ischaemia secondary to microemboli caused by prothrombin converters in the venom. This latter mechanism has been suggested by DUNCAN and TIBBALS (1982) as the reason for necrosis in FROST'S (1981) case. Both Mulga Snake and Tiger Snake venoms have coagulant action, as do nearly all Australian elapid venoms (WHITE, 1982). However, some species, such as the Brown Snake, which has potent direct prothrombin convertors, do not have significant local effects at the bite site, even in cases where first aid immobilization has been used. It is possible therefore, that some other venom component

is responsible for local tissue destruction, and that this is present in only some Australian elapid venoms, such as Mulga Snake, Tiger Snake and Red Bellied Black Snake venom. The myotoxic potential of these venoms is interesting in this regard, and further basic research into these venoms may answer the questions here raised.

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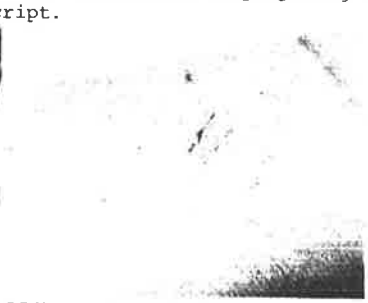
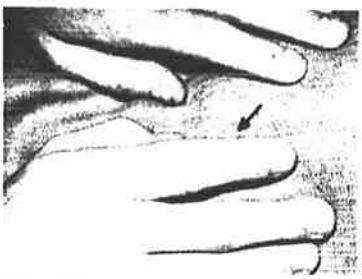
ACKNOWLEDGEMENTS

The author gratefully acknowledges the administrators and medical staff of the Adelaide Children's Hospital, Royal Adelaide Hospital and Flinders Medical Centre for permission to review case records; Brian Fotheringham for reviewing the draft of the paper; Jeremy Frost for detailed information on a case of Tiger Snake bite; the Department of Clinical Photography, A.C.H. for assistance in preparing illustrations; and Maureen Booth for typing the manuscript.

BROWN SNAKE BITE
Single linear fang mark on side of foot. No oedema.

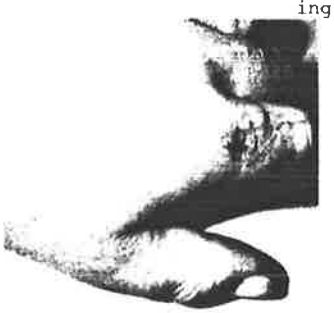


BROWN SNAKE BITE
Multiple linear fang marks on finger. No oedema.

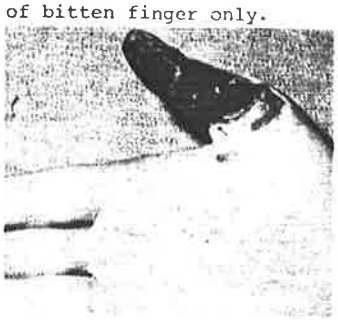


DEATH ADDER BITE
Fang entry on side of index finger. Slight but tense swelling of bitten finger only.

TIGER SNAKE BITE
Single bite in first web space, with local early necrosis and oedema of hand.



RED BELLIED BLACK SNAKE BITE
Multiple bite to index finger. Extensive oedema of hand.



MULGA SNAKE BITE
Necrosis of thumb after bite to thenar eminence.

Printed in Great Britain.

HAEMATOLOGICAL PROBLEMS AND AUSTRALIAN ELAPID ENVENOMATION

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INTRODUCTION

Australian terrestrial snakes include a number of species dangerous to man, all of which are members of the family ELAPIDAE. Unlike the vipers and crotalids, they cannot rotate their maxillae extensively, to fold fangs up, thus fang length is limited, and is much less in Elapids than in Viperids. Associated with this less efficient fang structure, at least in Australian Elapids, is the development of venoms with a predominantly systemic action rather than local action. Thus Australian Elapids have, on average, more potent venoms than the snakes of other continents. BROAD *et al*, (1979) in comparing the relative lethality of 23 species of snakes on the basis of LD50 for mice, have shown that some Australian Elapids have the most potent venoms of all land snakes, some species being more potent even than Sea Snakes. Most research and discussion of these venoms has dwelled on the very potent neurotoxins, contained in probably all of the dangerous Australian Elapid snake venoms. There are both postsynaptic, and numerous presynaptic neurotoxins. Prominent myotoxic activity has also been described for many of these venoms. However, relatively little work has been performed on those venom components with haematological activity. This is despite the importance of haematological problems in the clinical management of snakebite in Australia (WHITE, 1981).

MARTIN, in 1893 first split the effects of components of Australian Elapid venoms, demonstrating the thrombotic action of Red Bellied Black Snake and Tiger Snake venom. DENSON (1969), demonstrated incomplete thrombin activation by Tiger Snake venom, and complete thrombin activation by Taipan and Common Brown Snake venom. Clinically, such activity could cause defibrination, as shown for the Malayan Pit Viper, where the defibrinating dose may be much smaller than the whole venom lethal dose. The clinical problem of defibrination syndrome following snakebite in Australia has been discussed by WHITE (1981), and separately by SUTHERLAND *et al*, (1981), who investigated the coagulant activity of Australian elapid venom in Rhesus monkeys. They demonstrated coagulation disturbance following injection of Tiger Snake, Brown Snake, Taipan, Death Adder, Mulga Snake and Copperhead venom. Small Scaled Snake, Rough Scaled Snake and Red Bellied Black Snake venom showed no evidence of causing a significant coagulation defect.

Haemolytic activity of Australian elapid venoms have been investigated by DOERY and PEARSON (1961). They found that haemolytic activity was most prominent in the venoms of the Black Snake group, namely the Red Bellied Black Snake, the Mulga Snake, and the Papuan Black Snake. Both direct haemolysins and indirect haemolysis by phospholipase A was noted. VAUGHAN *et al*, (1981) have isolated a haemolytic phospholipase A, *Pseudexin* from Red Bellied Black Snake venom, with both direct and indirect haemolytic activity. They suggest it may also have presynaptic neurotoxic activity. TAKASAKI and TAMIYA (1982) have demonstrated a strong direct haemolytic activity of Mulga Snake venom, due to the lysophospholipase and phospholipase A_2 components of the venom.

A number of clinical reports of haematological problems following snakebite in Australia are extant, and have been reviewed by WHITE (1981). A fatality 2 hours after a Brown Snake bite, was described by FOXTON (1914). Coagulation function was apparently not tested in this case, but at autopsy there were haemorrhages in the floor of the lateral ventricles, pons, and medulla, these being the cause of death. SUTHERLAND *et al*, (1981) state that a similar fatality due to subarachnoid haemorrhage has been reported to C.S.L., and ascribe this to a coagulopathy. They also mention massive haematemesis occurring after snakebite. Haematemesis, haemoptysis, and haemoglobinuria have all been described in association with defibrination syndrome, most notably in CHAMPNESS' (1966) series from New Guinea. Other manifestations include scalp haematomas, cutaneous ecchymoses, bleeding from the peridental mucosa, and persistent ooze from venepuncture sites and tracheostomies. Available data suggests that most if not all of these cases of

coagulopathy are a defibrination syndrome, presumably caused by the direct and indirect prothrombin converters in venoms. The defibrination syndrome has been described following bites by the Taipan, Papuan Black Snake, Tiger Snake, and members of the Brown Snake group.

MATERIALS AND METHODS

Cases of snakebite admitted to the Adelaide Children's Hospital (1968-1982), Royal Adelaide Hospital (1970-1982) and Flinders Medical Centre (1976-1980) were reviewed, with special reference to local and systemic signs and symptoms of disturbance of haematologic function. Results of laboratory investigation for all cases were reviewed.

RESULTS

Several snakebites exhibiting coagulopathies were noted. A 2½ year old boy was bitten by an unidentified large snake beneath the buttocks, at least twice. In view of the position of the bite, and closeness to a hospital the parents elected not to institute first aid. At the hospital, about 15 minutes after the bite, the child collapsed and is variously reported as having had a cardiac arrest, respiratory arrest, and grand mal convulsion. Initial medical response to this was to insert an intravenous line, and give i.v. hydrocortisone. By this time he was conscious but drowsy, and irritable. Rationality prevailed and he was given Brown and Tiger Snake antivenom i.v., after blood had been taken for testing. This blood was unclottable. He was transferred to a major hospital where further coagulation studies were carried out, including factor assays. The blood was still unclottable, and there was clear evidence of a defibrination syndrome, with undetectable fibrinogen, greatly elevated fibrin degredation products, and a normal platelet count. Factors II, V, VII and VIII were all reduced. Further antivenom was given. Within 40 minutes there was some improvement in coagulation function, and within 2 hours there was acceptable clotting function, and the child was awake and playing with its parents. No treatment other than antivenom was used in this case, and return to normal coagulation function correlated well with a return to normal conscious state. The child went on to make a complete and uneventful recovery. There were no hard signs of classic neurotoxic envenomation in this case. In retrospective discussion with the parents it seems highly probably that the snake involved was a Common Brown Snake.

A similar clinical picture was noted in a 7½ year old boy bitten by a Brown Snake, confirmed by a positive ELISA test on his serum. He was catching reptiles when bitten by the snake. Thinking he had been bitten by a legless lizard, he instituted no first aid, but collapsed at home 15 minutes later. When first seen medically about 25 minutes after the bite he was pallid, sweaty, irritable and drowsy. Blood taken then showed no evidence of clotting over 30 minutes, and so he was given Brown Snake antivenom i.v. Some 4 hours later he was still drowsy, but there was slow clot formation, and a normal platelet count. 2 hours later there was near normal clotting function and he was awake and alert. No further treatment was instituted and he continued to improve, with no further evidence of defibrination syndrome.

Two cases of envenomation by Brown Snakes in adults developed renal failure and a coagulopathy, suggestive of deseminated intravascular coagulation (D.I.C.) rather than defibrination syndrome, a thrombocytopenia being a useful diagnostic differentiator. The first case, a 35 year old male was bitten by a Common Brown Snake of the Eyre Peninsula subspecies *(Pseudonaja textilis inframacula)* while inebriated on his way home from a funeral. He suffered no initial ill effects, but was later given Tiger Snake antivenom i.v., following which he became nauseous, vomited, and anuric. His acute renal failure necessitated haemodialysis for 8 days. Renal biopsy revealed extensive accumulation of IgM and fibrin in the glomeruli, and tubular necrosis. There was evidence of mild haemolysis and a coagulopathy with a thrombocytopenia of 88,000 and elevated fibrin degredation products, suggestive of D.I.C. These features resolved over the ensuing fortnight. The cause of the renal failure is not clear, but the glomerular pathology was attributed to the coagulopathy. The second case was a 26 year old man bitten by a large Western Brown Snake while changing a tyre. He killed the snake,

instituted no first aid, and went home to sleep. He awoke the following morning with diarrhoea, abdominal pain, vomiting and headache and passed a small quantity of dark urine. He then presented to the local hospital where he was given Polyvalent antivenom, about 15 hours after the bite. He then became anuric and on transfer to Adelaide, was noted to have a coagulopathy with a thrombocytopenia of 44,000 and raised fibrin degredation products. He was given further Polyvalent antivenom, and commenced on heparin There was a further deterioration in renal function and the platelet count dropped to 13,000. Fibrin degredation products further increased, and there was a microangiopathic haemolytic anaemia. There was no evidence of myoglobinuria. Treatment included haemodialysis, heparin, and repeated transfusions of blood. The non-resolution of the coagulopathy after 5 days occasioned the use of specific Brown Snake antivenom, with subsequent resolution of the coagulopathy. Haemodialysis was needed for 12 days. No renal biopsy was performed.

Of those cases of Tiger Snake envenomation in the study, several had evidence of a coagulopathy. A 23 year old reptile keeper bitten by his pet Tiger Snake while inebriated developed the defibrination syndrome with unclottable blood, raised fibrin degradation products, and normal platelet count, and without neurotoxic problems. He was successfully treated with Polyvalent antivenom and heparin, fresh frozen plasma, cryoprecipitate, and fibrinogen. A subsequent Black Snake bite in the same individual was associated with haemolytic anaemia, possible D.I.C. with thrombocytopenia, and a deep vein thrombosis in the bitten arm. Another Tiger Snake bite in a 27 year old woman was associated with neurotoxic envenomation and a transient and poorly documented coagulopathy which was noted after antivenom was administered, but had resolved by the time of transfer to Adelaide from a country hospital. Local manifestations of coagulopathy such as petichial haemorrhage on the bitten arm, and free persistent oozing from the bite site were reported following Brown Snake and Tiger Snake bites, respectively.

DISCUSSION

Several interesting points arise from these cases. For members of the Brown Snake group, a coagulopathy, usually a defibrination syndrome, may occur very rapidly after the bite, and its progress, at least in children, is correlated with conscious state. There may be a significant coagulopathy and impaired conscious state, without significant neurotoxic envenomation. The S.A. cases correlated well with other reported cases of defibrination syndrome following Brown Snake bite by all three major members of the group (WHITE 1981). The blood is unclottable, fibrin degredation products are increased, factors II, V, VII and VIII are all decreased, and platelet count is normal. There is rapid resolution after sufficient appropriate antivenom therapy, and other forms of treatment in these cases will usually not be needed. SUTHERLAND *et al*, (1981) demonstrated in Rhesus monkeys that the defibrination coagulopathy caused by Dugite and Western Brown Snake venom does not spontaneously resolve or respond to antivenom. This is diturbing, and further research into this is needed.

The relationship between the defibrination syndrome and the D.I.C. seen in the two cases with renal failure, is uncertain, as is the role of antivenom. In both cases it seems likely that significant envenomation had occurred, and that this was the cause of both the coagulopathy and renal failure, but the two may not be directly linked. In the second case, the resolution of the coagulopathy after massive doses of the correct specific antivenom is suggestive of a correlation. Thus correct antivenom treatment may be of value in correcting venom induced D.I.C. More detailed studies of any future cases will be needed to confirm this.

SUTHERLAND *et al's*, (1981) finding that the coagulopathy following Tiger Snake envenomation of monkeys is self limiting is neither confirmed nor denied by these cases, but those of FROST AND CAMPBELL suggest that in man the coagulopathy will only resolve after sufficient antivenom is given.

Inadequate studies of cagulation function in S.A. cases of Mulga Snake bite, and other reported cases make it impossible to confirm coagulopathy occurring in man, though it is prominent in monkeys. One S.A. case was said to have a coagulopathy, but details are insufficient for confirmation.

No Copperhead bites seen in Adelaide have been associated with a detected coagulopathy. A similar situation exists for Death Adders, with several reported bites, none with coagulopathy.

Compared with coagulopathy, haemolysis appears to be only a minor problem, and is only rarely detected.

Thus the direct and indirect prothrombin converters appear to be the most important haematologically active components of Australian elapid venoms, and in some cases they may cause more important clinical problems that the neurotoxins. The usual problem is defibrination syndrome, which can occur very rapidly, but usually resolves after administration of adequate appropriate antivenom.

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ACKNOWLEDGEMENTS

The author gratefully acknowledges the administrators and medical staff of the Adelaide Children's Hospital, Royal Adelaide Hospital, and Flinders Medical Centre for permission to review case records; Kevin Cheney and Isobel Speed for helpful comments on drafts of the paper; the Department of Clinical Photography, A.C.H. for assistance with illustrations; Rob Fassett and the Renal Unit, Queen Elizabeth Hospital for clinical details on the second case of renal failure; and Maureen Booth for typing the manuscript.

Acute renal failure and coagulopathy after snakebite

Julian White and Robert Fassett

ABSTRACT: A non-fatal case of acute renal failure with associated coagulopathy after envenomation by a western brown snake (*Pseudonaja nuchalis*) is described. The coagulopathy did not respond to the initial administration of polyvalent antivenom, but resolved rapidly after later infusion of large doses of specific antivenom. The renal failure necessitated haemodialysis for 12 days and then resolved completely. There were no signs of neurotoxic envenomation. Other Australian cases of snakebite associated with renal failure or coagulopathy are reviewed. An adequate dose of the correct antivenom, and infusion of fresh frozen plasma if indicated, is the best treatment of coagulopathy after snakebite.

(Med J Aust 1983; 2: 142-143)

RENAL FAILURE and coagulopathy have both been described as sequelae of snakebite in Australia.[1-8] However, no case of acute renal failure combined with severe coagulopathy has been described after envenomation by an Australian elapid snake. Elapid snakes (family Elapidae: front-fanged venomous snakes) have complex venoms with a variety of actions in humans, but no specific nephrotoxins have so far been isolated.[9,10] The non-fatal case, reported here, illustrates the combination of renal failure and coagulopathy after snakebite, and also some of the diagnostic and therapeutic problems which may be encountered in a serious case of snakebite.

Clinical record

At 6 p.m. on December 7, 1980, a healthy 26-year-old man was returning home from a hotel near Woomera, South Australia, where he had consumed alcohol. While filling a can with water from a tap, he was bitten on the right thumb by a large brown snake. He killed the snake, did not apply first aid, and went home to sleep. At this stage, he had no symptoms from the snakebite. On the following morning, he awoke with diarrhoea, crampy abdominal pain, vomiting, headache, general weakness, and passed a small quantity of dark urine. Fifteen hours after the snakebite, he sought medical attention at Woomera and received one ampoule of CSL polyvalent antivenom intravenously, together with adrenaline, promethazine hydrochloride (Phenergan), and hydrocortisone. He was then transferred to the Queen Elizabeth Hospital, Adelaide.

On arrival in hospital, about 24 hours after the bite, the patient was slightly icteric. On examination, he had no signs of neurotoxic paralysis. His blood pressure was 20/13.3 kPa (150/100 mmHg). The liver was palpable with a span of 14 cm. He passed 5 mL of dark urine which contained red cells, but no casts. Laboratory investigations showed serum levels of sodium, 137 mmol/L; urea, 11.6 mmol/L; potassium, 4.7 mmol/L; and creatinine, 0.48 mmol/L. The haemoglobin concentration was 151 g/L; white cell count, 18.6 × 10^9/L; and platelet count, 44 × 10^9/L. There was a coagulation abnormality with a thrombin clotting time of 12 s; partial thromboplastin time of 45 s; prothrombin time, 60%; fibrinogen level, 1.1 g/L; and fibrin degradation products, 64 μg/mL (normal, less than 8 μg/mL). The patient received two more ampoules of CSL polyvalent antivenom, which were administered intravenously about 36 hours after the bite, and treatment with heparin (750 units intravenously every hour) was commenced.

On the following day, the renal function had further deteriorated (serum creatinine level, 0.6 mmol/L). An arteriovenous shunt was inserted, and the patient received haemodialysis. The disseminated intravascular coagulopathy had intensified; the level of fibrin degradation products increased to 153.6 μg/mL and the platelet count fell to 13 × 10^9/L. There was evidence of microangiopathic haemolytic anaemia — haemoglobin concentration fell to 65 g/L, fragmentation of red cells was present and blood specimens were grossly haemolyzed. The result of Schumm's test was positive, haptoglobin levels were reduced to 0.1 g/L, the serum creatinine phosphokinase level was elevated to 494 u/L, but there was no evidence of myoglobinuria, urinary myoglobin concentration being less than 2 g/L. Multiple blood transfusions were necessary before the administration of CSL brown snake antivenom. Blood transfusions were given during haemodialysis.

Because of the renal failure, the patient continued to require dialysis, and his coagulopathy remained until the fifth day after envenomation, when, after positive identification of the snake as a western brown snake (*Pseudonaja nuchalis*) he received seven ampoules of CSL brown snake antivenom intravenously over 24 hours. The course of the renal failure and coagulopathy is illustrated in the Figure. After the administration of specific antivenom, the coagulopathy was reversed and did not recur. The patient received haemodialysis for 12 days until diuresis occurred, which was followed by rapid improvement in renal function. On discharge from hospital, three weeks after admission, his renal function was normal.

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Discussion

Renal failure after snakebite in Australia has been reported after bites of the dugite *(P. affinis)*,[1] western brown snake *(P. nuchalis)*,[1] tiger snake *(Notechis scutatus)*,[2] and small-eyed snake *(Cryptophis nigrescens)*.[3] In the cases of bites by the latter two species, there was an associated rhabdomyolysis and myoglobinuria. In a fatal case of mulga snake *(Pseudechis australis)* bite, rhabdomyolysis was also present, with congestion of the glomeruli at autopsy.[11]

There was an associated microangiopathic haemolytic anaemia in the cases of envenomation by the dugite and western brown snake, but not in those of envenomation by the tiger snake, small-eyed snake, and mulga snake. None of the patients in these cases developed a coagulopathy.

Coagulopathy is a frequent consequence of snakebite in Australia, particularly after bites by members of the brown snake complex *(P. textilis, P. nuchalis,* and *P. affinis)*,[5,7] and is most often manifested as a defibrination syndrome, with afibrinogenaemia, grossly elevated levels of fibrin degradation products, and a normal platelet count.[10,12] Detailed analysis of the venom components causing the coagulopathy has not been reported, but these venoms are known to be potent prothrombin converters.[10] While platelet counts are normal in the defibrination syndrome after brown snake bite, there is no information about platelet function, which might be impaired (A. H. Reid, personal communication). These coagulopathies respond rapidly to intravenous administration of specific antivenom with a prompt return to normal clotting function in most cases.[10] However, it has been shown that, in monkeys, antivenom may sometimes be ineffective in reversing coagulopathy caused by dugite and western brown snake venoms.[13]

In view of the common occurrence of both coagulopathy and renal failure after envenomation by the western brown snake, the concurrence of these two problems in a patient is not surprising, but the relationship between the two remains unclear. The renal failure in our patient had a clinical course suggestive of acute tubular necrosis, though confirmation by renal biopsy was not possible because of the coagulopathy. The clinical picture also suggested a microangiopathic haemolytic anaemia, and thus bears a close similarity to the previously reported cases of renal failure after envenomation by the western brown snake. The coagulopathy, however, was different to that usually seen after bites by this species, as, in addition to the afibrinogenaemia, there was a marked thrombocytopenia, and there was no response to the administration of antivenom in significant amounts early in the course of management. In view of this early failure of response, the apparently dramatic improvement after the administration of specific antivenom five days later is confusing. It may be that the resolution of the coagulopathy and the administration of specific antivenom were coincidental, but the other possibility, that the polyvalent antivenom contained an insufficient amount of brown snake antivenom, seems more likely. The ineffectiveness of heparin as a treatment for coagulopathy after snakebite is further illustrated by our case.

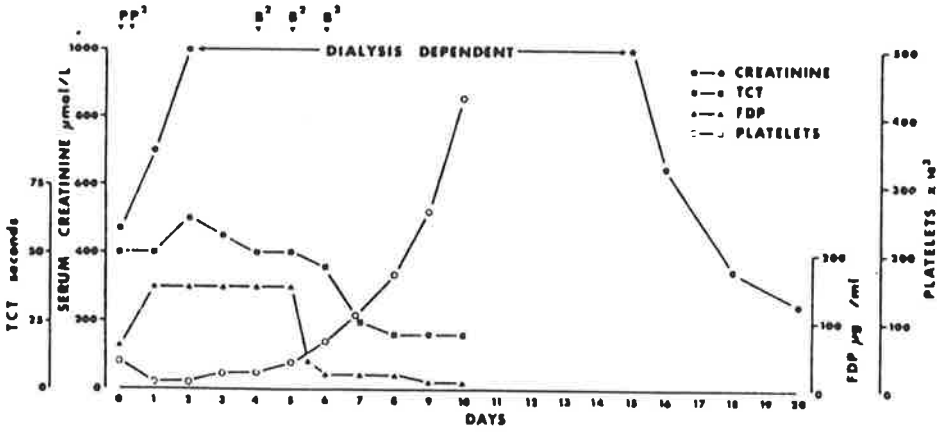

FIGURE 1: The course of acute renal failure and coagulopathy after the snakebite. It was not until an adequate amount of specific CSL brown snake antivenom was given that the coagulopathy began to resolve. P = CSL polyvalent antivenom (1 ampoule); P² = CSL polyvalent antivenom (2 ampoules); B² = CSL brown snake antivenom (2 ampoules); TCT = thrombin clotting time; FDP = fibrin degradation products.

Several important points about management are illustrated by our case. First, neurotoxic problems may not dominate the clinical course of snakebite envenomation, and may even be absent despite other serious venom-induced problems. Second, renal failure after snakebite envenomation is a significant hazard and should always be investigated, even in cases which are initially thought to be trivial. Third, coagulopathy after snakebite envenomation can be severe, and is best managed by adequate doses of specific antivenom and infusion of fresh frozen plasma if indicated. It may never be too late to give the specific antivenom. Last, the necessity to impress upon the general public the need for proper first aid after snakebite is reinforced. Our patient might have avoided a lengthy and costly stay in a renal unit if correct treatment for the snakebite had been instituted at an early stage.

Acknowledgements

We are grateful to Mr S. Narayan of Woomera who referred the patient; to the Clinical Photography Department of the Queen Elizabeth Hospital for preparing the graph; and to Mrs M. Booth for secretarial assistance.

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AUSTRALASIAN MEDICAL PUBLISHING COMPANY LIMITED
71-79 Arundel Street, Glebe, N.S.W. 2037

Records of the Adelaide Children's Hospital, Vol 3, No 2, 1983–84

Tiger Snake Bite

by Julian White,* David Tomkins,† Ian Steven,§ Vaughan Williams**

ABSTRACT

A case of confirmed envenomation by a Tiger Snake (*Notechis scutatus*) in a two-year-old girl is reported. Clinical problems in this case included neurotoxic paralysis, defibrination syndrome, convulsions, rhabdomyolysis and hyponatraemia. Complications from the hyponatraemia, which is not normally a problem after snakebite, included a further convulsion, a severe aspiration pneumonia, and a leukopenia. The cause of the hyponatraemia is uncertain. Specific antivenom stopped the progression of neurotoxic paralysis, and reversed the defibrination syndrome, but did not prevent the subsequent hyponatraemia. The child eventually recovered from this severe envenomation.

INTRODUCTION

A number of cases of Tiger Snake (*Notechis scutatus*) bite have been reported since Australia was first colonized by Europeans. In the period prior to antivenom, approximately 40% of Tiger Snake bites were fatal.[1] Recent reviews of snakebite in Australia,[2] and Tiger Snake bite[3] have identified a number of complications which may follow envenomation. These include neurotoxic paralysis, defibrination syndrome, rhabdomyolysis, and renal failure. Convulsions following snakebite are also reported, but rarely given prominence.[2] The case reported here demonstrated all of the above complications, except renal failure, and in addition a severe hyponatraemia, a complication hitherto unreported, though clinically of considerable importance, as this case illustrates.

CASE REPORT

On 15 September 1982, a two-year-old girl was playing in a sandpit, unsupervised, at her parents' property near Lucindale, in the South-East of South Australia. She came indoors at approximately 1200 hours and was seen by the mother to collapse, and then to have mild convulsions. A diagnosis of heat exhaustion was made and the child prepared for a cool bath. The mother then noted a small area of bruising with puncture marks on the left lateral calf, just below the knee. The diagnosis was revised to snakebite, and a compression bandage placed over the bite, and the child taken to the Naracoorte Hospital. The father searched the sandpit for a snake, and an 84 cm Tiger Snake (later positively identified in Adelaide) was found and killed.

On arrival at Naracoorte Hospital, the child was drowsy, but with stable vital signs, and no paralysis was noted. Blood was taken for venom testing and coagulation profile, and then at 1420 hours 3,000 units (one ampoule) of specific Tiger Snake antivenom was administered intravenously, slowly, diluted in Hartmann's Solution, and preceded by subcutaneous adrenaline and oral diazepam. The child showed initial improvement in conscious state. The coagulation profile showed gross prolongation of clotting time (Table I) and an ELISA test

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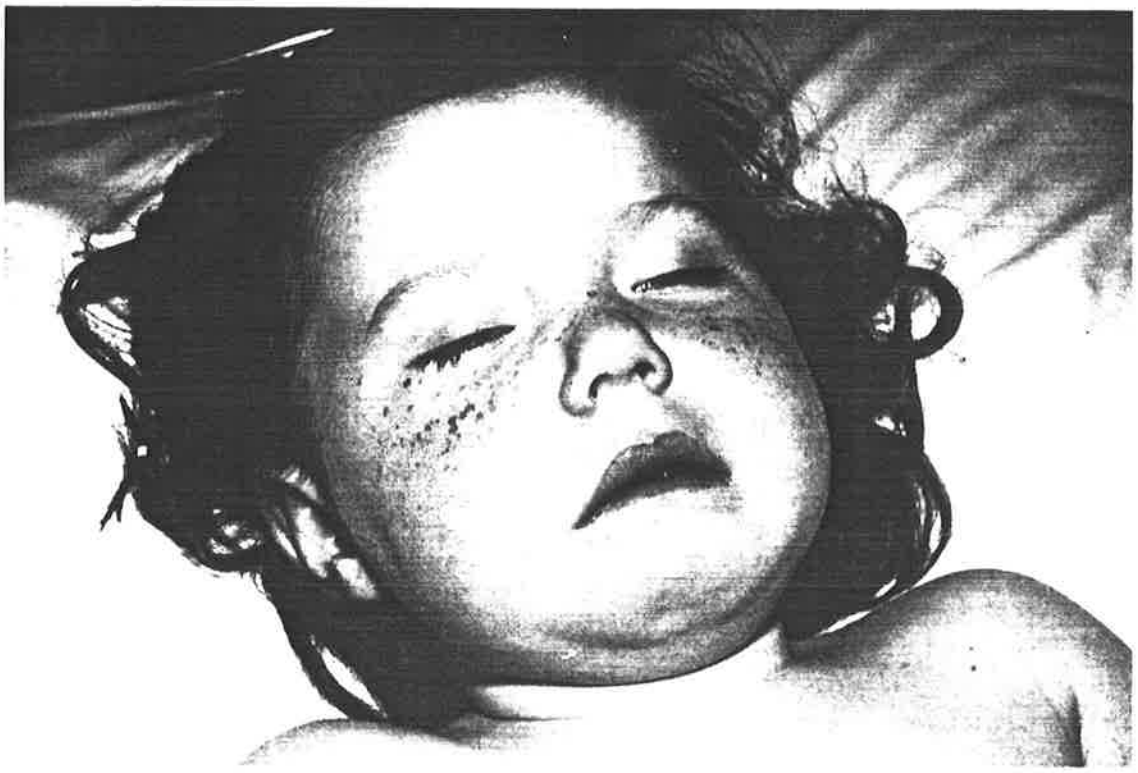

FIG. 1: Child's face approximately 8 hours after Tiger Snake bite, showing bilateral ptosis and lack of facial expression, indicative of neurotoxic paralysis.

FIG. 2: Child's left lateral calf, with double bite, and associated subcuticular haemorrhage and bruising.

confirmed Tiger Snake venom in the serum. A retrieval to Adelaide was requested, but while waiting, the child became more drowsy, and a bilateral ptosis was noted. A further 3,000 units of Tiger Snake antivenom was therefore given, at 1720 hours.

The retrieval team arrived at approximately 1820 hours, at which time the child was drowsy, irritable, but rousable, and would respond verbally to her parents. No dysarthria was noted. There was a marked bilateral ptosis, pupils were fixed dilated, and there was a lack of facial expression (Fig. 1). Examination of the bite site revealed an area of subcuticular haemorrhage and bruising in association with two distinct, but closely approximated, bites (Fig. 2). It was concluded that the child had been bitten twice. There had been no progression of signs since the second dose of antivenom, so further antivenom was withheld, blood was taken for coagulation profile, the drip changed to 4% dextrose in one-fifth normal saline, and the child transferred to the Adelaide Children's Hospital.

The child's condition remained stable, with no progression of paralysis or deterioration in conscious state. Further coagulation profiles, and electrolyte studies were performed in Adelaide, at 2230 hours. The coagulation profiles showed an initial picture of incoagulable blood in vitro with raised fibrin degradation products, and decrease in factors I, II, V and VIII. This picture is typical of defibrination syndrome sometimes seen after snakebite.[2] The blood sample of 2230 hours, some five hours after the second dose of antivenom, showed a substantial change towards normal clotting function, and was taken as an indicator, together with the clinical picture, that all circulating venom had been neutralized, and that further antivenom was unnecessary. The changing coagulation profile is shown in Table I.

Electrolytes at 2230 hours showed a mild hyponatraemia (Na = 130 mmol/l) and intravenous therapy was changed to half-normal saline. A creatine phosphokinase level at this time was 5225 U/l (normal < 110), but renal function was normal. The child was observed overnight and appeared stable. At 0620 hours on the following day, $18\frac{1}{2}$ hours after the bite, the child had a prolonged grand mal convulsion, with vomiting and aspiration of vomitus. There was no prior indication of change in her state, and the convulsion was an unpleasant surprise to those on duty. An airway was established, but it was clear she had aspirated vomitus, and a severe aspiration pneumonia developed, causing significant problems in maintaining adequate oxygenation over the subsequent few hours, and

TABLE I Coagulation parameters after Tiger Snake bite

	Hours after bite				
	2.5	*5.5*	*7.2*	*10.6*	*19.2*
Antivenom Administration	1 ampoule	1 ampoule			
Clotting time (N < 10 mins)	No clot		No clot	7 mins	$6\frac{1}{2}$ mins
Prothrombin time (NR = 67%–100%)	5%		5%	38%	56%
Activated partial thromboplastin time (N = 31–43 sec)	6 mins		5 mins	70.5 sec	42.5 sec
Factor I (Fibrinogen) (N = 1.80–4.0 g/L)	Undetectable		Undetectable	0.74	0.96
Fibrinogen Degradation products (N < 10 mg/L)			5000	1600	400
Factor II (Prothrombin) (N = 50–250%)			37%	65%	75%
Factor V (N = 50–250%)			6%	32%	70%
Factor VIII (N = 50–250%)			1%	50%	64%
Platelets				288,000	390,000

necessitating intubation and assisted ventilation. The girl also showed some decerebrate rigidity, and the possibility of a cerebral haemorrhage was considered. The coagulation profile was repeated, but showed a return to virtually normal, with good clot formation, and no evidence of a continuing defibrination syndrome. Urine was tested for Tiger Snake venom, but was negative, although urine the previous day was strongly positive. A C.T. scan of the brain showed some evidence of cerebral oedema, but no evidence of haemorrhage. Serum electrolytes showed a hyponatraemia (Na = 123mmol/l) but no other significant abnormality except a creatine phosphokinase of 6426 U/l. Renal function was normal.

On this evidence, it was concluded that the convulsion was not due to continuing envenomation, and antivenom was withheld. The intravenous drip was changed to normal saline to correct the hyponatraemia, and antibiotic therapy instituted for the aspiration pneumonia. The child made a slow recovery initially. The change in serum sodium concentration is shown in Figure 3.

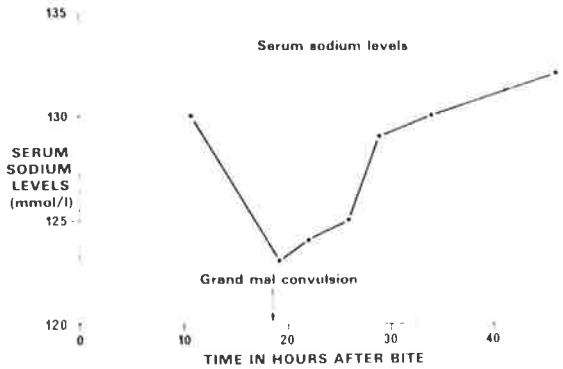

FIG. 3: Graph of serum sodium concentration, against time after Tiger Snake bite.

A repeat blood picture at 1400 hours, some $7\frac{1}{2}$ hours after the convulsion, revealed a leukopenia (W.C.C. = 1,300) and an anaemia (Hb. = 8.9 g%), but with a normal platelet count (213,000) and a continued improvement in the coagulation profile. (There had been no leukopenia immediately following the convulsion, the W.C.C. being 7,300).

Despite these numerous problems, clinically the child continued to improve. There was good resolution of the pneumonia over 48 hours. However, although mentally alert, she continued to have a bilateral ptosis and pupillary dilatation; this was not progressive but had not significantly resolved at discharge on 23 September 1982. Moreover, although she was playing happily at this time she remained unsteady in her gait, and had some general muscular weakness. The leukopenia resolved over 24 hours.

A subsequent progress report from her parents on 8 November 1982, nearly two months after the bite, revealed that the child was again behaving normally, but still appeared slightly weak in the legs, and the pupils remained dilated and only slightly reactive to light. The ptosis had resolved. The area at the bite site remained indurated, but no necrosis developed.

DISCUSSION

This case illustrates the rapidity with which snake venom may act, and the multitude of problems it may cause. A double bite is usually associated with significant envenomation, as in this case, and will usually require more than the standard amount of antivenom.

The rapid onset of convulsions in a child after snakebite has been observed before, as has the rapid onset of defibrination.[2] The significant extent of rhabdomyolysis is understandable in view of the myotoxic action of notexin, the presynaptic neurotoxin in Tiger Snake venom. The neurotoxic paralysis in this case is classic, with arrest of progression after the circulating venom was neutralized. As the neurotoxin responsible, notexin, is presynaptic in action, it can be expected that any paralysis established before antivenom is given, will not be reversed, and this is clearly illustrated in this case. The rapid response of the coagulation disturbance to neutralization of venom by antivenom has been reported before, and this may be a useful adjunct in titrating the antivenom requirement.[2] If adequate antivenom is given, then heparin, factor replacement, and similar therapies will usually be unnecessary.[4]

The cause of the second convulsion $18\frac{1}{2}$ hours after the bite is uncertain. It is possible that the hyponatraemia was responsible, but some other factor, undetected, may also have been responsible. The cause of the

hyponatraemia is also unknown, and such a problem has not previously been reported following snakebite in Australia, at least as far as the authors can ascertain. The evidence available suggests that all the circulating venom was adequately neutralized hours before the convulsion, and so a direct venom effect as cause seems very unlikely.

Although the reasons for the hyponatraemia and the convulsion remain unknown, these problems nearly resulted in the child's death. Had the child not been in an intensive care unit, with facilities rapidly available, then she would probably have succumbed. The lack of warning of impending disaster is worrying, and we will pay close attention to electrolyte imbalance in future cases of significant snakebite. We recommend that out colleagues do likewise.

ACKNOWLEDGEMENTS

The authors wish to thank their colleagues in the Adelaide Children's Hospital who assisted in managing this case, and the Photography Department for the illustrations. The authors are grateful to Dr. Vithianathan and his colleagues in Naracoorte for the information supplied about the early history of this case, and the excellent care they gave the patient. The authors thank the child's parents, for further information supplied, and permission to publish their child's photograph. Ms. Pauline Couch typed the manuscript.

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As it happens, I really do dislike the phrase "public image", not just because of its ugliness but because I'm convinced that what people call a good public image had something in common with other abstractions like dignity, a sense of humour, or sex appeal. If you find yourself worrying over whether you've got them you've probably got something to worry about. These surely are accolades that other people will award you if you are lucky. To try to award them to yourself is to court disaster.

Michael O'Donnell (1984):
One Man's Burden.
British Medical Journal
Vol. 288, page 652.

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Beyond Lanchi the road leaves the river-valley and turns off into the hills. Soon we were hurtling round the curves of a mountain pass. The scenery was superb, but we were too frightened even to look out of the window. Instead, Auden tried to distract our thoughts from the alarming Present by starting a conversation about eighteenth-century poetry. It was no good: we could remember nothing but verses on sudden death. Meanwhile, the road twisted and struggled, and the car clung to it like a mongoose attacking a cobra. Pedestrians screamed, cyclists overbalanced into paddy-fields, wrecked hens lay twitching spasmodically in the dust-storm behind us. At every corner we shut our eyes, but the chauffeur only laughed darkly as befitted one of the Lords of Death, and swung us round the curve with squealing brakes. Neither Major Yang nor Mr. Liu showed the least symptoms of nervousness. 'The road is very difficult,' Mr. Liu observed peacefully, as we shot across a crazy makeshift bridge over a gorge, rattling its loose planks like the bars of a xylophone. 'It wouldn't be difficult', I retorted, 'if we weren't driving at seventy miles an hour.'

W. H. Auden and Christopher Isherwood
(1939):
Journey to a War,
(Revised Edition 1973) Faber & Faber, London,
page 189.

The American Journal of Forensic Medicine and Pathology
Volume 5 Number 2
June 1984

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Fatal snakebite in Australia

ABSTRACT More than 20 venomous snakes found in Australia belong to the family Elapidae. Although their venom delivery system is less efficient than that of the Viperidae, their venoms are extremely potent. The active components of Australian elapid venoms include neurotoxins, myotoxins, hemolysins, and factors producing hypocoagulability. The groups at particular risk of snakebite are children, agricultural workers, bushwalkers, and herpetologists. A high index of suspicion must be maintained when investigating cases of sudden unexpected death in these groups. The bite wound itself may be particularly easily overlooked as there is little local reaction to many elapid bites. The necropsy findings in fatalities are predictable from the known properties of the venoms of the various species. However, these findings are nonspecific and the diagnosis ultimately rests upon the demonstration of the presence of venom by immunoassay. For this purpose swabs from the bite site, the overlying clothing, the excised bite site, the local and regional lymph nodes, urine, and blood may be used.

At least 20 Australian snakes, representing about 10% of Australian snake species, are highly venomous and regarded as potentially dangerous to man.[1] These venomous snakes are among some of the world's most dangerous; before the introduction of antivenom the fatality rate for bites was 50% for death adder, 45% for tiger snakes, and probably 100% for the Taipans.[2] Despite the fact that these venomous snakes are abundant, both in numbers and in geographic spread, few Australians die of snake bite and improved treatment with increased public awareness is likely to further reduce the number of deaths (Table 1). Nevertheless, an appreciation of the problems of diagnosing fatal envenomation at necropsy is of importance because, in the absence of a history of snakebite, the diagnosis is easily overlooked. Furthermore, it is hoped that reviewing the pertinent features of the Australian experience will be of value to both Australian pathologists and pathologists in those countries where fatal snakebite is more common.

TABLE 1.

Reported Deaths from Snakebite in the Australian States and Territories

State	1910–1926[a][2]	1945–1949[3]	1952–1961[4]
Queensland	74	18	18
New South Wales	57	6	9
Western Australia	6	3	6
Tasmania	8	1	—
South Australia	8	—	1
Victoria	43	—	9
Northern Territory	1	—	1
Australian Capital Territory	1	—	1

[a] Calculated on the assumption that 80% of deaths from venomous bites and stings were due to snakebite.

From the Adelaide Children's Hospital (JW), and the Institute of Medical and Veterinary Science (DJP), Adelaide, South Australia.

Snakes (Serpentes) are a suborder of reptiles, and the more than 3,000 species of snakes found worldwide are grouped into 11 families.[5] The family Viperidae includes vipers, adders, pit vipers, and rattlesnakes, and all members of this family are venomous and have a highly efficient apparatus for venom delivery. While the Viperidae account for almost half of all snakebite fatalities worldwide, they are not represented on the Australian continent (except in zoos and reptile parks). The important Australian venomous snakes (Table 2) belong to the family Elapidae which also includes the cobras, kraits, mambas, and coral snakes. All Elapidae are venomous, but they do not have as large fangs nor as effective a venom delivery mechanism as the Viperidae. As a consequence, not all Elapidae are dangerous to man, although worldwide the Elapidae family appear to be the largest single cause of snakebite fatality.[3] Sea snakes (family hydrophiidae) are also venomous and have fangs similar to the Elapidae. Common in the tropical waters of Australia, where they are represented by at least 30 different species, they are potentially dangerous to man, but only bite if handled. These snakes which might bite swimmers, divers, and persons in the fishing industry will not be discussed here.

TABLE 2.

Activities of Australian Elapid Snake Venoms

Snakes[a]	Venom Group[9]	LD50 for Mice in mg/kg	Local Tisue Damage[b]	Neuro-toxic Effects	Hemo-lytic Effects	Coagu-lopathy	Renal Failure	Myo-toxic Effects
Inland Taipan: *Oxyuranus microlepidotus*	Taipan	0.025	+	+	+	+	+?	+?
Taipan: *Oxyuranus scutellatus*	Taipan	0.099						
Eastern brown snake: *Pseudonaja textilis*	Brown	0.053						
Western brown snake: *Pseudonaja nuchalis*	Brown	0.473	−	+	+	+	+	−
Dugite: *Pseudonaja affinis*	Brown	0.660						
Spotted brown snake: *Pseudonaja guttata*	Brown	0.36						
Common tiger snake: *Notechis scutatus*	Tiger	0.118						
Black tiger snake: *Notechis ater*	Tiger	0.131–0.338	+	+	+	+	+	+
Copperheads: *Austrelaps superbus* group	Tiger	0.560	?	+	+	+?	−	+
Rough-scaled snake: *Tropedechis carinatus*	Tiger	1.36	+	+	+	+?	+?	+
Red-bellied black snake: *Pseudechis porphyriacus*	Tiger	2.52	+	+ (minor)	+	+	−	+
Mulga snake: *Pseudechis australis*	Mulga	2.38						
Spotted black snake: *Pseudechis guttatus*	Mulga/Tiger	2.13	+	+	+	+	+	+
Colletts snake: *Pseudechis colletti*	Mulga/Tiger	2.38						
Common death adder: *Acanthophis antarcticus*	Death adder	0.400						
Desert death adder: *Acanthophis pyrrhus*	Death adder	—	−	+	+	−	−	−
Stephens-banded snake: *Hoplocephalus stephensii*	? Tiger	1.36						
Broad-headed snake: *Hoplocephalus bungaroides*	? Tiger	—	?	+	?	?	?	?
Pale-headed snake: *Hoplocephalus bitorquatus*	? Tiger	—						
Small-eyed snake: *Cryptophis nigrescens*	? Tiger	2.67	?	?	?	+?	+	+

[a] The taxonomy of Australian elapid snakes is still in a state of flux. Several of the groups listed in this table are either being subdivided or elated new species, recently discovered, are being added. The copperheads (*Austrelaps*) will be subdivided into at least three species. There are new species of brown snake (*Pseudonaja*), mulga snake (*Pseudechis*), and death adder (*Acanthophis*), either described, or in the process of description. No details of their venoms or effects on man are published, hence they have not been included in this table.

[b] Local tissue damage means a tissue reaction, other than actual fang puncture or scratch marks, visible to the naked eye at the site of the bite.

Key: + = activity present either *in vitro* or *in vivo;* − = no activity known or insignificant activity; +? = activity probably present, but not confirmed; ? = no information available.

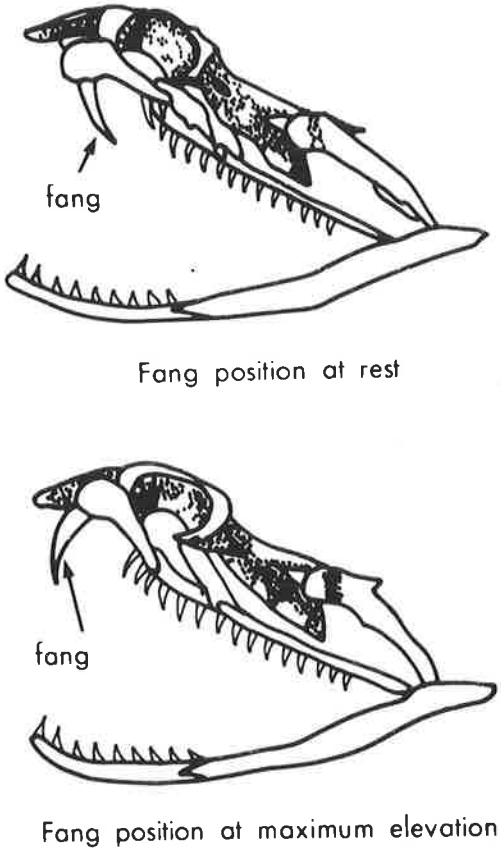

FIGURE 1
Rotation of the maxilla and attached fang in the death adder.

An understanding of the basic anatomy of the venom delivery system of the Australian Elapidae is essential to an appreciation of the mechanism by which the wound is inflicted. In these snakes, the venom gland is situated below and behind each eye, the gland duct passing forward beneath the eye. The venom duct leads into a channel in the fang which in turn opens to the exterior near the fang tip; an arrangement somewhat similar to the old quill pen. The fangs are paired and the snake is able to elevate them and rotate them forward (Fig. 1), although the degree of rotation that can be achieved is much less than that of the Viperidae, such as the rattle snake. When the elapid bites, the fangs enter the victim in the position of maximal elevation and pass into the tissues in a downward and backward direction.[7] The appearance of the resultant wound is extremely variable, ranging from a single fang puncture wound to multiple punctures and abrasions following repeated biting. It is probably the backward angling of the fang in the mouth of the snake that accounts for the thin scratch marks frequently seen on the skin of Australian snakebite victims (Fig. 2).

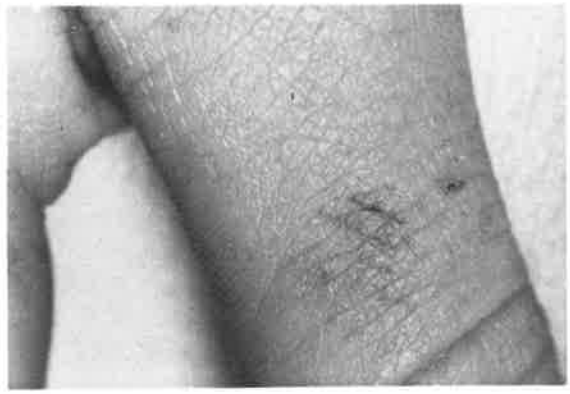

FIGURE 2
Thumb of a semiconscious boy 30 minutes after a bite from a brown snake (*Pseudonaja sp.*). He developed a severe coagulopathy.

Venom injection by the snake is under voluntary control in that the snake may inject none or most of its venom, unilaterally or bilaterally. Consequently, snakebite is not synonymous with envenomation. Furthermore, the degree of envenomation cannot be assessed by the bite for, while multiple bites usually indicate clinically significant envenomation, a victim may have only a minor scratch associated with the innoculation of a lethal dose of venom. The clinician, cognizant of the site of the bite, may easily overlook the marks and it is to be expected that the problem will be compounded at necropsy, especially if there is no history of snakebite available. The diagnostic problem is further compounded by the comparative lack of local reaction to many elapid bites.[5] This is particularly true of bites by brown snakes, and also some copperheads and death adders. A local reaction which may include some edema, erythema, and occasionally a small area of tissue necrosis, is seen after bites by tiger snakes (Figs. 3 and 4), mulga snakes (Fig. 5), and red-bellied black snakes. Thus, significant local tissue necrosis at the site of the snakebite is unusual in Australia and stands in marked contrast to the clinical picture seen in some elapid and viperid bites in other continents. However, one fatal case of mulga snakebite[8] was associated with gross edema and discoloration of the bitten hand and arm reflecting severe subcutaneous edema, hemorrhage, and infiltration with neutrophils. A second nonfatal mulga snakebite resulted in necrosis of the bitten thumb, requiring subsequent amputation.[5]

Australian elapid venoms contain a mixture of active components producing a wide variety of clinical effects, the pathology of which is now beginning to be unraveled. The most deadly compo-

FIGURE 3
Base of the index finger of a 24-year-old male 60 minutes after a bite from a tiger snake (*Notechis scutatus*). Note the hemorrhage and evolving necrosis which subsequently needed skin grafting.

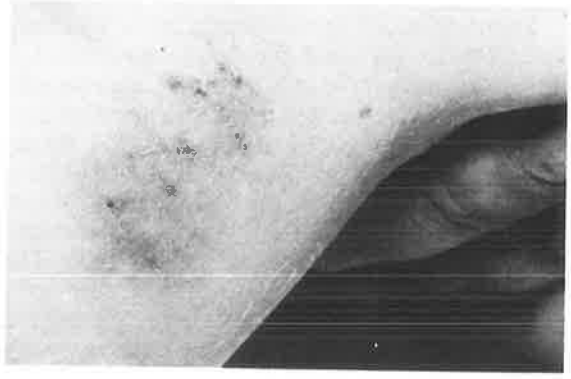

FIGURE 4
Leg of a 2-year-old girl who developed neuromuscular paralysis, coagulopathy, and rhabdomyolysis following two consecutive bites by a tiger snake (*Notechis scutatus*).

nents of the venoms are the neurotoxins, and the presynaptic neurotoxins of Australian elapids represent the most toxic substances isolated from any snake venom to date.[9] These presynaptic neurotoxins produce a progressive neuromuscular paralysis; the postsynaptic neurotoxins also present in these venoms produce a curare like neuromuscular block. Untreated, the neurotoxins will cause death as a consequence of paralysis of the muscles of respiration. Some of these neurotoxins may also cause myolysis. Evidence of myotoxins producing myolysis and myoglobinuria was found in fatalities from a mulga snake[8] and a small-eyed snake.[10] In the former, autopsy disclosed a rhabdomyolysis similar to that seen in paroxysmal myoglobinuria, most prominently in the bitten arm, respiratory muscles, and extraocular muscles. In the latter case, the myoglobinuria was associated with acute renal failure. It would seem that the acute renal failure

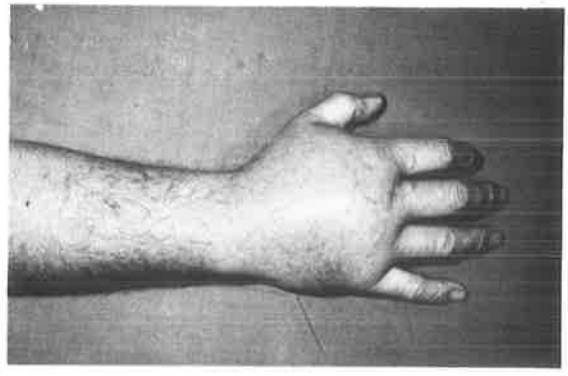

FIGURE 5
Residual edema of the hand of a 48-year-old male 3 days after a bite from a mulga snake (*Pseudechis australis*).

seen following Australian snakebite is most commonly represented pathologically by acute tubular necrosis secondary to myoglobinuria and shock.[8,11]

Hemolysis, a reported effect of several Australian venoms, is not a significant contributor to mortality even when associated with hemoglobinuria. Hematological interest is centered on the coagulopathy which is most commonly seen after bites by members of the brown snake group, and tiger snake group. Typical clinical laboratory findings include hypocoagulability of the blood, afibrinogenemia, elevated fibrin degredation products, and deficiencies of factors 2, 5, and 8, with a normal platelet count. The development of this coagulopathy may be evidenced by oozing from the bite wound or less commonly by hematemesis, cutaneous ecchymoses, or subcutaneous hematoma.[12] Catastrophic hemorrhage appears to be rare although fatal cerebral hemorrhage in association with snakebite has been reported.[13,14].

No part of Australia is free of venomous snakes, and while anyone in the community may be bitten three groups are at greatest risk[5]: 1) children as a consequence of their curiosity. There are two peaks of incidence among children,[15] the first peak in the toddler age group (1–3 years old) and the second peak in the 10–12-year-olds; 2) farm workers who have greater exposure to areas frequented by snakes; and 3) amateur and professional reptile keepers who handle venomous snakes. Bites are common in this group and may represent a significant percentage of snakebite admissions to major hospitals. It is not uncommon for amateur snake handlers to be bitten while under the influence of alcohol.[5] At necropsy there may or may not be a history of snakebite available. Since an adult poisoned by a snake is unlikely to become seriously ill immediately, and

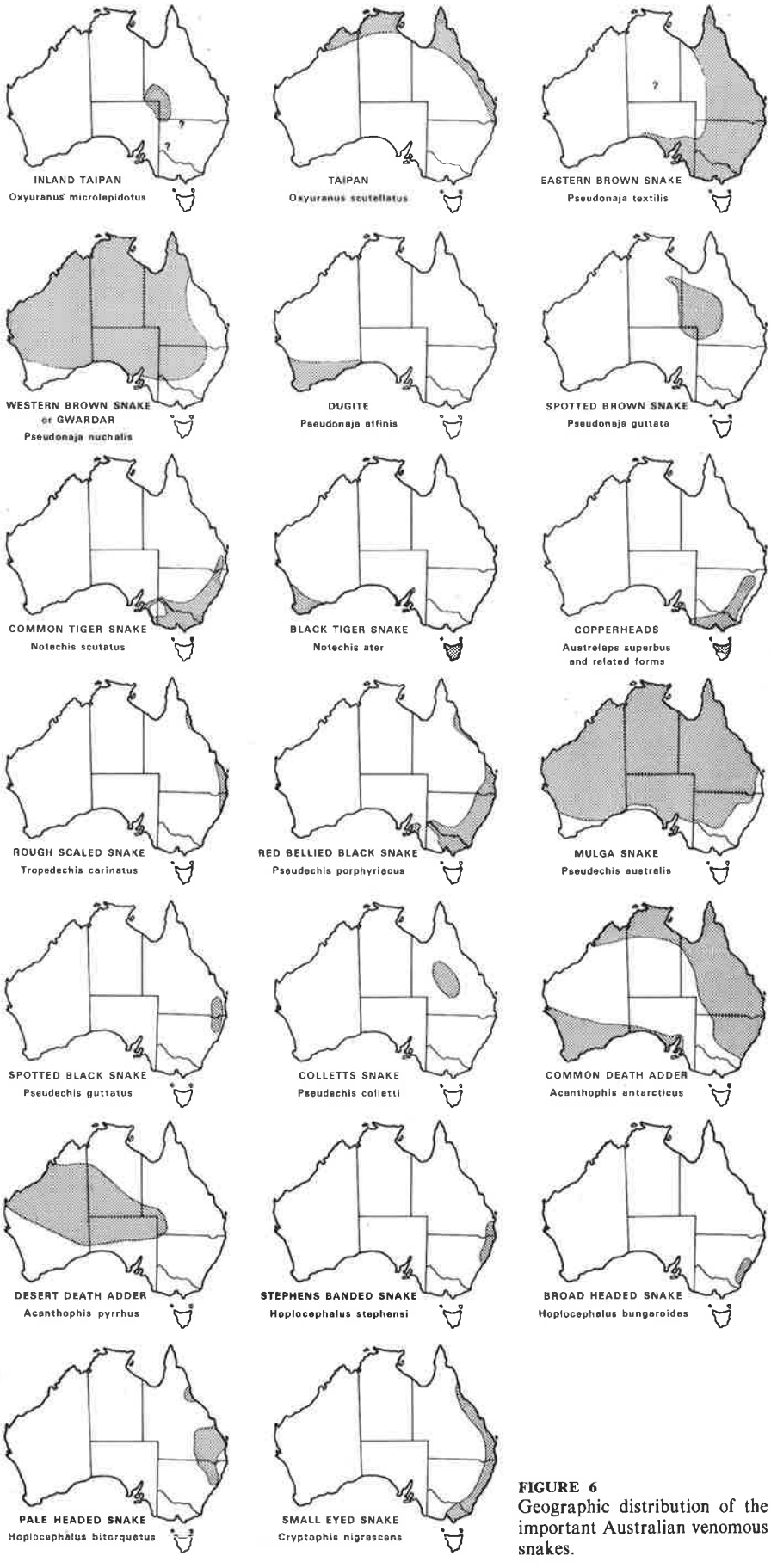

FIGURE 6
Geographic distribution of the important Australian venomous snakes.

unlikely to die within the first few hours,[2] aid has usually been sought and a history is available. However, it is not uncommon in significant cases of envenomation, especially in children, for loss of consciousness to occur before any other symptoms are reported to an observer. In particular, children may be bitten and ignore the bite because of its relative painlessness, or be unaware of having been bitten, and then gradually lapse into unconciousness from 15 minutes to an hour or more later.[5] Similarly, bush walkers passing through long and prickly vegetation may be bitten without realizing it. In the absence of a history in these cases it is likely that the diagnosis will be missed at necropsy unless a high index of suspicion is maintained. Envenomation should be considered in the differential diagnosis of all sudden unexpected deaths in children, agricultural workers, bushwalkers, and herpetologists. Failure to identify a bite wound should not discourage the pathologist from pursuing his suspicion (*vide supra*).

The possible pathological findings in snakebite are predictable from a knowledge of the toxins.[6] The combination of findings seen will vary in differnt parts of Australia according to the geographic distribution of the species of venomous snakes (Fig. 6) and the relative activities of their venoms (Table 2). The neurotoxins which cause death by respiratory paralysis will leave no anatomical markers. The coagulopathy may be evidenced by wound oozing, hemorrhage into viscera (particularly the gut), and fluid blood at necropsy.[16] Hemolysis may lead to hemolytic staining of the aorta and cardiac chambers, although this is often seen as a consequence of early putrefaction during the hot Australian summer. Unusually dark urine should arouse suspicion of hemoglobinuria and/or myoglobinuria. Myotoxic effects would remain undetected in a routine necropsy in which skeletal muscle biopsies were not taken. The kidneys are likely to be congested and edematous with histological evidence of acute tubular necrosis and hemoglobinuria. The spectrum of pathological findings might easily be interpreted as evidence of heat stroke,[17,18] although histological evidence of disseminated intravascular coagulation is expected in heat stroke, but is less likely in elapid envenomation.

Overall, the pathological findings in envenomation are nonspecific and the diagnosis ultimately rests upon the demonstration of the presence of venom. The development by the Commonwealth Serum Laboratory (Melbourne, Australia) of assays for the detection and differentiation of major Australian snake venoms have allowed a positive necropsy diagnosis to be made.[19] The initial radioimmunoassay has been superceded by an enzyme-linked immunosorbent assay which is cheaper, simpler, and more sensitive. Swabs from the bite site (if it can be identified) and from clothing over the bite site, provide the best source of material for assay,[22] but blood and urine should also be retained for this purpose. Urine is more likely to give a positive result than blood for two reasons; first, venom is excreted and concentrated in the urine and second, some components in serum occasionally interfere with the assay. Any suspected bite site should be photographed and then excised with the associated subcutaneous tissues for venom assay. Since the spread of venom occurs principally by the lymphatic system,[19,23] the local and regional lymph nodes are also extremely useful for venom assay and together with the other materials may be stored frozen.[22] In bodies developing putrefactive changes, the vitreous humour of the eye may offer a useful source of material for assay. In addition to the usual tissues retained for histological study, skeletal muscle samples should include the muscles of respiration, extraocular muscles, and muscles adjacent to the suspected bite site.[8] The urine should be tested for hemoglobinuria and myoglobinuria. Finally, the case report should be published as there is a sparsity of well-documented cases of fatal Australian elapid envenomation. □

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Acknowledgments

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OPHIDIAN ENVENOMATION IN AUSTRALIA:
Problems in the autopsy diagnosis

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SUMMARY

Snakebite fatalities are infrequently recorded from Australia despite the abundance of highly venomous elapid and hydrophiid snakes found in this continent. It seems likely that Ophidian envenomation is underdiagnosed and that some otherwise unexplained deaths may be due to snakebite. In Australia a snakebite may be easily overlooked because the wound is often inconspicuous with no local tissue necrosis or haemorrhage. The local tissue effects of Australian snake venoms are minor when compared with many non-Australian snake venoms. By contrast the systemic effects of the venoms are diverse and potent. The documented effects include neurotoxicity, myotoxicity, defibrination, haemolysis and nephrotoxicity. The pattern of pathological changes produced may arouse suspicion of snakebite and provide an indication of the species responsible. The demonstration of the presence of venom by an enzyme immunoassay of samples from the bite site, local lymph nodes and urine should allow a definitive autopsy diagnosis.

KEY WORDS:

Snake bite

Australia

Poisoning; snake venom

When compared with some other regions of the world death from snakebite is an uncommon event in Australia. Most recently published statistics suggest an average of 4 to 5 deaths per annum from snakebite, out of several thousands bitten annually. (1) By contrast, Theakston has calculated that there are at least 23,000 annual deaths due to snakebite in the savana regions of West Africa alone. (2) In South-East Asia, Swaroop and Grab calculated that in Thailand the rate of deaths from snakebite was 1.3 per 100,000, and in Burma 15.4 per 100,000 with some provinces being as high as 36.8 per 100,000, compared to Australian figures compiled at the same time, of 0.07 per 100,000. (3) However, Sutherland has suggested that some otherwise unexplained deaths in Australia may be due to snakebite. New investigative techniques to demonstrate the presence of venom at autopsy have only become available recently (4) and it remains to be seen whether otherwise unsuspected cases will be unmasked.

The low snakebite fatality rate recorded in Australia seems all the more remarkable in view of the fact that Australia has some of the world's most toxic snakes. (5) All of Australia's potentially dangerous venomous snakes are frontfanged and belong to the families Elapidae (land snakes) and Hydrophiidae (Seasnakes).

In contrast to the lethal snakes of other regions, the predominant actions of their venoms in man are systemic rather than local. (6) It is this absence of a reaction around the bite site which makes the wound difficult to identify and the diagnosis likely to be missed at autopsy.

The venom is produced in a modified parotid gland situated behind the eye, and ensheathed in muscle. The gland is connected to the hollow, syringe like fang, by a duct and the snake appears to have voluntary control over venom flow. Consequently a bite may result in anything from no or minimal venom innoculation (the most common situation in man) to devastating envenomation, which is fortunately rare. The fangs themselves are usually small and partially mobile. The combined action of strike and fang motion tends to produce a variety of marks at the site of the bite ranging from classic puncture wounds, to faint scratches. (7)

The extent of local bite marks and reaction is not a good indication of the quantity of venom innoculated where there is a single bite, but where multiple bites have occurred, then significant venom innoculation is very likely. Some or most of the venom may be left on the skin surface, where it is absorbed onto clothing or bandages used as first aid. (8)

Once the venom is innoculated, it will move centrally, mostly via the lymphatics, with concentration in lymph glands being

reported. (4) Having entered the bloodstream, the venom rapidly reaches its various sites of action. Excretion of venom appears to be rapid, with high concentrations appearing in the urine where it is quite stable. Enroute some venom-substrate complexes may deposit in the kidney.

All the venoms are complex mixtures of toxins with both highly monospecific and polyspecific actions. (6) The actions of venom of Australian Elapid snakes varies from species to species, with intra species variation also possible. However, some basic effects are common to most if not all species.

The most studied components are the neurotoxins, which are complex phospholipase A based substances. They may be classified into two groups. The most toxic are the presynaptic neurotoxins such as Notexin from the Tiger Snake, and Textilotoxin from the Brown Snake, this latter toxin being the most lethal snake venom component currently known to man. (9) The presynaptic neurotoxins cause irreversible neuromuscular paralysis, and electron microscopic examination has demonstrated that the synaptic vesicles are reduced in number or morphologically abnormal. Once they have entered the nerve terminal they are unaffected by antivenom. Fatal paralysis may take 24 hours or more to develop. Some, but not all of these presynaptic toxins also

cause severe myolysis. The less toxic but more abundant post-synaptic neurotoxins act more swiftly, but are destroyed in-situ by antivenom.

In addition to the myolytic action of the presynaptic neurotoxins, some species produce specific myolysins which can induce massive rhabdomyolysis throughout the skeletal muscle system. This phenomenon may be associated with gross myoglobinuria, and consequent renal damage. Serum creatinine phosphokinase levels will invariably be grossly elevated.

Many elapid venoms have prothrombin converters which cause severe defibrination with consequent hypocoagulability or non-coagulability of blood, at least in-vitro. When this has been documented clinically, in addition to severe derangement of routine clotting time analyses, there is usually severe to complete depletion of factors II, V, VII and VIII, and a gross elevation of fibrin degredation products. (6,10) Fatal cerebral haemorrhages have been reported after snakebite, (11, 12) and are presumably a result of the coagulation defects.

Some venoms also have a haemolytic action, either direct or indirect, and while in most cases intravascular haemolysis is not a clinical problem, haemoglobinuria may be detected in severe cases.

No specific nephrotoxic venom components have been isolated from Australian venoms, but acute renal failure requiring haemodialysis has been reported after envenomation by several species. The precise cause is unknown in most instances, although acute tubular necrosis has been described, where biopsy was performed. Many of these cases of acute renal failure were not associated with either myolysis or myoglobinuria. (13)

Though Australian venoms do contain numerous enzymes, some of which are clearly cytotoxic, local tissue damage is a relatively mild problem in Australian snakebite compared to the experience with non-Australian species, including some related elapids such as the cobras, especially the Spitting cobras. There are clear patterns now emerging in the type of local reaction after Australian snakebite, related to species group. (7)

The Australian dangerously venomous snakes fall into 6 main groups. The first group are the Taipans. There are two species, the common Taipan (Oxyuranus scutellatus) of Queensland, and the Inland Taipan (O. microlepidotus) of central eastern Australia. Both have large fangs, and are capable of injecting a large quantity of venom, which is highly toxic. The Inland Taipan has the most toxic snake venom known. These venoms, which are both neutralized by

Taipan antivenom, are strongly neurotoxic, myolytic, and cause defibrination, but have produced relatively little local reaction in most, but not all, reported cases.

The second group are the Brown Snakes (genus Pseudonaja), whose members collectively are found throughout Australia, except Tasmania. The Eastern Brown Snake (P. textilis) has the second most toxic snake venom known. They have small fangs, inject little venom, and the bite marks, which are often only scratches, are frequently hard to identify (Figure 1). The venom is highly neurotoxic, defibrinating, and has caused renal failure, but is not significantly myotoxic. There is no significant local reaction at the bite site (Figure 2). The venoms are neutralized by Brown Snake antivenom.

The third group are the Death Adders (genus Acanthophis), which are found throughout most of mainland Australia. They have moderate sized fangs, and can inject a considerable amount of venom, which is strongly neurotoxic (post-synaptic only), but not myolytic or defibrinating, although some coagulant activity has been noted experimentally. There is usually little or no visible local reaction, but local joint stiffness and pain has been reported. The venoms are neutralized by Death Adder antivenom.

The fourth group are the Black and Mulga Snakes (genus Pseudechis), the most important being the Mulga Snake

(P. australis). This group have moderate to large fangs, and can deliver large amounts of venom. The Mulga Snake delivers more venom, on average, than any other Australian snake. The venoms are not significantly neurotoxic, but at least in the case of the Mulga Snake, is very strongly myolytic, anticoagulant (probably defibrinating), and haemolytic. Bites from this species group usually cause local pain and oedema, the latter sometimes being severe and lasting many days (Figure 3). Local tissue necrosis around the bite site has been described. There is a specific antivenom, Black Snake antivenom, for the whole group, but all except the Mulga Snake also respond to Tiger Snake antivenom.

The fifth group are the Tiger Snakes (Notechis scutatus and N. ater), which have a wide distribution in eastern and southern non-arid areas; the Copperheads (Austrelaps superbus group), from south east Australia; the Rough-Scaled Snake (Tropedechis carinatus) from the central and north-eastern coastal region; and the more unusual and less well studied species of uncertain medical importance, of genus Hoplocephalus and Cryptophis. In many of these species the fangs are small, but considerable quantities of highly toxic venom may be injected, especially by the Tiger Snakes. Tiger Snake venom is highly neurotoxic, (with pre- and post-synaptic activity), myolytic, defibrinating, and may cause renal failure. Bites are frequently associated with a

significant local reaction, and local tissue necrosis has been described (Figure 4). Copperhead venom is less well understood, but bites have many similarities to Tiger Snake bites, although a local reaction is more commonly absent in bites by specimens from the Mount Lofty Ranges in South Australia. Rough-scaled venom is also neurotoxic and myolytic, but significant local reactions at the bite site appear to be less common.

The sixth group are the sea-snakes, of which there are numerous species recorded from waters of the northern half of Australia's coastline. Those venoms studied have potent neurotoxic and myotoxic potential, and appear to be neutralised by Tiger Snake antivenom, which may indicate a close phylogenetic relationship between the two groups. The numerous long teeth of sea-snakes leave a large number of distinctive puncture wounds, quite different to that encountered with terrestrial elapid snake bite.

The pathologist presented with an unexplained death, especially in a child, in an area frequented by snakes, should retain a high index of suspicion for snake bite. While with some species, the area of the bite may be readily visible, with many, as we have indicated above, the bite site may either be difficult to locate, or even unidentifiable (eg. Brown

Snake bites).

Specific testing of tissue samples for snake venom has been described, and has proved useful in making the diagnosis of snake bite at autopsy. (4) Two such tests both developed at the Commonwealth Serum Laboratories in Melbourne, are currently available in Australia. The earlier and most sensitive is a radioimmunoassay. (4) This has been superseded by the now readily available enzyme immunoassay (EI) technique, which may detect venom levels as low as 5 nanograms. (14) Venom appears to be stable for detection purposes despite repeated freezing, and samples from the bite site, local lymph nodes, and urine are most likely to yield positive results. (8)

In addition to the standard autopsy procedures, particular attention should be given to evidence of specific venom related pathology. There may be evidence of a coagulopathy with visceral haemorrhages and wound oozing. Local and central lymph nodes should be examined, and may be used for E.I. venom detection. Skeletal muscle should be examined macro and microscopically for evidence of myolysis. The kidneys should be assessed for evidence of tubular or glomerular damage, and haemorrhage. The brain, particularly in children, should be examined for evidence of macro or microscopic haemorrhages and the C.S.F. sampled. If venom

is detected elsewhere in the body, C.S.F. should be sampled for E.I., as there is currently no published information on venom penetration of the blood-brain barrier in man. The lungs may show evidence of pulmonary oedema and haemorrhage. Urine must be examined with E.I., and for myoglobinuria and haemoglobinuria. The vitreous humour of the eye may be useful for E.I. examination, especially in putrefying bodies when other tissues and bodily fluids are unsuitable.

The differential diagnosis, in these cases will include other forms of poisoning such as envenomation by a spider, and more importantly, heat stroke. In the latter, histological evidence of disseminated intravascular coagulation (DIC) is expected (15, 16); DIC is much less likely in snakebite.

All cases of autopsy confirmed snakebite in Australia should be reported, as there is a paucity of well documented cases. The reporting of pathological findings in Asian snakebite fatalities would also aid international comparisons.

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LEGEND FOR ILLUSTRATIONS:

Figure 1.

Upper arm of a 23 year old man bitten by a Western Brown Snake, with consequent collapse and neuromuscular paralysis. Note the absence of visible puncture marks at the bite site.

Figure 2.

Thigh of a 2½ year old female bitten twice by a Brown Snake, with subsequent collapse and severe coagulopathy.

Figure 3.

Marked oedema of the hand of a 48 year old male bitten 3 days previously by a Mulga Snake, with consequent collapse and myolysis. No bite mark visible.

Figure 4.

Toes of a 28 year old female bitten by a Tiger Snake, with consequent minor local tissue necrosis, coagulopathy, and neurotoxic paralysis.

SNAKEBITE IN SOUTH AUSTRALIA

Compared to the many other ways of achieving one's demise, and the apparent popularity of certain of these methods, such as coronary artery insufficiency, malignant disease (not including herpetology, or it's more extreme variant, herpetological photography), and of course road trauma, death due to snakebite in South Australia is a very exclusive method. Unfortunately for those wishing to apply for membership of such an exclusive club, the spoilsports of the medical profession have, through chance, fortune, and occasional good management, completely defeated all attempts to die from snakebite in S.A., for at least 10 years. The reason for such success is not entirely apparent, as S.A. certainly has a healthy (or perhaps unhealthy) number and variety of deadly reptiles, and until recently, doctors in S.A. did not recieve any training in snakebite and its management. Furthermore, there were no studies on the pattern of snakebite in S.A.

These and related enigmas and inadequacies prompted me to undertake a retrospective review of snakebite in S.A. The review was not concieved as a comprehensive study, but rather an initial pilot study. I hope that a more complete, statewide survey may be undertaken in the future.

Before embarking on discussion of this retrospective review, I would like to refresh everyones knowledge of the snakes and their venoms. The principal potentially lethal snakes in S.A. break up into five groups, in terms of antivenom requirements. The first group are the Brown Snakes, principally the Common Brown Snake, Pseudonaja textilis, very common in the southern

half of the state, and the Western Brown Snake, Pseudonaja nuchalis, common in the northern areas.

The second group are the Tiger Snakes and Copperheads. The common Tiger Snake, Notechis scutatus occurs principally along the Murray corridor and in the southeast. The Black Tiger Snake, Notechis ater, is found on a number of islands, and the tips of Eyre and Yorke peninsula's, and the southern Flinders Ranges. The Common Copperhead, Austrelaps superbus, is found in the southeast, and another variety, of distincitve form, is found in the Mount Lofty Ranges, and Kangaroo Island.

The third group are the Black Snakes, of which the most important is the Mulga Snake, Pseudechis australis, common in arid S.A. The Red-Bellied Black Snake, Pseudechis porphyriacus, is common in the Mount Lofty Ranges.

The fourth group are the Death Adder, typified by the Common Death Adder, Acanthophis antarcticus, but also represented by the Desert Death Adder, Acanthophis pyrrhus.

The last group are the Taipans, represented legitimately in S.A. by the Inland Taipan, Oxyuranus microlepidotus, in the far northeast, and illegitimately by the numerous Queensland Taipans now in captivity in S.A.

The venoms of all these species are complex, and none are completely understood. The most important actions clinically, are neurotoxicity, myotoxicity, defibrination, and to a lesser extent, local cytotoxicity. Alan Broad and his colleagues at C.S.L. have shown that these snakes are amongst the most toxic

in the world. However some less toxic species, such as the Carpet Viper, Echis carinatus, which is now known to kill at least 23,000 people a year in savana West Africa alone, are clearly more important epidemiologically. The low fatality rate from snakebite, currently running at about 1 a year, out of probably 2000 or more bites throughout Australia, is due in part to the ready availability of high-tech medicine, but also, I think, due in large part to the fact that our snakes often inject little or no venom, as recently confirmed by John Morrison's studies in Brisbane.

The retrospective review of snakebite was based on admissions to three Adelaide teaching hospitals, and cases were located using these hospital's admission coding system, which lumps all bites and stings together as one group. All such case files were examined, and only those listed as snakebite included. The varying efficiency of the filing system determined the period of review for each hospital.

Each case file in the history was examined with specific reference to description of the snake and bite, circumstances of the bite, onset and nature of symptoms and signs, investigation results, first aid and medical treatment and its outcome, and complications of envenomation. Unfortunately, for many cases, details in some or all of these categories were either sparse or non-existant. Even the area bitten was omitted in some cases.

RESULTS

At the Adelaide Children's Hospital, in a 10 year period 23 cases of definite or suspected snakebite were admitted, and there were no fatalities. The average age of patients was

8.2 years, with a range of 1.6 years to 13.8 years, and a sex ratio of 19 male to 4 female. There was a definite increased incidence in the toddler age group, and in the early teen years. In only 12 of the 23 cases was any reasonable identification of the snake made, and in these, 10 were Common Brown Snakes and 2 were Red Bellied Black Snakes. The area bitten was evenly divided, with 11 to the upper limb, 11 to the lower limb, and 1 to the lower lip. 12 of the bites occurred during spring and 8 in the summer. Of the 23 cases, only 7 developed any signs or symptoms of envenomation. Of these, only 2 were significantly envenomated, and both received polyvalent antivenom as the species of snake involved was in doubt. One had initial collapse, followed by nausea, vomiting and restlessness. The other had definite neurotoxic envenomation, with impaired concious state, diplopia, dysarthria, ptosis and general facial weakness, all of which were well established when antivenom was given over 24 hours after the bite. There were no haematological problems documented. Of the 7 patients with possible evidence of envenomation, 4 received antivenom. More puzzling is the fact that 5 patients were given antivenom in the absence of either signs or symptoms of envenomation, and without a snake even having been seen.

At the Royal Adelaide Hospital in a 9 year period, 16 cases of snakebite or possible snakebite were admitted, and there were no fatalities. The average age of patients was 26 years, with a range of 14 years to 46 years, and a sex ratio of 15 male to one female. 4 of the cases were in amateur reptile keepers.. Of the remaining 12 bites, 7 were due to Brown Snakes, 2 to Tiger Snakes and 2 to Red Bellied Black Snakes, with 1 uncertain. The area bitten was the upper limb in 6 cases and the lower limb

in 5. Surprisingly, the area bitten was not stated in 5 cases. Of the 16 cases, 8 developed some symptoms or signs suggestive of envenomation, but none were severe, although one case developed renal failure requiring haemodialysis for several weeks. This case is interesting for several reasons. The man concerned was apparently well known in his country town for his frequent and excessive imbibing of alcohol. While thus intoxicated one night, he was bitten by a snake later positively identified as a Brown Snake. In retaliation he replied in kind by biting the snake, and succeeded in biting its head off, which grizzly momento he pocketed. He than presented to the local hospital for treatment, but was not taken seriously until the snake's head was produced. However he developed no signs or symptoms of snakebite and was transported to another country hospital where he was given Tiger Snake antivenom. This certainly was followed by problems, culminating in acute renal failure, from which he eventually recovered. Other complications seen following snakebite were minor. Of the 16 cases, 10 received antivenom, but in 4 of these, there were very doubtful grounds for use of antivenom. Also, 3 patients who did not receive antivenom, did have symptoms or signs suggestive of envenomation. Of the four bites in reptile keepers, making 25% of all snakebite admissions, 2 were the same keeper. This young man also accounted for 5 of the 14 admissions at the Flinders Medical Centre, and I believe he also has had multiple admissions to other hospitals not surveyed by me.

At the Flinders Medical Centre in a 5 year period 14 cases of snakebite or possible snakebite were admitted, and there were no fatalities. The average age of patients was 18.5 years, with a range from 1 year to 30 years old, and a sex ratio of 12 males

to 2 females. 6 of the cases, or 43% were in reptile keepers, although 5 of the 6 were just one individual, as mentioned earlier. Of the remaining 8 bites, all were due to Brown Snakes or unidentified snakes which in retrospect, were probably Brown Snakes. The area bitten was the upper limb in 7 cases, and the lower limb in 6. 8 of the 14 bites were at night, but 6 of these were in reptile keepers. Significant problems following snakebite only occurred in the reptile keepers, and these accounted for the only 2 cases given antivenom.

Obviously this study has limitations reducing its applicability to S.A. as a whole. Firstly, the method of locating cases is open to error, for incorrectly coded files will be missed. Secondly, the pattern of snake distribution in S.A. does not mirror the local hinterland of these hospitals. However, most serious cases would gravitate to one of these hospitals. Statistics from C.S.L., which only account for those cases given antivenom, and then reported to C.S.L., show 207 cases of snakebite in S.A. from 1957 to 1982, averaging at 8 cases a year. I estimate that there are between 20 and 50 cases of snakebite in S.A. annually, but that only 1 or 2 of these are potentially lethal.

The C.S.L. statistics show that of those cases where the snake was identified 63% were due to Brown Snakes, and 21% due to Tiger Snakes. This is certainly in accord with the results of the study, and my own personel experience in managing snakebite.

New investigative methods should enable my group to accurately determine the species group for most cases of snakebite referred to me. Principal amongst these investigations is the enzyme immunoassay, which is accurate to a few nanograms of venom, and should yeild a positive swab from the bite site in 70% or more snakebites, that is once the bugs in this C.S.L. test are eliminated. In addition, I hope that we can develop a more accurate enzyme immunoassay in Adelaide to allow precise identification of individual species, and quantitate the degree of envenomation. This will allow refinement of the current understanding of envenomation caused by each species.

Based on the review, and personal experience, I believe it is possible to make some useful observations on snakebite in S.A. Firstly, the majority of bites are from members of the Brown Snake group, and the majority of these bites do not cause significant envenomation. Serious envenomation by Brown Snakes is more likely to occur in children, where it may cause massive rapid envenomation, resulting in unconciousness and unclottable defibrinated blood within 15 minutes of the bite. Impaired concious state and the coagulopathy rather than neurotoxic paralysis tend to dominate the clinical picture. However severe neurotoxic paralysis may occur, particularly in the active adult. The site of the bite is ususally unremarkable, with no pain, oedema, erythema, or evidence of local tissue necrosis. An almost invisible bite may still result in potentially lethal envenomation. (degree to care)

At least two cases of Brown Snake envenomation in S.A. have resulted directly or indirectly, in renal failure requiring extended periods of haemodialysis. The mechanism of renal

failure in these cases appears to be acute tubular necrosis, but the relationship to envenomation is unclear.

Tiger Snakes cause far fewer bites in S.A., but have a higher rate of serious envenomation. Most legitimate Tiger Snake bites occur in the southeast. As with the Brown Snake, devastating systemic envenomation may occur quite rapidly after the bite. Impaired concious state, coagulopathy due to defibrination, and neurotoxic paralysis all occur, and in addition rhabdomyolysis also occurs. Unlike Brown Snakes, neurotoxic paralysis is a dominant feature. Renal failure may occur, although there have been no recent S.A. cases. The bite site is painful, often oedematous, and with subcuticular haemorrhage, and local tissue necrosis is not infrequently seen.

Mulga Snakes are the only other group consistantly causing serious envenomation in S.A. They cause severe local reactions, varying from gross oedema of the bitten area, to necrosis at the bite site, or even of the whole digit. The victim may also be rendered unconcious for extended periods, and the severe rhabdomyolysis may mimic myocardial infarction. Severe coagulopathy may occur. Red-Bellied Black Snake bites are similar to, but less severe than those of the Mulga Snake.

SLIDE OFF

Bites from other species do occur, but too infrequently to make detailed analysis relevant as yet.

It is clear from the review that the absence of medical teaching on the management of snakebite has resulted in many instances of mismanagement, though none have had a fatal outcome. The poor correllation of evidence of envenomation, and systemic use of antivenom is typical of this lack of correct education. Unfortunately many doctors still believe that any person bitten by a dangerous snake needs antivenom. This is clearly mistreatment. Last year, formal instruction on the management of snakebite was introduced into the final year of the medical course at the University of Adelaide, and it is to be hoped that this will, in time, improve the information base on which treatment is based.

Unfortunately, this does not help the majority of doctors already in practice, who are either uninformed on the subject, or misinformed by doctors with an interest in envenomation, not supported by the required depth of knowledge and breadth of experience. This has resulted in the continued promulgation of the idea that a definite bite mark means antivenom is needed; that no obvious bite mark, means no envenomation has occurred; and that due to the diversity of S.A.'s snakes, only polyvalent antivenom can be used. A consultant toxinologist with appropriate understanding of our snake fauna and envenomation will avoid all these traps and fallacies, and enable the use of monovalent antivenoms in nearly all cases needing antivenom. A state funded venom and envenomation research unit would certainly improve the overall care of snakebite in S.A.

Lastly, the number of significant bites occurring in amateur reptile keepers is disturbing, though not really unexpected. Similar experience is well documented overseas. Nevertheless,

where a very few individuals sustain a high number of serious bites, repeatedly, due to carelessness and irresponsibility, I believe the question of control should be raised. These invididuals cost the State a significant sum of money, and it may be justified to introduce regulations to effectively control, but not prohibit, the non-institutional keeping of dangerous snakes, with licence fees being used to offset the cost of antivenoms and treatment. Obviously there are many problems with such an approach and I personally do not have a satisfactory solution to the problem.

CORRELATION OF PHYLOGENY OF AUSTRALIAN ELAPID SNAKES, DETERMINED BY IMMUNOLOGIC METHODS, WITH VENOM STRUCTURE AND CLINICAL ENVENOMATION.

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For herpetologists, an accurate taxonomy reflecting phylogeny is of the utmost importance in many diverse areas of research. For toxinologists taxonomy is equally important in seeking to understand the venoms of apparently related species. For clinicians involved in the management of envenomation, an accurate taxonomy is a vital cornerstone to assessing the clinical effects and outcome of envenomation.(1)

Australia has a large number of potentially lethal elapid snakes, whose venoms are amongst the most toxic snake venoms known.(2,3) Yet, the taxonomy, let alone phylogeny, of these snakes is still in a state of flux. As interest in reptiles increases, with a consequent increase in bites by unusual species for which no clinical data are available, it is becoming vital that both taxonomy and phylogeny be clarified, to allow at least some prediction of the possible hazards of envenomation by these species.

Conventional methods of taxonomy have relied on analysis of morphology, with little reference to ecology or other parameters. McDowell published an extensive analysis of

Australian elapid snakes based on morphologic criteria, and proposed a phylogeny.(4) This analysis placed Australian genera into four major groupings; the *Glyphodon* type which included *Pseudonaja*; the *Oxyuranus* type, which also included *Notechis*, *Tropidechis*, *Acanthophis* and *Hoplocephalus*; the *Pseudechis* type, which included *Austrelaps*; and the *Demansia* type. This grouping does not accord well with either ecological or toxinological information.

We have commenced a study of the phylogeny and taxonomy of Australian elapid genera using protein analysis, and in particular analysis of the serum proteins, especially transferrin, using immunodiffusion patterns developed on ouchterlony plates of trefoil design, and microcomplement fixation.(5,6) Sera from representative species of most, but not all Australian and some foreign elapid genera were available in the South Australian Museum Frozen Tissue Collection, and similar collections elsewhere.

Antisera to transferrins of 4 species of Australian elapids were prepared in rabbits (3 per antigen) and compared with sera from representatives of 1' Australian, 2 Papua-New Guinean, 1 African, and 2 South East Asian Genera. Using previously reported methods of analysis we have shown in these preliminary studies, that there are at least 4 major lineages of Australian terrestrial elapids, of relatively recent origin. Using the molecular clock, it would appear that all have evolved in the last 10 million years.

The proposed new tree correlates well with both morphologic and ecologic data, but is substantially different from McDowell. However, only four genera have been studied with full cross-reaction of individual antisera, and to further refine and confirm this phylogeny, more specific antisera will be

raised and cross-reacted. The results are also rendered difficult to analyse because of the apparently short time over which so many genera have evolved, and the consequent inevitable small differences in immunogenic distances. Nevertheless, correlation of current results, between antigenic distances and microcomplement fixation values is high (r=0.93; n=23).

Our proposed phylogeny may represent recent episodes in the biogeography of elapids. For instance, viviparity, which has been independantly evolved by many reptile groups as an apparent response to low temperature climatic extremes, might be expected to have arisen in those species radiating during such periods, and that these species would now have largely southern distributions. Those genera in our phylogeny showing viviparity all appear closely related, in a single lineage, centering on the *Notechis* genus, and may be the most recently evolved genera, whose ancestors may have radiated during recent periods of climatic change. Similarly, in this lineage, there is some morphological homology, the subcaudal scales being single. No members of this lineage appear to be represented in New Guinea.

In contrast, members of the other 3 lineages are all oviparous, have wide continental distributions, and all 3 lineages have representatives in New Guinea. Similarly, there is some limited morphological homology in all 3 lineages, with subcaudal scales divided.

Nevertheless, the relatively recent origin of all 4 lineages is evidenced by the ability of *Notechis* antivenom to cross-react to venoms of species in every lineage.

One lineage contains 8 genera, 3 of which are of major importance medically, namely: *Notechis*, the Tiger Snakes, which have a southern and eastern distribution; *Tropidechis*, the Rough-Scaled Snake, with an easten distribution; and *Austrelaps*, the Copperheads, with a south-eastern distribution. All are smallish wetland snakes, and all have venom neutralized by Tiger Snake antivenom (C.S.L.).

The venoms of the *Notechis* group have been studied extensively, and contain both physpholispase A_2 pre-synaptic neurotoxins and post-synaptic neurotoxins of the long and short chain types.(2,3)

Clincally, neurotoxic paralysis is often a dominant feature, and convulsions have been recorded. In addition, a coagulopathy is frequently observed, and ascribed to potent indirect prothrombin converters in the venom. Rhabdomyolysis may also be a prominent feature in some cases, with grossly elevated serum creatinine phosphokinase levels. In our experience, a significant local reaction is common, sometimes associated with a small area of necrosis.(2,9)

Tropidechis venom is less well studied, but recent work suggests that clinical envenomation is associated with neurotoxic paralysis, coagulopathy, and rhabdomolyiss, though the extent of coagulopathy is not documented. However, no major local reactions have been recorded, similar to those seen with *Notechis* envenomation. (Morrison, J.J.; Pers. Com). There is some evidence of pre-synaptic neurotoxic activity, but no pre-synaptic neurotoxin has been so far identified. If our proposed phylogeny is correct, then there should be homology between the neurotoxins of *Notechis* and *Tropidechis*.

The *Austrelaps* group, though further divergent from *Notechis*, could also be expected to show clinical and toxinological homology. Again, detailed fractionation studies of the venom are sparse, although myolytic activity has been established. Clinically, there is a similar paucity of information, but it appears that paralysis, coagulopathy, and rhabbomyolysis may occur. Local tissue reaction, and even necrosis can occur, at least after bites by the Victorian *Austrelaps superbus*. (Sutherland, S.K.; Pers. Com.)

In terms of toxicity, *Notechis* is most potent, then *Austrelaps*, which in many areas is sympatric, and least toxic is *Tropidechis*.

None of the other members of this lineage have been studied clinically. Some are wetland animals, but others extend well into arid habitats. All members of the lineage appear to feed principally on reptiles or amphibians. All are viviparous.

Two lineages contain only one genus each, *Demansia* and *Pseudechis* respectively. There is little information about *Demansia* venom. The most common species, *D. psammophis* is involved in a number of snake bites each year, all of which appear minor. There is usually a significant degree of local oedema and pain, the former taking several days to resolve. (2) No data on the venom is available. *D. olivacea (atra)* apparently causes similar symptoms, and Kellaway noted the venom to be feebly coagulant. More recent studies have shown an LD_{50} of greater than 14.2 mg/kg s.c. mouse. (3) This is much less toxic than those Australian elapids known to be dangerous to man.

Genus *Pseudechis* has received considerable attention from toxinologists. The largest member of the genus, *P. australis*, can deliver more venom than any other Australian elapid. However, with an LD_{50} of 2.38 mg/kg s.c. mouse its venom is less toxic than many other Australian elapids. Kellaway noted an absence of curariform action in this venom, but a potent single polypeptide chain myotoxin, *Mulgatoxin*, has been isolated. This causes significant myolysis, and is potentially lethal. In addition, the venom cau es a coagulopathy, and is more powerfully haemolytic than most other Australian elapid venoms. It causes significant local pain and oedema, and potentially, necrosis.(2,9) Clinical experience suggests that this venom can cause ptosis generalized weakness, and impaired concious state.

P. porphyriacus has a venom of similar toxicity invitro, but

clinically bites are much less significant. Evidence so far suggests that the venom can cause a coagulopathy, haemolysis, and myolysis, but not neurotoxic paralysis. Clinically, local oedema and pain are significant, and cases resemble *P. australis* envenomation, though less severe. (2,9)

P. guttatus and *P. colletti* both have similar venoms, though clinical information on envenomation by these species is sparse. Myolytic phospholipase A polypeptides have now been isolated from all four species. (3,7)

P. papuanus, found in New Guinea, is considered one of the most lethal snakes in that country, causing severe coagulopathy, and apparently, an irreversible neurotoxic paralysis, similar to that seen with *O. scuttelatus canni* envenomation. It's venom, with an LD_{50} of 1.09 mg/kg s.c. mouse, is more toxic than other members of the genus. Unfortuantely, further information on this venom is unavailable. All members of this group have venoms neutralized by *P. australis* antivenom (C.S.L.), but *P. porphyriacus*, *P. guttatus* and *P. colletti* can also be neutralized by *Notechis* antivenom. *P. papuanus* venom is neutralized by a specific antivenom.

- The last lineage contains both *Pseudonaja* and *Oxyuranus*, and possibly a number of other genera, including 2 genera of sea snakes, *Aipysurus* and *Aspidomorphus*. Further research will be undertaken to verify this relationship.

- The snakes of genus *Pseudonaja* are found on mainland Australia, with a wide distribution, and a bewildering variety of colour morphs, currently divided into at least 7 species. *P. textilis* venom has both pre. and post-synaptic neurotoxins, the former being the most potent such toxins known, with an LD_{50} of 0.6 mg/kg

mouse. Like other presynaptic neurotoxins, it contains phospholipase A_2, but has 4 subunits with a total MW 88,000, thus it is quite different from *Notexin*, from *Notechis* venom, but shows some similarities to *Taipoxin* and *Paradoxin* from *Oxyuranus* venoms. *Pseudonaja* envenomation clinically causes a severe coagulopathy, and impaired concious state, but frequently, neurotoxic paralysis is not a prominent feature.(2,8) Myolysis has not been detected clinically in any cases of *Pseudonaja* envenomation, but experimentally has occurred in association with *P. nuchalis* and *P. affinis* venoms. (3) Local oedema and pain is usually absent after *Pseudonaja* bites.(9) A recent case of *P. nuchalis?* envenomation in South Australia had severe neurotoxic parlaysis without evidence of either myolysis or coagulopathy. There are several varieties of *P. nuchalis* in the area in which the victim was bitten, and they are of unusual morphology, and may represent new species. However, despite the atypical sequelae of envenomation in this case, the venom was strongly positive for *Pseudonaja* on EIA testing. All *Pseudonaja* venoms are neutralized by specific *P. textilis* antivenom.

- The two members of *Oxyuranus* are large snakes with a north-eastern distribution, and a subspecies is found in New Guinea. Both have very toxic venoms, with pre and post-synaptic neurotoxins, and clinically cause severe neurotoxic paralysis, severe coagulopathy, occasional myolysis, impaired concious state, convulsions, and occasional, but usually minor local oedema and pain. All are neutralized by *O. scuttelatus* antivenom.

- The position of the other clinically important Australian genus, *Acanthophis*, is not clear from our research to date. It is possibly an early divergence. The genus is also found in New Guinea. Unlike the other genera discussed, *Acanthophis* venom, though

having neurotoxic action, has no apparent clinically significant action on coagulation. Furthermore, its neurotoxins appear to be post-synaptic only, with both long and short chain types.

SLIDE OFF

Conclusions on the phylogeny of Australian elapids, based on morphology, immunology, and toxinology must still be tentative only. However, the results of our research to date does appear to mesh well with toxinologic and other data.

It would therefore seem probable that elapid ancestors, entering through New Guinea, have spread across the continent in waves probably associated with climatic changes in recent times. Some lineages are still represented in New Guinea, ie. *Acanthophis, Pseudechis, Demansia, and Oxyuranus,* but not *Pseudonaja* which may have arisen from a common ancestor with *Oxyuranus,* within *Australia.* Another ancestor has resulted in the *Notechis* lineage. The *Oxyuranus - Pseudonaja* lineage and the *Notechis* lineage may therefore have evolved presynaptic neurotoxins and pro-coagulants separately, accounting for the known differences between these venoms.

The similarities between sea snakes and Australian elapids, and the distinct differences between these groups, and elapids in other regions of the world, may indicate a common origin, and it has been recently suggested that Australian elapids should be placed in family, *Hydrophiidae* rather than *Elapidae.* Our research to date correlates well with such a phylogeny.

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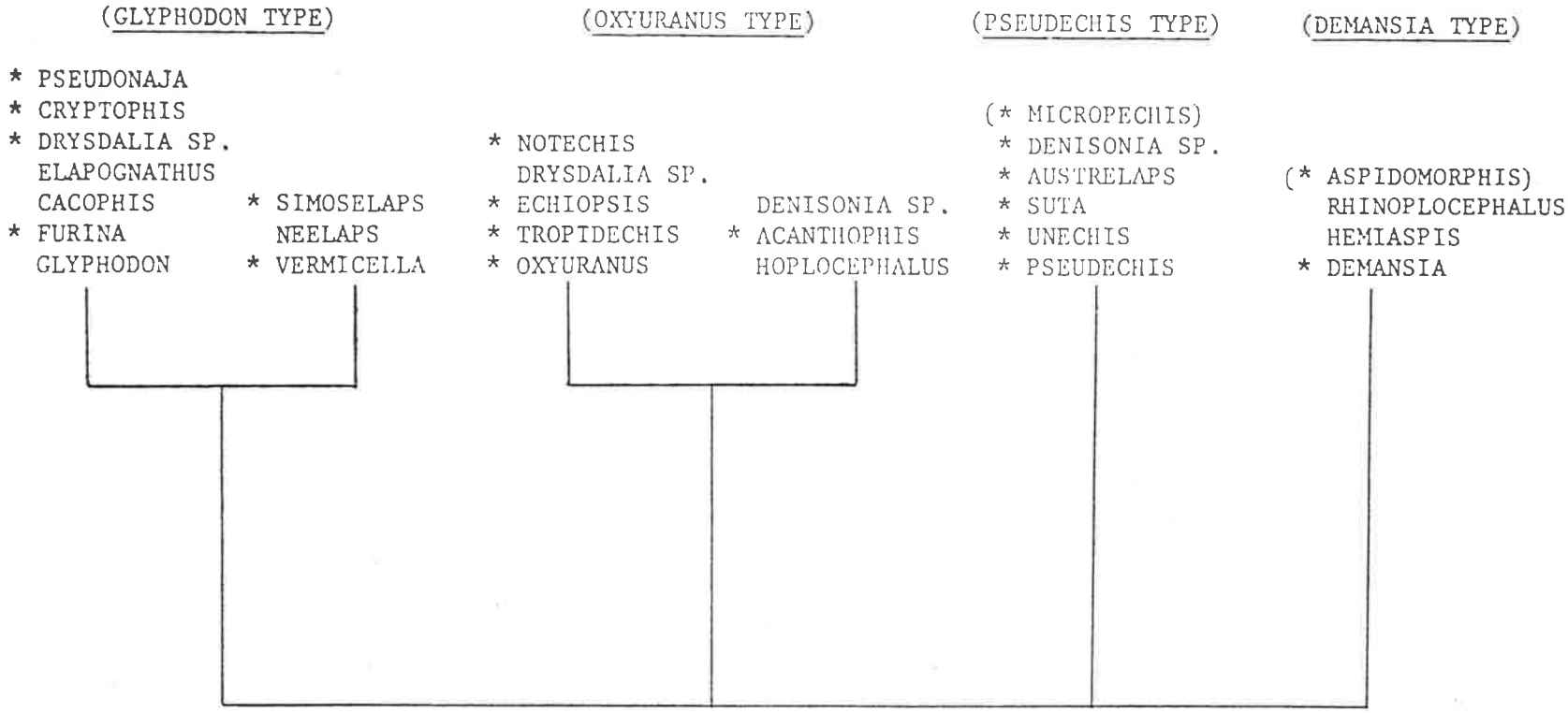
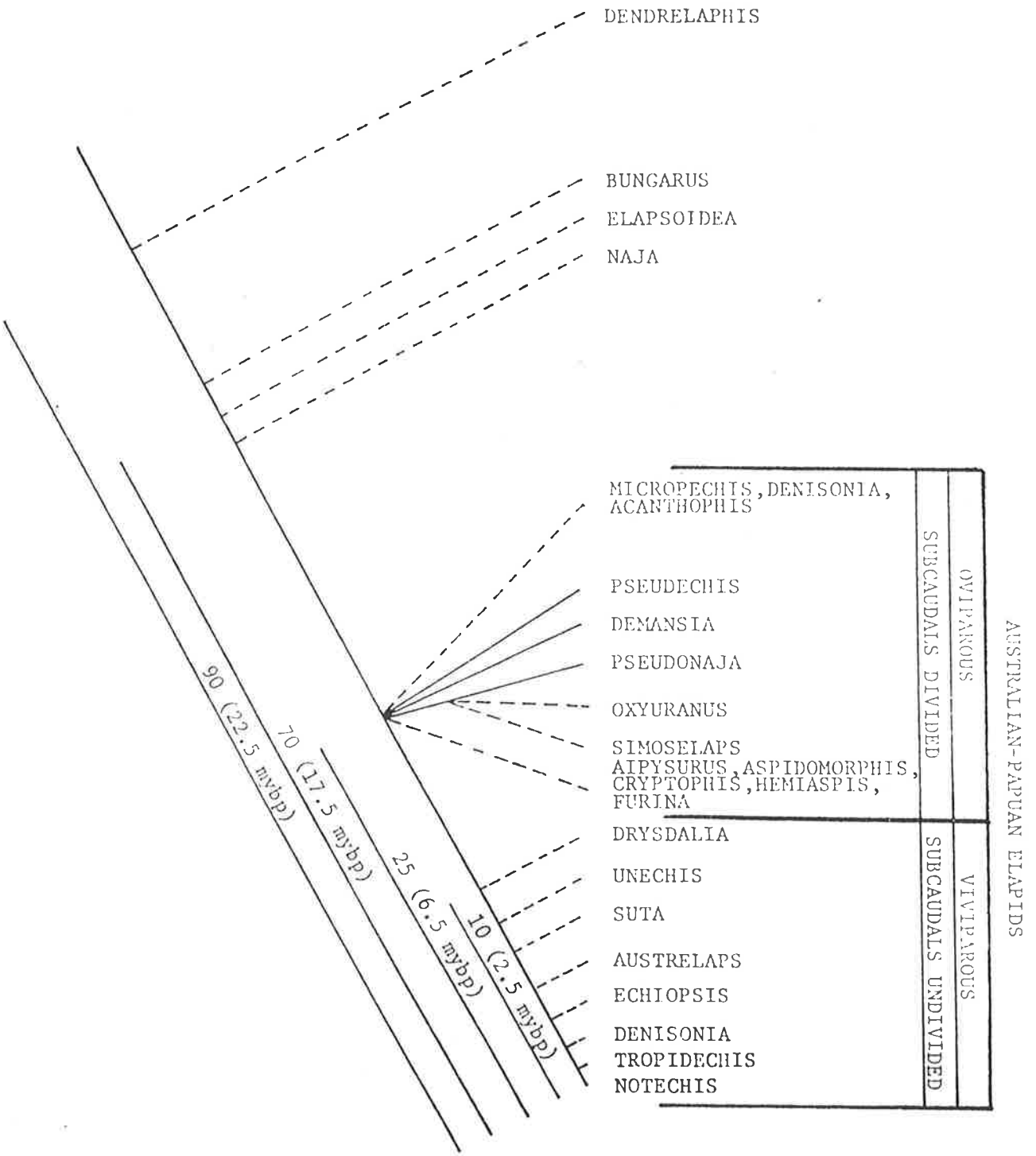


PHYLOGENETIC RELATIONSHIPS OF
AUSTRALIAN ELAPID SNAKES
(After McDowell, 1967)

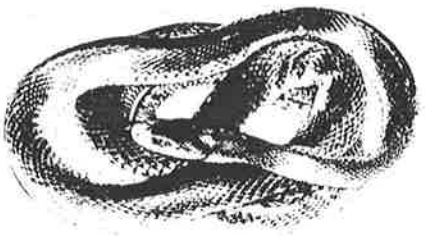
*Groups for which plasma has been stored in the Collection of Viable Reptile Tissues, The South Australian Museum. Genera in brackets are Papuan groups.


DENDRELAPHIS
BUNGARUS
ELAPSOIDEA
NAJA
MICROPECHIS, DENISONIA, ACANTHOPHIS
PSEUDECHIS
DEMANSIA
PSEUDONAJA
OXYURANUS
SIMOSELAPS
AIPYSURUS, ASPIDOMORPHIS, CRYPTOPHIS, HEMIASPIS, FURINA
DRYSDALIA
UNECHIS
SUTA
AUSTRELAPS
ECHIOPSIS
DENISONIA
TROPIDECHIS
NOTECHIS
SUBCAUDALS DIVIDED
OVIPAROUS
SUBCAUDALS UNDIVIDED
VIVIPAROUS
90 (22.5 mybp)
70 (17.5 mybp)
25 (6.5 mybp)
10 (2.5 mybp)
AUSTRALIAN-PAPUAN ELAPIDS

A PERSPECTIVE ON THE PROBLEMS OF SNAKEBITE IN AUSTRALIA

J. WHITE[1], D. POUNDER[2], J. H. PEARN[3] and J. J. MORRISON[3]

Poisonous snakebite in Australia is a medical emergency which is rarely encountered by most doctors (1000-3000 cases/year), and clinical data acquisition is problematic. The Australian Snakebite Study Group has been formed to improve data acquisition. Initial study of snakebite fatalities reveals an apparent decrease in mortality from 4-5/year to 1-2/year over the last nine years. The majority of deaths occurred more than 24 hours after the bite. The problems of management of snakebite are discussed, the two major problems being decision on the type of antivenom required; and when and how much antivenom to give. Detailed knowledge of the distribution of dangerous snakes combined with data on clinical differences between bites by the various species/venom groups should allow use of monovalent or mixtures of monovalent, rather than polyvalent antivenom in many cases. Antivenom should only be given when systemic envenomation is present. ELISA venom detection techniques have an undefined but potentially valuable role in the management of snakebite.



Pages 511-14 *in* BIOLOGY OF AUSTRALASIAN FROGS AND REPTILES, ed. by Gordon Grigg, Richard Shine and Harry Ehmann, Royal Zoological Society of New South Wales, 1985.

INTRODUCTION

IN TERMS of the total range of potential medical emergencies presenting for treatment in Australia, snakebite is relatively rare overall, with between 1000 and 3000 cases each year (Sutherland 1983). In a few regional centres, such as Darwin and parts of Queensland (Munro and Pearn 1978), cases of snakebite present frequently, enabling medical expertise in management to be readily acquired and maintained. For most medical practitioners, however, the infrequency of cases makes acquisition of skills in management difficult. Similarly, the acquisition of clinical data for research into snakebite is hampered by the geographically scattered and sporadic nature of cases. In an attempt to consolidate available clinical data, both retrospectively and prospectively, a research group, the Australian Snakebite Study Group, has been formed. The group will attempt to define problems in the management of snakebite in Australia, and develop solutions. This paper will discuss the initial results of a survey of fatal snakebite in Australia, and outline currently perceived problems in the medical management of snakebite, and available solutions.

SURVEY OF FATAL SNAKEBITE

Method

Registrars of Births, Deaths and Marriages in all states of Australia, in association with the Australian Bureau of Statistics were asked to supply lists of fatal cases of snakebite occurring since January 1st, 1970, and in addition, forensic pathologists in all states were individually asked to supply available information on cases of fatal snakebite dealt with by them. These lists will make it possible to obtain autopsy reports, which will be analysed to give demographic data, and data on the snake species involved, site and type of bite, onset and progression of symptoms and signs (not all cases), and major autopsy findings including cause of death, evidence of coagulopathy, rhabdomyolysis, central nervous system involvement, and renal damage.

Results

The survey is only in its initial stages, and therefore results are incomplete. No detailed autopsy data are available for most cases at this stage (though they should be forthcoming later). Some states have provided data prior to 1970. One state has declined to provide any data at present.

Fatal cases of snakebite notified are listed in Table 1. Information from Queensland is incomplete, but 15 deaths ascribed to snakebite occurred from 1970 to 1982. Figures for Western Australia may be incomplete. No deaths occurred in the Australian Capital Territory from 1968 to 1983.

Most detailed information so far is from New South Wales and Victoria. Currently available data on New South Wales cases (1967-1983) are shown in Table 2, and similar data for Victoria (1969-1983) in Table 3. One of the three cases from Tasmania was a middle-aged man who was bitten on the right index finger while exhibiting a Tiger Snake (probably *Notechis ater*). He collapsed a few minutes later, and died within two hours. He had previously been bitten by Tiger

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Table 1. Snakebite Fatalities in Australia; 1967-1983.

	NSW	VICTORIA	SA	WA	TASMANIA	QLD	TOTAL+
1967	2	NA	NA	NA	NA	NA	NA
1968	0	NA	1	NA	0	NA	1
1969	1	2	1	1	0	NA	5
1970	1	1	0	0	1	0	3
1971	2	2	0	0	0	4	8
1972	1	1	0	2	0	1	5
1973	0	1	0	0	0	2	3
1974	2	1	0	1	0	1	5
1975	1	1	0	1	0	2	5
1976	0	0	0	0	0	0	0
1977	0	1	0	1	1	3	6
1978	0	1	0	0	1	0	2
1979	0	0	0	0	0	1	1
1980	0	0	0	1	0	0	1
1981	1	0	0	0	0	0	1
1982	0	0	0	0	0	1	1
1983	1	0	0	0	0	0	1
TOTAL	12	11	2	7	3	15	48

(NA = not available).

\+ Total for each year excludes the Northern Territory, which is unlikely to contribute a significant number of snakebite fatalities due to its low population.

Table 2. Snakebite Fatalities in NSW; 1967-1983

Age of victim	Sex	Time from bite to death	Month of year when bitten
11	M	N.A.	9
16	F	1 hour	12
17	F	9 days	2
19	M	12 days	1
20	M	less than 1 day	1
25	M	2 days	5
32	M	N.A.	5
45	M	13 days	12
48	M	less than 1 day	10
49	M	2 days	9
62	F	19 hours	12
88	F	2 days	11

Table 3. Snakebite Fatalities in Victoria; 1967-1983.

Age of victim	Sex	Month of year when bitten
2	F	1
3	F	12
9	M	10
11	M	8
19	M	10
19	M	1
23	F	10
38	M	12
53	F	1
60	M	2
67	M	12

Snakes, and in view of the clinical picture, he is presumed to have had an acute hypersensitivity reaction to the venom. Detailed information on only two cases from Western Australia is currently available:

a) a two-year-old girl was bitten by an unidentified snake, and died less than 16 hours later, without receiving antivenom;
b) a 20-year-old man was bitten by an unidentified snake, and suffered rapid collapse, brain death, and actual death 14 days later.

Discussion

The currently available data are insufficient for complete analysis, however several interesting facts emerge. Firstly, there would appear to be a reduction in fatality rate since 1978, when new methods of first aid were introduced. The current fatality rate in Australia has fallen below the 4-5 deaths per year quoted in a previous survey (Trinca 1963). It is not possible to determine the cause of this reduction in mortality though several factors may be responsible, including improved public education, improved first aid, and improved medical education in the management of snakebite.

Secondly, it is apparent from the New South Wales data that the majority of deaths occur more than 24 hours after the bite, in some cases 10 days or more post bite. Detailed examination of autopsy findings and other records will be required to determine the cause for such delayed death. The case from Western Australia may give some clue, in that brain damage occurred rapidly, and death may possibly have occurred much sooner without medical intervention and the use of life support systems.

MANAGEMENT OF SNAKEBITE IN AUSTRALIA

The management of snakebite naturally falls into two stages: a) immediate management or first aid; b) medical management, usually in a hospital setting. First aid for snakebite in Australia is now generally accepted as the use of a broad constrictive bandage and immobilization, following the technique of Sutherland *et al.* (1979a). This safe and simple technique has been shown to be effective experimentally (Sutherland

et al. 1981) and clinically (Pearn *et al.* 1981; Murrell 1981) though the latter is less rigorously proven owing to inherent difficulties in designing and executing such a study.

The medical management of snakebite in Australia is relatively clearcut, in that significant envenomation requires the administration of antivenom (Sutherland 1983; White 1981). However, this results in two major areas of uncertainty; a) which antivenom is appropriate, in turn determined by the type of snake involved; b) when and how much antivenom to give, which is a reflection of the extent of envenomation.

There is ample evidence that polyvalent antivenom has a higher incidence of untoward reactions, and is also higher in cost. In one series, untoward reactions occurred in 7% of patients given monovalent antivenoms, and 27% of patients given polyvalent antivenom (Sutherland *et al.* 1979b). Therefore, for both medical and economic reasons, it is desirable to give monovalent, or a mixture of two monovalent antivenoms rather than polyvalent antivenom (Sutherland 1983). To do this it is necessary to know the venom group of the snake involved, or to use a combination of other data to limit the possibilities to no more than two different venom groups. The snake itself is rarely available, and without an expert herpetological opinion, errors in identification are likely (Morrison *et al.* 1983).

Direct determination of the venom group, and therefore the monovalent antivenom required, can be made by analysing the venom, either from the bite site, associated clothing, patient serum, or patient urine (the latter two are only relevant if the patient has systemic envenomation at the time of sample acquisition). Three methods are available for determination of venom, namely radioimmunoassay, an accurate, sensitive, but clinically impractical technique; enzyme linked immunosorbant assay (ELISA), either using the commercial kit (Commonwealth Serum Laboratories), or lastly; a laboratory based quantitative derivative of the ELISA. As only the commercial kit ELISA, which gives qualitative results, is freely available, the other methods can be discounted for practical purposes.

Using recommended times, the commercial ELISA takes approximately one hour to give a result. The most reliable sample is a swab from the bite site, as serum has proved an unreliable sample (Hurrell *et al.* 1982). In practical terms, many cases of snakebite where significant envenomation is present will occur in areas remote from ELISA kits. Even in areas well serviced with ELISA kits a decision on the type of antivenom needed will often be required before the results of the ELISA are available. Therefore, while the kit is certainly a significant advance, its definitive role in the management of snakebite has yet to be established, and other options for deciding antivenom choice are also needed by the medical practitioner.

The options available to determine the monovalent antivenom(s) needed in the absence of an ELISA kit result are limited. Firstly, in certain regions of Australia the only naturally occurring dangerous snakes fall into one or two groups (Sutherland 1983; White 1981). The two most common such groupings are Brown Snake and Tiger Snake Antivenom (covering *Pseudonaja* spp. *Notechis* spp. *Austrelaps* spp. *Tropidechis carinatus, Pseudechis* spp. except *P. australis* and *P. butleri*), and Brown Snake and Black Snake antivenom (covering *Pseudonaja* spp. and *Pseudechis* spp.). Access to an expert on the distribution of Australian dangerous snakes is usually required to determine the likely snake group(s) that may occur at a locality. Where three or more groups are likely at a locality, it will usually be more practical to use polyvalent antivenom rather than a mixture of monovalent antivenoms.

Secondly, given a limited range of potential species of snake, based on geographic locality, it may be possible to give a "most probable" diagnosis of the snake species involved on the basis of clinical findings. It has previously been assumed that there are no significant differences between bites of Australian dangerous snakes, at least as far as bite site appearance and pattern of systemic envenomation are concerned. However, as series of cases are studied in more detail, and it is possible to retrospectively determine the venom group using the ELISA, some significant patterns are emerging for each venom group (White 1983 a, b, c).

Bites by members of the *Pseudonaja* group are usually associated with minimal or no local pain, or tissue reaction, while manifestations of systemic envenomation include collapse, headache and abdominal pain, and coagulopathy, but not rhabdomyolysis. In contrast, bites by the *Notechis* group are usually associated with significant local pain and reaction, including minor necrosis on occasion. Manifestations of systemic envenomation include collapse, headache and abdominal pain, coagulopathy, paralysis, and rhabdomyolysis. Bites by members of the *Pseudechis* group are associated with variable local pain, but local oedema is a common finding. Systemic manifestations include collapse, malaise, coagulopathy (*P. australis*) and rhabdomyolysis, but true paralysis is not seen.

The problem of determining when and how much antivenom to give shares some solutions with the determination of antivenom type. As more information is collected on the clinical manifestations of snakebite, and systemic envenomation in particular, so it will become clearer what criteria should be used to guide therapy with antivenom.

Certain guidelines are already clear. Antivenom should only be given when there is systemic envenomation, and should not be given where snakebite is limited to a local reaction (White 1981; Sutherland 1983). A positive ELISA kit test for venom at the bite site is not an indication for antivenom (White 1981). Systemic envenomation is usually evident from symptoms and signs. These include general manifestations such as headache, nausea, abdominal pain, and collapse, and specific manifestations such as progressive paralysis, muscle movement pain, and oozing blood from the bite wound and venepuncture sites. Most laboratory tests will take longer than is practical for decision-making in severe cases, but simple tests of clotting time may be done rapidly, and may show early coagulopathy when signs are equivocal. Monitoring the progress of the coagulopathy and its resolution, especially basic tests of the coagulation pathway (Prothrombin ratio, P.T.T.K.) and fibrinolysis (Fibrinogen, Fibrin degradation products), can be a very useful pointer to the neutralization or otherwise of circulating venom (White 1981c).

The decision on the quantity of antivenom required is best determined by combining clinical and laboratory findings. Where a patient has sustained multiple bites, or is severely envenomated, multiple doses of antivenom will be needed. Where an initial dose of antivenom causes only short term improvement, followed by deterioration, more antivenom will be needed. As mentioned earlier, the resolution of coagulopathy may act as a good guide to the adequacy of antivenom dosage.

While these methods do allow effective treatment to be given in most cases, it is clear that the assessment of systemic envenomation is less than an exact science. At least for major toxinology units, the future development of quantitative ELISA assays for venom in plasma will allow more accurate titration of antivenom requirements. This may permit the development of more comprehensive guidelines for antivenom administration.

ACKNOWLEDGEMENTS

We gratefully acknowledge the assistance of forensic pathologists, Registrars of Births, Deaths and Marriages, and staff of the Australian Bureau of Statistics who have provided information on fatal cases of snakebite. Pauline Pahl typed the manuscript.

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CLINICAL PROBLEMS IN THE MANAGEMENT OF AUSTRALIAN SNAKEBITE - THE CHOICE OF ANTIVENOM

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Australia has a large and diverse collection of front fanged venomous snakes, variously placed in the families ELAPIDAE and HYDROPHIIDAE[1,2]. Some of these snakes are amongst the most dangerous serpents known[3], yet despite up to 3,000 bites annually in Australia only about 1 to 3 Australians die from snakebite each year[1,4]. Nevertheless, at least 200 patients require antivenom each year, and the major medical decisions in managing snakebite in Australia involve choices about antivenom. Issues arising from this will be discussed in this paper. A full discussion of the presentation, symptoms and signs, complications, and management of snakebite in Australia has been adequately covered elsewhere[1,4].

Antivenom (AV), produced by the Commonwealth Serum Laboratories (CSL), Melbourne, Australia, is readily available throughout the country, and is well proven and accepted as the treatment of choice for systemic envenomation by Australian snakes[1,4]. It is available as five "monovalent" AVs, covering the five main venom groups for Australian snakes, and

also as a "polyvalent" AV, covering all five groups. There is a separate sea snake AV.

In considering the use of AV the physician must ask three basic question:

(I) When to use antivenom?

(II) Which type of antivenom?

(III) How much antivenom?

This paper will focus on an answer to question (II), namely which type of AV should be used.

In brief, the answer to question (I) is that AV should be used only in cases of systemic envenomation, and to question (III), that enough AV must be given to neutralize all circulating venom, which may mean giving several times the normal recommended dose. More detailed discussion may be found elsewhere[1,4].

Given the availability of polyvalent AV, it might be assumed that monovalent AVs are redundant. However perusal of CSL's own figures reveals that the incidence of untoward side effects is significantly higher with polyvalent AV than with monovalent AVs (table I), reflecting the higher volume involved[5]. It is therefore now seen as preferable to use monovalent AVs wherever possible, an attitude strengthened by

the large cost differential between the two most commonly required monovalent AVs (Tiger Snake AV and Brown Snake AV) and the far more expensive polyvalent AV.

However, to correctly use monovalent antivenom, the type of snake (or venom group) involved in each bite must be known. There are several ways this might be determined.

1) **Direct Identification of the Snake**

Unfortunately, the snake responsible for the bite is rarely brought in with the patient, and if it is brought in, it is frequently badly damaged and difficult to identify. Furthermore, it has been shown that most Australians, including doctors, are very inexpert at identifying types of snakes, even when examining well preserved dead specimens, and at best will be correct only 20-25% of the time[6]. As the snake is seen in only 75% or less cases of snakebite[7], and then often only fleetingly, it is clear that reliance on correct identification of an actual snake specimen, either presented with the patient or, worse still, as a description of sighting only, is unacceptable as a routine method of determining antivenom type.

2) **Identification of the Venom**

CSL have for some years pursued research into venom identification from tissue samples and wound swabs of snakebite victims. They have produced a highly reliable RIA which unfortunately takes too long (when including specimen transport time) to be of practical value in the management of snakebite, except in rare instances[4]. However, the brilliant work of David Theakston and colleagues in developing an ELISA method for venom (and antibody) detection has been expanded on by CSL. This has resulted in the development and distribution of an ELISA based Venom Detection Kit (VDK)[8,9]. This kit, which is self contained and requires no laboratory facilities to use, will determine which of the five venom groups (corresponding to the 5 monovalent AV types), a given venom sample belongs to. The sample is taken from a swab of the bite site and is capable of giving results with as little as 5-10 ngms of venom in the sample. For maximum accuracy the test takes about 30-40 minutes to perform. It costs approximately A$20 per test, and comes in kits of 3 tests, with a shelf life of about 6 months (though efforts are being made to extend this).

In theory this VDK should now allow the type of snake to be determined in virtually all cases of snakebite in Australia, and thus allow use of monovalent AV rather than polyvalent AV.

In practice the VDK has not yet caused a major change in AV usage (as determined by AV sales). Figures kindly supplied by CSL show that in the third year of availability of the VDK, the ratio of monovalent to polyvalent AV sold (5.8:1) has not changed significantly from the period prior to the introduction of the VDK (5.6:1).

CSL have assessed the usage of the VDK[9,10]. In the two year period 1980-82 137 instances of VDK usage were reported, and of these 31% had clinical criteria for the administration of AV. In the same period 385 instances of AV administration were reported. Although these two sets of statistics were not jointly analysed it is clear that at best little over 10% of those requiring AV have a VDK assessment of the AV required.

In view of these figures, it is clear that the VDK has not gained wide acceptance, and several factors may have contributed to this.

(a) **Logistics**

Many snakebites occur in remote areas, and by the time the patient reaches medical attention, systemic envenomation, if it is going to develop at all, will be well established. In this situation it is unethical to delay treatment with AV until a VDK result is available.

In the author's experience, no case of major envenomation has had primary AV requirements determined by VDK results. However, subsequent doses of AV have, on occasion, been determined by VDK results, and the VDK has proved invaluable in confirming clinical judgment on the snake involved in many cases.

(b) **Availability**

Two aspects of availability have inhibited use of the VDK in South Australia. Firstly, there have been too few kits available at times of peak demand. Secondly, the distribution of kits to country centres does not match with the list of country centres treating snakebites. Thus in analysing cases of snakebite treated at country hospitals in the calendar year 1981, and comparing with distribution of VDK kits to these same hospitals, many country hospitals had kits

and no snakebites (9 centres), or vice versa, had snakebites and no kits (7 centres), and only 7 had both kits and snakebites.

(c) **Cost**

Given the short shelf life and cost, and the relative infrequency of snakebites, many country centres, where the VDK could be particularly useful, have decided they are not cost effective. This relates back to availability and logistics.

(d) **Reliability**

The VDKs produced over the first 2-3 years gained a reputation as unreliable, in that the control failed, thus invalidating any results. This problem has been rectified, and the author has not had any kit failures in the last 2 years.

In summary, the VDK has not been as useful as was first hoped, but the author believes it is potentially a very valuable tool for correlating the pattern of systemic envenomation with the type of snake involved, and as such deserves a much higher profile than currently afforded it.

3) Correlation of Herpetological and Clinical Information

The distribution of each species of snake in Australia is increasingly well known. For many areas the range of species may be narrowed down to only 2 or 3 venom groups. In this situation it is often possible to use a mixture of 2 monovalent AVs rather than polyvalent AV[1,4]. The author is currently developing this concept for both South Australia, as illustrated by the prototype map (Figure 1) and for the whole continent. In constructing such maps judgments must be made about which snakes will cause confusion in identification, and which snakes are unlikely to cause bites, either due to behaviour or density of distribution. Thus in South Australia there are large inland arid areas where snakes of 3 separate venom groups occur: Brown Snake Group, Black Snake Group and Death Adder Group. However, due to behaviour, density of distribution, and appearance, the Desert Death Adder will not usually cause a problem to differentiate from the other two groups, and no bites by this species have been recorded from this area (as far as records show). Therefore the Death Adder group is not included in the venom types for this area, as in practice all legitimate snakebites from this area will be from snakes in the Brown Snake group or Black Snake group (i.e. Mulga Snake).

In the author's experience, detailed assessment of clinical cases of snakebite shows consistent findings for bites by each venom group of snakes. A preliminary analysis shows that the differences between each group may be sufficient to allow the treating clinician to determine the type of snake involved without recourse to the VDK (Table II). Of particular relevance is the pattern of local reactions at the bite site, and in cases with systemic envenomation, the presence or absence of coagulopathy and paralysis. Thus for bites by members of the Brown Snake group, there will be little or no local pain or other local reaction, clearly distinguishing them from bites by members of the Tiger Snake or Black Snake (Mulga Snake) groups, the latter being readily differentiated by the lack of true paralysis in bites by the Black Snake group. Unfortunately the number of cases accumulated so far is small, but the VDK is greatly assisting in improving the situation. Nevertheless it is probably too early to universally adopt these guidelines though they are certainly a useful guide for the experienced toxinologist.

By combining this detailed clinical knowledge with similarly detailed herpetological knowledge, the savvy toxinologist should in most situations be able to recommend either a single, or mixture of two appropriate monovalent AVs rather than polyvalent AV.

Conclusions

Polyvalent AV should no longer by the universal choice for snakebite by an unidentified snake in most regions of Australia. The availability of the VDK, and the consultant services of toxinologists with both relevant herpetological skills and appropriate experience in assessing and managing snakebite should allow the use of either a mixture of two or a single monovalent AV in the majority of snakebite cases. Implicit in such a recommendation is the assumption that such expert toxinologists will be directly involved in the assessment and management of virtually all cases of snakebite, to ensure the establishment and maintenance of relevant skills.

Acknowledgements

The author wishes to thank the many medical colleagues whose referral of cases has allowed the present data base to be established, and those other colleagues who have assisted in the management of some of these cases.

Several members of CSL have been very helpful and provided invaluable information: Wendy Cowling, Kevin Healey, John Hurrell, Alan Coulter, Struan Sutherland, Bob O'Grady.

Staff of the Adelaide Children's Hospital have contributed immensely to this snakebite project, and specifically, the Photography Department assisted with the illustration, and Mrs Olivia Lammas, Department of Haematology, expertly typed the manuscript.

Table I - Incidence of untoward reactions following Antivenom

(after Sutherland and Lovering, ref. 5)

Antivenom	Average Volumes	Untoward Reactions	(Total No. of cases/No. with reaction)
Brown Snake AV	4.2 mls	5%	(20/ 1)
Tiger Snake AV	10.2 mls	12%	(16/ 2)
Above 2 AV combined	13.8 mls	8%	(36/ 3)
Overall for all monovalent AVs		7%	(87/ 6)
Polyvalent AV	69.6 mls	27%	(86/23)

Table II - Clinical effects of envenomation by Australian snakes

Snake/Venom Group	Local Problems			Systemic Problems			
	Pain	Swelling	Ecchymosis (± mild necrosis)	Collapse	Paralysis	Coagulopathy	Rhabdomyolysis (muscle movement pain)
Brown Snakes (*Pseudonaja sps*)	- (or very mild)	-	-	+	+ (some cases only)	+	-
Tiger Snakes (*Notechis sps*)	+	+ (mild)	+	+	+	+	+
Mulga Snake (*Pseudechis australis*)	+/-	+ (often severe)	-/+	+	-	+ (often mild only)	+
Black Snake (*Pseudechis porphyriacus*)	+/-	+	+/-	- (only rarely)	-	- (or very mild)	+/-
Death Adders (*Acanthophis sps*)	+	- (minimal)	-	-	+	-	-
Taipans (*Oxyuranus sps*)	+	+/-	+/-	+	+	+	+

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ELAPID SNAKES: VENOM PRODUCTION AND BITE MECHANISM

Julian White

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◄ Skull of Taipan, *Oxyuranus scutellatus*.

Venom is a substance which is produced by an animal and is capable of producing toxic reactions when introduced into the body of another animal. Many different animal groups have evolved venom production, associated with a diverse array of venom delivery mechanisms. However, of all the many venomous creatures inhabiting this planet, snakes are undoubtedly the best known, and probably the most feared for their venomous bite. Since many thousands of people die from the effects of snake bite every year throughout the world such fear and notoriety are understandable, but it seems unreasonable to suppose that snakes have developed this efficient killing mechanism just to threaten man.

In view of the wide variety and apparent success of venomous snakes it would seem reasonable to suppose that the development of a venomous capacity has given these reptiles a selective advantage, and this is reflected in our current concepts of the taxonomy of all reptiles, where the venomous snakes are treated as the most advanced groups of reptiles now in existence.

What purpose does snake venom serve? Investigations suggest that there are three major roles for snake venom (Gans 1978). The first of these is to incapacitate prey. To swallow prey of significant size often requires the snake to swallow something with a larger diameter than the normal resting diameter of the snake itself. This remarkable feat is accomplished because of the highly modified lower jaw of the snake which can distend and stretch at the join at the front of the mouth between the right and left halves of the lower jaw. However, when large prey is swallowed, it puts much stress on many organs in the snake which are compressed and subjected to considerable pressure by the prey during swallowing. If the prey being swallowed is still moving, this further increases the degree of stress and increases the likelihood of damage to the snake itself. Thus if the prey is at first immobilised by venom it will decrease significantly the chance of damage to the snake during the act of swallowing. This in turn will allow the snake to swallow larger prey than would otherwise be possible, thus increasing the range of prey that it may accept, and decreasing the frequency with which it may need to feed. It

is obvious that this capacity to immobilise prey using venom gives the venomous snake a distinct advantage over its non-venomous cousins.

The second major value of venom for the snake is in facilitating digestion of the prey. Indeed it can be said that venom assists in digesting prey from the inside out. Like all reptiles, snakes are ectothermic animals. They are unable to maintain a constant inner body temperature by internal mechanisms, and thus are subject to changes in temperature of the external environment. Many stomach enzymes involved in the digestion of prey only work efficiently above certain temperature levels. If any reptile, including snakes, suffers a temperature drop to below the critical level, digestion of any food in the stomach will cease. Bacteria will continue to grow however, and the food in the stomach will probably putrefy, forcing the snake to either regurgitate the prey and therefore lose its food value, or risk causing significant internal damage to its digestive system. For any reptile the speed with which food can be digested is therefore of considerable importance. If venom can digest prey from the inside, while at the same time stomach enzymes are digesting the prey from the outside, complete digestion of the prey will be significantly faster than would otherwise occur. This is a clear advantage to the animal so equipped.

The third use of venom is as a deterrent for predators. This function of venom is perhaps the hardest to define and prove, but certainly a number of potential predators on snakes avoid contact with venomous snakes, and with some harmless reptiles which apparently mimic the colouration and/or behaviour of venomous snakes.

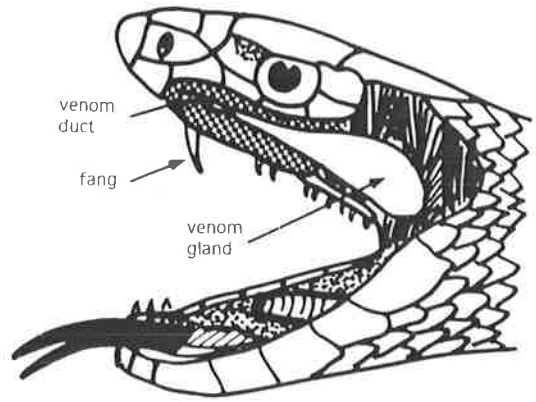
Thus it seems clear that the evolution of the venom gland and venom apparatus in snakes has conferred on them a considerable selective advantage. In some groups of venomous snakes this has been refined by the development of other sophisticated methods of prey detection and highly efficient venom delivery systems such as long mobile fangs, and an ability to strike at prey very rapidly. This has enabled these snakes to routinely feed on prey not usually available to non-venomous snakes found in the same habitat.

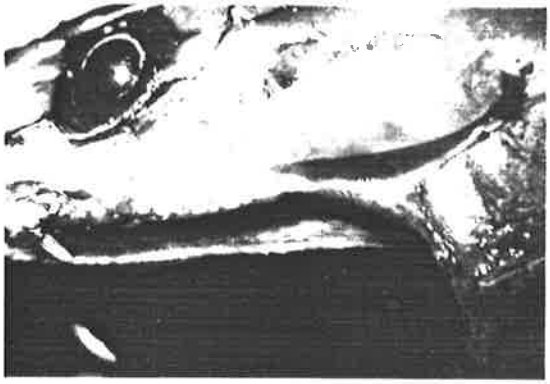
How venom glands have evolved is of course an area of scientific speculation. One interesting and well argued theory is that of Gans (1978). He has suggested that in ancestral snakes, long before the development of venom glands or fangs, most of the prey items would have been arthropods or molluscs. Many of these either contain or produce substances which are sticky or in other ways a nuisance in consuming this sort of prey. In particular they may damage the lining cells of the mouth and adhere to and possibly impair the function of the teeth. Therefore the secretion of any saliva-like substance which would break down such materials and keep the teeth clean and effective would clearly be an advantage. As such a system of secretions became more developed and refined it is possible that enzymes may have also been produced as these would help in breaking down the sticky mucin and other undesirable components from the prey. Through random change some of these would also become toxic for some prey items, and thus would have enhanced the snake's ability to catch and kill such prey. This would also facilitate the sampling of a wider variety of prey types. These factors would give snakes so equipped an advantage over their cousins, and set the stage for the development of even more complex salivary secretions with more efficient immobilisation and digestion of the prey, and ultimately predator deterrence. Thus we have the step-wise evolution of the modern venom gland from the oral secretions in the mouths of ancestral reptiles.

THE VENOM GLAND

The venom gland appears to have originated from some form of salivary gland. The position and structure of the venom gland vary in and between each of the three major groups of venomous snakes found in the world today. However, in nearly all venomous snakes the gland is located in the upper jaw, with the bulk of the gland being situated behind the eye.

All the important venomous snakes found in Australia belong to the family Elapidae. This wide-ranging family also includes the Cobras, Kraits, Mambas, and Coral Snakes. It is closely related to the Sea Snakes, and recent work by eminent Australian taxonomists suggests that the elapid snakes found in Australia may be





▲▲ Position of venom gland, duct and fang in a typical Australian elapid snake (after White 1981).

▲ Dissection of the head of a Brown Snake (*Pseudonaja textilis*), with removal of the scales, to show the venom gland behind the eye, the venom duct, and the exposed fang. A portion of the duct and adjacent venom gland have been stained to assist visualization.

more closely related to the Sea Snakes than they are to elapid snakes in other parts of the world (Cogger 1983; and Schwaner *et al.* 1985).

The anatomy of elapid venom glands has been reviewed by Kochva (1978) and Bdolah (1979). The glands consist of a main venom gland situated just beneath the scales of the posterior part of the head, posterior to the eye; and the more anteriorly placed accessory venom gland. The main gland is enclosed in a tough capsule of connective tissue and within this are several lobules with a serous secretory epithelium discharging venom into numerous small tubules which converge towards the centre of the gland, ultimately opening into a central duct which has only a small lumen. This then runs forward to the front of the head where the venom is ultimately discharged at the base of the fangs whence it enters the duct or groove in the fang which directs it near the tip of the fang. As the duct leaves the main venom gland it passes through the accessory (mucous) gland which contains a mucus secreting epithelium with much shorter tubules, again discharging into the main venom duct.

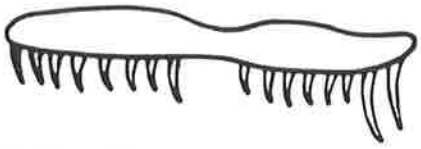
It appears that once venom is produced in the cells of the gland it is stored in the lumina of the tubules rather than intra-cellularly, and control of venom production is not fully understood at this stage. However, it appears that when a snake ejects venom from the fang, the venom used is only drawn from a proportion of the tubules of the gland, and it is in these that the cells lining the tubules are stimulated to produce more venom and so replenish the supply. Like most other aspects of the metabolism of reptiles, venom production is influenced by the temperature of the snake, and a cold snake is probably unable to produce venom in significant quantity.

The main venom gland overlies a number of muscles of the posterior part of the skull, and in particular is closely associated with the adductor superficialis muscle which is divided into superior and inferior segments. When this muscle contracts the venom gland is pulled and compressed against underlying structures, forcing venom to be expelled from the tubules into the duct and thus to the fangs. It would therefore appear to be possible for the snake to control the quantity of venom that it ejects at any given bite.

FANGS

Snake fangs have clearly evolved from the normal tooth structure in the upper jaw of primitive snakes. A variety of different fang positions and mechanisms have evolved, and these can be simply divided into three groups. The most primitive fangs are those of the Colubrid snakes which have fangs towards the back of the mouth, hence the common name of Back-fanged Snakes. Australia has several representatives of this family found in northern and eastern Australia, but none appears capable of causing significant injury to man. Probably the best known and most feared back-fanged snake in the world is found in southern Africa, namely the Boomslang (*Dispholidus typus*).

Colubrine aglyphous maxilla (no fangs)



Colubrine opisthoglyphous maxilla (back fanged)

Elapid proteroglyphous maxilla (front fanged)

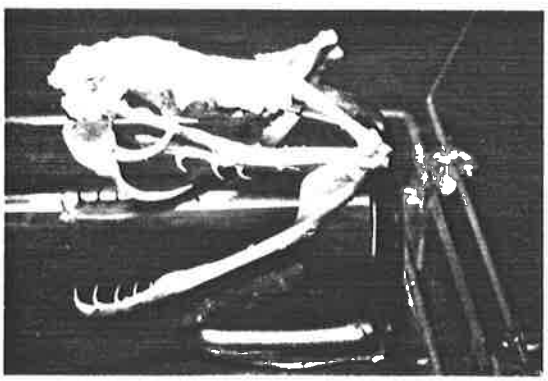
Viperid proteroglyphous maxilla (front fanged)

◄ The maxilla and fangs in modern snakes (after Webb *et al.* 1978).

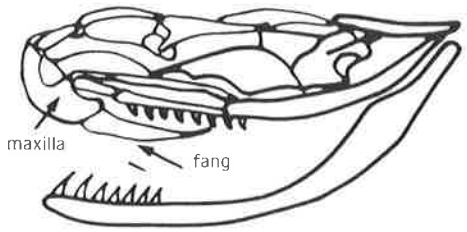
The most advanced fang structure is found in the Viperid snakes which include Rattlesnakes, Pit Vipers, Puff Adders, and Asps. This family is not represented at all in Australia. As can be seen in the associated figure, these snakes have a highly evolved upper jaw which allows very considerable rotation of the maxilla and attached fangs. This enables the fang to be folded up parallel to the roof of the mouth when not in use, and then moved into an erect position for biting prey. This high mobility has enabled these snakes to develop very long fangs and coupled with this many species also have large venom glands capable of producing large volumes of venom. The mobility and rotation of the fang is illustrated.

Intermediate between these two groups, though more closely associated with the Vipers, is the family Elapidae. Elapid snakes have fangs at the front of the mouth as do Vipers, and the fangs also arise from a modified maxilla. However, it is not nearly as mobile as that found in Vipers, and this has probably significantly limited the size of the fangs. Thus in elapid snakes fangs are only of moderate length. Fairley (1929), investigated the dentition and biting mechanism of Australian elapid snakes. A figure adapted from Fairley's pioneering work is presented, which illustrates the degree of rotation of fangs possible in the Australian Death Adder (*Acanthophis antarcticus*). This species, which despite the common name is not an adder at all, but a true elapid snake, appears to have the largest degree of rotation of the fang of any Australian elapid snake so far studied. The extent of rotation for this and other Australian species which have been studied is given in Table 1.

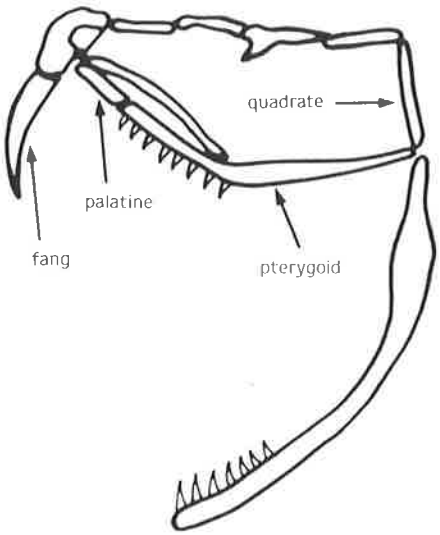
The fang itself is attached to the modified maxilla, and has a groove running almost its entire length. This is used to facilitate the movement of venom from the end of the venom duct at the base of the fang, through the groove, down to the tip of the fang where it enters the victim. In many species this groove is completely enclosed for most of its distance,



◄ The Western Diamond-Back Rattlesnake (*Crotalus atrox*), a viperid snake from North America. **a**. Live specimen with fangs moved into erect or strike position. **b**. Skull showing long fang and adjacent reserve fang, on the highly modified maxilla.



Mouth closed with fang parallel to palatine and pterygoid bones



Mouth open with fang erection as maxilla rotates as quadrate pulls prefrontal, frontal, parietal and supratemporal backwards

Table 1: Elevation angle of the fangs of various snakes (after Fairley 1929).

Snake	Elevation Angle
Death Adder *(Acanthophis antarcticus)*	40-50°
Tiger Snake *(Notechis scutatus)*	30-35°
Copperhead *(Austrelaps superbus)*	25-30°
Red-bellied Black Snake *(Pseudechis porphyriacus)*	25-30°
Brown Snake *(Pseudonaja textilis)*	10-15°

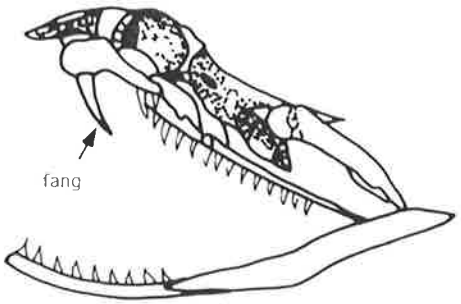
thus rendering the fang a highly efficient method of injecting venom, very similar to that of the medical hypodermic needle.

Clearly the length of the fang in any individual snake will depend on two major factors. The first is the species of snake involved, as some species tend to have very small fangs, while others are at the opposite extreme. The second factor is obviously the size of the snake, and clearly the larger the snake within any given species, the longer its fangs are likely to be. Details on the average fang length and expected range of length of fangs in adult snakes for several major Australian species are given in Table 2. From this it is clear that the common Brown Snake (*Pseudonaja textilis*) on average has the smallest fang of any of the species responsible for serious cases of snake bite in Australia. The Taipan (*Oxyuranus scutellatus*) has the largest fang.

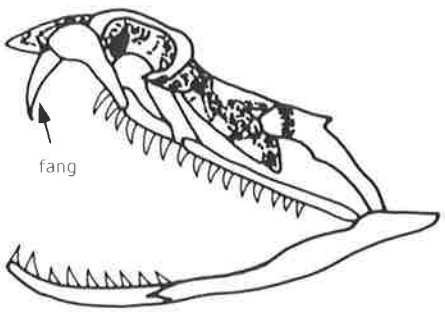
The distance between fangs will also vary between different species of snakes, and in theory might offer an attractive method of determining what snake was involved in any

◄ Dynamics of fang erection in the Rattlesnake (after Webb *et al.* 1978).

▼ Rotation of maxilla and attached fang and post maxillary teeth in the Death Adder (after Fairley 1929 and White 1981).



Fang position at rest



Fang position at maximum elevation

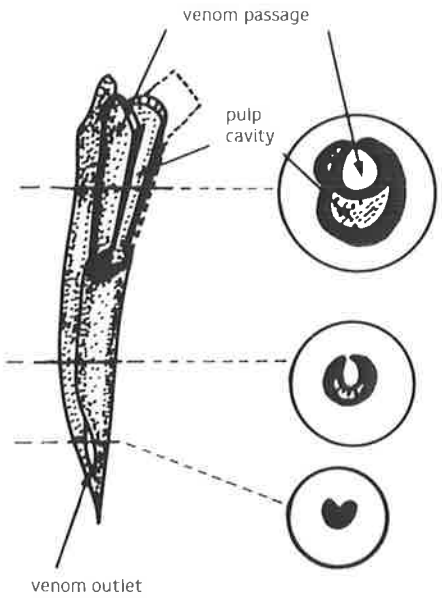


Table 2: Length of fangs of various snakes (after Fairley 1929; Covacevich *et al.* 1981; Kellaway and Thompson 1930).

Snake	Average length of fang (mm)	Range of length of fang (mm)
Taipan *(Oxyuranus scutellatus)*		7.9-12.1
Inland Taipan *(Oxyuranus microlepidotus)*		3.5-6.2
Mulga Snake *(Pseudechis australis)*	6.5	
Death Adder *(Acanthophis antarcticus)*	6.2	5.0-8.3
Red-bellied Black Snake *(Pseudechis porphyriacus)*	4.0	3.5-5.0
Tiger Snake *(Notechis scutatus)*	3.5	2.0-5.5
Copperhead *(Austrelaps superbus)*	3.3	3.0-4.5
Brown Snake *(Pseudonaja textilis)*	2.8	2.0-4.0

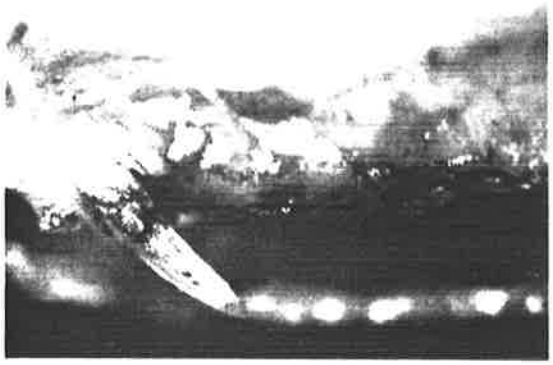
Table 3: Average distance between fangs (after Fairley and Splatt 1929).

Snake	Distance (mm)
Death Adder *(Acanthophis antarcticus)*	16
Tiger Snake *(Notechis scutatus)*	14
Red-bellied Black Snake *(Pseudechis porphyriacus)*	12
Copperhead *(Austrelaps superbus)*	11
Brown Snake *(Pseudonaja textilis)*	9

given snake bite. However, in practice this is not a reliable method, as many other factors are involved in the bite and determine the distance between fang puncture marks. The average measured distance between fangs in adult specimens of several major Australian species is given in Table 3.

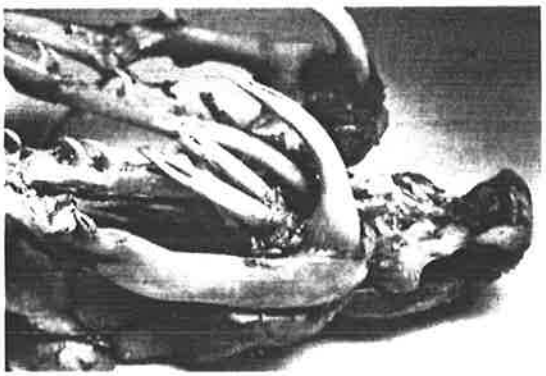
◄ Typical proteroglyphous fang of Australian elapid snake, with enclosed venom groove.

▼ The fang of an adult Brown Snake (*Pseudonaja textilis*), with the fang sheath removed. Note the exit point of the venom channel, near the tip of the fang.

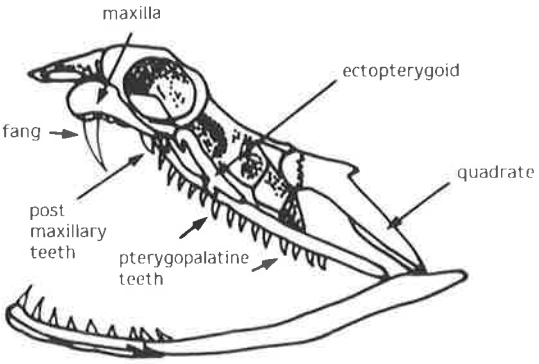


Fangs are occasionally lost through accident or natural attrition, but the snake has an effective back-up set of fangs which rapidly move into position. This is clearly seen in the Taipan (*Oxyuranus scutellatus*) as illustrated. Because of the back-up fang system it is occasionally possible for two fangs to be in action on one side, though this appears to be only rarely seen in Australian snakes.

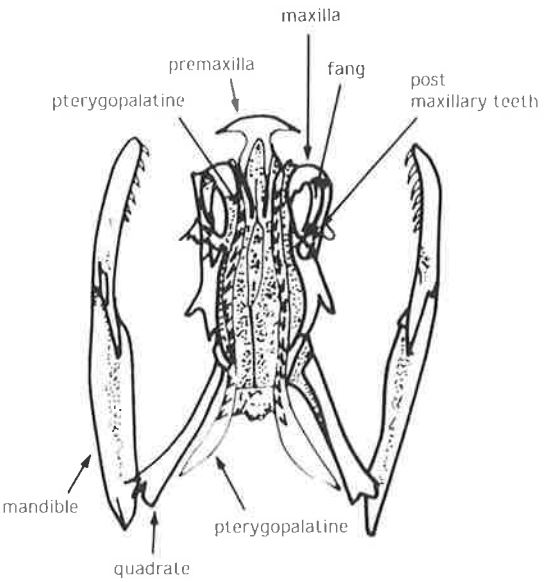
Fangs are by no means the only teeth found in the skulls of venomous snakes. In fact there is a wide variety of teeth found in a complex array which mirrors the complex osteology of the elapid snake skull. Thus in the upper jaw the modified maxilla which holds the fang also has several posterior maxillary teeth. In addition there are a large number of more medially placed pterygo-palatine teeth. Each of the bony elements containing these teeth may move separately, and this allows the prey to be systematically pulled into the mouth and passed on down the gullet of the snake. This sequential stepping motion in the movement of prey is very obvious if one watches a snake swallowing prey. In addition there are a number of teeth in the



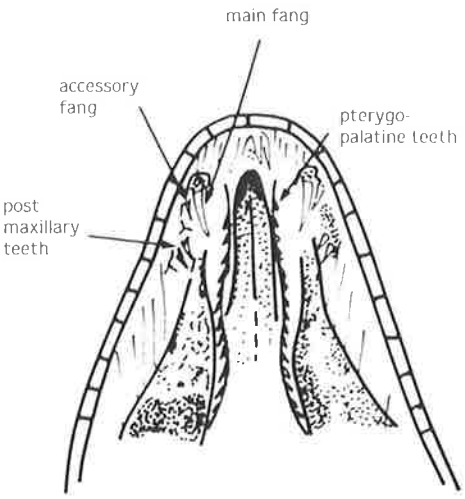
▲ Skull of a Taipan (*Oxyuranus scutellatus*) showing the main fang and several reserve fangs, ready to move into position should the main fang be lost. Note also the other teeth in the upper and lower jaws. These teeth may also enter the victim, helping to cause the complex pattern of tooth marks and scratch marks often seen at the site of a snake bite.



◄ Lateral view of the skull of a Death Adder (after White 1981).



► Ventral view of skull of a Death Adder. Note independent maxillae, mandibles and pterygopalatine bones and attached teeth (after White 1981).



▲ Ventral view of mouth of Death Adder showing fangs, post maxillary and pterygopalatine teeth (after White 1981).

lower jaw and as mentioned earlier the two halves of the lower jaw are joined by a highly elastic band at the front, which allows wide stretching of the lower jaw for ingestion of large prey. The trachea or airway is very mobile and is positioned towards the front of the lower part of the mouth and readily allows continued breathing while the snake is ingesting large prey. The forked tongue typical of all snakes is housed in a sheath just beneath the trachea.

VENOM DELIVERY

As mentioned earlier it appears that snakes have a considerable control over the quantity of venom they can eject through the fang. This is related to contraction of muscles around and connecting to the venom gland. In some species venom can be ejected at considerable pressure, and this is most dramatically illustrated in the Spitting Cobras found in Africa. These elapid snakes have the exit point at the tip of the fang facing forward and shaped in such a way that a fine stream of venom can be squirted forwards from the snake's open mouth a distance of two or three metres towards the victim. This clearly is a very effective method of deterring potential predators of these highly dangerous Cobras.

The mechanism of action of elapid snake bite was studied in detail by Fairley (1929), and the following description draws heavily on his important work. He concluded that there were four phases of biting and noted then that the local lesion caused by snake bite may show entry by only one fang, both fangs, or a variety of marks or scratches due to the movement of fangs and other teeth during the course of the bite. Obviously if the victim has been bitten more than once the pattern of punctures and scratches will become even more complicated and hard to interpret. Of all Australian elapid snakes Fairley believed that the Death Adder had the fastest strike and that all species except the Brown Snake (*Pseudonaja textilis*) strike with the jaws closed until the time of impact. It is known that Australian snakes will on occasion make a strike without opening the mouth at all, and the author has personally seen such apparent feint, or warning, strikes by several Australian snakes.

The actual strike of the snake is the first phase of the biting mechanism. Usually a snake will need to have its head and neck contracted into a form of S posture before the strike and this is a typical threat posture of many Australian snakes. The degree of S formation varies from species to species, and is particularly prominent in snakes such as the Brown Snakes (*Pseudonaja* species) and the Taipans (*Oxyuranus* species). It tends to be less prominent in the Tiger Snakes, which show considerable flattening of the neck just behind the head, in similar fashion to that seen in some Cobras, though in no way comparable in extent to that of the Cobras. Hissing and prolonged flicking of the tongue will often occur in this situation.

The second phase of the biting mechanism occurs as the snake's head reaches the victim. The mandibles are depressed by rapid contraction of the digastric, cervico-mandibular and vertebro-mandibular muscles, and simultaneously the fangs are rotated forward by the forward swing of the pterygo-palatine transverse arch. This is brought about by the simultaneous contraction of the spheno-pterygoid and parieto-pterygoid muscles.

The third phase is closure of the mouth brought about by the simultaneous contraction of the anterior, middle, and posterior temporal muscles which strongly elevate the mandibles.

Brown Snake, *Pseudonaja textilis*, aggressive stance. (Photograph: Chris Pollitt).

With closure of the jaws the fangs simultaneously penetrate the victim and immediately inoculation of venom occurs. As discussed earlier this is brought about by the contraction of muscles attaching to the gland which apply torsion to the gland and compress it against surrounding structures. The venom passes along the duct through the dental papilla into the vagina dentis which, by the tense approximation of its edges to each other, and to the surface of the fang, prevents escape of venom except into the fang channel, whence it is conveyed under pressure into the victim.

The fourth phase occurs immediately following the entry of the rotated and elevated fang. Accompanying the discharge of venom there occurs another set of movements due to the contraction of the retractor muscles operating on the pterygo-palatine transverse arch which results in the distribution of venom along an oblique, posteriorly directed, fang track. The fangs enter the tissue of the victim in a position of maximal elevation, and continue their subsequent course in a downward and backward direction, producing the scratch mark typically seen in cases of Australian snake bite. Small animals can be drawn into the mouth by this movement, while on large animals the snake's head is drawn forward over its victim. Also occurring during this movement is the contraction of the superior bundle of the parieto-palatine muscle, which tenses the vagina dentis, and contraction of the external pterygoid muscle which thus compresses the inferior surface of the venom gland. This, acting synchronously with the contraction of the adductor superficialis muscle, facilitates injection of venom under pressure.

Both the forward rotation and elevation of the fangs, and venom injection, are under voluntary control, so that the snake can inject no venom, some venom, or all its venom, unilaterally, or bilaterally. This degree of control explains why experiments in the past, using live snakes to bite test animals, are unreliable, and why the range of effects and degree of envenomation in man are so variable.

The quantity of venom produced has been the subject of research over many years. Most recent of these investigations are those carried out by Morrison and colleagues at Pearn's laboratories in Brisbane. Using modern techniques for identifying the type and quantity of venom, they have constructed controlled situations for measuring snake bite. Their work (Morrison *et al.* 1982, 1983, 1983-84) has built on the work of early researchers such as Fairley and Splatt (1929) and more recently Broad *et al.* (1979). The data on average and maximum amount of venom milked, range of venom injected at a first bite, and relationship between venom injected into the victim, and venom spilt on the skin, are listed in Tables 4, 5 and 6. It can be seen that most snakes leave a small sample of venom on the skin surface, and it is this finding which has allowed the development of sensitive clinical tests to determine the type of snake involved in clinical cases of snake bite (Theakston *et al.* 1977; Sutherland 1979; Coulter *et al.* 1980). It is also clear that the quantity of venom injected at any given bite is highly variable, and while not absolutely proven, it seems very probable that on occasion snakes will bite without injecting any venom at all. On the other hand it must be remembered that it is possible for any given snake to inject a large proportion of its available venom, which may be considerably more than the average venom yield milked from the particular species of snake. As antivenom doses are based on the average milking yield, it can be readily understood why some severe snake bites require several times the normal dose of antivenom.

Of course the quantity of venom produced is by no means the only measure of how potentially dangerous the snake may be. Of even more importance is the toxicity of the venom, and the assessment of the relative danger of the various Australian snakes must be based on a joint assessment of venom produced, and the toxicity of that venom. (See White, 'Australian Elapid Snakes: Venom Toxicity and Actions', this volume).

Table 4: Range of venom injected on first bite in an experimental model (after Morrison *et al.* 1982, 1983, 1983-84).

Snake	Range of venom injected (mg)
Inland Taipan *(Oxyuranus microlepidotus)*	0.7 to 45.6
Taipan *(Oxyuranus scutellatus)*	0.6 to 68.9
Death Adder *(Acanthophis antarcticus)*	3.4 to 99
Tiger Snake *(Notechis scutatus)*	1.1 to 31.9
Brown Snake *(Pseudonaja textilis)*	0.03 to 9.10
Rough-Scale Snake *(Tropidechis carinatus)*	0.23 to 22.57

Table 5: Venom injected by various elapid snakes, on first bite, in an experimental setting (after Morrison *et al.* 1982, 1983, 1983-84)

Snake	Mean Venom Injected (mg)	Mean Venom left on skin (mg)	Mean Total venom injected (mg)
Inland Taipan *(Oxyuranus microlepidotus)*	17.3	0.6	17.9
Taipan *(Oxyuranus scutellatus)*	20.8	0.9	21.7
Tiger Snake *(Notechis scutatus)*	12.7	0.8	13.5
Brown Snake *(Pseudonaja textilis)*	4.5	0.22	4.7
Rough-Scale Snake *(Tropidechis carinatus)*	6.0	0.1	6.1
Mulga Snake *(Pseudechis australis)*	61.6	0.07	61.7
Red-bellied Black Snake *(Pseudechis porphyriacus)*	1.3	0.9	2.1
Death Adder *(Acanthophis antarcticus)*	36.0	2.0	42.0

Table 6: Venom production in various snakes based on milking of venom (after Broad *et al.* 1979; Fairley and Splatt 1929).

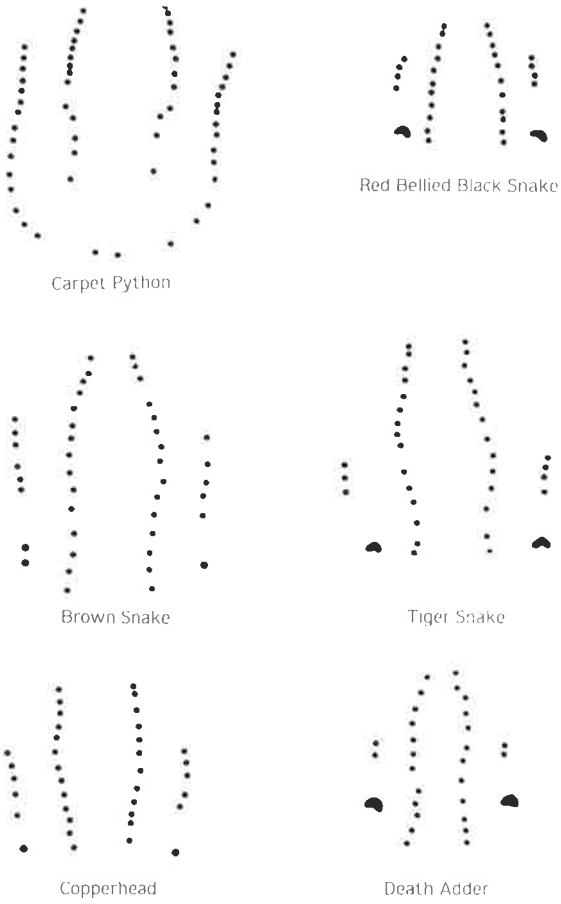
Snake	Average Yield (mg)	Maximum Yield (mg)
Mulga Snake *(Pseudechis australis)*	180	
Taipan *(Oxyuranus scutellatus)*	120	400
Death Adder *(Acanthophis antarcticus)*	78	236
Chappell Island Black Tiger Snake *(Notechis ater serventyi)*	75	388
Inland Taipan *(Oxyuranus microlepidotus)*	44	110
Red-bellied Black Snake *(Pseudechis porphyriacus)*	40	75
Tiger Snake *(Notechis scutatus)*	35	189
Copperhead *(Austrelaps superbus)*	20	85
Rough-Scale Snake *(Tropidechis carinatus)*	6	
Common Brown Snake *(Pseudonaja textilis)*	2	67
Indian Cobra *(Naja naja)*	169	610
Eastern Diamond Back Rattlesnake *(Crotalus adamanteus)*	410	848

Snake bite, as already mentioned, may result in a variety of scratch or puncture marks at the site of the bite. The clinical pattern seen for a variety of Australian dangerous snakes is discussed in detail in another paper in this volume (White, 'Australian Elapid Snakes: Management of Snake Bite'). It is, however, worth mentioning the theoretical bite patterns seen for a variety of Australia's dangerous snakes (illustrations based on the work of Fairley (1929)). It was noted by Fairley in 1929 that the greater the distance between the two fangs, the larger was the potential venom yield from the snake. It has been stated many times in the past in popular literature that non-venomous snakes such as pythons have a quite different pattern of bite marks from that seen in venomous snake bite. The reason for this can be seen in the bite impressions made in plaster. However in practice snake bite is often not a clean event, and such differentiation may not always be practical. A typical bite by a Brown Snake (*Pseudonaja* sp.) on the thumb is also illustrated. The mass of tiny scratches seen in this case certainly do not fit the classical description of a snake bite, yet the patient in this case received a substantial amount of venom, and without prompt administration of antivenom, the ultimate outcome may have been fatal.

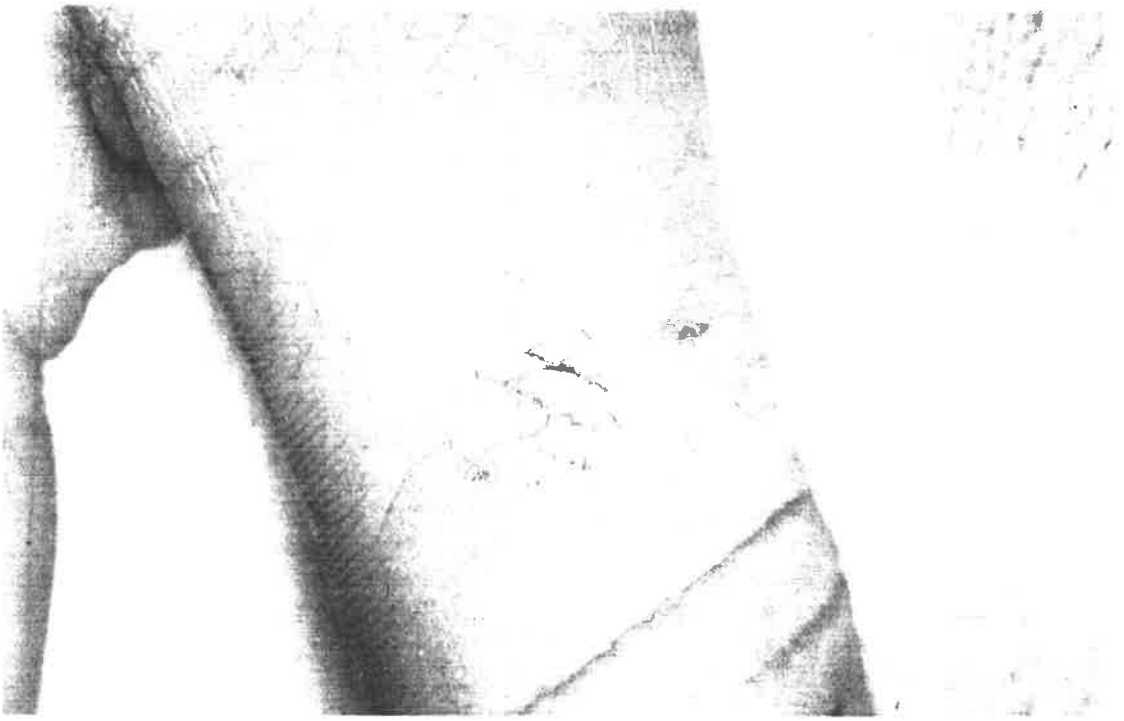
ACKNOWLEDGEMENTS

The author wishes to thank the many people who have contributed in various ways to the completion of this work. In particular, Drs B. Byok, K. Cheney, E. Sims, I. Speed and I. White have given valuable advice on drafts of the text, and their considerable help is gratefully acknowledged.

Dr D. Pounder was the prime mover of the survey of fatal snakebite in Australia, and he was responsible for obtaining many of the case records. His efforts in this regard are most gratefully acknowledged. The staff of the Photography Department, Adelaide Children's Hospital, have given excellent service in



Ideal upper jaw bite patterns of a non-venomous python and five dangerous Australian elapid snakes (after Fairley 1929 and White 1981).



The thumb of a 7 year old boy, seriously bitten by a Brown Snake (*Pseudonaja sp.*), showing multiple fine scratch marks where fangs, and possibly other teeth, have been pulled through the skin during the biting process.

preparation of prints (Chris Sprod, Mark Stevens) and preparation of art work (Colleen Lloyd, Jan Nichols).

The author is grateful for the generous support of Dr K. Cheney and his staff in the Haematology Department, Adelaide Children's Hospital; and of Mrs Olivia Lammas.

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ELAPID SNAKES: VENOM TOXICITY AND ACTIONS

Julian White

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◄ Skull of Taipan, *Oxyuranus scutellatus*.

Snake venoms contain a complex mixture of components which have a diverse array of actions both on prey and on human victims of snake bite. The primary functions of venom are to kill or immobilise the prey, and presumably to assist in digestion of the prey. In considering the various actions of venom, it is helpful to remember these basic functions of venom. Each species of snake will have its own particular requirements for the actions of venom, and the proportionate mix of actions will at least in part be determined by the types of prey it seeks. Inevitably, however, there will be considerable similarities between the venom actions of related species of snake, and this can certainly be seen in the Australian fauna.

TOXICITY

The toxicity of venom is in essence a measure of how powerful the venom is in causing a particular action. The standard way of assessing the degree of danger posed by a particular species of snake is to define the toxicity of the venom in terms of its ability to kill a test animal. This outwardly simple test is, of course, far from simple. Inevitably, different animals react to any given venom in different ways. Therefore some test animals may be more susceptible to a given venom than others. In the early years of venom testing this led to great confusion about which venoms were more toxic, as it depended to a large extent on the laboratory animal selected for the experiment. In recent years, however, some degree of standardisation has occurred. The World Health Organisation has produced a standardised formula for assessing the toxicity of venoms, both in terms of their lethal action, and in terms of some of their more specific actions; such include the ability to destroy muscle, to destroy tissue at the site of the bite, and to cause destruction of blood cells. (Theakston and Reid 1983).

The standard measure of lethal toxicity of any venom is the LD_{50} (lethal dose, 50%) measurement. This in fact means the quantity of the test venom required to kill on average 50% of the test animals given that particular dose of venom. Another less commonly used measure of toxicity is the LD_{100}, or minimum dose whch will kill 100% of the test animals. To determine an LD_{50} it is necessary to select a

range of venom doses and administer each dose to a large pool of test animals to ensure that the overall result will be statistically significant. Unfortunately this can consume a large number of test animals, which at the present time are usually laboratory mice. The route of injection of the venom or venom component under test is of course important. Common routes used include sub-cutaneous (SC) (that is, injected just beneath the skin); intravenous (IV) (that is, injected directly into the blood stream); intraperitoneal (IP) (that is, injected into the abdominal cavity of the test animal); and lastly intramuscularly (IM) (that is, injected into the muscle of the test animal, usually selecting a muscle of some size, such as a large muscle of the leg). Thus when an LD_{50} assessment of the toxicity of venom is made it must always be stated what the test animal is; usually an average weight for the test animal is given, and the route of injection of the venom must be stated. A further problem has recently been introduced, and that is the solution used to dilute the venom under test. It has been found that for some Australian snake venoms, standard saline solution may not successfully remove all venom components from binding to laboratory glassware; the addition of a small amount of blood protein (namely, bovine serum albumin) to the saline solution, significantly enhances retrieval of all venom components, and therefore increases the tested toxicity of the venom (Broad *et al.* 1979).

Besides the fact that venom components may bind to laboratory equipment (thus removing those components so bound from any testing of the toxicity of the venom) there is also the possibility that some venom components may be rapidly destroyed after milking of the snake. This means that testing of a venom toxicity in a laboratory situation may not completely reflect the true toxicity of the venom in either the snake's prey, or in a human victim of snake bite. Nevertheless, modern techniques in toxinology have certainly greatly increased the reliability of testing for venom toxicity. New methods of testing for toxicity are still being pioneered. It is to be hoped that important test procedures such as the LD_{50} which currently require the death of some laboratory animals, may be replaced with newer tests which do not require such use of laboratory animals. It must be stressed, however, that such alternative tests are not yet

Common Brown Snake, *Pseudonaja textilis*, being milked.

standardized, and that the use of the LD_{50} is still justifiable in scientific terms, providing certain guidelines are met (I.S.T. 1986).

Most LD_{50} testing relates to whole snake venom, and thus is a way of comparing the relative lethality of each species of snake. In terms of the toxicity of their venoms, Australian snakes have the dubious distinction of being technically the most dangerous snakes in the world. Table 1 is adapted from the work of Broad *et al.* (1979). These workers performed a comprehensive survey of Australian dangerous snakes and selected overseas snakes to determine the LD_{50} (S.C. 18–21 g mice) in both normal saline dilutions, and in bovine serum albumin/saline dilutions. From this table it can be seen that the Inland Taipan, otherwise known as the Small-scaled Snake (*Oxyuranus microlepidotus*) is technically the most venomous snake in the world. It is closely followed by the Common Brown Snake (*Pseudonaja textilis*) and then the Taipan (*Oxyuranus scutellatus*). No overseas venomous snakes appear to have venom as toxic as these Australian species, and indeed the venom of most Vipers, such as the North American Rattlesnakes, appears to be far less toxic than that of the Australian elapid snakes.

However, the actual toxicity of the venom cannot be used as the sole determinant of how dangerous the snake is likely to be. Obviously other factors are important, and particularly important is the quantity of venom that the snake is likely to inject. Obviously factors such as the length of the fang, how much venom the snake injects on the first bite, and how much venom is available at different times, are all

Table 1: Relative toxicity of snake venoms in mice (after Broad *et al.* 1979).
(S = sea-snake; E = elapid snake; EA = Australian elapid snake; V = viperid snake.)

Snake		LD_{50} (Saline) in mg/kg	LD_{50} (bovine serum albumin)
Inland Taipan (*Oxyuranus microlepidotus*)	EA	0.025	0.010
Common Brown Snake (*Pseudonaja textilis*)	EA	0.053	0.041
Taipan (*Oxyuranus scutellatus*)	EA	0.099	0.064
Tiger Snake (*Notechis scutatus*)	EA	0.118	0.118
Reevesby Island Tiger Snake (*Notechis ater niger*)	EA	0.131	0.099
Beaked Sea Snake (*Enhydrina schistosa*)	S	0.164	0.173
W.A. Tiger Snake (*Notechis ater occidentalis*)	EA	0.194	0.124
Chappell Island Tiger Snake (*Notechis ater serventyi*)	EA	0.338	0.271
Death Adder (*Acanthophis antarcticus*)	EA	0.400	0.338
Western Brown Snake (*Pseudonaja nuchalis*)	EA	0.473	0.338
Copperhead (*Austrelaps superbus*)	EA	0.560	0.500
Indian Cobra (*Naja naja*)	E	0.565	0.500
Dugite (*Pseudonaja affinis*)	EA	0.660	0.560
Papuan Black Snake (*Pseudechis papuanus*)	E	1.09	1.36
Stephens Banded Snake (*Hoplocephalus stephensii*)	EA	1.36	1.44
Rough-scaled Snake (*Tropidechis carinatus*)	EA	1.36	1.09
King Cobra (*Ophiophagus hannah*)	E	1.80	1.91
Blue-bellied Black Snake (*Pseudechis guttatus*)	EA	2.13	1.53
Collet's Snake (*Pseudechis colletti*)	EA	2.38	—
Mulga Snake (*Pseudechis australis*)	EA	2.38	1.91
Red-bellied Black Snake (*Pseudechis porphyriacus*)	EA	2.52	2.53
Small-eyed Snake (*Cryptophis nigrescens*)	EA	2.67	—
Eastern Diamond Back Rattlesnake (*Crotalus adamanteus*)	V	11.4	7.70

determinants of how dangerous any given snake bite will be. Nevertheless, the two most important determinants remain the toxicity of the venom as defined by the LD_{50}, and the average quantity of venom delivered at a bite.

The average and maximum milked yields of venom for several species of snake have been analysed by Broad *et al.* (1979) and correlated with the LD_{50} to give an estimate of LD_{50} doses. This information is summarized in Table 2.

Morrison *et al.* (1984) have proposed a toxicity index relating these two pieces of information, to try to determine the real level of danger posed by a variety of Australian venomous snakes. Information based on this is given in Table 3. This predicted lethality index clearly shows that the Inland Taipan (*Oxyuranus microlepidotus*) is potentially the most dangerous snake in the world. However, the Common Brown Snake (*Pseudonaja textilis*), which has a very toxic venom, produces a small amount of this venom. Thus it can be seen that the combination of moderately to highly toxic venom combined with large amounts of venom delivered rank Australian snakes such as the Tiger Snake (*Notechis scutatus*) and the Death Adder (*Acanthophis antarcticus*) very high on the practical scale of danger. Practical experience with snake bite has shown that such a lethality index does indeed correlate well with the likelihood of a fatal outcome following a bite by a particular species of snake. Table 4 shows the death rate for bites by several Australian species of snake, based on statistics gathered in the early part of this century, prior to the availability of antivenom. This clearly shows that Tiger Snakes and Death Adders caused death far more frequently than Brown Snakes. Fortunately there have been only two reported cases of bites by the Inland Taipan, both in reptile fanciers, both of whom survived following antivenom treatment (Trinca 1969; Mirtschin *et al.* 1984).

In one sense, however, it is slightly unreal to consider the lethality of snakes purely in terms of their potential for killing at any given bite. It is now well established that many thousands of people die from snake bite around the world each year (Swaroop and Grab 1954; Theakston *et al.* 1977). Fortunately, very few of these deaths occur in Australia. Some species of Viper, such as the Carpet Viper (*Echis carinatus*) of Africa and South West Asia, and Russell's Viper (*Vipera russellii*) found across Asia, probably kill many thousands of people each year; thus although they may be technically less dangerous than the Australian species, they are a far more important cause of snake bite (Pugh and Theakston 1980).

VENOM ACTIONS

Venom is not a single lethal substance, but a complex mixture of components, which have a diverse array of actions on their victim. The classification of these venom components and their actions is not currently subject to rigid guidelines, and there are therefore several different ways of classifying these components and their actions. It is beyond the scope of this paper to canvass this topic completely, but some basic concepts of relevance particularly to the treatment of snake bite will be discussed.

In considering venom components it is perhaps worth briefly mentioning how these components may be separated. Attempts at separating venom into different components

Table 2: Average venom yields and various species of snake (LD_{50} for mice) (after Broad *et al.* 1979).

Snake	Average Venom Yield		Maximum Venom Yield	
	mg	LD_{50} doses	mg	LD_{50} doses
Inland Taipan (*Oxyuranus microlepidotus*)	44	217,821	110	544,554
Taipan (*Oxyuranus scutellatus*)	120	94,488	400	314,961
Brown Snake (*Pseudonaja textilis*)	2	2,469	67	80,426
Chappell Island Tiger Snake (*Notechis ater serventyi*)	75	13,838	388	71,587
Indian Cobra (*Naja naja*)	169	16,900	610	61,000
Death Adder (*Acanthophis antarcticus*)	78	11,538	236	34,911
King Cobra (*Ophiophagus hannah*)	421	11,050	(500?)	13,123
Eastern Diamond Back Rattlesnake (*Crotalus adamanteus*)	410	2,662	848	5,505

Table 3: Predicted lethality index (after Morrison *et al.* 1983–84) for various snakes, based on S.C. LD_{50} (saline) in mice (after Broad *et al.* 1979), published average venom milkings (after Broad *et al.* 1979), and experimental studies on venom injected at a first bite (after Morrison *et al.* 1982, 1983, 1983–84).

Snake	Lethality Index based on:	
	Mean experimental venom	Average venom milked
AUSTRALIAN ELAPIDS		
Inland Taipan (*Oxyuranus microlepidotus*)	692	1760
Taipan (*Oxyuranus scutellatus*)	210	1212
Tiger Snake (*Notechis scutatus*)	108	297
Death Adder (*Acanthophis antarcticus*)	90	195
Brown Snake (*Pseudonaja textilis*)	85	38
Mulga Snake (*Pseudechis australis*)	26	77
Rough-scaled Snake (*Tropidechis carinatus*)	4.4	4.4
Copperhead (*Austrelaps superbus*)	—	36
Red-bellied Black Snake (*Pseudechis porphyriacus*)	0.5	16
EXOTIC ELAPID		
Indian Cobra (*Naja naja*)	—	299
EXOTIC VIPER		
Eastern Diamond Back Rattlesnake (*Crotalus adamanteus*)	—	36

Table 4: Mortality rate for various snakes, if no antivenom used (after Fairley 1929).

Snake	No. of Patients	Deaths	% Mortality
Taipan* (*Oxyuranus scutellatus*)	8	6	75%
Death Adder (*Acanthophis antarcticus*)	10	5	50%
Tiger Snake (*Notechis scutatus*)	45	18	40%
Brown Snake (*Pseudonaja textilis*)	70	6	8.6%
Red-bellied Black Snake (*Pseudechis porphyriacus*)	125	1	0.8%

*Based on review of published cases, prior to availability of antivenom. This may well be an underestimate of the mortality rate for untreated cases of Taipan snakebite.

have been the subject of much toxinologic scientific endeavour for at least a hundred years. Many methods are available, and predictably the most modern techniques allow the most complete degree of separation of components. The majority of venom components of importance in snake venoms have at least some protein content, and separation may be performed to differentiate between different sized components, differently charged components, or antigenically different components. Unfortunately many components will be very similar in all three areas, and therefore what is studied as a single component

may in truth be a mixture of several similar components.

This situation is further complicated by the fact that the exact content of components in the venom and their relative mix within that venom show significant variations at least between different members of the same species of snake, and possibly within any individual snake from time to time (Glenn *et al.* 1983). In the past much venom research has been performed on large quantities of venom pooled from venom milked from many specimens of a single species of snake. As not all the snakes of a given species will have a particular component, pooled samples of venom from any given species will vary. Thus important components isolated from one pooled sample of venom may be completely absent from further samples of pooled venom from the same species of snake. This is obviously a source of great frustration to the researchers involved, and also has relevance in the management of snake bite, for it cannot be said with any absolute certainty that a particular species of snake will have an absolutely defined and constant set of actions on its victims.

Having defined a particular venom component, one cannot then assume it will have one and only one action. Thus some major components of snake venoms may have a variety of actions on both prey and human victims of snake bite. As an example, the phospholipase A2 toxins, which are of importance in a number of Australian snake venoms, may cause a variety of effects in man including paralysis and destruction of voluntary muscle. Inevitably then any classification of venom components and actions must be at least partially artificial.

BIOCHEMICAL/PHYSIOLOGICAL CLASSIFICATION OF VENOM COMPONENTS

This classification, based on Karlsson (1979), Lee and Ho (1982), and Iwanga and Suzuki (1979), is not definitive.

1. PRESYNAPTIC NEUROTOXINS (PHOSPHOLIPASE A2)

Presynaptic neurotoxins are found in several snake venoms, and are particularly prominent in some Australian snake venoms. Their mode of action will be discussed in more detail under the functional classification (below) but in essence they work presynaptically at the neuromuscular junction to inhibit the release of transmitter from the nerve ending thus causing muscle paralysis. They all appear to be based on a phospholipase A2 enzyme, the exact mode of action of which is still uncertain. They are probably the most powerful and lethal components of any snake venom.

2. POSTSYNAPTIC NEUROTOXINS

These neurotoxins also cause paralysis of voluntary muscle at the neuromuscular junction, acting postsynaptically by binding to the transmitter receptor on the muscle. Specifically they bind to the nicotinic acetylcholine receptor and as such behave in a similar fashion to the well known South American poison curare. They are proteins, and are subdivided into short and long chain varieties. While certainly lethal, they are not as toxic as the presynaptic neurotoxins.

3. MEMBRANE TOXINS

Membrane toxins appear to change the permeability of cell membranes in a wide range of tissues and as such may cause a wide range of problems. They may act as cardiotoxins, affecting the function of the heart, or as lytic factors, or cytotoxins, causing destruction of cells. It is possible that at least part of the action of the presynaptic neurotoxins may also be related to a membrane toxin action.

4 .ENZYMES

An enzyme is an organic catalyst, that is, a substance which promotes a particular chemical reaction. Strictly speaking a phospholipase A2 is a form of enzyme, but because of its fairly specific action in relation to snake venoms, it is usually classified separately. Looking at the action of other enzymes known to occur in snake venoms they may cause:

a)Local capillary damage and tissue necrosis, e.g. caused by proteinases, other phospholipases, arginine esterhydrolases, and hyaluronidases.

b)Diverse coagulant and anticoagulant actions e.g. proteinases and phospholipases.

c)Induce acute hypotension and pain due to release of vasoactive peptides such as kinin releasing enzymes.

d)Other non-specific actions.

5. OTHER COMPONENTS

Inevitably a number of venom components cannot be classified under any of the above headings and these include peptides, nucleosides, and metal-ions, the roles of which are at present uncertain.

FUNCTIONAL CLASSIFICATION OF VENOM ACTIONS

For medical practitioners trying to understand the complexity of problems occurring in a patient with serious snake bite it is probably most helpful to think of venom components in terms of their effects on the patient. Obviously a single venom component may cause a variety of effects in the snake bite victim, but essentially it is the effects which are important for the patient, not how many components may be causing a given effect. There are major differences in the effects of venoms from various groups of snakes, and attention in this chapter will be focussed on those effects of importance in treating Australian snake bite.

1. NEUROTOXINS

Neurotoxins are substances which have the capacity to paralyse or otherwise disrupt transmission of information by the nervous system. Obviously this may occur at many levels starting with the most complex in the central nervous system, and in particular the brain. From there information will be transmitted via a variety of nerves and nerve interchange systems (ganglions) culminating in transmission from the end of the nerve cell to whichever organ may be involved. However, snake venom neurotoxins appear to have most of their action at just one site in the nervous system, namely the neuromuscular junction. This is the region where the nerve impulse is transmitted to voluntary muscle, and when that impulse is received the muscle will contract.

Actual transmission of information to contract from the nerve ending to the muscle is carried out by release of a neurotransmitter substance, acetylcholine (ACh) (Fig. 1). Thus on receipt of an impulse from the brain to cause a given muscle fibre to contract, the nerve ending releases ACh stored in the nerve terminal in synaptic vesicles. This ACh then crosses the very narrow gap to the surface of the muscle cell where it interacts with a specific ACh receptor in the surface membrane of the muscle cell. This then initiates the complex series of events which rapidly cause the muscle fibres in the muscle cell to contract. Having effected this process, the ACh is then quickly destroyed by the enzyme acetyl cholinesterase (there are in fact several acetyl cholinesterase enzymes).

The exact mechanisms involved in the release of ACh from the nerve terminal are still incompletely understood, though obviously of crucial importance. At the final stage synaptic vesicles (containing the ACh), stored within the end of the nerve, fuse to the internal surface of the nerve cell membrane, and transport the ACh across this membrane to exit the nerve cell and thus reach the receptor on the muscle cell. The binding of ACh to a receptor on the muscle cell is somewhat better understood, largely as a result of research into neurotoxins in snake venoms. The pre- and postsynaptic neurotoxins found in snake venoms, including some Australian snake venoms, have already proven to be of inestimable value as research tools in understanding the function of the normal neuromuscular junction in man and its disorders.

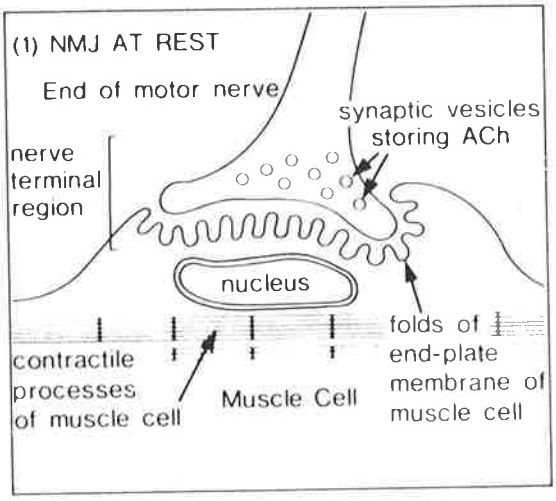
Postsynaptic neurotoxins from snake venom have been used to isolate the ACh receptor on the muscle cell, and this in turn has allowed understanding of a number of important disease states of neuromuscular transmission, the best known of which is myasthenia gravis (Dawkins *et al.* 1979). Thus research into snake venoms has implications and a value far beyond the narrow but important confines of the actual treatment of victims of snake bite.

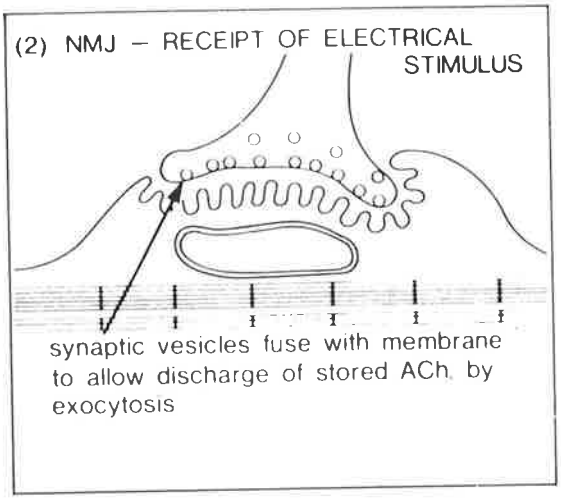
As mentioned earlier snake venom neurotoxins can be divided into two principal varieties, both of which appear to act principally at the neuromuscular junction. The first are the presynaptic neurotoxins which appear to have reached their zenith of development in Australian snake venoms. The second are postsynaptic neurotoxins which are widely represented in Australian snake venoms (Fig. 1).

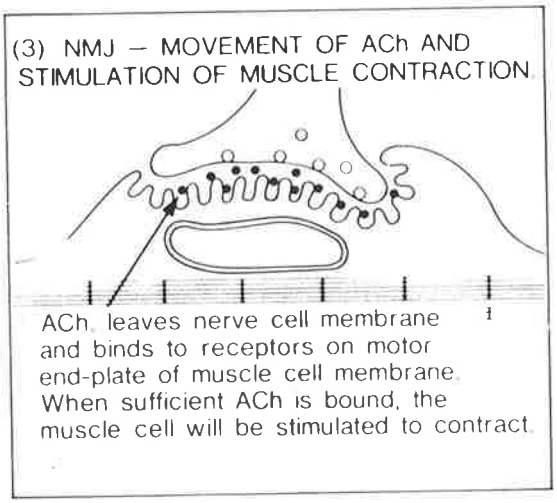
2. PRESYNAPTIC NEUROTOXINS

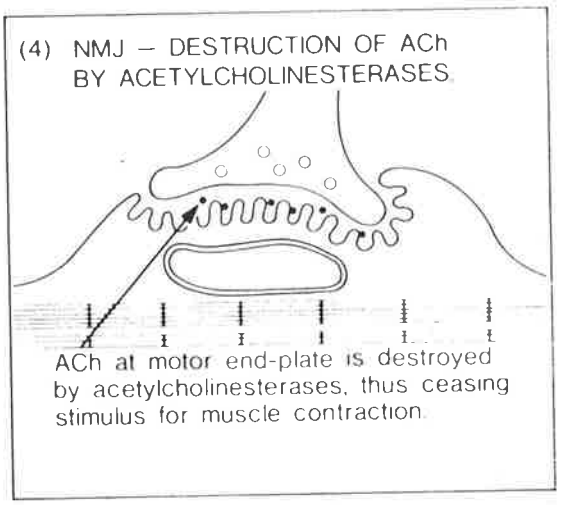
The first presynaptic neurotoxin discovered was β bungarotoxin from the Taiwanese Krait (*Bungarus multicinctus*) (Chang and Lee 1963). This powerful presynaptic neurotoxin has a molecular weight of approximately 22,000; has two major sub-units, of which the active sub-unit is a basic phospholipase A2; and has an LD_{50} in mice of 20 micrograms/kilogram IV. Since the discovery of this very potent neurotoxin, presynaptic neurotoxins have been found in the venom of several other elapid snakes and a few viperid snakes.

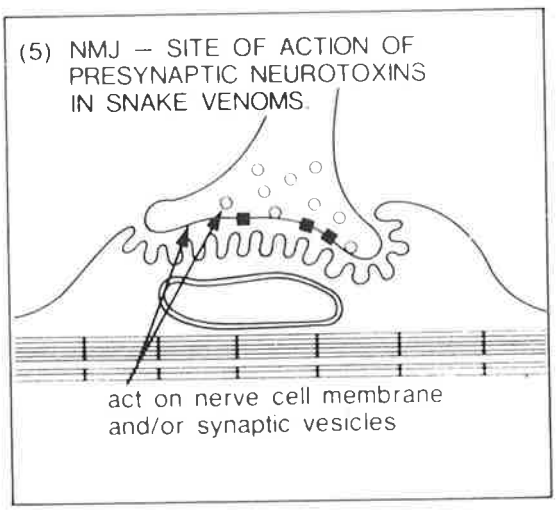
The first presynaptic neurotoxin isolated from

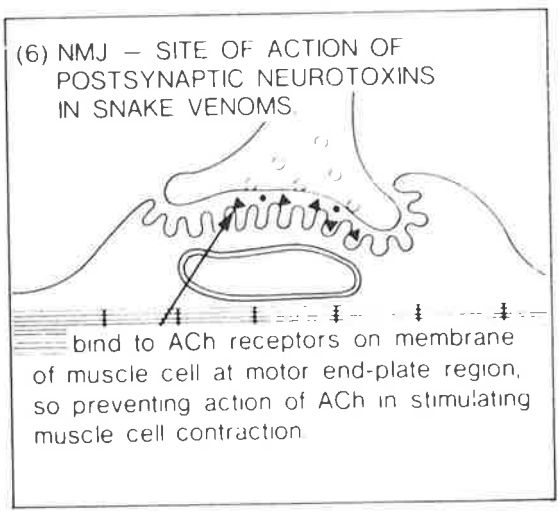

(1) NMJ AT REST
End of motor nerve
synaptic vesicles storing ACh
nerve terminal region
nucleus
folds of end-plate membrane of muscle cell
contractile processes of muscle cell
Muscle Cell


(2) NMJ – RECEIPT OF ELECTRICAL STIMULUS
synaptic vesicles fuse with membrane to allow discharge of stored ACh by exocytosis


(3) NMJ – MOVEMENT OF ACh AND STIMULATION OF MUSCLE CONTRACTION
ACh leaves nerve cell membrane and binds to receptors on motor end-plate of muscle cell membrane. When sufficient ACh is bound, the muscle cell will be stimulated to contract.


(4) NMJ – DESTRUCTION OF ACh BY ACETYLCHOLINESTERASES
ACh at motor end-plate is destroyed by acetylcholinesterases, thus ceasing stimulus for muscle contraction.


(5) NMJ – SITE OF ACTION OF PRESYNAPTIC NEUROTOXINS IN SNAKE VENOMS.
act on nerve cell membrane and/or synaptic vesicles


(6) NMJ – SITE OF ACTION OF POSTSYNAPTIC NEUROTOXINS IN SNAKE VENOMS.
bind to ACh receptors on membrane of muscle cell at motor end-plate region, so preventing action of ACh in stimulating muscle cell contraction.

an Australian elapid snake was Notexin, isolated from venom of the Tiger Snake (*Notechis scutatus*). (Karlsson *et al.* 1972) Notexin constituted 5% of the crude venom, and like β bungarotoxin contains a basic phospholipase A2 with 119 amino acids in a single peptide chain, with 7 di-sulphide bridges. The molecular weight is approximately 13,500, and the LD_{50} in mice is 20 micrograms/kilogram IV.

A presynaptic neurotoxin has also been found in the venom of the Taipan (*Oxyuranus scutellatus*), and has been named Taipoxin (Fohlman *et al.* 1976). A similar component, Paradoxin has also been found in the closely related Inland Taipan (*Oxyuranus microlepidotus*) (Fohlman 1979). Taipoxin is a 1:1:1 ternary complex of sub-units labelled alpha, beta and gamma. The alpha and beta sub-units are each of 120 amino acids with 7 di-sulphide bridges. The gamma sub-unit has 135 amino acids with 8 di-sulphide bridges. The alpha sub-unit is the only sub-unit with lethal neurotoxicity, but is much less toxic than the whole Taipoxin complex (LD_{50} equals 300 micrograms/kilogram for the alpha sub-unit compared to 2 micrograms/kilogram for whole Taipoxin, IV, mouse). The molecular weight of Taipoxin is 45,600.

A further presynaptic neurotoxin has been isolated from the venom of the Common Brown Snake (*Pseudonaja textilis*) (Coulter *et al.* 1979; Su *et al.* 1983; Coulter *et al.* 1983). This toxin, textilotoxin, constituted 4–5% of the crude venom, but possessed nearly 70% of the total venom toxicity. Its structure is not presently defined, but the molecular weight is 88,000. Its LD_{50} in mice is 0.6 microgram/kilogram, and it would therefore appear to be the most lethal snake venom neurotoxin known.

Two toxins with both pre- and post-synaptic neurotoxic activity have been isolated from Rough Scaled Snake (*Tropidechis carinatus*) venom, though their structures have not yet been elucidated (Morrison *et al.* 1984).

A schematic representation of the structure of some of these presynaptic neurotoxins is given in Fig. 2. The common feature of all of these neurotoxins is the presence of a basic phospholipase A2 component, and it is becoming

◄ Fig. 1. A schematic diagram of the mammalian Neuro-Muscular Junction (NMJ), illustrating: **1**. the normal resting anatomy; **2–4**. the process of stimulation and muscle contraction; **5**, **6**. the site of action of snake venom neurotoxins.

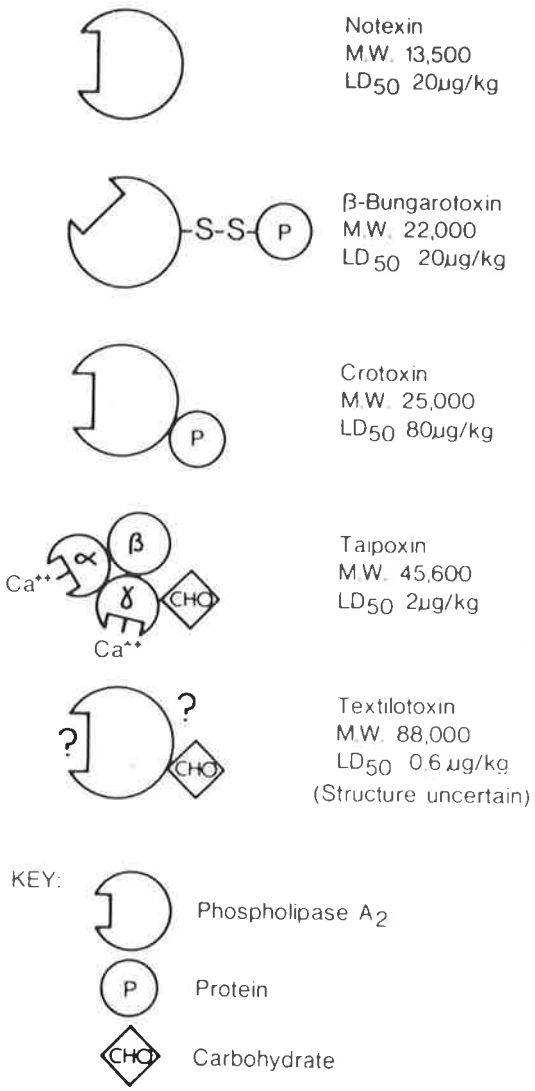


Fig. 2. Structure, molecular weight and toxicity (mice) of presynaptic neurotoxins (after Eaker 1978).

increasingly clear that the phospholipase activity is crucial to the overall toxicity of these compounds (Fohlman *et al.* 1979; Eaker 1978; Lee and Ho 1982; Su *et al.* 1983; Su and Chang 1984). These presynaptic neurotoxins cause a progressive neuromuscular paralysis, which may take two to three days to kill the test animal. Paralysis sufficient to be lethal may, however, occur much more rapidly in some cases.

When these toxins reach the neuromuscular junction, presumably carried in the blood stream, they apparently bind to the membrane

of the nerve ending. There is then a latency period of 30-60 minutes before actual paralysis occurs (Cull-Candy *et al.* 1976; Thesleff 1979). The length of the latency period is reduced by nervous activity, though not by increased toxin concentration (Thesleff 1979). The toxin is rapidly and irreversibly bound to the membrane and binding may take as little as 3-5 minutes (Thesleff 1979). There is an apparent immediate inhibition of the high affinity choline transport system in synaptosomes (Dowdall *et al.* 1977). During the latency period, spontaneous transmitter release is increased, evidenced by an increase in miniature end plate potentials (Thesleff 1979). Nerve impulse evoked release is also altered, with a rapid fall in amplitude. At the time of paralysis there is a marked reduction in the number of vesicles containing ACh, and there is a wide variation in the morphology and size of the remaining vesicles, and swelling and disruption of mitochondria has also been observed. (Eaker 1978; Fohlman *et al.* 1979: Thesleff 1979; Lee and Ho 1982).

Various mechanisms have been invoked to explain the mechanism of action of these presynaptic neurotoxins. Thesleff (1978) has suggested that these 'presynaptic neurotoxins rapidly bind with a high degree of specificity and irreversibility to the axolemma of cholineric nerve terminals, possibly to the membrane protein involved in the high affinity choline transport system. Upon binding the neurotoxins enter the nerve terminal axoplasm by the endocytic mechanism. Once inside the nerve terminals the phospholipase A2 activity of the toxin exerts its hydrolytic action on the vesicle membrane and on other intracellular membranous constituents. This causes a reduction in synaptic vesicle number, a fusion of vesicles to form large synaptic vesicles, and damage to mitochondria and other intracellular organelles storing calcium thereby increasing the level of free calcium inside the nerve terminal. Paralysis results when the number of synaptic vesicles is sufficiently reduced by the hydrolytic phospholipase A2 activity of the toxin molecule'.

It seems probable that whatever the precise mechanisms, the considerable structural complexity of these toxins is reflected in multiple binding sites reflecting different modes of the toxin's action (Lee and Ho 1982). It is still

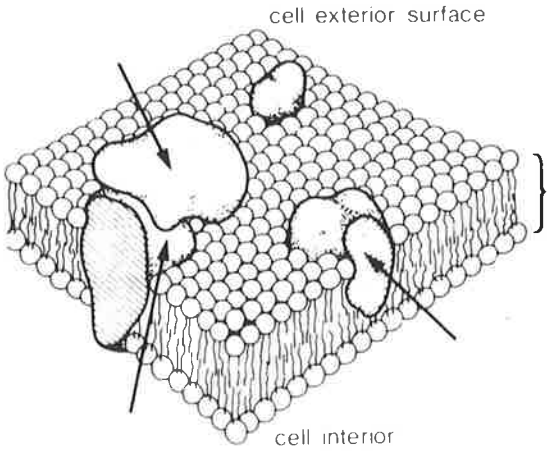
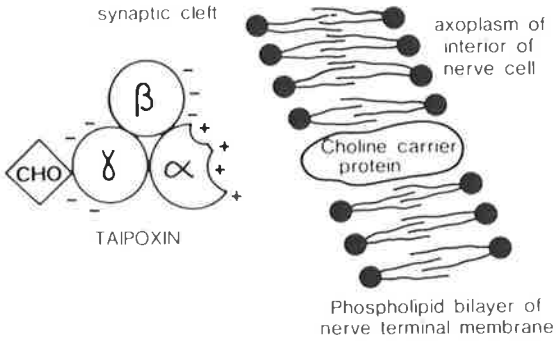
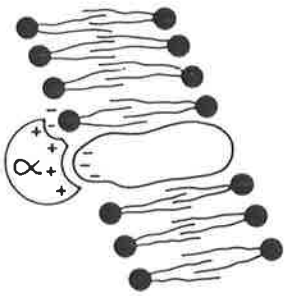


Fig. 3. Schematic diagram of cell membrane (based on Singer-Nicholson (1972) fluid mosaic model) to show binding of toxin to membrane receptor.



(1) ELECTROSTATIC ORIENTATION



(2) DISSOCIATION, BINDING, CATALYSIS

Fig. 4. A tentative model for the mode of presynaptic action of taipoxin (after Fohlman *et al.* 1979).

uncertain whether the toxin actually passes through the membrane of the nerve ending, or whether its effects are exerted via binding to receptor proteins on the external surface of the nerve ending membrane, with toxic effects occuring due to perturbation and damage to this membrane, possibly associated with enzymic action of the phospholipase A2 on the phospholipid bi-layer of the membrane (Figs 3,4).

It is worth noting that once paralysis has been established in human victims of bites by snakes containing these presynaptic neurotoxins, antivenom is unlikely to reverse this paralysis (Campbell 1967). This has also been demonstrated experimentally (Datyner and Gage 1973). This is in contrast to the situation frequently observed following paralysis by venoms containing purely postsynaptic neurotoxins, where the paralysis may be reversed by the administration of appropriate antivenom. (Campbell 1966).

3.POSTSYNAPTIC NEUROTOXINS

Though less potent than the presynaptic neurotoxins, powerful postsynaptic neurotoxins are found in many elapid venoms, and their actions are better understood. Kellaway and Holden (1932), Kellaway *et al.* (1932), and Kellaway (1932) showed a curare-like neuromuscular blocking action of Australian snake venoms on both the frog neuromuscular and rabbit diaphragm preparations. The venoms of the Death Adder (*Acanthophis antarcticus*) the Common Copperhead (*Austrelaps superbus*), the Taipan (*Oxyuranus scutellatus*), the Tiger Snake (*Notechis scutatus*) and the Common Brown Snake (*Pseudonaja textilis*) all showed this effect. The postsynaptic block was rapidly established, though subsequent work has shown it is less severe than that caused by presynaptic neurotoxins. It is nevertheless quite lethal, as illustrated by the venom of the Death Adder (*Acanthophis antarcticus*) for which untreated bites have a significant mortality rate, principally due to the effects of paralysis caused by postsynaptic neurotoxins (Campbell 1966).

These elapid postsynaptic neurotoxins bind to and inhibit the function of the nicotinic acetylcholine receptor at the neuromuscular junction in voluntary muscle (Patrick 1979). One of the best characterised postsynaptic neurotoxins also came from the Taiwanese Banded Krait (*Bungarus multicinctus*) (Chang and Lee 1963), and this component has been labelled Alpha Bungarotoxin. From this the label Alpha Neurotoxins has been derived, and these toxins have been instrumental in defining the nature of the normal acetylcholine receptor at the neuromuscular junction (Patrick 1979; Jeffrey 1979). Though curare-like in some respects at least some of these postsynaptic neurotoxins show irreversible block (Chang and Lee 1963; Mebs 1978). From a practical clinical point of view it appears that these postsynaptic neurotoxins having bound to the acetylcholine receptor on the surface of the muscle cell, may at least in some cases be susceptible to attack by antivenom (Datyner and Gage 1973). Experience with Death Adder bites in Papua New Guinea has clearly illustrated that at least for this species of snake, which has no detected presynaptic neurotoxins, antivenom therapy is able to completely reverse paralysis caused by these postsynaptic neurotoxins (Campbell 1966, 1967).

Postsynaptic neurotoxins have been found in many Australian elapid venoms, including those which also contain presynaptic neurotoxins. They may be divided into long and short chain varieties (Mebs 1978).

In fractionating Tiger Snake (*Notechis scutatus*) venom four postsynaptic neurotoxins were noted (Karlsson *et al* 1972). The first of these had an LD_{100} (mouse) of 100 micrograms/kilogram and comprised 60 amino acids. The second had an LD_{100} of 150 micrograms/kilogram with about 70 amino acids, and the third an LD_{100} of 600 micrograms/kilogram and 120 amino acids. The fourth toxin was not examined in such detail. All caused respiratory distress in test animals, and the first two appeared similar to Cobra toxin from the Common Indian Cobra (*Naja naja*). The third in sub-lethal doses caused respiratory distress for up to 48 hours.

Similarly in fractionating Taipan (*Oxyuranus scutellatus*) venom postsynaptic neurotoxins were found (Fohlmann *et al.* 1976). Fraction 3 showed neurotoxin paralytic effects, with an LD_{100} of 100 micrograms/kilogram, and Fraction 4 caused respiratory paralysis in mice with an LD_{100} of 120 micrograms/kilogram.

Fractionation of Death Adder (*Acanthophis antarcticus*) venom has revealed five separate lethal fractions, including a postsynaptic neurotoxin, Acanthophin A (Scheumack *et al.* 1979). It is a single chain of 63 amino acids with

4 di-sulphide bridges, an approximate molecular weight of 7,700, and an intraperitoneal LD_{50} (mice) of 0.16 milligram/kilogram, and is thus characterized as a short chain neurotoxin. A second short chain neurotoxin, Acanthophis antarcticus C has been isolated from this venom, and has 62 amino acids and an LD_{50} of 0.08 microgram/kg (IM, mouse) (Kim and Tamiya 1981a). A long chain neurotoxin has also been isolated from this venom, called Acanthophis antarcticus B, and has 73 amino acids, a MW of 8135, and an LD_{50} of 0.13 microgram/g (IM mouse) (Kim and Tamiya 1981b).

As most attention is focussed on presynaptic neurotoxins at present, the postsynaptic neurotoxins in Australian snake venoms do not appear to be the subject of current intensive research. However, recently the venom of the Rough Scaled Snake (*Tropidechis carinatus*) has been investigated, and Fraction 4, with a molecular weight less than 10,000, had strong irreversible neurotoxic action at the neuromuscular junction, which may be either presynaptic or postsynaptic or both in mode of activity (Morrison *et al.* 1984).

MYOTOXINS

The myolytic or muscle destroying properties of some snake venoms have been well documented, especially for Sea Snakes (Mebs 1978). The precise mode of action of these myolysins is still incompletely understood, and is the subject of intense research by several groups, especially the Newcastle-upon-Tyne group led by Harris. Their research suggests that the myotoxic components of elapid snake venoms possess phospholipase A2 activity, and are capable of hydrolysing the major phospholipids represented in the surface membrane of muscle cells (Harris 1983). It is possible that hydrolysis of the phospholipids in the membrane is causally related to the initiation of the muscle destruction. The degree of muscle destruction depends on the metabolic variety of muscle fibre involved, and immature muscle cells appear resistant to these toxins.

Table 5: Myotoxic activity of snake venoms based on testing of fractionated venom in mice (Mebs and Samejima 1980) and whole venom in primates (Sutherland *et al.* 1981).

Snake	Venoms Containing Myolytic Fractions	Whole Venoms With Myolytic Activity
Taipan (*Oxyuranus scutellatus*)	–	+
Inland Taipan (*Oxyuranus microlepidotus*)	–	mild
Brown Snake (*Pseudonaja textilis*)	–	–
Western Brown Snake (*Pseudonaja nuchalis*)	not tested	mild
Dugite (*Pseudonaja affinis*)	not tested	mild
Tiger Snake (*Notechis scutatus*)	–	+
Black Tiger Snake (*Notechis ater*)	–	not tested
Copperhead (*Austrelaps superbus*)	+	+
Mulga Snake (*Pseudechis australis*)	+	+
Red-bellied Black Snake (*Pseudechis porphyriacus*)	+	+
Collet's Snake (*Pseudechis colletti*)	+	not tested
Death Adder (*Acanthophis antarcticus*)	–	–
Rough-scaled Snake (*Tropidechis carinatus*)	not tested	+

Although the destruction of the muscle cell may be quite significant, it appears that the cell will regenerate, commencing about 3 days after exposure to the toxin and the regeneration is usually complete after 3-4 weeks. While these regenerated muscles are functionally normal, there are some detectable differences from the original muscle structure.

A review of the myotoxic actions of Australian snake venoms has recently been undertaken (Mebs and Samejima 1980; Sutherland *et al.* 1981) (Table 6). These studies using laboratory animals, with measurement of plasma creatine kinase levels as an indicator of muscle injury (Sutherland *et al.* 1981), confirmed that the venoms of the Tiger Snake (*Notechis scutatus*), Mulga Snake (*Pseudechis australis*), the Rough Scaled Snake (*Tropidechis carinatus*), the Copperhead (*Austrelaps superbus*), and the Red Bellied Black Snake (*Pseudechis porphyriacus*) were all powerfully myolytic and the venom of the Taipan (*Oxyuranus scutellatus*) was less so. The venoms of the Inland Taipan (*Oxyuranus microlepidotus*), the Dugite (*Pseudonaja affinis*), and the Western Brown Snake or Gwardar (*Pseudonaja nuchalis*) all showed some myolytic activity, although the significance of this was equivocal in the opinion of the researchers. The Common Brown Snake (*Pseudonaja textilis*), and the Death Adder (*Acanthophis antarcticus*) did not show any myolytic activity. In the clinical situation evidence of muscle destruction has occurred following bites by the Tiger Snake (*Notechis scutatus*) (Hood and Johnson 1975), the Mulga Snake (*Pseudechis australis*) (Rowlands *et al.* 1969), the Small Eyed Snake (*Cryptophis nigresens*) (Furtado and Lester 1968) and the Taipan (*Oxyuranus scutellatus*) (Bridgen and Sutherland 1981).

As might be expected the phospholipase A2 components of some Australian snake venoms, in addition to their presynaptic neurotoxic action, also appear to be potent myolysins (Karlsson 1979). This certainly appears to be true of Notexin from Tiger Snake (*Notechis scutatus*) venom, and Taipoxin from Taipan (*Oxyuranus scutellatus*) venom (Harris *et al.* 1975; Harris *et al.* 1977; Harris and Johnson 1978; Harris *et al.* 1980; Harris and MacDonnell 1981; Harris and Maltin 1982). A lethal myotoxin has also been isolated from Mulga Snake (*Pseudechis australis*) venom (Leonardi *et al.* 1979). One of the four fractions identified from this venom, and labelled Mulgatoxin A, is a basic single polypeptide chain of 122 amino acids, with 7 di-sulphide bridges. It causes myoglobinuria in mice, and has an LD_{50} (mouse) of 200 micrograms/kilogram; its activity appears specific for skeletal muscle, causing massive cell damage both in vitro and in vivo. The molecular weight is 13,700. This component also appears to be a phospholipase A2.

The role of myotoxins in killing prey, and in human victims of snake bite, remains unclear. However, it seems likely that severe muscle destruction with consequent myoglobinuria, may lead to significant kidney problems, and even acute kidney failure (Rowland *et al.* 1969; Hood and Johnson 1975; Sutherland *et al.* 1981; Brigden and Sutherland 1981). Obviously major destruction of skeletal muscle in a prey animal would significantly assist digestion of the prey by the snake.

HAEMOTOXINS

Haemotoxins are venom components either interfering in some way with the normal processes of blood clotting including platelet function, or causing actual destruction of circulating blood cells, particularly red blood cells. The latter action, haemolysis, is usually far less important than the effect on blood clotting which may cause major problems for the victim of snake bite (White 1981, 1983b). Nearly all snake venoms have some effect on the blood clotting system although in some cases this may be minor.

In understanding the effect of venom on these systems the complexity of the systems involved in normal blood clotting must be appreciated. Platelets, very small non-nucleated cells circulating in the blood stream in large numbers, have an important role in the clotting process, both mechanically as they bind together to plug any hole in the blood vessel, and pharmacologically as they produce or assist in the production of several key components in the clotting process, some chemical reactions of which occur on the phospholipid surface membrane of platelets. The complex step-wise chemical processes leading to the formation of a stabilised blood clot are known as the coagulation cascade (Fig. 5) and this involves

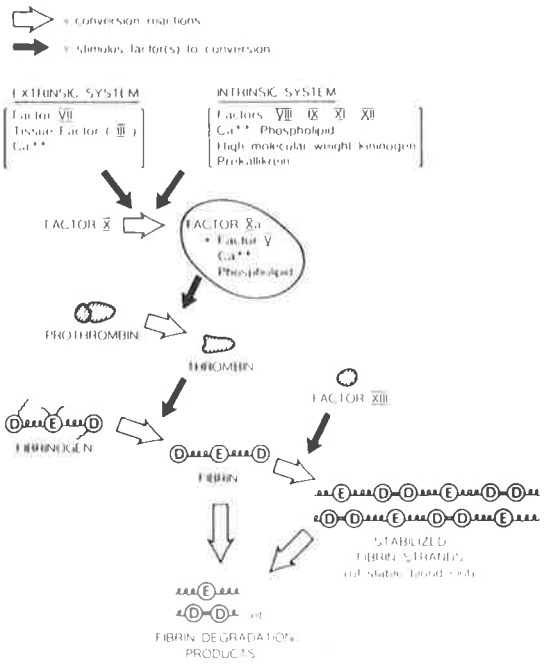


Fig. 5. Schematic diagram illustrating the basic reactions of the coagulation cascade, leading to the formation of a stabilized fibrin clot.

sequential conversion of various clotting factors with the penultimate production of thrombin from prothrombin, which leads to conversion of fibrinogen to fibrin, and in the normal situation stabilisation of fibrin into a stable fibrin blood clot. This clot will eventually be broken down, and the fibrin reduced to fibrin degradation products. It is however possible to bypass the production of a stable clot, moving from fibrinogen through to production of fibrin degradation products. The homeostatic mechanisms involved in control of this process include positive and negative feedback effects of various factors produced during activation of the cascade. In particular it should be noted that fibrin degradation products themselves act as powerful anticoagulants, that is, inhibiting further activation of the clotting process. As many of the steps in this cascade are essentially enzymatic, it is not surprising to find that snake venoms, with their potent enzyme components, can mimic various factors, and cause activation of selected parts of the coagulation cascade.

COAGULANT ACTION

The most important haemotoxins in Australian snake venoms have a coagulant action. This implies that they may cause activation of part or all of the coagulation cascade leading to blood clot formation. In practice, however, only a portion of the cascade in its final stages is activated, and frequently the clinical effect is production of large quanitites of fibrin degradation products and depletion of other factors, leading to a clinical situation of anticoagulation (White 1981, 1983b). Nevertheless, as the actual effect of the venom component is a coagulant one, it is probably more appropriate to classify their action as coagulant rather than anticoagulant.

Such action may occur at three major points in the coagulation cascade. The first is the formation of a prothrombin converter which may be either physiological such as factor Xa, or entirely artificial such as a factor Xa analogue. Obviously such activation can occur anywhere in the cascade above factor X.

The second potential point of action is the direct conversion of prothrombin to thrombin. The third potential point of action is the direct conversion of fibrinogen either to fibrin or to fibrin degradation products.

Table 6: Clotting times of factor V-deficient plasma, with four Australian elapid venoms, showing complete and incomplete prothrombin activation (after Denson 1969).

Snake	Clotting Time of Normal Plasma (sec.)	Clotting Time of Factor V Deficient Plasma (sec.)	Type of Prothrombin Activator
Tiger Snake (*Notechis scutatus*)	10	67	Incomplete
Death Adder (*Acanthophis antarcticus*)	12	170	Incomplete
Taipan (*Oxyuranus scutellatus*)	8	8	Complete
Brown Snake (*Pseudonaja textilis*)	6	6	Complete

Table 7: Coagulant action of venoms on human plasma (after Marshall and Herrmann 1983). *Venom diluted due to potency.

Snake	Clotting Time in Normal Plasma (secs.)	Clotting Time in Factor V Deficient Plasma (secs.)	Comment
TIGER SNAKE GROUP			
Notechis scutatus	11.5	36	Factor V dependent
Notechis ater occidentalis	10.5	58	
Notechis ater niger	11	62	
Austrelaps superbus (Copperhead)	190	>300	
Tropidechis carinatus (Rough-scaled Snake)	8	31.5	
TAIPAN GROUP			Factor V independent
*Oxyuranus scutellatus**	11	18	
Oxyuranus microlepidotus	7	7.5	
BROWN SNAKE GROUP			
*Pseudonaja textilis**	7.5	12	
*Pseudonaja affinis**	8	12	
Pseudonaja nuchalis	10	11.5	
BLACK SNAKE GROUP			Factor V dependent
Pseudechis australis	139	>300	
Pseudechis porphyriacus	32	115	
Pseudechis papuanus	137	>300	
DEATH ADDER GROUP			
Acanthophis antarcticus	123	>300	

Table 8: Ability of whole venoms to induce a coagulopathy in experimental animals (primates) (after Sutherland *et al.* 1981).

Snake	Evidence of coagulopathy, based on abnormally prolonged (+) clotting in:	
	Prothrombin Time	Partial Thromboplastin Time
Brown Snake (*Pseudonaja textilis*)	+	+
Western Brown Snake (*Pseudonaja nuchalis*)	+	+
Dugite (*Pseudonaja affinis*)	+	+
Tiger Snake (*Notechis scutatus*)	+	+
Copperhead (*Austrelaps superbus*)	+	+
Rough-scaled Snake (*Tropidechis carinatus*)	–	–
Taipan (*Oxyuranus scutellatus*)	+	+
Inland Taipan (*Oxyuranus microlepidotus*)	+	–
Mulga Snake (*Pseudechis australis*)	+	+
Red-bellied Black Snake (*Pseudechis porphyriacus*)	–	+
Death Adder (*Acanthophis antarcticus*)	+	+

Australian elapid venoms with a coagulant action appear to possess powerful prothrombin converters, thus bypassing all of the earlier parts of the coagulation cascade. Martin (1893) first demonstrated the thrombotic action of Red-bellied Black Snake (*Pseudechis porphyriacus*) venom, and Tiger Snake (*Notechis scutatus*) venom. Subsequent work by Kellaway (1929a, b, c, d, e, 1930, 1931); and Kellaway and Williams (1929) showed that only some Australian elapids have significant effects on coagulation. Later Denson (1969) studied some Australian elapid venoms with thrombotic effects, finding that Tiger Snake (*Notechis scutatus*) venom was an incomplete prothrombin activator, and that the Taipan (*Oxyuranus scutellatus*) and Common Brown Snake (*Pseudonaja textilis*) venoms were complete prothrombin converters (Table 6). More recently Chester and Crawford (1982) and Marshall and Herrmann (1983) have surveyed the coagulant activity of most important Australian snake venoms. The earlier work of Denson has been confirmed. A summary of these results is provided in Table 7. It must be emphasised however that the key clinical result of this coagulant action is a defibrination syndrome (Champness 1966; White 1981; 1983). This has largely correlated with clinical findings in laboratory animals experimentally envenomated by Australian snakes (Sutherland *et al.* 1981). A summary of their conclusions is given in Table 8

Isolation of the actual venom components causing prothrombin activation has proved difficult. A potent prothrombin activator from Taipan (*Oxyuranus scutellatus*) venom has been partially characterised (Walker *et al.* 1980). This group estimated that the prothrombin activator was a large protein complex with a molecular weight of 380,000, in a two chain structure with a large chain of 220,000 and a smaller chain of 160,000. The relationship in activity of the two chains was uncertain. It appeared enzymatic in action, and was able to activate prothrombin in the absence of any other agent including phospholipid, calcium, and factor V. Coulter *et al.* (1983) have also reported a large molecular weight venom component with potent coagulant activity from the venom of the Common Brown Snake (*Pseudonaja textilis*). The nature of this component has yet to be fully elucidated. Further research into this area will require highly sophisticated fractionation techniques to isolate the active components of significance.

Table 9: Anticoagulant activity of Australian elapid venoms (after Kaire 1964).

Snakes — in descending order of anticoagulant activity	
1	Mulga Snake (highest anticoagulant activity) (*Pseudechis australis*)
2	Papuan Black Snake (*Pseudechis papuanus*)
3	Red-bellied Black Snake (*Pseudechis porphyriacus*)
4	Copperhead (*Austrelaps superbus*)
5	Death Adder (*Acanthophis antarcticus*)
6	Tiger Snake (*Notechis scutatus*)
7	Taipan (*Oxyuranus scutellatus*)
8	Brown Snake (lowest anticoagulant activity) (*Pseudonaja textilis*)

ANTICOAGULANTS

As mentioned previously the coagulant components of snake venoms may clinically cause a situation of anticoagulation, and this has led to considerable confusion about anticoagulants in snake venom. However, it is possible for venom components, including enzymatic components, to block a portion or portions of the normal coagulation cascade pathway thus preventing any coagulation, and acting as true anticoagulants. Kaire (1964) surveyed the apparent anticoagulant activity in a number of major Australian snake venoms (Table 9). More recently Marshall and Herrmann (1983) selected those venoms which in their survey had not shown any evidence of coagulant activity and tested these for anticoagulant properties using a tissue thromboplastin inhibition test, and an agarose gel technique for detection of coagulation inhibitors. They specifically looked for evidence of antithrombin activity, and fibrinogenolytic activity. Neither of these activities was found in any of the venoms tested, but they reported that venoms of the Copperhead (*Austrelaps superbus*), the Mulga Snake (*Pseudechis australis*), the Papuan Black Snake (*Pseudechis papuanus*), and the Death Adder (*Acanthophis antarcticus*) all apparently possessed powerful anticoagulants. It must be admitted, however, that these results are somewhat confusing as both the Mulga Snake

and the Papuan Black Snake either experimentally (Sutherland *et al.* 1981), or clinically (Champness 1966; Campbell and Chesterman 1972), have shown definite evidence of a coagulopathy which is apparently due to defibrination.

PLATELET ACTIONS

The action of Australian elapid venoms on human platelet function has been rarely reported. It is known that some Viper venoms have components with potent effects on human blood platelets, in particular affecting platelet aggregation (Davey and Esnouf 1969; Ouyang *et al.* 1983; Marlas 1982). Preliminary work in our own laboratory suggests that at least some Australian venoms have a potent platelet aggregating action. However, the clinical role of this activity is far from clear. Indeed in the majority of cases of coagulopathy following Australian snake bite platelet numbers are apparently normal (White 1981, 1983b; Sutherland *et al.* 1981). It is possible, however, that the potent defibrinating action of some snake venoms may be related at least in part to platelet activation.

HAEMOLYSINS

Haemolysins occur in many snake venoms and have been noted to exist in Australian elapid venoms for many years. Kellaway and others made extensive examinations of the haemolytic properties of these venoms (Kellaway and Williams 1933a; Kellaway and Williams 1933b; Holden 1934) and their work was extended by Doery and Pearson (1961). Using the venom of the Red-bellied Black Snake (*Pseudechis porphyriacus*) as a control the haemolytic activity of most major Australian elapid venoms was documented (Table 10). From this it is clear that members of the Black Snake (*Pseudechis*) group have the most potent haemolytic activity, and that the venoms of the Brown Snake (*Pseudonaja*) group are only very weakly haemolytic.

Two forms of haemolytic activity are evident. There is direct haemolysis by venom components, and indirect haemolysis by phospholipase A components which appear to work by converting lecithin to lysolecithin, which will then cause haemolysis.

The exact relationship of these haemolytic components to other venom components has yet to be established. However, clinically haemolysis is not a significant contributor to mortality following envenomation by Australian elapid snakes. Experimental envenomation studies where evidence of severe coagulopathy and/or muscle destruction has occurred, have not revealed any evidence of haemolysis, and the conclusion of the researchers was that haemolysis is unlikely to be a significant clinical problem except in exceptional circumstances (Sutherland *et al.* 1981).

Table 10: Haemolytic activity of Australian elapid venoms, as a percentage of activity of Red-bellied Black Snake venom (after Doery and Pearson 1961).

Snake	Phospholipase A	Direct Haemolysis
Red-bellied Black Snake (*Pseudechis porphyriacus*)	100	100
Mulga Snake (*Pseudechis australis*)	150	50
Papuan Black Snake (*Pseudechis papuanus*)	150	100
Copperhead (*Austrelaps superbus*)	50	50
Tiger Snake (*Notechis scutatus*)	50	2-3
Taipan (*Oxyuranus scutellatus*)	100	<1
Death Adder (*Acanthophis antarcticus*)	50	<1
Brown Snake (*Pseudonaja textilis*)	<20	<12

LOCAL ACTIONS OF VENOM

Many non-Australian snake venoms, particularly those of vipers such as the Rattlesnakes, are known to cause potent local tissue destruction. Similarly the venom of some Cobras may cause significant local tissue destruction. However, in Australia major local tissue injury is very rarely seen and cannot be considered a significant component of the snake bite problem (White 1981, 1983a). Obviously a variety of enzyme components of snake venoms may cause local tissue destruction, and the spreading action of hyaluronidase as found in snake venom is widely reported. Probably because of the relative insignificance of local tissue injury in Australian snake bite, there appear to be no reports of research into components of Australian venoms capable of causing such injury. Nevertheless, minor local injury does occur following bites by some species. This is to be discussed in the chapter on envenomation (White, this volume).

OTHER ACTIONS

Clinically a number of responses to envenomation other than those already discussed may occur following Australian snake bite. In particular the collapse of the victim, associated with either unconsciousness, or some degree of impaired consciousness or irritability is frequently seen, particularly in children (White 1981). While it is possible that initial collapse may be secondary to a severe hypotension (low blood pressure) induced by the snake venom, in the author's experience such collapse or irritability is most usually seen without an associated detectable hypotension. The mechanism of action of the central nervous system problems is unknown, and there is therefore no information on venom components which may cause such action. Apparent epileptic fits following envenomation do occur, and have been reported after a bite by the Red-bellied Black Snake (*Pseudechis porphyriacus*) (Sutherland 1979), and in the author's personal experience following bites by the Common Brown Snake (*Pseudonaja textilis*), and the Tiger Snake (*Notechis scutatus*) (White 1981; White *et al.* 1984). The role of snake venom neurotoxins in such effects is most unclear, but early experiments clearly demonstrated that direct application of venom to the central nervous system required concentrations of venom to produce actions on the central nervous system much higher than those normally requried to kill the test animal peripherally (Kellaway *et al.* 1932). These workers demonstrated that direct application of venom to the fourth ventricle in the central nervous system could not produce respiratory paralysis and death at venom concentrations well in excess of those required to cause paralysis of respiratory muscles and death following intravenous or intraperitoneal administration of the same venom. Furthermore, it is far from certain that some of these quite large molecular weight toxins would successfully pass the blood/brain barrier. However, other pharmacologically active substances released by venom components from normal body cells might in some way explain the central nervous system effects seen clinically. Unfortunately this important area remains largely speculative.

Renal failure following envenomation by a variety of Australian elapid snakes has been reported, and one case of nephrotic syndrome secondary to envenomation has also been described (Harris *et al.* 1976; Steinbeck 1960; White and Fassett 1983). Such renal failure has always been in association with a coagulopathy, and the relationship of the two is uncertain. In addition, the relationship of muscle destruction to renal failure is unclear, though as with coagulopathy it is conceivable that high levels of circulating myoglobin could impair renal function and potentially lead to acute renal failure. No venom components with specific actions against the kidney have been described from Australian snake venoms.

No specific cardiotoxins have been identified from Australian elapid venoms, but some features suggestive of cardiac actions have been reported. However, the role of cardiotoxins in cases of envenomation by Australian elapids remains unresolved, though it seems probable that if cardiotoxins are present they play only a minor part. Kellaway (1933) noted that the Mulga Snake (*Pseudechis australis*) venom had a direct effect on cardiac muscle, which he concluded was important in the lethality of this venom. He also noted a weak cardiotoxic action in the venoms of the Copperhead (*Austrelaps superbus*) and the Red-bellied Black Snake (*Pseudechis porphyriacus*). He found Mulga Snake venom caused cessation of the beat in

isolated rabbit auricle in about one hour, at concentrations of one in 10,000 (Kellaway and Thompson 1930). He considered this venom to be similar in cardiac action to Cobra venom.

A pure cardiotoxin has been isolated from Cobra venom (Mebs 1978) and causes systolic arrest in the frog heart. It irreversibly reduces the resting membrane potentials of the cardiac muscle. There is an increase in the QT interval of the electrocardiogram. Purified neurotoxin and phospholipase A from the same venom do not produce these cardiac changes. However, there does appear to be a synergistic action between phospholipase A and cardiotoxin. This cardiotoxin is considered to be a membrane toxin.

In addition to a direct action on the heart, cardiotoxins may also act on the vascular system. The venoms of the Red-bellied Black Snake (*Pseudechis porphyriacus*) and the Copperhead (*Austrelaps superbus*) can cause severe progressive hypotension if administered intravenously (Sutherland 1979). This may be due to a direct effect of a cardiotoxin, or to the release of pharmacologically active substances such as histamine or slow-reacting substance A, both of which can be liberated by Red-bellied Black Snake venom. It is likely that a phospholipase A in the venom is responsible for the release of these substances indirectly, by forming lysolecithin which in turn damages cells causing the release of these substances (Lee and Lee 1979).

The role of snake venoms in causing the release of pharmacologically active substances such as bradykinins has not been extensively investigated for Australian snake venoms, although kinin releasing enzymes are apparently found in some snake venoms. (Iwanga and Suzuki 1979).

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ELAPID SNAKES: ASPECTS OF ENVENOMATION

Julian White

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◄ Skull of Taipan, *Oxyuranus scutellatus*.

Envenomation may be defined as the spectrum of problems which occur in the victim as a result of a bite or sting by a venomous animal. Envenomation will only occur when enough venom has been inoculated into the victim to cause detectable abnormalities. In medical terms a patient has been envenomated by a venomous snake when sufficient venom has been injected to cause detectable problems in the patient. Thus when a snake strikes and injects little or no venom, and no detectable problems arise, that patient has not been envenomed. This is an important distinction, because only patients who have been envenomed, and have sufficient medical problems as a result of envenomation, actually require treatment with antivenom.

In a significant number of cases of snake bite too little venom will have been injected into the patient to cause medical problems severe enough to justify treatment. All too often in the past it has been assumed that because a person has been bitten by a snake, he or she will necessarily suffer dire consequences unless given antivenom treatment. It is now understood that this is not necessarily the case. However, for significant snake bites, treatment with antivenom will often be life saving, and remains the mainstay of treatment of snake bite in Australia.

In considering the effects of venom in human snake bite victims, the complex and overlapping actions of snake venom and its various components, as discussed in the preceding paper, must be remembered. It is therefore not uncommon to see a patient with serious envenomation exhibit multiple complex problems involving many different organ systems in the body. Nevertheless, certain basic symptoms seem common to nearly all victims of significant snake bite in Australia.

Our understanding of envenomation by snakes in Australia, and thus the management of snake bite, depends heavily on the experience of doctors involved in treating snake bite, as reported in the medical literature. As in any field, the most likely route to major advances involves experts with extensive experience. In the management of snake bite this means just a few individuals who see virtually every case of snake bite presenting in the region in which they consult. In practice in Australia this means probably a single expert, or perhaps a team of

two experts, for each State in Australia. Unfortunately this situation rarely occurs, and the potential experience available from managing cases of snake bite is diluted by the large number of medical practitioners who individually manage cases. While this is understandable in the rural setting, it seems hard to justify in the capital cities of Australia, where rivalry between major hospitals in each city may prevent any one individual or team of individuals seeing all cases of snake bite presenting.

EPIDEMIOLOGY OF SNAKE BITE

In considering snake bite as a problem in Australia, it is also worth considering the problem of snake bite worldwide. Information on the true incidence of snake bite is incomplete, and the most comprehensive worldwide study presently available is of deaths from snake bite recorded in hospital and other official records (Swaroop and Grab 1954). This World Health Organisation study conducted in the latter part of the 1940s, estimated that between 30,000 and 40,000 people die from snake bite each year. The breakdown of deaths per geographic region is given in Table 1. From this it may be seen that the Asian region has the largest number of deaths by far, and the geographic region with the smallest number of total deaths from snake bite is Oceania, which of course includes Australia. As will be seen later, it is probable that the figures for the Australian region are now even lower. Therefore despite the potential lethality of our many venomous snakes, Australia does not rate highly in the snake bite death statistics. Recent work on the epidemiology of snake bite in savanna Nigeria, Africa (Pugh and Theakston 1980) casts doubt on the validity of the W.H.O. statistics. For all of Africa the W.H.O. statistics suggest between 400 and 1,000 deaths annually, yet Pugh and Theakston estimated that there were 23,000 deaths annually in savanna West Africa alone, mostly due to bites by the Carpet Viper (*Echis carinatus*). In this same region (savanna Nigeria) the W.H.O. figures noted only 9 deaths in a period of 5 years. This reflects the disparity between official hospital statistics, and the real death rate in many parts of the world, where non-medical treatment is often sought for snake bite and where the vast majority of deaths will never be included in hospital statistics.

Table 1: Total snake bite mortality worldwide (after Swaroop and Grab 1954).

Region	Estimated Mortality
Asia	25,000–35,000
South America	3,000– 4,000
Africa	400– 1,000
North America	300– 500
Europe	50
Oceania (includes Australia)	10
TOTAL	30,000–40,000 deaths per year

In a study of snake bite in Waorani Indians in Ecuador, where the treatment of snake bite had never involved modern medical methods, Theakston *et al.* (1981) demonstrated that 78% of all adults tested had detectable antibody against at least one of six snake venoms from dangerously venomous snakes found in that region of Ecuador. To produce such antibody it seems reasonable to assume that a significant snake bite must have occurred, and the inference is that virtually all of the inhabitants of this region are bitten by snakes during the course of their life. It was further estimated that 4.9% of all deaths in the region were due to snake bite. In a further study (Theakston *et al.* 1983) it was found that some of these Indians had sufficient antibody to snake venom to potentially provide them with some protection in the event of further snake bites!

Unfortunately, the accuracy of epidemiological studies of snake bite, using ELISA techniques for antibody detection, as used in Nigeria and Ecuador (Pugh and Theakston, 1980; Theakson *et al.* 1981; Theakson *et al.* 1983), has recently been challenged by workers in Thailand (Ho *et al.* 1986). Ho and his co-workers have shown that a variety of technical problems with the ELISA technique may cause false positive results, thus a considerable over-estimate of population exposure to snake bite may occur. It is not possible to state, at this time, whether such over-estimation has occurred in relation to the Nigerian and Ecuador snake bite studies.

It is possible also to consider snake bite in terms of incidence of snake bite deaths per 100,000 people per year. On this basis the W.H.O. figures show that Burma has a snake bite mortality of 36.8 cases per 100,000 population per year in the Sagaing province, with

Table 2: Reported snake bite fatalities in the Australian States and Territories (after Fairley 1929; Swaroop and Grab 1954; Trinca 1963; White *et al*. 1985).

State	Pre-Antivenom Era 1910–1926	Antivenom Era		
		1945–49	1952–61	1968–82
Queensland	74	18	18	15
New South Wales	57	6	9	9
Victoria	43	—	9	11
Western Australia	6	3	6	7
South Australia	8	—	1	2
Tasmania	8	1	—	3
Northern Territory	1	—	1	9
Australian Capital Territory	1	—	1	—
TOTAL	198	28	45	56
Fatality Rate Per Year	12.4	5.6	4.5	3.7

an overall rate for that country of 15.4 per 100,000. In contrast, the Netherlands has a rate of .004 per 100,000, the United Kingdom has a rate of .02 per 100,000, and France a rate of .07 per 100,000 population. During the same period Australia had a rate of .07 per 100,000 population. Thus even allowing for the small population of Australia, it can be seen that the chance of dying as a result of snake bite in Australia is quite small by world standards.

In Australia it has been estimated that approximately 3,000 snake bites occur each year, and at least 200, and possibly as many as 500 of these receive antivenom treatment (Sutherland 1983). Deaths from snake bite in Australia have obviously varied over the last century, and were clearly much higher in the era before antivenom was available. Recorded deaths from snake bite for each State of Australia are given in Table 2, showing figures from the era prior to antivenom availability, and 3 sets of figures following the advent of antivenom, the most recent statistics being from the 15 year period 1968 to 1982. It can be seen from these figures that Queensland has consistently the highest number of deaths from snake bite, and this doubtless reflects the relative abundance of potentially dangerous snakes in rural and semi-urban areas where significant human populations occur. In particular, snakes such as the Taipan (*Oxyuranus scutellatus*) are not infrequently found in cane growing areas of Queensland, where the potential for accidental snake bite while working in cane fields is significant. The low number of deaths from snake bite in South Australia reflects the large concentration of population in a single urban complex, with only a relatively small rural population. Overall it can be seen that the total fatality rate per year has steadily dropped since the introduction of antivenom. The recent introduction of improved first aid procedures due to the efforts of Dr Sutherland, of the Commonwealth Serum Laboratories (C.S.L.), Melbourne (Sutherland *et al*. 1979) will undoubtedly assist in further reducing the

Table 3: Mortality rate for various snakes, if no antivenom used (after Fairley 1929).

Snake	Number of Patients	Deaths	% Mortality
Taipan* (*Oxyuranus scutellatus*)	8	6	75%
Death Adder (*Acanthophis antarcticus*)	10	5	50%
Tiger Snake (*Notechis scutatus*)	45	18	40%
Brown Snake (*Pseudonaja textilis*)	70	6	8.6%
Red-bellied Black Snake (*Pseudechis porphyriacus*)	125	1	0.8%

*Based on review of published cases, prior to availability of antivenom.

number of deaths from snake bite in Australia.

An important factor in determining the number of deaths from snake bite in each state per year is of course the snake fauna found in areas of human habitation. Some snakes have a much higher chance of killing a snake bite victim than others, and this is best represented using figures compiled before any antivenom treatment was available. This may be seen in Table 3. From this it can be seen that the Taipan was most likely to kill its victims, and that a common cause of snake bites in Victoria, the Tiger Snake (*Notechis scutatus*) had approximately a 40% lethality rate. In contrast the Brown Snake (*Pseudonaja textilis*) and presumably related species in this genus, had a much lower lethality rate. As these snakes are the predominant cause of snake bite in South Australia, and probably in Western Australia as well, it is perhaps not surprising that these states recorded a lower number of snake bite fatalities. The disproportion in fatalities between various states seen in the most recent figures probably also reflects the problems of snake bite treatment in remote areas, where the victim may be unable to reach medical attention in time to ensure survival. This doubtless is of relevance in the snake bite statistics particularly from the Northern Territory and Western Australia, and to a lesser extent from Queensland.

A further review of the most recent mortality figures is given in Table 4. It can be seen that during this period there was a significant drop in the total number of deaths from snake bite each year, and while this may of course be coincidental, it is tempting to speculate that the vigorous work of Sutherland and others is at least in part responsible for this improved situation.

In reviewing snake bite fatalities in Australia it is pertinent to note that death may not necessarily occur rapidly. Indeed, in analysing snake bite fatalities in the era prior to antivenom, it is clear that nearly half of all cases died 24 hours or more after the bite, and only about 16% died in the first 6 hours after the

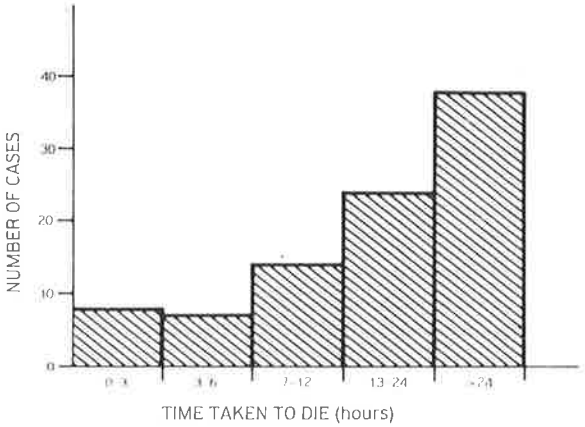


Fig. 1. Time between snake bite and death in 81 cases of fatal snake bite not treated with antivenom (after Fairley 1929).

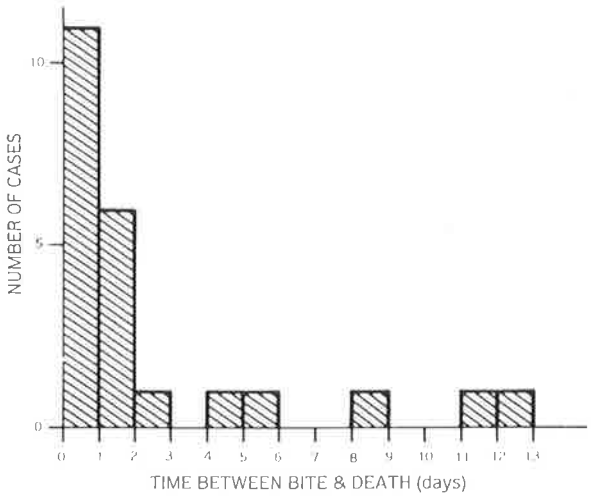


Fig. 2. Time taken to die following snake bite (23 cases).

Table 4: Fatal snake bite in Australia 1968–1982* (after White *et al.* 1985).

	Qld	Vic	NSW	NT	WA	Tas	SA	Total	Deaths Per Year
1968–72	5	6	5	2	3	1	2	24	4.8
1973–77	8	4	3	4	3	1	0	23	4.6
1977–82	2	1	1	3	1	1	0	9	1.8
Total	15	11	9	9	7	3	2	56	3.7

*Based on statistics from State Registrars of Births, Deaths and Marriages. May include some cases ascribed to snake bite without definite proof, but equally may exclude some cases where cause of death was never defined.

Table 5: Problems associated with envenomation in 21 cases of fatal snakebite. Some cases had more than one problem*.

Problem	Probable Cause of Death (no. of cases)	Total Cases where Problem noted
Coagulopathy	1	12
Cerebral haemorrhage	4	4
Renal failure	4	5
Paralysis	1	8
Muscle destruction	0	2
Anaphylaxis following antivenom	2	2
Anaphylaxis to venom	1	1
Cardiac problems	2	3
Delayed treatment	2	3
Uncertain	4	—

*Based on retrospective analysis of case records and/or autopsy reports.

bite. This is illustrated in Fig. 1. An analysis of snake bite fatalities in the 15 year period 1968 to 1982 also yields interesting information. At present data is only available on 23 cases, and the time between bite and death is illustrated in Fig. 2. It can again be seen that over half of those who died took more than 24 hours to do so. The cause for such prolonged death is still a matter of investigation. However, initial evidence suggests that in a number of these cases a major injurious event associated with the snake bite occurred relatively early during the course of the illness, and that delayed death in part reflected the availability of modern intensive care life support systems (White *et al.* 1985).

In 21 cases in this latter series sufficient information is available at present to determine a probable cause of death, and this is summarised in Table 5. Information has been gained from a retrospective analysis of case records including autopsy reports where available. From this information it is clear that severe respiratory paralysis is now an unusual cause of death from snake bite in Australia. Delay either in seeking treatment or administration of treatment is still an occasional cause of death. Severe cerebral haemorrhage associated with a coagulopathy, and in all probability therefore ascribable to the coagulant actions of some snake venoms, appears to be a significant cause of death. Similarly renal failure and the problems associated with it account for several deaths. It is somewhat disturbing to note that two of the 21 deaths appear attributable to an anaphylactic reaction to the antivenom administered as treatment for snake bite. The potential for anaphylaxis following antivenom treatment is well established, and modern guidelines for the administration of antivenom should render such deaths extremely rare. It is, however, too early to make final judgment on the information from this survey of fatal snake bite in Australia, as much data has yet to be assessed.

It is difficult to determine the precise ratio between ineffective snake bites not requiring treatment, and effective snake bites with symptoms of envenomation (and so requiring some form of therapy). In a study of snake bite in children in South East Queensland, Munro and Pearn (1978) noted 71 cases of snake bite incidents in children over a 5 year period, and in only 12 of these did the child have unequivocal evidence of envenomation. One case was fatal, secondary to a coagulopathy, and a large intracranial haemorrhage. Only 2 of the cases in Munro and Pearn's series received antivenom.

A retrospective survey of snake bites admitted to three major teaching hospitals in Adelaide, South Australia, yielded similar data, which are summarised in Tables 6, 7 and 8 (White 1983a). At the Adelaide Children's Hospital 23 cases were noted over a 10 year period and only 2 of these showed signs of significant envenomation. Where it was possible to make at least some determination of the snake involved, the Brown Snake (*Pseudonaja* species) was the most frequent cause of bites. Similarly figures for adults at the Royal Adelaide Hospital showed

Table 6: Snake bites treated at the Adelaide Children's Hospital over a 10-year period (after White 1983).

1	Number of cases = 23; no fatalities	
2	Age range 1.6 to 13.8 years; mean 8.2 years	
3	Sex ratio M:F = 4.75:1	
4	Identification of snake:	
	Brown Snake (*Pseudonaja spp.*)	10
	Red-bellied Black Snake (*Pseudechis porphyriacus*)	2
	Unknown	11
5	Location of bite:	
	Upper limb	11
	Lower limb	11
	Lip	1
6	Evidence of systemic envenomation:	
	Mild	5
	Significant	2

Table 7: Snake bites treated at the Royal Adelaide Hospital over a 9-year period (after White 1983).

1	Number of cases = 16; no fatalities	
2	Age range 14 to 46 years; mean 26 years	
3	Sex ratio M:F = 15:1	
4	Identification of snake:	
	Brown Snake (*Pseudonaja textilis*)	7
	Tiger Snake (*Notechis scutatus*)	2
	Red-bellied Black Snake (*Pseudechis porphyriacus*)	2
	Unknown	1
5	Location of bite:	
	Upper limb	6
	Lower limb	5
	Uncertain?	5
6	Evidence of systemic envenomation:	
	Mild	7
	Significant (acute renal failure)	1

Table 8: Snake bites treated at the Flinders Medical Centre over a 5-year period (after White 1983).

1	Number of cases = 14; no fatalities	
2	Age range 1 to 30 years; mean 18.5 years	
3	Sex ratio M:F = 6:1	
4	Identification of snake:	
	Illegitimate bites	6 (5 in one individual)
	Brown Snake (*Pseudonaja textilis*)	8
5	Location of bite:	
	Upper limb	7
	Lower limb	6
6	Evidence of systemic envenomation:	
	Mild	1
	Significant	1

only one case of significant envenomation in 16 cases of snake bite, and of these, the majority of 'legitimate' snake bites appeared due to some variety of Brown Snake. However four of the 16 bites were classed 'illegitimate', that is, bites received by a reptile keeper or handler while handling a venomous snake. At the Flinders Medical Centre over a shorter period, 6 of 14 bites were 'illegitimate', and of the remainder, all appeared ascribable to Brown Snakes. Only one of the bites had evidence of significant envenomation. Strikingly, 5 of the 6 'illegitimate' snake bites at the Flinders Medical Centre involved just one amateur reptile keeper who presented on numerous occasions. Further investigations revealed that he had presented with snake bite on several occasions to virtually every other public hospital in the city. At the very least it could be suggested that this individual was accident prone!

The data from this survey were incorporated with other cases of snake bite the author has been involved with, and in 76 cases it was possible to state either definitely, or with a significant degree of probability, the type of snake involved in the bite. This information is summarised in Table 9. It can therefore be seen that at least in South Australia members of the Brown Snake group are responsible for the majority of bites, followed by Tiger Snakes, Red-bellied Black Snakes, Mulga Snakes, and Copperheads. All 6 Death Adder bites seen were illegitimate bites in reptile keepers. Of the various cases in this series, about 30% of those ascribable to Brown Snake bites had some evidence of systemic envenomation. For Tiger Snake bites, approximately 60% had some evidence of systemic envenomation, with corresponding figures for Mulga Snake bites being 83%, Black Snake bites being 70% and Death Adder bites being 50%. It must be stressed, however, that for most of these species this is a small and selected series, and therefore may not represent the true average incidence of envenomation for each species. A perusal of C.S.L. information on snake bites assessed using the venom detection kit, which equally will be a highly selected series, suggests a higher incidence of patients presenting with definite symptoms of envenomation (Table 10). Overall, however, it is the author's experience that the majority of patients presenting with a

Table 9: Snake responsible in 76 cases of snake bite in South Australia, where the identity of the snake was at least moderately certain.

Snake	Total No. of Cases	No. of cases due to illegitimate bites
Brown Snake (*Pseudonaja spp.*)	36	7
Tiger Snake (*Notechis scutatus*)	13	7
Red-bellied Black Snake (*Pseudechis porphyriacus*)	10	4
Mulga Snake (*Pseudechis australis*)	6	4
Death Adder (*Acanthophis antarcticus*)	6	6
Copperhead (*Austrelaps sps*)	5	1

Table 10: Severity of bite for various species of snake based on data kindly provided by C.S.L. on the use of Venom Detection Kits (1980-1984).

Snake	Definite Symptoms	Doubtful Symptoms	No Symptoms
Tiger Snake group (*Notechis spp.*)	32	7	2
Brown Snake group (*Pseudonaja spp.*)	22	1	12
Mulga Snake group (*Pseudechis spp.*)	10	1	3
Death Adder group (*Acanthophis spp.*)	2	—	—
Taipan group (*Oxyuranus spp.*)	4	—	1

history of snake bite or possible snake bite, do not develop evidence of systemic envenomation, and therefore do not ultimately need antivenom treatment. This is certainly in line with the findings of Munro and Pearn (1978).

Although anyone can be bitten by a snake, certain groups in the community are more likely to be bitten.

Young children

Munro and Pearn (1978) identified the toddler age group (1 to 3 years old) as the most common group of children involved in snake bite. This age group will readily play with any snakes they encounter in the garden, especially small snakes such as juvenile Brown Snakes. These may be encountered in some outer metropolitan areas and gardens, particularly in the late summer and autumn.

Late primary school age children

Munro and Pearn (1978) found a second peak incidence in the 10 to 12 year old group. These children are inquisitive about wild animals and have relatively little fear of animals such as snakes. This situation is confirmed by the author's own experience in South Australia. Sometimes these youngsters can catch small snakes and bring them to school, where display of their prize specimen often results in one or more people being bitten. Nearly all such victims in this age group are boys.

Farm workers

Persons working in areas frequented by snakes, such as around pastoral station homesteads, haystacks, swamps, and drainage channels, have a greater exposure to snakes and are more likely to be bitten. The species involved will obviously depend on the location, but in most areas of Australia the Brown Snakes (*Pseudonaja* species) are the most common venomous species found around human habitation. In south east Australia and Tasmania, Tiger Snakes (*Notechis* species) would be most likely to be encountered.

Reptile workers

Despite the official protection of all reptiles, including venomous snakes, in all states of Australia, and the consequent bureaucratic impediments to obtaining snakes as pets, there are many amateur reptile keepers throughout the country. In South Australia alone it has been estimated there are between 150 and 200 people who keep or study reptiles on an amateur basis (White 1981) and for Australia as a whole, this figure may exceed 1,000. Perhaps 10% of these individuals will have frequent contact with dangerously venomous snakes. Clearly this group, who range in age from about 14 years to retired people over 65 years, have an overall increased likelihood of snake bite, due to increased exposure. It must be stressed that many people in this group are very responsible about keeping and handling venomous snakes, and may never be bitten, but inevitably there are some who are either less careful than they should be, or who are simply irresponsible. The mixing of alcohol consumption with handling of venomous snakes is a disturbingly frequent association with snake bite in this latter group. Another common cause of bites is over-familiarity with the pet reptile, leading to bites occurring at feeding time when the keeper's hand all too commonly sustains the bite meant for the proffered food. Some species (e.g. the Rough-scaled Snake) feature prominently in case-series of reptile fanciers who are bitten.

Professional reptile keepers

There are an unknown number of people in Australia who make some or all of their income from catching, keeping and exhibiting dangerously venomous snakes. This includes those individuals who catch and maintain large numbers of venomous species to milk them of snake venom. This is mostly for the production of antivenom by C.S.L. in Melbourne. There are, of course, a number of researchers investigating snake venom, some of whom milk their own reptiles, and there are numerous zoos and wildlife parks with displays of venomous reptiles.

These professional snake keepers may handle large numbers of venomous snakes, and although their safety consciousness is high, a few are bitten occasionally. Some are more careful than others, and while the director of one major park has been bitten many times by a variety of venomous snakes, another senior reptile keeper working in a zoo, with 30 years' experience in this field, has apparently never been significantly bitten by a venomous snake in this entire period. However, this group may provide special problems for the treating physician, for those who have sustained multiple bites may easily develop hypersensitivity to both the venom and the antivenom. At least one death due to an acute hypersensitivity reaction to snake venom has occurred in Australia (Sutherland 1983). The victim was a professional herpetologist who was bitten while displaying a Tiger Snake, and died shortly afterwards, apparently due to anaphylaxis. He had been bitten by Tiger Snakes previously. The author has seen an acute hypersensitivity reaction in a 15 year old bitten for the second time by a juvenile Red-bellied Black Snake. Fortunately, severe anaphylaxis with shock did not occur, and this patient survived.

Other risk factors

Munro and Pearn (1978) identified risk factors. They found 36% of victims were bitten whilst in paddocks, fields or open country, and a further 29% were bitten in gardens or yards. Only 2 cases were bitten in creeks, and 2 whilst in a house. In 75% of cases with reliable data there was no intentional provocation of the snake. They found a peak seasonal incidence in summer (42%), and in the afternoon (51%). There was a random distribution throughout the week with no weekend peak.

A review of cases at the Adelaide Children's Hospital (White 1981, 1983(a)) showed that the majority of victims are bitten in fields, paddocks and especially areas with long grass (65%), the remainder being bitten in yards or gardens. Data on provocation were incomplete, but at least 60% showed no history of provocation. The majority of bites were in spring, later winter (August) or early summer. The majority of bites were in the afternoon. Males were involved more than three times as commonly as females.

GENERAL SYMPTOMS AND SIGNS OF ENVENOMATION

The symptoms and signs of snake bite in Australia will vary for each species of snake involved, and will depend on the nature and extent of envenomation. Some features seem common to bites by all species, and for a

significant number of cases where insufficient venom has been injected into the victim, no symptoms or signs of envenomation will occur. However, it must be remembered that sometimes an apparently trivial or doubtful snake bite may be associated with major and life threatening envenomation, particularly in children. It is necessary for medical practitioners treating suspected cases of snake bite in Australia to maintain a high index of suspicion for this diagnosis, and ensure that adequate time has elapsed before declaring the patient safe from the effects of snake venom. There is at least one report in the medical literature of a patient declared unaffected by snake bite by hospital staff after only a few hours' observation, and therefore sent home, only to be found in a coma due to snake bite some hours later (Sutherland *et al.* 1975). She subsequently died.

Symptoms of Envenomation Including History of the Bite

CIRCUMSTANCES OF THE BITE

As discussed earlier most of the actions of Australian snake venom are systemic. They cause generalised problems throughout the body, rather than a single problem or problems isolated to the area bitten. The local reaction to a snake bite may be negligible, with little or no local pain, swelling, or redness around the bite. At least for some species, small fangs dragged through the skin for a short distance may leave fine scratches rather than puncture marks, and for the first 15 to 30 minutes after a bite these may be extremely hard to see with the naked eye. (White 1981) A snake may also inject little or no venom, despite leaving obvious marks of a bite. These two factors result in a considerable amount of uncertainty in determining whether a serious snake bite has occurred. It is therefore most important in talking to the victim of a snake bite (and also witnesses to the bite if available), to determine details of the actual bite episode. It is very relevant how the snake strikes, and for how long it hangs on. A prolonged bite with chewing is very likely to result in significant envenomation, whereas a glancing strike may even occur with the snake's mouth closed, when obviously no envenomation will ensue. If a snake strikes rapidly and with only a single glancing blow, it is quite possible that only a very small amount of venom may be injected. However, if a snake strikes repeatedly it is virtually certain that at least one of the strikes will be associated with a significant amount of venom being injected into the victim. As discussed below, examination of the wound may help to determine whether a snake bite has occurred, and if multiple bites have occurred.

Only a few Australian snakes can successfully inject venom through thick clothing or, even more improbably, boots or other stout footwear. The Taipan (*Oxyuranus scutellatus*) with its exceptionally long fangs for an Australian snake is the only species which has definitely proved capable of successfully biting through a boot (Reid and Flecker 1950).

Similarly the position of the strike may give some clues. While the Taipan may be able to strike quite high (Campbell 1967) the Death Adders (*Acanthophis* spp.) being relatively short snakes can usually only bite at a fairly low level, and bites above the ankle are unlikely to be due to a Death Adder (Campbell 1966).

Because minimal or no significant pain may be associated with snake bite, victims may be unaware that they have been bitten (White 1981). Thus they may feel something brush up against their leg, or there may be a brief and minor stinging sensation, as if scratched by a thorn. This may be ignored, so that no history of snake bite will be available, and no first aid will be instituted following the bite. Occasionally when walking in groups, it may be someone behind the victim who actually sees the snake and the snake bite incident. If a snake is seen it is certainly worthwhile obtaining as detailed a description as possible of the snake, although it must be admitted that Australians are not skilled at the identification of Australian poisonous snakes, even in the most ideal of circumstances (Morrison *et al.* 1983). Furthermore, the wide variety and overlapping range of body colours for Australian venomous snakes make accurate identification difficult. A portion of a reptile glimpsed briefly during the bite episode (and its quick departure from the scene) is a virtually hopeless task in identification in most cases, even for an expert.

Fortunately the range of snakes found in many areas of Australia is limited, and an expert herpetologist may be able to limit the range of potential species which might have been involved, using a knowledge of their distribution

and habits, and even a relatively poor description of the snake from the victim or another witness (White *et al.* 1985).

GENERAL SYMPTOMS OF SNAKE BITE

The range of symptoms indicative of systemic envenomation is wide, and in part the pattern of symptoms will depend on the victim's own attitude to snakes and snake bite, particularly the extent of fear. A person terrified of snakes and snake bite is likely to become extremely agitated, or even hysterical, and this in itself will produce certain changes. Thus the pulse rate may increase, the patient may breathe too rapidly, and this in turn may cause a degree of dizziness or even fainting. As a result of the fear nausea and even vomiting can also occur, and in some individuals increased activity of the gut muscles will cause abdominal pain. As many of these symptoms also occur as a genuine result of envenomation, it can be seen that distinguishing between fear induced symptoms, and snake venom induced symptoms may be difficult. In such a situation a careful discussion with the patient and witnesses may give a clue to the influence of fear in production of observed symptoms.

Commonly described early symptoms of systemic envenomation following Australian snake bite include dizziness, nausea and vomiting, sweating, and headache. Severe frontal headache is often encountered in patients following snake bite. These symptoms may then progress to include pain in regional lymph nodes draining the site of the bite, and abdominal pain which may be crampy in nature and quite severe. Diarrhoea sometimes occurs.

The conscious state of the patient is often affected, so he/she may be slow to answer questions, or ignore questioning. There may also be signs of irritability, and restlessness, with frequent tossing and turning on the bed. In many cases there may be a period of complete loss of consciousness, and during this period witnesses may describe a patient as having convulsions. Indeed it is not uncommon for the first noticed symptom of snake bite to be the sudden collapse into unconsciousness of the victim, sometimes followed by convulsions. This may happen in both children and adults, though in the author's experience it is more likely to occur in children. Such a collapse may occur within 10–15 minutes of the bite, or may be delayed up to an hour or so. Where a bite has gone unnoticed, such a sudden and unexplained loss of consciousness may not be immediately related to the possibility of snake bite, and medical practitioners treating patients who have presented in this way, and where there is a possibility that snake bite might have occurred, should maintain a high index of suspicion for the diagnosis of snake bite.

SYMPTOMS OF NEUROTOXIC ENVENOMATION

Symptoms of neurotoxic envenomation are usually those of a progressive paralysis. The patient may first notice double vision or blurring of vision. Speech may start to change, often subtly at first, as the tongue and soft palate, velo-pharyngeal sphincter, and vocal cords, are all progressively paralysed. The voice may become nasal as the velo-pharyngeal sphincter ceases to close completely for vowel sounds. Speech is distorted as the tongue becomes paralysed. The facial muscles may also show early evidence of paralysis, with an apparent lack of facial expression, and this may be noticed by a witness. One of the earliest signs of paralysis noted is ptosis, or drooping of the upper eyelids, and the patient may describe this as heaviness of the eyelids or eyes. If antivenom is not given at an early stage, the paralysis will often progress with development of a flaccid paralysis of all voluntary muscles. This may be first noted by victims when they attempt to move or write. The most detailed descriptions of the progression and extent of paralysis following Australian snake bite are those of Campbell (1969, 1979a). Campbell noted that the first muscles to show paralysis are often the extrinsic ocular muscles, causing double vision, and the last to be paralysed are the muscles of the diaphragm, with consequent cessation of respiration and death of the victim.

As paralysis of the facial muscles progresses, a patient cannot open his mouth or protrude the tongue, the jaw becomes relaxed, and falls backwards along with the tongue. At this stage the tongue may fall back in the mouth and cause respiratory obstruction and rapid death unless treatment is given to re-establish the airway. The progressive weakness of the respiratory muscles will impair the ability to cough up secretions, which may become more profuse as a result of systemic envenomation. The eventual outcome of the progressive paralysis is a

complete paralysis of all muscles including all of those involved in respiration, so that the patient is unable to breathe.

SYMPTOMS OF HAEMATOLOGICAL DISTURBANCE

A patient with systemic envenomation may have a severe coagulopathy, in which the blood is unable to clot as a result of venom action, yet symptoms from this problem may not be detectable, at least in the early stages. Careful questioning may reveal that scratches, or the bite site itself have continued to ooze, and this is certainly indicative of a coagulopathy. The patient may also vomit or expectorate blood or pass blood-stained urine (Campbell 1964, 1969). Severe internal bleeding may occur as a result of coagulopathy, and this may manifest as pain, or if it occurs in the brain, as loss of consciousness and convulsions. It should be stressed that this is unlikely to occur at an early stage after the bite, and it is important to differentiate the loss of consciousness and convulsions occurring as a result of a major bleed into the brain, from those seen very early following snake bite purely as a result of the effects of circulating venom.

SYMPTOMS OF MYOPATHY

The muscle destruction caused by myotoxins in some Australian snake venoms may cause generalised muscle pain, and especially pain on movement of major muscle groups. It may also cause an apparent muscle weakness, which may be difficult to distinguish from true paralysis. The patient may note that urine passed is very dark in colour, and as this may be due to muscle pigment being excreted via the kidneys, such a history of dark urine should always raise the suspicion of muscle destruction. It may, however, be due to destruction of some red blood cells causing a haemoglobinuria, and these two causes of dark coloured urine should be separated.

Signs of Envenomation

While symptoms of envenomation are problems which the patient may himself complain of, signs are basically problems noted when the patient is examined, usually by a medical practitioner.

THE BITE SITE

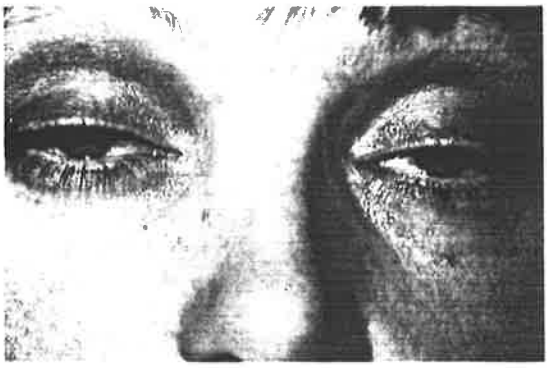
The appearance of bite marks at the bite site is both potentially and in reality quite variable, but it may give a clue to the species of snake involved, at least to an experienced toxinologist (White 1981, 1983b). There may be two classic fang puncture marks, or a single fang puncture mark, or a mixture of puncture marks and scratches, or just a single scratch where one fang has been dragged through the skin (Campbell 1979b; White 1981, 1983b). If the snake has bitten more than once there may be an even more complicated pattern of tissue injury, but the different site or orientation of the second and subsequent bites is usually obvious, and as multiple bites usually indicate serious envenomation, evidence of multiple bites should always be searched for. It must be reiterated that while fang marks may be obvious, this does not indicate that venom has been injected. The differences in appearance of bites from different species of snakes in Australia have been reviewed (White 1981, 1983b, 1986; White and Pounder 1984; Pearn and Covacevich 1981) and will be further discussed with illustrations later in this chapter.

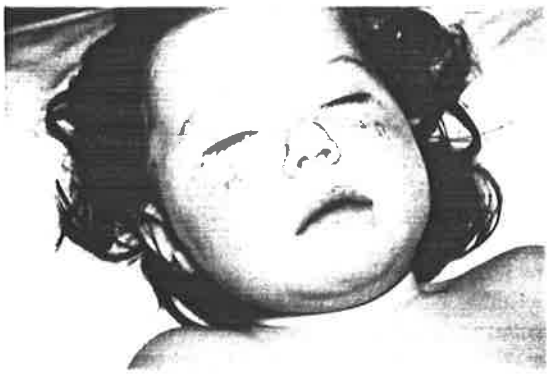
INVOLVEMENT OF LOCAL LYMPH NODES

The majority of venom components appear to be absorbed into the blood stream via the lymphatic channels, and this has become the basis of first aid measures (Sutherland *et al.* 1979; Sutherland *et al.* 1981). At least in some cases then it may be expected that if venom is injected in sufficient quantity to cause a problem for the victim, one of the earliest signs of this will be changes in the lymph nodes draining the region of the bite. In the case of a bite in the hand this is likely to be lymph nodes in the armpit, and in the case of bites to the lower limb, lymph nodes in the groin. These may become enlarged or tender, and if such a sign is discovered, it is strongly suggestive that systemic envenomation will occur. Campbell (1964) reported that the earliest sign of snake bite in patients envenomated in Papua New Guinea was tender enlarged regional lymph nodes, which were usually involved about one to two hours after the bite. It must be noted, however, that lymph node involvement does not occur in all cases of snake bite where systemic envenomation develops, and the absence of tender or enlarged lymph nodes draining the bitten region does not exclude envenomation.

GENERAL SYSTEMIC INVOLVEMENT

The pattern of systemic signs like all other signs of snake bite is highly variable. General systemic signs observed in snake bite include elevated pulse rate, decreased or increased blood





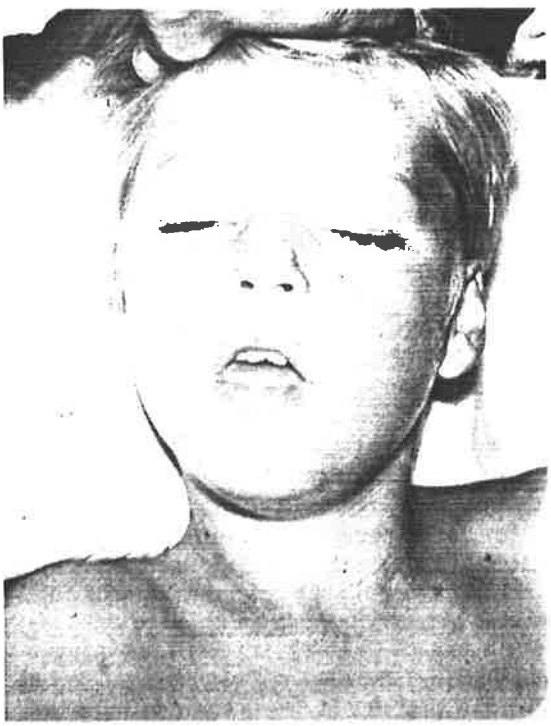
▲▲ Fig. 3. Early signs of neurotoxic paralysis in a 27 year old woman bitten by a Tiger Snake (*Notechis scutatus*). Note the very obvious ptosis, or drooping of the upper eyelids, a classic early sign of paralysis.

▲ Fig. 4. Early stage neurotoxic paralysis in a 2 year old girl following a Tiger Snake (*Notechis scutatus*) bite. Note the expressionless appearance of the face, and the obvious ptosis. Particularly in a child these early signs may be mistaken for non-specific drowsiness. In this case the child was conscious but irritable, and was unable to open her eyes further than displayed.

► Fig. 5. Marked neurotoxic paralysis in an 8 year old boy following a bite by an unidentified snake. Note the expressionless face, open mouth, with jaw apparently relaxed. There is obvious ptosis.

pressure, vomiting, restlessness, and impaired conscious state. The coughing up or vomiting of blood, or the passing of blood in the urine may occur, and is indicative of a coagulopathy. Patients may be feverish, and sometimes may sweat profusely. Of particular relevance in determining if systemic envenomation has occurred is evidence of developing paralysis caused by neurotoxins.

Neurotoxic signs usually develop and progress over several hours, and the early signs may be subtle and easily missed. The most highly innervated muscles are usually the first to show neurotoxic paralytic effects. Thus the muscle elevating the upper eyelid, the extrinsic ocular muscles, and ciliary muscles controlling the lens usually provide first evidence of paralysis. There is frequently ptosis or drooping of the upper eyelid (Figs 3, 4 and 5). Vision may be blurred, and careful testing of ocular movement may show abnormalities. There may be double vision on lateral or upward gaze. The muscles associated with speech are often affected early, with changes in the character of the voice due to distortions produced by nasal escape, and then slurring as the tongue becomes progressively paralysed. All other voluntary muscle groups will usually be involved in the untreated or inadequately treated case, and tendon reflexes may be decreased or abolished. As the paralysis advances, paralysis of the tongue and facial muscles and those at the back of the mouth may lead to respiratory obstruction. Limb muscles and muscles of respiration will then gradually become paralysed, and this may at first be evident as an increased respiration rate and use of accessory muscles of respiration. Finally the diaphragm will be the only muscle assisting in respiration, and once this, too, is paralysed the



patient will die from respiratory arrest unless given assisted ventilation (Campbell 1979a).

The signs of myopathy and muscle destruction are similar to the symptoms previously mentioned, and pain on muscle movement should be checked for. In late stages of muscle destruction severe wasting of voluntary muscle may occur (Hood and Johnson 1975).

Changes in the conscious state should be carefully observed, and irritability and restlessness are positive signs of envenomation. If late stage unconsciousness and convulsions occur, particularly in association with a coagulopathy, some form of intracranial bleed into the brain should be considered. Serum electrolyte disturbance may cause a similar clinical picture (White *et al.* 1984).

THE TIMING OF ONSET OF SYSTEMIC ENVENOMATION

As with every other aspect of snake bite, the rapidity with which envenomation may occur is highly variable. The author is aware of children who have become severely envenomated within 15 minutes of a bite, and yet has seen others who have shown no evidence of envenomation for several hours. If no symptoms have developed within 6 hours of the bite, then it is unlikely that problems will ensue, but not impossible, and any patient definitely bitten by a snake, or suspected of having suffered a snake bite, should be observed in a hospital situation for at least 18–24 hours. Tragedies have occurred when patients have presented early following a snake bite, and have been assessed as showing no signs of envenomation, and so have been discharged only to die some hours later at home as a result of the snake bite (Sutherland *et al.* 1975). Coagulopathy following snake bite may occur within 30 minutes of the bite (White 1981, 1983c) as may collapse and convulsions (White 1981; White *et al.* 1984).

Peripheral paralysis due to neurotoxins may take longer to become established. Certainly experimentally there is a latent period of between 30–60 minutes between application of venom to the neuromuscular junction and established block of transmission with consequent paralysis (Thesleff 1979). This latent period is decreased by muscle activity, such as the victim running or in other ways being active following the bite. Clearly in a human victim, unless venom is injected directly into a vein, there will be a delay between the bite and the passage of the venom through the lymphatic network via the thoracic duct to the general blood circulation. The speed of passage of the venom to the blood will depend on the effectiveness of the first aid undertaken by the victim or companion. Thus a child bitten on the leg who continues to play and run around after the bite will sustain much faster venom movement than an adult who immediately has correct compression-immobilization first aid applied, and is then kept motionless until medical care is reached.

In a series of 52 cases of snake bite in Papua New Guinea Campbell (1964) reported that 33% developed symptoms within one hour or less of the bite, 15% developed symptoms from one to two hours after the bite, and the remaining 52% developed symptoms in two to twelve hours after the bite. The earliest signs noted by Campbell, lymph node tenderness, appeared about one to two hours after the bite. In the author's experience in South Australia, headache and impaired conscious state are frequent presenting signs of systemic envenomation, and usually occur within one to two hours of the bite.

Complications of Envenomation

PARALYSIS

The neurotoxins are probably the most distinctive, important and lethal components of Australian snake venoms. They act at the neuromusclar junction, either postsynaptically or presynaptically. If untreated by appropriate antivenom administration, paralysis caused by these toxins may well lead to death of the victim due to complete respiratory paralysis. However, such extensive paralysis is now rarely seen in Australia, because antivenom is readily available and is often given before major paralysis can occur. Thus detailed analyses of neurotoxic envenomation in man are rarely reported in the Australian medical literature. By far the best accounts have come from Papua New Guinea and were reported by Campbell (1969, 1979a). Progression of symptoms and signs of this paralysis has already been discussed. Campbell noted that paralysis was symmetrical, and in severe cases the victims 'lay as if dead, and the only movement detectable was an ineffective twist of the pelvis'. The diaphragm appeared

most resistant to paralysis, and if the patient was put on respiratory support mechanisms, it apparently took between 24–30 hours for complete paralysis of the diaphragm to occur. This paralysis remained complete for about 6 hours, after which weak diaphragmatic movements recommenced. It took a further one to four days for sufficient recovery to enable the patient to breathe unaided. Next to recover were eye muscles, which showed signs of recovery about 48 hours after the bite. Within two to five days of the first sign of return of eye movement, most of the muscle function was recovered. A further week or more was required for recovery of muscle power.

In the author's experience even early stage paralysis may be associated with changes or complete loss of the senses of smell and taste, occurring after bites by the Tiger Snake (*Notechis scutatus*), and similar findings have been reported after bites by the Taipan (*Oxyuranus scutellatus*). In one case there was complete loss of taste and smell, but two months later there was partial return of smell, which was altered, everything smelling the same and disagreeable (Flecker 1944). Taste likewise returned, but was altered. Salt and sugar tastes returned to normal but sauces and the like all tasted the same. In another case of Taipan bite there was short term disturbance of smell and taste with later complete recovery (Reid and Flecker 1950). Loss of smell has occurred after envenomation by the Red-bellied Black Snake (*Pseudechis porphyriacus*) (White 1981).

Severe paralysis caused by neurotoxins has been described following bites by the Taipan (*Oxyuranus scutellatus*) (Flecker 1940; Flecker 1944; Reid and Flecker 1950; Benn 1951; Brigden and Sutherland 1981); the Inland Taipan (*Oxyuranus microlepidotus*) (Trinca 1969); the Tiger Snake (*Notechis scutatus*) (Frost 1980; White *et al.* 1984; White 1981; Frost 1981; Gaynor 1977); the Brown snake (*Pseudonaja* species) (Fairley 1929); and the Rough Scaled Snake (*Tropidechis carinatus*) (Trinca *et al.* 1971; Patten *et al.* 1985). This list is by no means exhaustive.

CENTRAL NERVOUS SYSTEM INVOLVEMENT

As discussed earlier the action of Australian elapid venoms on the central nervous system is not well understood, although it has been suggested that toxicity to the brain is of little significance compared with the other effects of venoms (Kellaway 1933).

Nevertheless, the early onset of unconsciousness has been recorded by a number of authors for a variety of snake species including Brown Snakes (*Pseudonaja* spp. White 1981, 1985; Harris *et al.* 1976; Foxton 1914); Taipans (*Oxyuranus* spp.) (Flecker 1940, 1944; Reid and Flecker 1950; Benn 1951; Sutherland *et al.* 1980; Trinca 1969); Tiger Snakes (*Notechis* species) (Hood and Johnson 1975; Frost 1980; Frost 1981; Gaynor 1977); Death Adders (*Acanthophis* species) (Flecker 1940; Fairley 1929); Rough Scaled Snake (*Tropidechis carinatus*) (Patten *et al.* 1985; Trinca *et al.* 1971); Red-bellied Black Snake (*Pseudechis porphyriacus*) (Sutherland 1979). In addition the author has seen it following bites by Brown Snakes, Tiger Snakes, and Mulga Snakes (*Pseudechis australis*). The extent of central nervous system problems noted in these cases varies from drowsiness, through irritability, to unconsciousness, and convulsions, the latter having been reported for virtually all the species noted. While the cause of the convulsions is far from clear, review of the literature suggests that convulsions following significant snake bite are a relatively common occurrence.

Long term brain damage following Australian snake bite has only been described following the bite of a Tiger Snake (Symons 1960) but this case appears to be the result of cerebral anoxia following a severe anaphylactic reaction to the administration of antivenom. The long and short term effects of venom on taste and smell have already been mentioned.

Coagulation Disturbance/the Defibrination Syndrome

The coagulation problems following Australian snake bite have been documented for a number of species and this has been reviewed (White 1981, 1983c; Sutherland 1983).

The most important clinical coagulation problem in snake bite is the defibrination syndrome. This occurs in cases of systemic envenomation, probably as a result of prothrombin converters in the venom. These in turn cause formation of thrombin, then conversion of fibrinogen into fibrin, which is then destroyed causing elevated titres of fibrin degradation products, and associated very low levels of fibrinogen. This results in blood which is

very hard to clot, or is completely unclottable in the laboratory situation. However, as significant size blood clots are not formed, platelets do not appear to be bound into clots in large numbers, and platelet levels are usually normal. Thus the term defibrination syndrome is probably more appropriate than the term disseminated intravascular coagulation.

Even though the defibrination may be severe, clinical evidence of this on examining the patient may be scarce or non-existent. The classic feature is prolonged bleeding from the bite site, or a venepuncture site where blood has been taken for various tests. Far more unusual is the presentation of multiple bruises, or even large collections of blood under the skin, although a scalp haematoma has been reported (Frost 1980). If treatment is delayed, however, bleeding may occur internally in the gastrointestinal tract with consequent vomiting of blood or passing of blood per rectum (Champness 1966; Frost 1980); or bleeding may occur into the kidneys and bladder, with passing of blood urine (Knyvett and Molphy 1959; Champness 1966; Schapel *et al.* 1971; Crawford 1980; Trinca 1969; Frost 1980). Coughing up blood in snake bite patients with defibrination syndrome has been described from Papua New Guinea (Champness 1966). Continuous ooze from tracheostomy wounds in the neck in those patients with a co-existent paralysis requiring ventilatory support using a tracheostomy has also been reported (Knyvett and Molphy 1959; Champness 1966). There is one case report of abnormally increased menstrual flow in a mild case of defibrination syndrome (Herrmann *et al.* 1972). The laboratory findings in defibrination syndrome following snake bite vary with the species of snake involved. Champness (1966) reported experience with defibrination in snake bites in Papua. The snakes implicated here were the Taipan (*Oxyuranus scutellatus*), and the Papuan Black Snake (*Pseudechis papuanus*). In a series of 22 cases of alleged snake bite, 11 had clinical signs of envenomation, and 6 of these had defibrination. Five of these 6 also had severe paralysis. In three cases there was bloody urine. One of the patients died of paralysis. In 5 of the cases the blood would not clot at initial testing. In only one of these cases was there definite haemolytic destruction of red cells. Platelets were normal in all cases. In only one case was there sufficient blood loss to require treatment with blood replacement. In all of the cases

Table 11: Defibrination syndrome in a 2½ year-old male following envenomation by an unidentified snake, presumed to be a common brown snake. (*Pseudonaja textilis*) (after WHITE 1981).

	TIME AFTER BITE						
	35 mins	3 hours	4 hours	4 hours 40 mins	6 hours	19 hours	43 hours
Antivenom — Tiger Snake	3000u	—	3000u	—	—	—	—
Antivenom — Brown Snake	1000u	—	2000u	—	—	—	—
Clotting time	Unclottable	Unclottable	—	Very weak clot with lysis	6½ mins weak clot	7¾ mins	7¾ mins
Prothrombin time	>150 secs	>15 mins	—	>15 mins	120 secs	14.5 secs	12.5 secs
A.P.T.T.	>90 secs	>15 mins	—	>15 mins	55 secs	34 secs	34 secs
Fibrinogen (mg/ml)	—	Not detected	—	Not detected	Not detected	74	174
Fibrin degradation products (mg/L)	—	>5000	—	>5000	2500	800	30
Platelet count	—	250000	—	—	—	250000	224000
Factor II	—	54%	—	58%	55%	—	—
Factor V	—	15%	—	34%	72%	—	—
Factor VII	—	44%	—	49%	55%	—	—
Factor VIII	—	8%	—	23%	50%	—	—
Factor IX	—	100%	—	100%	100%	—	—
Factor X	—	50%	—	80%	80%	—	—
Clinical state	Irritable drowsy	Irritable drowsy	Irritable drowsy	Irritable drowsy	Fully conscious, playing with parents	Normal	Normal

Table 12: Defibrination syndrome in a 7½ year-old male following envenomation by a brown snake (*Pseudonaja* sp.) (after WHITE 1981).

	TIME AFTER BITE						
	25 mins	1 hour	5 hours	7 hours	approx 18 hrs	approx 40 hrs	approx 64 hrs
Antivenom — Brown Snake	—	1000u	—	—	—	—	—
Clotting time	Unclottable	—	20 mins	5½ mins	5 mins	5½ mins	5½ mins
Prothrombin time	—	—	3½ mins	30 secs	18 secs	13 secs	13 secs
A.P.T.T.	—	—	74 secs	51 secs	46 secs	34 secs	32 secs
Fibrinogen (mg/ml)	—	—	14	19	—	99	156
Fibrin degradation products (mg/L)	—	—	2000	1000	500	10	6
Platelet count	—	—	265000	—	—	187000	253000
Clinical stage	Irritable drowsy	Irritable drowsy	Irritable — less drowsy	Awake, not irritable	Normal	Normal	Normal

Table 13: Comparison of initial coagulation studies in cases of defibrination syndrome following envenomation by members of the brown snake genus (after WHITE 1981).

	Common Brown Snake (*Pseudonaja textilis*) (after SCHAPEL *et al.* 1971; WHITE 1981)			Dugite (*Pseudonaja affinis*) (after HERRMANN *et al.* 1972)			Gwardar or Western Brown Snake (*Pseudonaja nuchalis*) (after CRAWFORD 1980)
	Case 1	Case 2	Case 3	Case 1	Case 2	Case 3	Case 1
Time after bite	3 hrs	5 hrs	?	2 hrs	18 hrs	2 hrs	?
Whole blood clotting time	Unclottable	20 mins	Unclottable	Unclottable	6 mins	Unclottable	Unclottable
Prothrombin time	>15 mins	3½ mins	>20 mins	>120 secs	16 secs	>120 secs	Grossly prolonged
A.P.T.T.	>15 mins	74 secs	—	—	—	—	Grossly prolonged
Fibrinogen assay (mg/100 ml)	Not detected	14	Not detected	<10 mg	<10 mg	Not detected	Not detected
Fibrin degradation products (μg/ml)	>5000	2000	—	Increased	Slightly increased	Increased	Markedly increased
Factor II	54%	—	—	25%	—	12%	28%
Factor V	15%	—	—	—	—	19%	18.5%
Factor VII	44%	—	—	—	—	55%	76%
Factor VIII	8%	—	—	55%	60%	2%	6.5%
Factor IX	100%	—	—	—	—	78%	44%
Factor X	50%	—	—	100%	—	100%	—
Factor XI	—	—	—	—	—	—	85%
Platelet count	250000	265000	nil	190000	185000	250000	Normal

fibrinogen titres were very significantly reduced, consistent with defibrination.

In Australia the defibrination syndrome is most commonly reported after envenomation by members of the Brown Snake (*Pseudonaja* species) group. Herrmann *et al.* (1972) have described the defibrination syndrome following envenomation by the Dugite (*Pseudonaja affinis*). They reported 3 non-fatal cases, all in adults, and 2 of these cases had initially incoagulable blood, with very low or undetectable fibrinogen, and marked elevation of fibrin degradation products. A number of other factors in the coagulation cascade were also reduced, particularly prothrombin and factor V. Their third case had definite symptoms of envenomation, but was apparently not seen until 18 hours after the bite, by which time symptoms were receding, and clotting time was normal with good clot retraction, though the fibrinogen titre was reduced.

Schapel *et al.* (1971) also reported a case of envenomation by a Brown Snake with defibrination syndrome and neurotoxic paralysis. Crawford (1980) reported a case of envenomation by a juvenile Western Brown Snake or Gwardar (*Pseudonaja nuchalis*), in which there was a definite defibrination, but no

paralysis. White (1981) reported two cases of envenomation by a Brown Snake where significant systemic envenomation occurred, associated with a marked defibrination syndrome. A summary of the findings of these cases is given in Tables 11, 12 and 13. It is clear from these cases that after adequate antivenom has been administered, the defibrination can be expected to resolve within a matter of hours to the point where the blood is again normally clottable.

The defibrination syndrome following bites by the Tiger Snake (*Notechis scutatus*) appears quite similar to those seen following Brown Snake envenomation, with similar resolution after adequate antivenom is given.

The potential for patients with a defibrination syndrome following snake bite to develop fatal haemorrhages has not been widely emphasised in the past. However, a review of the literature reveals at least 3 case reports of apparently fatal haemorrhages into or around the brain in association with a coagulopathy caused by snake bite (Foxton 1914; Sullivan 1979; Munro and Pearn 1978). More recently a review of fatal snake bites in Australia in the 15 year period 1968 to 1982 has been commenced, and as quoted earlier in this chapter, and elsewhere (White *et al.* 1985), such fatal brain haemorrhages associated with snake bite appear to be a potentially significant cause of death. Therefore while the coagulopathy may resolve spontaneously, it now appears appropriate to treat this complication as potentially lethal, and so treat aggressively with appropriate antivenom.

Muscle Destruction

Muscle destruction may follow the bites of a number of Australian species of snake. Clinically, however, severe muscle destruction following Australian snake bite has only been reported infrequently. In a fatal case of Mulga Snake (*Pseudechis australis*) envenomation Rowlands *et al.* (1969) noted all muscles examined at autopsy showed swelling and acute coagulative necrosis of numbers of the muscle fibres. The most severely affected muscles were those of the bitten limb, the respiratory muscles, and the extra-ocular muscles around the eye. An inflammatory reaction was only observed in the muscles of the right arm, adjacent to the site of the bite. Heart muscle showed many small regions where muscle fibres were swollen with vacuole formation, and absent nuclei. These findings are suggestive of a severe and generalised muscle destructive effect of the snake venom. The victim, who was a 20 year old male farm labourer, died 40 hours after the snake bite. Early symptoms in this case included nausea and vomiting, and some hours later he developed lethargy and weakness of limb movement. Dark urine was passed at this stage and it must be presumed this was evidence of myoglobinuria. He developed signs suggestive of a progressive paralysis including drooping of the eyelids, poor chest expansion, limited jaw opening, and partial paralysis of the tongue. He was unable to maintain any voluntary muscle contraction of the limbs against more than slight resistance. All deep tendon reflexes were decreased. His condition progressively deteriorated, and he died following a cardiac arrest. It is not recorded if muscle movement pain was present. It is unclear whether the reported paralysis in this case was a true paralysis, or merely secondary to the massive destruction of muscle throughout the body.

Hood and Johnson (1975) reported a severe case of Tiger Snake (*Notechis scutatus*) bite associated with massive muscle destruction, myoglobinuria, and severe renal failure. In this case the victim, a 47 year old male, who had been bitten by snakes many times previously, was bitten on the left hand. He was given Tiger Snake antivenom quite promptly, but the following day developed aching tender muscles and dark discolouration of the urine typical of myoglobinuria. The next day his urine output had decreased significantly, and he was noted to be delirious, with diminished deep tendon reflexes. Creatine phosphokinase levels were grossly elevated, indicative of muscle destruction. His renal failure required dialysis for about 10 days, and muscle wasting was noted to be gross. Biopsies of these muscles at that time showed a focal necrotising myopathy consistent with the myolytic effect of Tiger Snake venom. In this case, however, recovery was complete eventually, with an apparent return to normal muscle bulk and power.

Furtado and Lester (1968) reported a fatal case of acute renal failure and myoglobinuria in a 20 year old man bitten by a Small-eyed Snake (*Cryptophis nigrescens*). Before this event this

snake had not been considered as potentially dangerous, and no treatment was either sought or given. However 3 days after the bite the victim developed jaw muscle weakness and pain, followed over the next 2 days by intense muscular pain in both lower limbs with decreased power. His urine became dark at this time, and by the eighth day following the bite he had developed renal failure, with severe muscle weakness, impairing his ability to breathe unaided. He died shortly afterwards, and autopsy confirmed areas of significant muscle destruction.

Brigden and Sutherland (1981) described a Taipan (*Oxyuranus scutellatus*) bite to a 39 year old man, with consequent multiple problems of envenomation including defibrination, severe paralysis, and significant muscle destruction with grossly elevated levels of creatine phosphokinase. Renal failure apparently occurred in this case, as peritoneal dialysis was required.

Patten *et al.* (1985) have recently described a case of Rough Scaled Snake bite (*Tropidechis carinatus*) in a 9 year old boy with evidence of defibrination, paralysis, and muscle destruction with grossly elevated levels of creatine phosphokinase. Again renal failure occurred.

It is clear from these cases that where massive muscle destruction does occur following snake bite, renal failure is a potential accompaniment, presumably due to the effect of large quantities of muscle protein being filtered by the kidneys.

Kidney Problems

Kidney failure has been reported following envenomation by a variety of Australian snakes and one case of nephrotic syndrome secondary to envenomation has also been described. The mechanisms of renal failure in snake bite are varied, and the renal lesions reported worldwide include glomerulonephritis, glomerulitis, arteritis, interstitial nephritis, tubular necrosis, cortical necrosis, and renal infarction (Sitprija and Boonpucknavig 1979). However, no specific nephrotoxins have been isolated from Australian venom. In cases of renal failure reported from Australia following snake bite, acute tubular necrosis is the lesion most often seen. Harris *et al.* (1976) reported 3 cases of acute renal failure with recovery after envenomation by members of the Brown Snake group (*Pseudonaja* spp.) in Western Australia. In 2 of their 3 cases no antivenom was given. All 3 cases had blood pictures consistent with microangiopathic haemolytic anaemia and remained oliguric for between 14-21 days. None of these cases developed a documented defibrination syndrome or evidence of muscle destruction, and certainly major muscle destruction would not be expected following bites by these snakes. None of the cases showed significant paralysis. Needless to say all cases required dialysis, but renal recovery was complete in 2 cases and in the third case there was some evidence of residual kidney damage twelve months after the bite.

Renal failure has been associated with myoglobinuria and muscle destruction following bites by a variety of species including the Tiger Snake (*Notechis scutatus*), the Taipan (*Oxyuranus scutellatus*), the Mulga Snake (*Pseudechis australis*) and the Rough Scaled Snake (*Tropidechis carinatus*). A further case of renal failure following a bite by a Brown Snake (*Pseudonaja* species) has been reported in association with a true disseminated intravascular coagulation, without evidence of any muscle destruction (White and Fassett 1983).

Nephrotic syndrome following snake bite has also been reported (Steinbeck 1960). This case was thought to be due to envenomation by either a Brown Snake or Taipan, in a 23 year old female, who was uncertain about having been bitten by a snake. However, later during the course of the day on which she was thought to have been bitten, she became lethargic, and developed swelling of the bitten foot. She later developed swelling of the feet, legs, abdomen and around the eyes. On the third day she developed increased thirst and decreased urine output, and this and the swelling became worse. She sought medical aid on the fifth day by which time further swelling had developed. There was a definite proteinuria with low blood albumin and protein levels. She then developed bilateral pleural effusions and ascites, with an elevated blood urea level. The oliguria persisted for 7 weeks, but by the 15th week following the bite, her urine was consistently free of protein.

Respiratory Problems

Neurotoxins may cause a progressive and fatal respiratory paralysis, requiring ventilatory support in some cases in addition to the use of antivenom. Obviously this is a major respiratory

problem, but has already been discussed. However, in addition to these problems, pulmonary oedema, (excessive fluid in the lungs) may also occur and be severe enough to require treatment. In one fatal case (Knyvett and Molphy 1959) autopsy showed a fibrinous and diffuse polymorphonuclear cell exudate in grossly congested and oedematous lungs. The lower lobes of the lungs were consolidated. The severe pulmonary oedema prior to death undoubtedly contributed to the difficulties in this case in the use of assisted ventilation, and death was considered to be due to the acute pulmonary oedema. In a fatal case of Mulga Snake (*Pseudechis australis*) bite (Rowlands *et al.* 1969) autopsy findings included basal congestion of the lungs and pulmonary oedema. In a non-fatal case of Tiger Snake (*Notechis scutatus*) bite (Hood and Johnson 1975) with renal failure, extensive bilateral bronchopneumonia developed, again requiring ventilatory assistance for several days.

The unconscious or semiconscious snake bite victim is also at risk of inhalation of vomitus, and consequent aspiration pneumonia. This may particularly occur if the patient has sudden and unexpected convulsions associated with vomiting, and such a case has been described following Tiger Snake bite (White *et al.* 1984). In this case the aspiration pneumonia was quite severe, and in addition to other problems was potentially life threatening.

Local Tissue Injury

In many discussions of snake bite on other continents, particularly where bites by vipers are involved, local tissue destruction around the site of the bite is frequently seen. However, in Australia major local tissue destruction around the bite site is apparently extremely rare (White 1981, 1983b). The local reaction to envenomation varies with the species of snake involved. Brown Snakes (*Pseudonaja* spp.) usually cause little or no local pain, swelling, or redness, and the same applies to the Adelaide Hills variety of Copperhead (*Austrelaps* sp.).

Death Adder (*Acanthophis* spp.) bites may cause significant local pain and minor degrees of swelling, the pain being present for several weeks on some occasions.

At the other extreme bites by Tiger Snakes (*Notechis* spp.), Red-bellied Black Snakes (*Pseudechis porphyriacus*), and Mulga Snakes

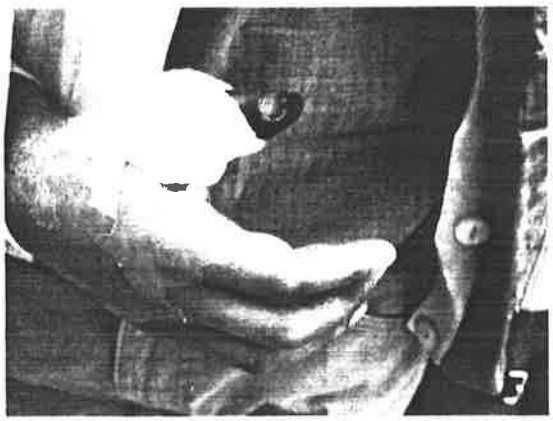


Fig. 6. The right hand of a 24 year old amateur herpetolgoist several days after a bite to the base of the thumb by a Mulga Snake (*Pseudechis australis*). Gangrene of the thumb is already obvious.

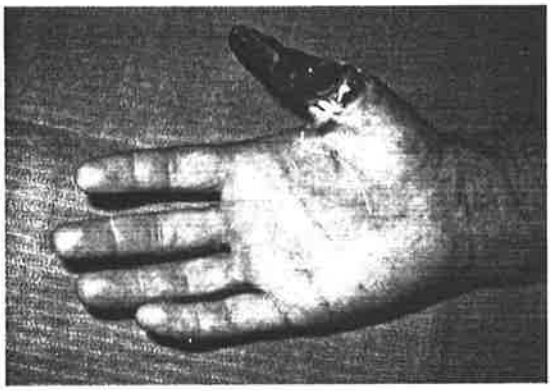


Fig. 7. Same case as in Figure 6, approximately two weeks later, showing well demarcated necrosis of the right thumb.

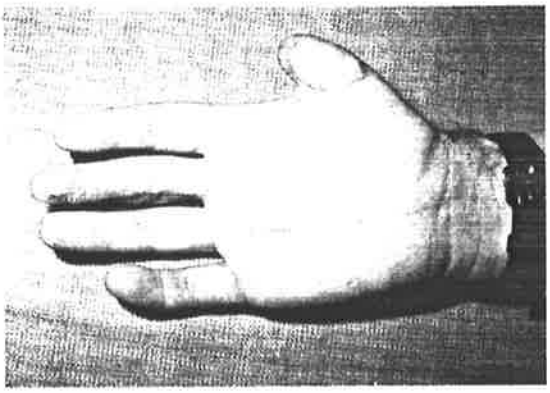
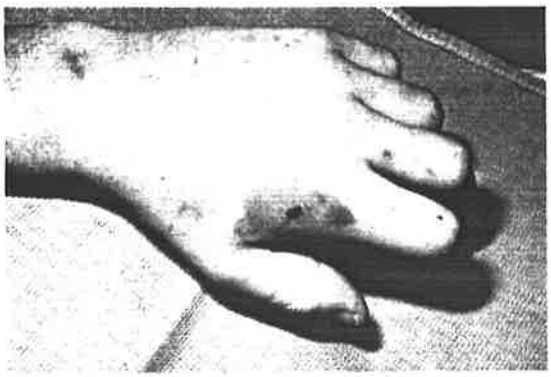


Fig. 8. Same case as in Figures 6, and 7. Approximately 6 months later, the dead thumb having been amputated, and minor reconstruction of the thumb having been achieved.

(*Pseudechis australis*), are often associated with local pain and swelling, and on occasion small areas of bruising or even necrosis.

Two cases of Mulga Snake bite with local damage have been reported. In a fatal case there was gross oedema and discolouration of the bitten hand and arm, with severe subcutaneous oedema, haemorrhage and infiltration with polymorphonuclear cells at autopsy (Rowland *et al.* 1969). In a non-fatal case the victim was bitten at the base of the thumb and subsequently developed gangrene of the thumb requiring amputation and plastic surgical reconstruction (Vines 1978; White 1981, 1983b). This case is illustrated in Figs 6, 7 and 8.

In the author's experience small areas of bruising, and occasionally necrosis leading to small ulcers or even more extensive areas requiring skin grafting occur following bites by Tiger Snakes (Fig. 9). These are illustrated in more detail under the section on Tiger Snake bites. Frost (1980, 1981) has made similar observations following Tiger Snake bite, although in at least one of these cases a pressure/immobilisation bandage was left in place over the bite site for a considerable period of time, and the consequent local immobilisation of venom may have contributed in part to the extent of local tissue destruction seen in this case (Sutherland 1981). Harvey *et al.* (1982) have reported a similar case, with necrosis at the bite site, associated with prolonged pressure/immobilization first aid, following a bite by a Black Tiger Snake (*Notechis ater humphreysi*). Local tissue necrosis of the bite site has also been reported following the bite of a Taipan (*Oxyuranus scutellatus*) (Benn 1951). A case of severe snake bite with paralysis caused by an unidentified snake was reported by Fotheringham (1971) and in this case some necrosis of skin between the supposed puncture wounds on the leg occurred, with subsequent infection and cellulitis, but ultimate response to antibiotic therapy. This case is illustrated in Fig. 10.



▲ Fig. 9. Tiger Snake bite to the first web space of the right hand of a 22 year old amateur herpetologist. This photo was taken several hours after the bite. Note the mild oedema of the whole hand, and a sizable area of skin discolouration around the site of the bite. This well demarcated area subsequently became necrotic and required a skin graft.

► Fig. 10. A small area of ulceration on the leg of an 8 year old boy bitten by an unidentified snake. This ulceration, pictured here approximately one week after the bite, developed between two apparent fang puncture marks.

Despite the list of cases, it must be emphasised that local tissue injury is a relatively insignificant part of snake bite problems in Australia.

Other Clinical Problems Associated with Snake Bite

The major problems associated with systemic envenomation following Australian snake bite have already been discussed, but occasionally other problems of significance in managing cases of snake bite do occur, though often they are not reported. White *et al.* (1984) in reporting a serious case of Tiger Snake (*Notechis scutatus*) envenomation noted that their patient

developed a severe and unexplained hyponatremia (low level of sodium in the blood) and this appeared to be the immediate cause of a major convulsion the consequences of which were potentially life threatening to their patient. As the patient was receiving adequate and appropriate intravenous therapy at the time, the cause of this hyponatremia was considered uncertain by the authors, and it would seem reasonable to ascribe this effect in some way to the action of venom, either directly or far more likely indirectly. Other disturbances of blood electrolytes may potentially occur, and while not well described, the potential for cardiac abnormalities, particularly arrhythmias, cannot be excluded.

Death

Obviously the most serious potential outcome of any snake bite is the death of the victim. In Australia's early history death following snake bite was probably a frequent outcome, although as discussed earlier the actual death rate varied from snake to snake. Even so, in the pre-antivenom era the majority of people bitten by snakes did not die, and death from snake bite is becoming increasingly rare in Australia as modern methods of treatment, including first aid, are improved. As part of an ongoing research project, fatal snake bites in Australia since 1968 are being reviewed by the author. An initial analysis of 21 cases of fatal snake bite during this period was presented in Table 5. It can be seen from this that a wide variety of problems may be responsible for fatality following snake bite, but the author's present impression on reviewing these records is that delayed treatment, often because the victims themselves do not or are unable to seek early treatment, is a significant cause of fatality. This has been further discussed elsewhere (White and Pounder 1984; White *et al.* 1985).

EFFECTS OF BITES BY AUSTRALIA'S VENOMOUS SNAKES

Here, each major snake group is treated separately with a discussion of the clinical pattern of systemic envenomation. The information for each of these groups is combined in a summarised table, Table 14 (White 1981, 1986). Brief histories of cases of snake bite are given wherever possible. The author has had direct involvement in the management of each of these cases, though not necessarily from the outset of medical managment.

Brown Snakes (*Pseudonaja* species)

The major species in this group are: Common or Eastern Brown Snake, *Pseudonaja textilis*; Western Brown Snake or Gwardar, *Pseudonaja nuchalis*; Dugite, *Pseudonaja affinis*.

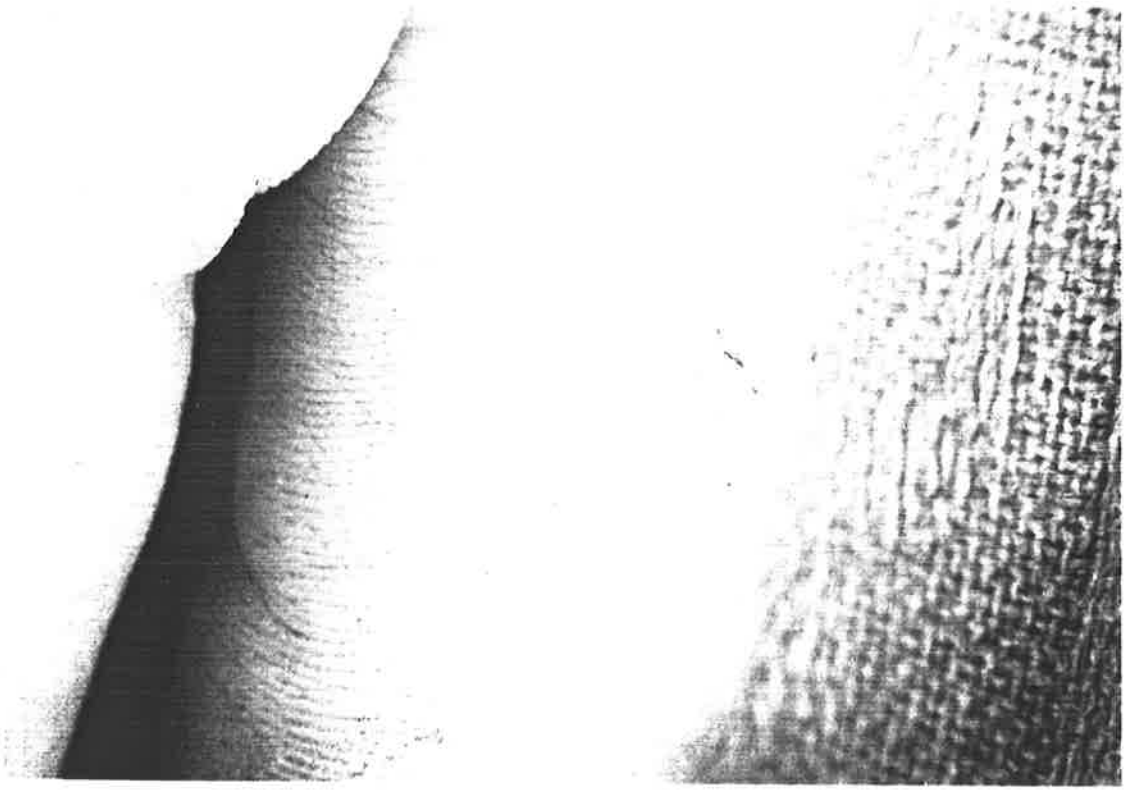
It is likely in the near future that the Brown Snake group will be split into many more species (Mengden 1985).

Table 14: Summary of clinical effects of envenomation by Australian elapid snakes (after WHITE 1981, 1986: WHITE and POUNDER 1984).

Snake/Venom Group	Local Problems at the bite site			Systemic Problems			
	Pain	Swelling	Bruising (± mild necrosis)	Collapse or Unconsciousness	Paralysis	Coagulopathy	Muscle destruction (Muscle movement pain/myoglobinuria)
Brown Snakes (*Pseudonaja* spp.)	Absent or minimal	Nil	Nil	Usual (± convulsions)	Uncommon	Usual, severe	Nil
Tiger Snakes (*Notechis* spp.)	Frequent	Frequent but mild	Often present	Usual (± convulsions)	Usual and sometimes severe	Usual, severe	Frequently present
Mulga Snake (*Pseudechis australis*)	Often minor	Usually severe	Usually absent or minimal but necrosis may occur	Usual	True paralysis not seen	Frequent, but often mild	Usual and sometimes severe
Red-bellied Black Snake (*Pseudechis porphyriacus*)	Often minor	Usual	Variable	Occasionally	Not seen	Unlikely	Frequently present, though often minor
Death Adders (*Acanthophis* spp.)	Frequent	Minimal or absent	Absent	Unusual	Usual and sometimes severe	Unlikely	Not seen
Taipans (*Oxyuranus* spp.)	Variable, sometimes absent	Often minimal	Unusual	Usual (± convulsions)	Usual and sometimes severe	Usual, severe	Probably frequently present

Table 15: Summary of reported cases of Brown Snake (*Pseudonaja* spp.) bites.

Reference	Age of victim	Sex	Impaired conscious state	Convulsions	Paralysis	Coagulopathy	Muscle destruction	Renal failure	Local tissue injury	Outcome
FAIRLEY (1929)	29	F	?	?	+	+	–	–	–	Fatal
FOXTON (1914)	29	M	+	+	–	+	–	?	–	Fatal
SCHAPEL *et al.* (1971)	19	M	–	–	–	+	–	–	–	Survived
HERRMANN *et al.* (1972)	50 35 45	M F M	– – –	– – –	– – –	+ + +	– – –	– – –	– – –	Survived Survived Survived
HARRIS *et al.* (1976)	35 60 53	M M F	– + +	– – –	– – –	+ + +	– – –	+ + +	– – –	Survived Survived Survived
CRAWFORD (1980)	57	M	–	–	–	+	–	–	–	Survived
SUTHERLAND *et al.* (1982)	27	F	–	–	?	?	–	–	–	Fatal
WHITE (1981)	2 7	M M	+ +	+ –	– –	+ +	– –	– –	– –	Survived Survived
WHITE & FASSETT (1983)	26	M	+	–	–	+	–	+	–	Survived
SUTHERLAND *et al.* (1975)	42 66	F F	+ –	+? –	– –	+ –	– –	– –	Induration –	Survived Survived



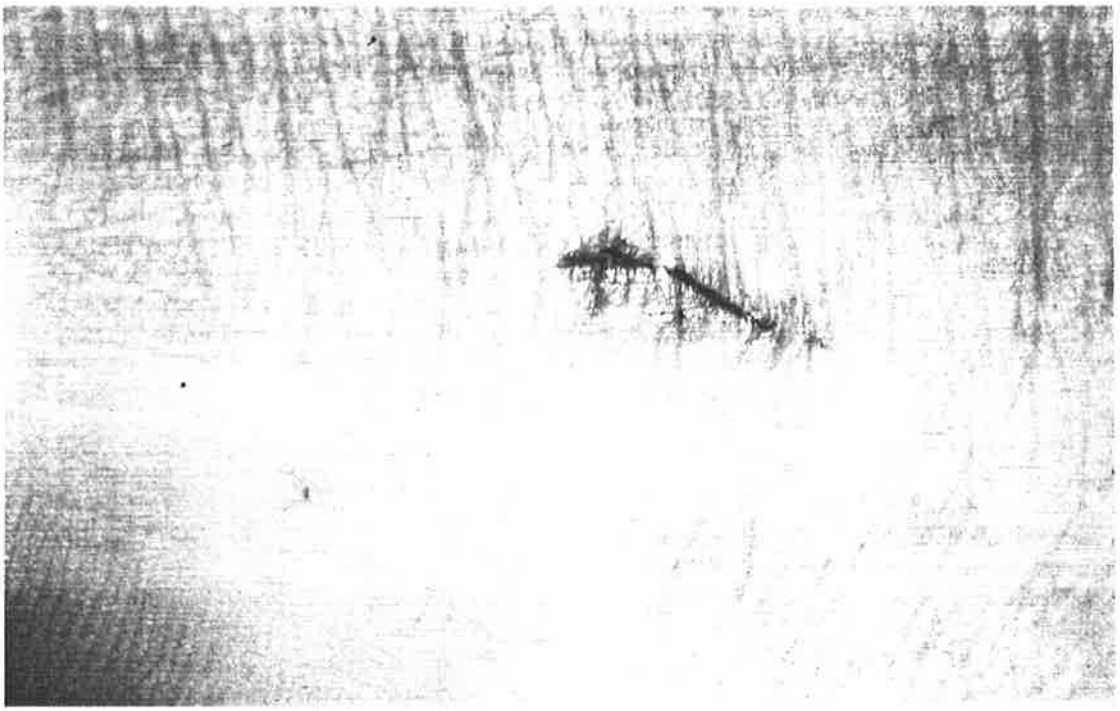


Fig. 13. Same case as Figure 12. Note the absence of local reaction, and a laceration between the two fang marks, made as part of first aid treatment used by the patient.

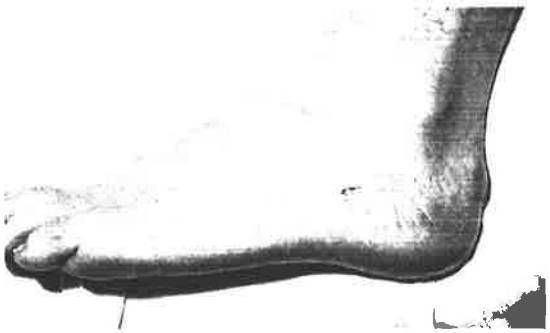


Fig. 12. Brown Snake (*Pseudonaja* sps) bite to the foot of a 21 year old man, associated with mild systemic envenomation.

Brown Snakes are found throughout most areas of mainland Australia, and are probably the most significant cause of snake bite overall throughout Australia. In general they produce only a small amount of venom, through a small fang, but this venom is highly toxic, and may cause a variety of problems including coagulopathy (defibrination syndrome), and paralysis. A summary of findings in reported cases is given in Table 15 (see also Tables 11, 12, and 13). In the author's experience in South Australia about 30% of bites show evidence of systemic envenomation, with approximately a third of these or more showing evidence of a defibrination syndrome, and over 60% showing evidence of decreased conscious state, loss of consciousness and even on occasion convulsions. Renal failure can occur, and is apparently associated with a true disseminated intravascular coagulation, and a microangiopathic haemolytic anaemia, and is often associated with delayed treatment. The bite site itself is usually either pain free, or there is minimal local pain, and local swelling or necrosis is not seen. As the fangs are small, fang entry marks may be hard to identify, at least for the first 30 to 60 minutes following the bite, and scratches are probably more common than actual fang marks. Typical cases are illustrated in Figs 11 to 18.

◄ Fig. 11. A Brown Snake (*Pseudonaja* sp.) bite to the thumb of a 7 year old boy. Note the linear scratches without obvious fang marks. Despite the small extent of the local lesion, this child developed significant systemic envenomation, with a marked defibrination syndrome (see Table 12, after White 1981).

Systemic symptoms may develop rapidly following Brown Snake bite, with headache,

nausea, vomiting and collapse to unconsciousness sometimes occurring within 15 minutes of the bite, at least in children. In adults the events may be less sudden or severe, and intense abdominal pain is more frequently seen. Though rarely seen paralysis may occur after several hours, but muscle destruction does not appear to occur. Because the fangs are small, persistent oozing from fang puncture marks as a result of a defibrination is not usually seen. The author has seen one case of Brown Snake bite in a 19 year old man, associated with paralysis but no coagulopathy, this being the reverse of the normal situation in Brown Snake bites. However, the bite occurred in a remote area of South Australia where unusual varieties of Brown Snake are known to occur.

CASE 1: BROWN SNAKE BITE

A 21 year old man was bitten on his left foot by a 1 metre snake (subsequently identified as a Common Brown Snake, *Pseudonaja textilis*), while he was picking a hay bale up off the road at 1.00 am, on a warm summer night, in a rural area.

He killed the snake, applied a 'tourniquet' to his lower leg, and sought help from friends at a nearby residence. Here he incised between the two puncture wounds.

Within an hour he felt dizzy, developed a headache, blurred vision, nausea, and vomited. He was taken to a country hospital, where his BP was 130/80, pulse 80 regular. He was given i.m. antihistamine and transferred to a city hospital.

On arrival there, some 4 hours after the bite, he was still dizzy, but alert, without evidence of paralysis, but complained of minor pain in the bitten foot. There was no evidence of local reaction at the bite site (Figs 12, 13). Shortly thereafter he developed severe crampy abdominal pain, causing him to writhe on the bed. Clotting studies were normal. There was no evidence of haemolysis.

He was given one ampoule of polyvalent AV i.v. slowly, preceded by i.v. antihistamine. The abdominal pain subsided approximately 3 hours after commencement of AV, and thereafter he made an uneventful recovery. No further pain, swelling or other local reaction was observed at the bite site.

CASE 2: BROWN SNAKE BITE

A 42 year old woman was bitten on the mid-calf of her right leg one evening while attending to her market garden. She saw a brown coloured snake, 1 metre or more in length. She did not apply first aid, but sought help at the local hospital.

There two sets of puncture wounds were noted, without associated oedema or bruising (Figs 15,16). Initially she was symptomatically well but anxious, but then developed a headache. Clotting studies at 1 hour post bite showed a marked coagulopathy, with raised FDPs (1380 mg/1; normal 10). Brown Snake venom was detected at the bite site by C.S.L. Venom Detection Kit (V.D.K.; see P.437).

One ampoule (1000 units) of Brown Snake AV

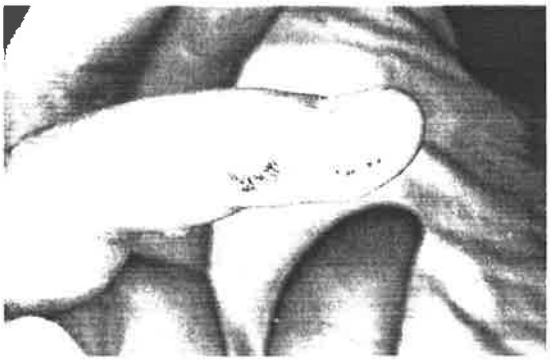


Fig. 14. A Brown Snake (*Pseudonaja textilis*) bite to the tip of the ring finger of a 24 year old herpetologist. Note the lack of local reaction. Significant systemic envenomation did not occur.

was given i.v. slowly, diluted, commencing 2 hours post bite. Headache continued most of the night, and there was occasional pain at the bite site, but no evidence of local reaction. The coagulopathy showed rapid resolution following AV, with return to normal parameters by 48 hours post AV, and blood was again clottable in vitro by 2 hours post AV (though still abnormal of course).

The patient made an uneventful recovery, and no necrosis or other problems developed at the bite site.

CASE 3: BROWN SNAKE BITE

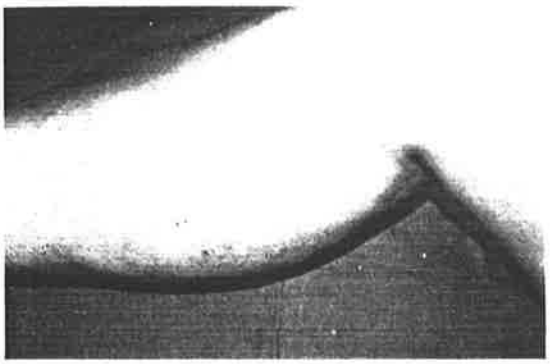
A 19 year old man was working under his house, at a remote mining settlement, when he felt a minor sting on his arm. No snake was seen. He ignored this sting, but about 45 minutes later he developed a headache, felt dizzy, and became nauseated. He presented to the first aid station for treatment, where a tentative diagnosis of snake bite was made. The Royal Flying Doctor

Service was called, and on their arrival, about 2 hours later, he had generalized muscle weakness, blurred vision, and was hypersensitive to touch. Neither VDK results nor clotting studies were available.

A diagnosis of snake bite was made, and as the only potentially dangerous snakes in the area were Mulga Snakes and Brown Snakes, it was recommended that Brown Snake AV and Black Snake (Mulga Snake) AV be given. These were administered slowly, diluted, and without apparent reaction. However, one hour later he developed acute crushing chest pain, cyanosis, and became clammy. This occurred while in the R.F.D.S. aircraft returning to the city hospital, and BP was not noted, though presumably low. Adrenaline 1:1000 was given s.c. and the symptoms rapidly resolved.

On arrival in Adelaide he was alert, but exhibited general muscle weakness. The bite site was not swollen, no bite marks were visible, but it was slightly tender to touch. The bite area was tested for venom by VDK and was positive for Brown Snake venom. Blood taken prior to AV administration showed no evidence of coagulopathy, and later coagulation studies in Adelaide were also normal. The serum creatine phosphokinase levels remained normal. Over the following 24 hours he slowly regained muscle power which was virtually normal by day 3.

This case is interesting as it represents a Brown Snake bite with paralysis and no coagulopathy, the reverse of the usual clinical picture in South Australia. The snakes in the



◄ Fig. 15. A Brown Snake (*Pseudonaja* sps) bite to the calf of a 42 year old woman. Systemic envenomation developed, with a defibrination syndrome.

▼ Fig. 16. Same case as Fig. 15. Note the apparent fang and teeth entry marks with slight scratching, and lack of associated local reaction.

area from which the patient came had only recently been extensively surveyed by the author. Two distinct varieties of Western Brown Snake, *Pseudonaja nuchalis*, were found in the region, in addition to Mulga Snakes. It may be that one of these snakes has distinctively different venom composition from that of its better known brethren. This is the subject of current research in the author's laboratory.

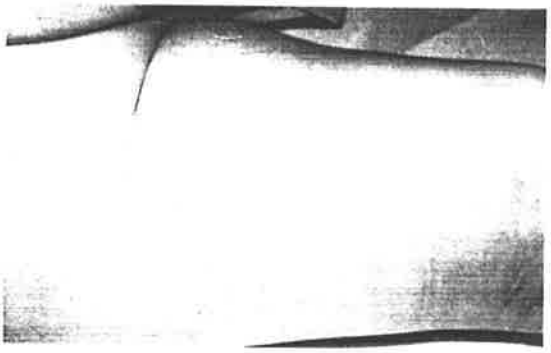
Tiger Snake Group (*Notechis* spp.)

This group includes the Mainland Tiger Snake (*Notechis scutatus*) and the Black Tiger Snake (*Notechis ater*).

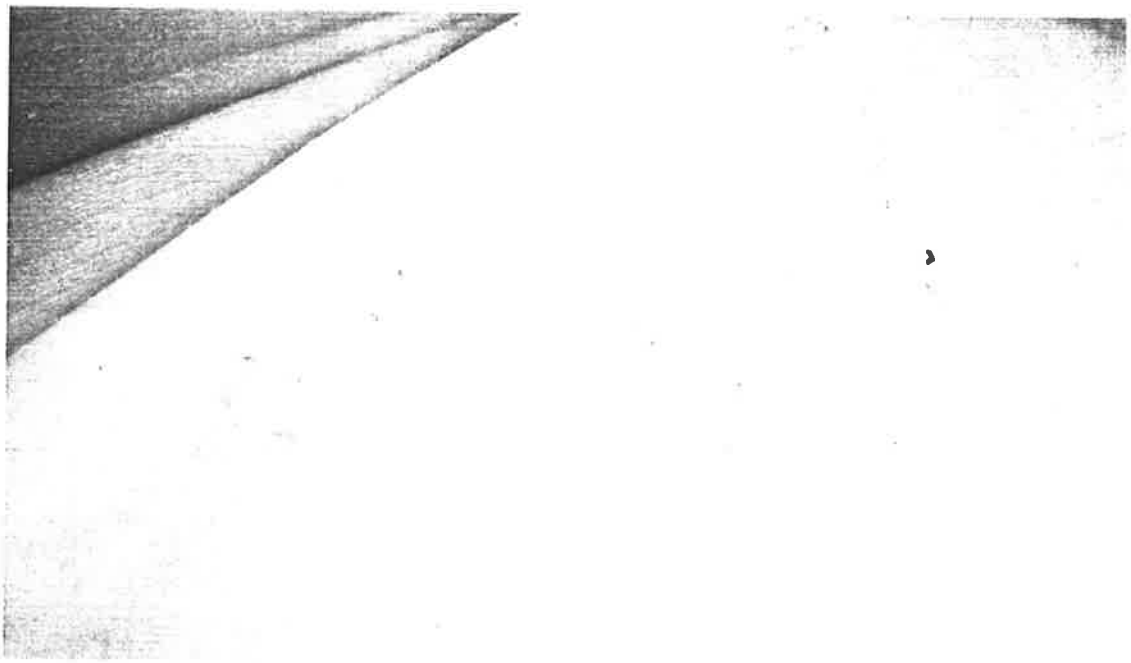
The taxonomy of the Tiger Snake group is still the subject of some discussion. Tiger Snakes are frequently responsible for bites in Australia and there are numerous reports of their bites in the literature, some of which are summarised in Table 16.

Tiger Snake venom contains components capable of causing both presynaptic and postsynapthic paralysis, marked coagulopathy, and muscle destruction; it has also been noted to cause renal failure and disturbances in serum electrolytes. The fangs are moderate in size, and a significant amount of venom can be injected. Thus the Tiger Snake is a very dangerous snake, and without treatment up to 40% of victims might be expected to die.

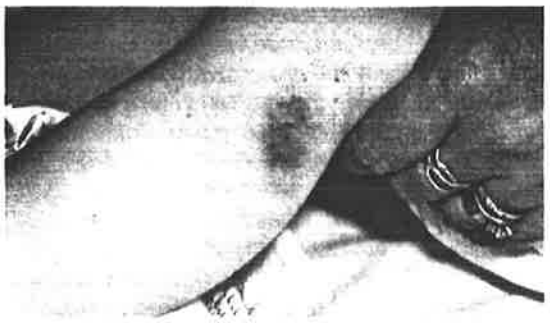
The Tiger Snake is a wetlands snake, most usually associated with swamps, rivers or creeks, and while principally active during the daytime, may also be active on warm nights. When threatened it can become aggressive, and multiple bites may occur.



◄ Fig. 17. A Brown Snake (*Pseudonaja* spp.) bite to the upper thigh of a 2 year old boy. This child developed severe systemic envenomation with convulsions, and a severe defibrination syndrome (see Table 11, after White 1981).



▼ Fig. 18. Same case as Figure 17. Note the small fang puncture marks and associated scratches. Two distinct areas of teeth entry marks in this case indicate that the snake has bitten twice.



▲ Fig. 19. A Tiger Snake (*Notechis scutatus*) bite just beneath the knee in a 2 year old girl. This case developed severe systemic envenomation with initial convulsions paralysis, defibrination, and some evidence of muscle destruction (White *et al.* 1984).

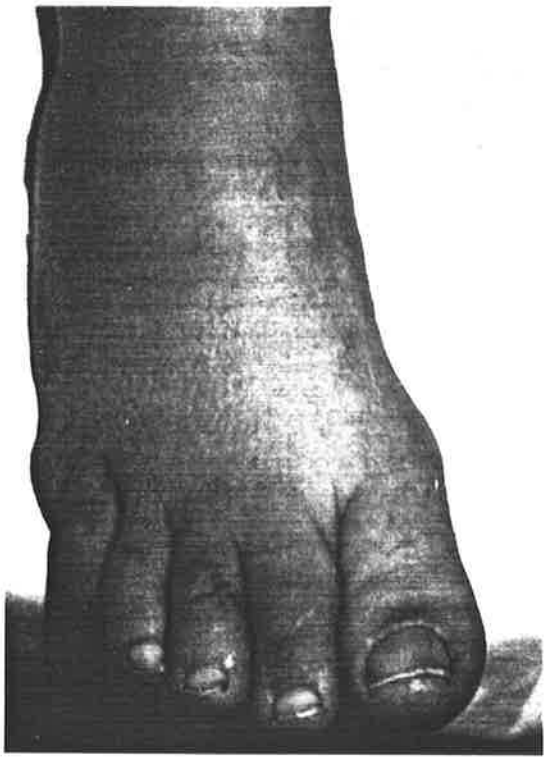
► Fig. 20. Same case as Fig. 19. Note the two distinct sets of teeth and scratch marks indicating a double bite. There is also associated bruising and tissue reaction around the bite site, as often seen with Tiger Snake bites.

In the author's experience Tiger Snake bites are usually associated with local pain, and oedema, and bruising around the bite site is frequently seen. Small areas of necrosis are also seen on occasion. (Figs 19 to 22, and Figs 3, 4 and 9). Early symptoms of systemic envenomation include nausea, vomiting, headache and sudden loss of consciousness. Convulsions can occur. The onset of symptoms may be within minutes of the bite, or delayed several hours. There may be profound shock with hypotension. Persistent oozing of blood from the bite site is also commonly seen, in association with a defibrination syndrome.

Without treatment neurotoxic manifestations will often ensue, following a typical pattern with early development of visual disturbance and ptosis, progressing to more generalised paralysis if treatment is delayed. Muscle destruction may also occur with Tiger Snake bites, and renal failure has been described.

Table 16: Summary of some reported cases of Tiger Snake (*Notechis* spp.) bites.

Reference	Age of victim	Sex	Impaired conscious state	Convulsions	Paralysis	Coagulopathy	Muscle destruction	Renal failure	Local tissue injury	Outcome
FAIRLEY (1929)	Adult	M	?	?	+	+	–?	–	Bruises	Fatal
FROST (1980)	4 28	M M	? ?	– –	+ +	+ +	? +	– –	? ?	Survived Survived
FROST (1981)	15	M	+	?	+	–	–	–	Necrosis	Survived
GAYNOR (1977)	7	F	+	–	+	–	+	–	Bruise	Survived
SUTHERLAND *et al.* (1975)	2	F	+	–	+	?	?	?	Necrosis	Fatal
SUTHERLAND *et al.* (1977)	9 10	M F	+ +	? ?	? +	?+ ?	?+ +	? +	?+ ?	Fatal Survived
CAMPBELL (1977)	42	M	–	–	–	+	–	–	Oedema	Survived
WHITE *et al.* (1984)	2	F	+	+	+	+	+	–	Bruises	Survived
HOOD & JOHNSON (1975)	47	M	+	–	+?	–	+	+	Oedema	Survived
ROLLISON (1928)	41	M	+	–	+	+	+?	–	Oedema, bruising	Fatal
SUTHERLAND (1983)	12	F	+	+	+	+	?	–	?	Survived



▲ Fig. 21. The foot of a 27 year old woman bitten by a Tiger Snake (*Notechis scutatus*). This patient developed paralysis and defibrination.

► Fig. 22. Same case as Fig. 21. Note the virtually joined teeth entry and scratch marks on the toe, with a tiny area of superficial necrosis around one fang entry area. There is mild swelling of the toes.

CASE 4: TIGER SNAKE BITE

A 27 year old woman in a rural area was bitten on her left middle toe while walking barefoot on her property in the early evening. A snake was seen and described as brown in colour. She promptly sought help at the local hospital, arriving there approximately 30 minutes after the bite.

At this stage she complained of headache, vomiting, and pain in the bitten toe, which was swollen and black. Some 9 hours after the bite she was noted to have ptosis and difficulty in swallowing and speaking, and there was a subjective sensation of numbness in the mouth. At this stage she was given one ampoule of polyvalent AV i.v., diluted and given slowly, preceded by s.c. adrenaline. AV administration was uneventful. Blood taken at this time failed to clot.

The patient was subsequently transferred to a major city hospital where she made a steady recovery, without the need for further AV. The coagulopathy rapidly resolved. No electrolyte abnormality was detected. Creatine phosphokinase was not measured, but serum LD and AST were both initially elevated. Renal function was normal. Tiger Snake venom was detected in early urine specimens.

At time of transfer she was noted to have bilateral ptosis (Fig. 3); equal, reacting pupils; some loss of taste (to salt); restricted tongue movements; reduced muscle tone; normal reflexes. There were several scratch and puncture marks on the bitten toe, with mild swelling, and a small area of necrosis (Figs 20, 21). The necrosis resolved without need of surgical intervention. The ptosis and loss of taste took several weeks to recover fully, and the patient noted she was weak for many months later.

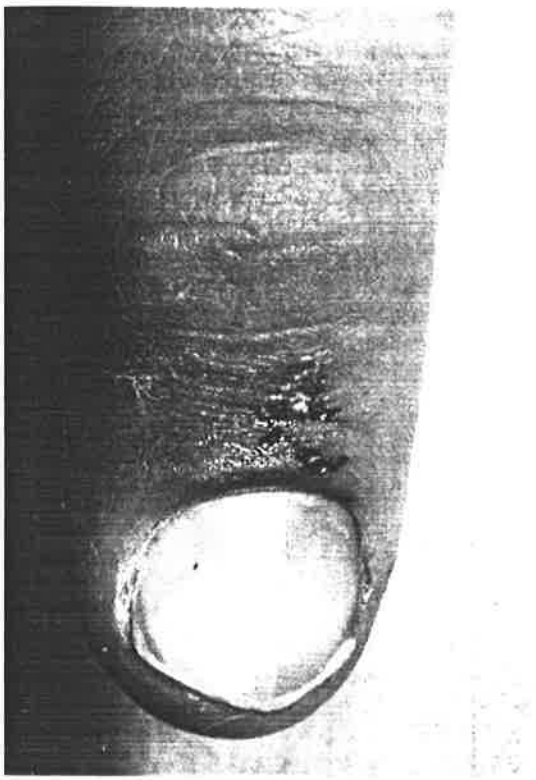
In the region of Australia in which this patient lived, Tiger Snakes are common, including unbanded brown colour phases, which are superficially similar to Common Brown Snakes.

CASE 5: TIGER SNAKE BITE

A 66 year old man was bitten by a Tiger Snake on the right index finger while working on his rural property. He apparently did not initially consider the bite significant, and so did not apply first aid. A short while later (within 30 minutes) he developed a headache, and felt faint. He then presented to the local hospital where he was given one ampoule (3000 units) of Tiger Snake AV i.v. preceded by antihistamine and hydrocortisone, the infusion commencing approximately one hour after the bite. Blood

taken at this time would not clot, and was positive for Tiger Snake venom (VDK).

He became drowsy over the next hour, although he showed no sign of ptosis or other features of early paralysis. Repeat clotting studies at 2 hours post bite again showed a coagulopathy, with failure of the blood to clot. He was therefore given a further one ampoule of Tiger Snake AV i.v. at 4 hours post bite, without incident. He was subjectively well by this stage, and remained so from then on. However, repeat



clotting studies at 8 hours post bite showed continued coagulopathy, so he was transferred to a major city hospital.

Further clotting studies performed at 11 hours post bite and some 6 hours after the second dose of AV showed virtually normal clotting, but with decreased fibrinogen (0.27 g/l; normal 1.5–4.0) and grossly raised FDPs (5,120 mg/l; normal 10). Subjectively he was well, and there was no evidence of paralysis, haemorrhage, muscle damage, or local tissue injury at the bite site, although this showed mild swelling and a single puncture mark.

Clotting function remained acceptable, with gradual return to normal of all parameters over the following 48 hours.

This case illustrates the need to titrate AV against evidence of envenomation, and the principle that once sufficient AV has been given, a venom-induced coagulopathy will reverse.

Mulga Snake (*Pseudechis australis*)

The Mulga Snake is a large member of the Black Snake genus, found in arid areas throughout mainland Australia. It has large fangs and can inject a very large amount of venom.

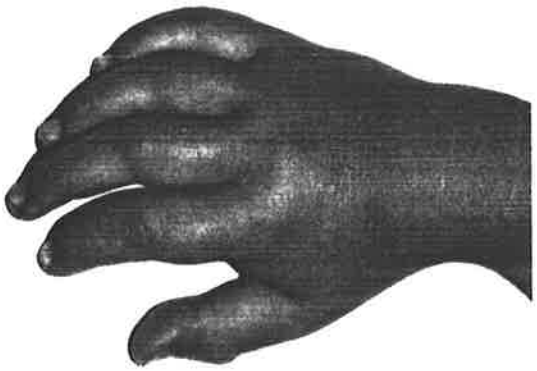
Relatively few bites from this species have been reported (Rowlands *et al.* 1969; Vines 1978; White 1981; Sutherland 1983; Balmain and McClelland 1982).

The venom of the Mulga Snake is less toxic than many other Australian species, but as a large amount of venom may be injected, it must still be considered as a very dangerous snake. The venom does not appear to contain any neurotoxins, so true paralysis is unlikely to be seen following bites by this species, but it contains potent myolysins, and the consequent severe muscle destruction may mimic paralysis. The venom may also cause a marked coagulopathy.

In the author's experience Mulga Snake bites are associated with minor to significant local pain, but are always associated with marked

◄ Fig. 23. The thumb of an 11 year old boy bitten while trying to catch a small Mulga Snake (*Pseudechis australis*). Note the few small puncture and scratch marks around the bite site with mild local reaction.

▼ Fig. 24. Same case as Fig. 23. Note that there is a significant swelling of the entire hand extending to involve the forearm. Obvious extensive swelling of this type is frequently seen following Mulga Snake bite.



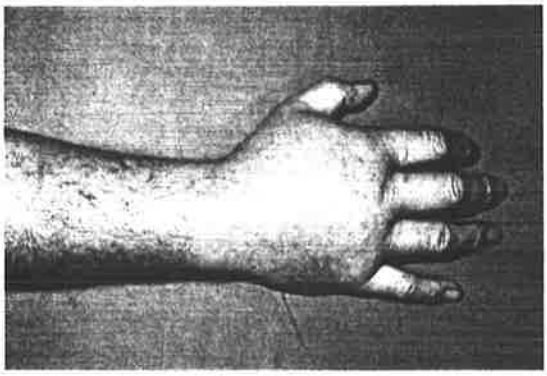


Fig. 25. The hand of a 48 year old man approximately 4 days following a bite by a Mulga Snake (*Pseudechis australis*). The bite was not noticed by the patient, the diagnosis being made 3 days later by venom assay. Note the marked swelling of the entire hand and distal forearm. No acutal bite site could be identified.

local swelling of the area around the bite extending up the bitten limb, and lasting several days (Figs 23-25). While local necrosis has been described in one case, the author has not seen it in other cases of bites by this species. There may be impaired conscious state, though this is not a universal finding. Some form of coagulopathy, often mild, is seen in the majority of cases. As muscle destruction is the prominent feature of Mulga Snake bites, myoglobinuria is to be expected. There is therefore a potential for renal failure.

CASE 6: MULGA SNAKE BITE

A 48 year old man in a remote country area was found unconscious by his son at 9.00 am and taken to the local hospital, where he was noted to be drowsy, cold, BP 90/65, Pulse 100 regular, with otherwise normal general examination, except for a swollen right hand. On later questioning the patient noted he had been working on the floor of a disused house, pulling up floorboards all the previous day. He had seen no snakes, and felt no bites. By 9.30 pm that night he felt unwell and nauseated, and went to bed, and next remembered being at the hospital the following morning.

He had a past history of myocardial infarction 5 years previously, and therefore a further episode of myocardial ischaemia was suspected as the primary diagnosis on this occasion. However, the ECG was normal, and enzyme estimation the following day showed a massive increase in creatine phosphokinase (6705 u/l; normal 60-270), and clinically the right hand was more swollen and painful. Urine examination at this time was positive for myoglobin.

In view of these findings, and despite the absence of either a history of snakebite, or signs of fang marks on the swollen hand, the astute medical officer in charge considered snake bite a possible diagnosis. The urine was tested for venom using the VDK (C.S.L.) and was positive for Mulga Snake. (The Mulga Snake is the most common dangerous snake in the locality). Clotting studies done at this time were normal and as the patient was symptomatically well, and kidney function was normal, it was decided to withhold AV.

The patient was transferred to a major city hospital for further observation, and made an uneventful recovery over the following few days, though the right hand remained swollen for over one week (Fig. 25). The serum enzymes were followed serially, with the creatine phosphokinase reaching a peak 3 days post bite of 13,758 u/l, and dropping back to the normal range by day 10.

This case illustrates how a significant snake bite may occur without the victim being aware of it, and the consequent need of medical practitioners to maintain a high index of suspicion for the diagnosis of snake bite.

CASE 7: MULGA SNAKE BITE

An 11 year old boy in a remote rural area was bitten on the right thumb by a small (?900 mm) Mulga Snake while trying to catch the snake. He did not apply first aid. He rapidly developed headache, abdominal pain, nausea, vomiting, and local pain in the bitten hand with associated swelling. He was given one ampoule of polyvalent AV i.v. one hour post bite at the local hospital (no Black Snake AV available) and then a further one ampoule (18,000 units) of Black Snake AV i.v. at 4 hours post bite (brought up by a retrieval team).

He remained drowsy overnight, but by the following morning, after transfer to a major city hospital, he was subjectively well apart from a swollen painful right hand and forearm (Figs 23, 24). At no stage was there evidence of paralysis or haemorrhage. Serial clotting studies on specimens collected at the local hospital and later at the city hospital showed an initial mild elevation of FDPs (30 mg/l; normal 10) and

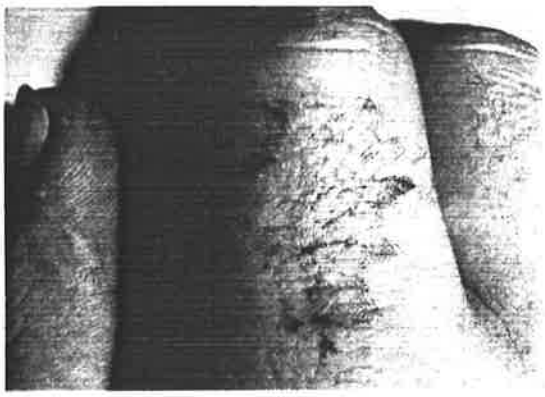
marginal decrease in fibrinogen (1.5 g/l; normal 1.5–4.0), but other clotting parameters were normal, and both FDPs and fibrinogen were normal within 12 hours of the bite. Creatine phosphokinase showed mild elevation, maximal at 17 hours post bite (400 u/l; normal 25–200), and myoglobin was not detected in the urine.

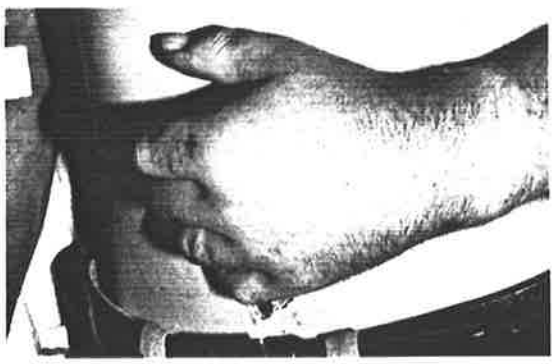
He made a complete recovery, although the swollen right hand took a week to settle. No necrosis developed.

Red-bellied Black Snake (*Pseudechis porphyriacus*)

This species is usually found in association with water. It is a moderate to large sized snake, with moderate sized fangs, but injects only a modest amount of relatively weak venom, and is probably the least dangerous of Australia's dangerous snakes.

There is usually local pain and oedema at the site of the bite, and indeed swelling may involve much of the bitten limb (Figs 26–28). Symptoms of systemic envenomation vary, including headache, nausea and vomiting, and occasionally oozing from the bite site associated with mild coagulopathy. Major coagulopathy following bites by this species is uncommon. Significant muscle destruction may occur, and there is therefore a potential for renal failure. No neurotoxin has been described from the venom, and true neurotoxic paralysis is unlikely to occur, but certainly the patient may have an impaired conscious state or even may become unconscious, and convulsions have been reported.





▼Fig. 26. Fore-finger of a 28 year old amateur herpetologist bitten twice by a Red-bellied Black Snake (*Pseudechis porphyriacus*). Note the evident scratch marks, and swelling of the finger.

◄Fig. 27. Same case as Fig. 26. There is obvious swelling of the entire hand.

CASE 8: RED-BELLIED BLACK SNAKE BITE

A 28 year old man was bitten on his left index finger by his pet Red-bellied Black Snake while displaying the snake to his friend. The snake apparently bit him twice, and was 1 metre long. The incident occurred at the end of an evening spent consuming alcohol with his friends. He had a history of a previous Tiger Snake bite, for which he had received AV.

Apparently no notice was taken of the bite initially, but about one hour later he became dizzy, short of breath, vomited, then lapsed into unconsciousness for a brief period. He was promptly taken to hospital, by which time he had regained consciousness and was symptomatically well apart from some mild 'blurred vision' and pain in the left hand and forearm, which was swollen (Figs 26, 27).

Enzyme screen and clotting studies performed at this time were normal. Apart from several further episodes of vomiting overnight, he made an uneventful recovery. AV was not used because (1) he was bitten by a snake unlikely to cause death in an adult; (2) he was already showing improvement in clinical condition by the time he reached hospital; (3) he had been previously exposed to horse serum, and the risks of reaction to the AV outweighed the risk of no AV treatment in this clinical situation. This was reinforced by his history of snake bites associated with his hobby of keeping snakes, and the likelihood that he would require further AV in the future.

CASE 9: RED-BELLIED BLACK SNAKE BITE

A 9 year old boy was bitten on his right middle toe by a black snake while walking near a creek. He was taken to the local hospital,

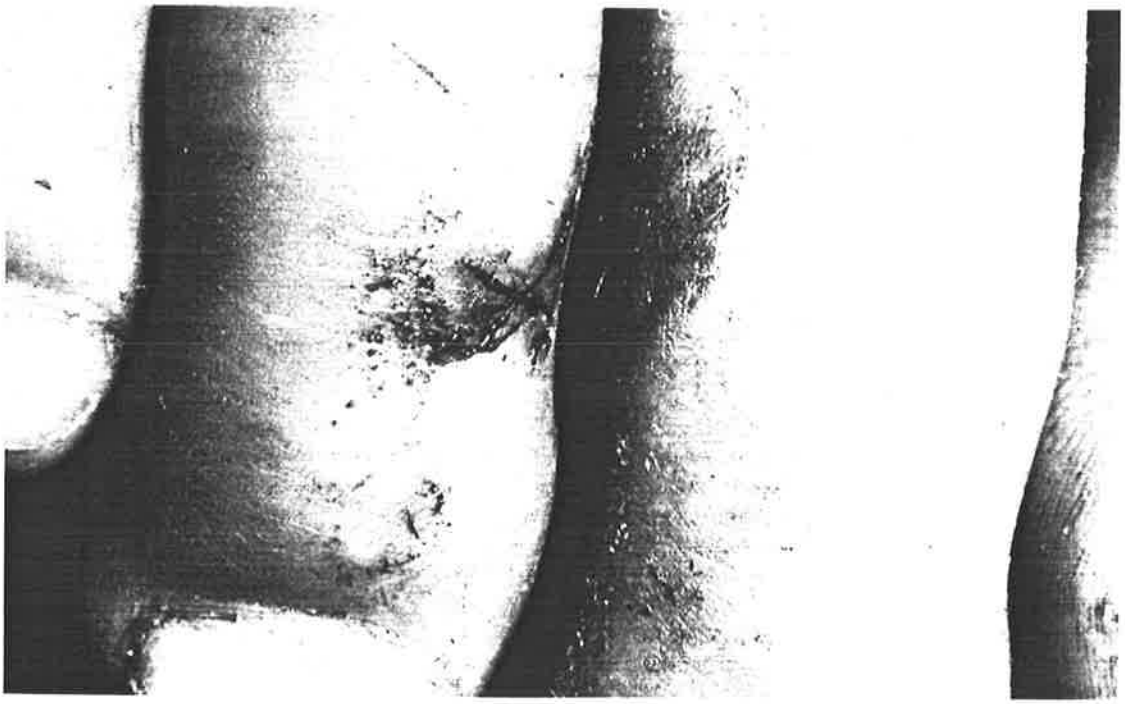


Fig. 28. The foot of a 9 year old boy bitten by a Red-bellied Black Snake (*Pseudechis porphyriacus*). Note the mild swelling.

arriving there approximately ½ hour post bite, by which time he was nauseated, and had abdominal pain, and shortly afterwards he commenced vomiting. A VDK on a swab from the bite site was performed and was positive for Tiger Snake venom. As no Tiger Snakes are found in this area, but Red-bellied Black Snakes are common (and give a positive reaction in the Tiger Snake tube of the VDK), it was decided that the latter snake was the culprit.

He was promptly transferred to a city hospital for further management, and on arrival there, about 2 hours post bite, he was alert but anxious, complained of some pain at the bite site, but was otherwise well. There was no evidence of paralysis or bleeding. Clotting studies on specimens taken at about one hour post bite showed minimal evidence of coagulopathy (PR 1.45; APTT 35 secs; FDPs 80 mg/l; Fibrinogen 2.3 g/l). Further clotting studies taken at 2 hours and about 20 hours post bite were essentially normal, with normal FDPs and normal platelet count and function. Creatine phosphokinase was normal. There was no evidence of myoglobinuria.

In view of the minimal clinical evidence of envenomation and the type of snake involved, no AV was given. At about 4 hours post bite he developed a further episode of crampy abdominal pain, nausea, vomiting, and irritability, lasting 2 hours and settling spontaneously. The pain in the bitten foot settled over 6 hours, but the toe became more swollen, maximal at about 20 hours post bite (Fig. 28), and settling uneventfully over the next few days.

Following the second bout of abdominal pain he remained symptomatically well, and made an uneventful recovery.

Death Adders (*Acanthophis* spp.)

The several species in this group are: Common Death Adder (*Acanthophis antarcticus*); Desert Death Adder (*Acanthophis pyrrhus*); and Northern Death Adder (*Acanthophis praelongus*).

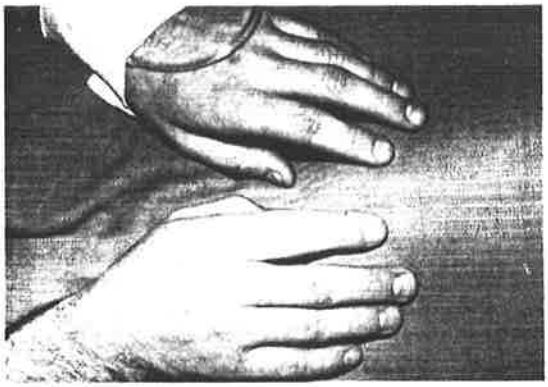
Death Adders have long mobile fangs and are capable of injecting a large amount of venom. The venom is principally neurotoxic, and paralysis is the major feature of envenomation.

The largest series of Death Adder bites reported are those of Campbell (1966) from New Guinea. The Death Adder usually conceals itself in leaf litter and other debris, and will not usually move on the approach of humans. Thus it

is more likely to be trodden on than most other Australian snakes. It is usually active at night, in contrast to all other dangerous snakes in Australia which are active during the daytime, and are only active at night on warm nights. It usually strikes very low so that bites above the ankle on an adult are unlikely to be due to a Death Adder (Campbell 1966). Death Adder bites are probably uncommon on mainland Australia, apart from bites to the hand in reptile keepers who are bitten by their pets. All 6 cases of Death Adder bite in South Australia reviewed by the author were illegitimate bites to reptile keepers, and half of these cases showed some evidence of envenomation, mostly mild.

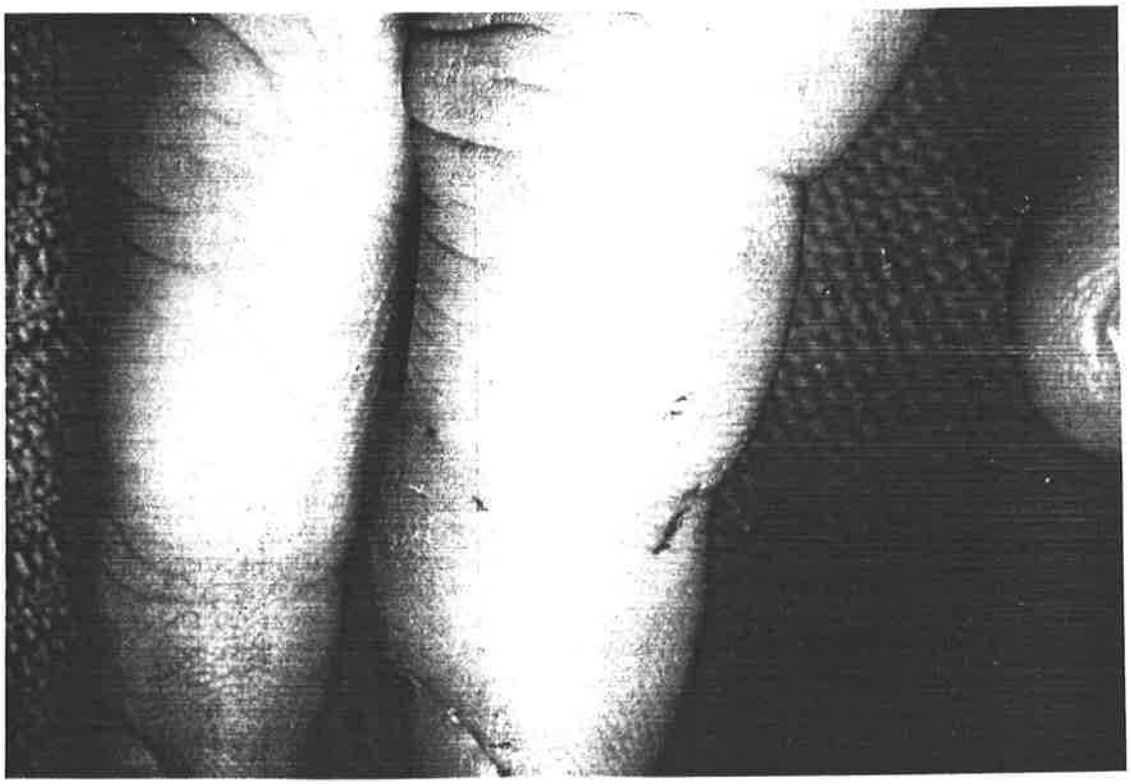
The bite site may be painless, or mildly painful, but sometimes there is apparently mild but tense swelling which may cause pain persistent for days or even weeks following the bite (Figs 29–31). If the bite is on a finger, near a joint, there may be considerable limitation of movement in that finger. Bleeding from the bite site does not occur.

Symptoms are usually mild, until severe neurotoxic paralysis occurs. Early mild headache and occasional vomiting are seen in some cases. There may be some local lymph node pain, which can become severe. The earliest signs of



◄ Fig. 29. The hand of a 24 year old amateur herpetologist bitten by a Death Adder (*Acanthophis antarcticus*). The bite was sustained on the right index finger which shows only very mild local swelling.

▼ Fig. 30. Same case as in Fig. 29. There is mild swelling of the bitten finger only without evidence of other local reaction around the rather small fang marks. This patient developed mild systemic envenomation.



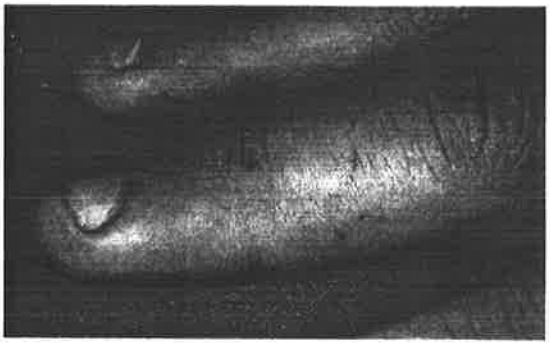


Fig. 31. The right middle finger of a 35 year old amateur herpetologist bitten by a Death Adder (*Acanthophis antarcticus*), showing mild swelling and minuscule fang entry marks, not associated with any other local reaction. This patient developed very mild systemic envenomation not requiring antivenom.

envenomation may develop within one hour of the bite, and include tenderness of local lymph nodes and ptosis as signs of early paralysis (Campbell 1966). In severe cases the paralysis may be total for all voluntary muscles, but cardiac muscle appears unaffected.

The neurotoxic paralysis is reversed readily by antivenom. Coagulation disorders do not appear to occur, and defibrination syndrome has not been reported. Renal failure likewise has not been reported, and the venom does not appear to possess myolytic activity. Local tissue destruction is unlikely.

CASE 10: DEATH ADDER BITE

A 24 year old man was bitten on his right index finger by his pet Death Adder while transferring it between cages. The bite was immediately painful. A compression bandage was promptly applied, the snake secured in its new quarters, and the patient then proceeded to hospital.

Over the next hour he developed right axillary lymphadenopathy, and nausea and vomiting, and the finger became painful, with tense swelling, and then became numb (Figs 29-30). No signs of neurotoxic paralysis were noted. Coagulation studies were normal.

Some 2 hours after the bite 6000 units of Death Adder AV (diluted) were administered i.v. over 40 minutes, preceded by s.c. adrenaline and i.v. antihistamine. The AV infusion was uneventful, and all symptoms of envenomation disappeared. The bitten finger remained slightly swollen and painful for several days.

Taipans (*Oxyuranus* spp.)

These include the Common Taipan (*Oxyuranus scutellatus*), and Inland Taipan (*Oxyuranus microlepidotus*).

The Taipans are undoubtedly the most feared venomous snakes in Australia, and with good reason. They have long fangs and are capable of injecting a large amount of venom which is amongst the most toxic snake venoms known. Indeed, that of the Inland Taipan ranks as *the* most toxic snake venom. Until antivenom became available, the majority of Taipan bites resulted in death. A summary of most of the reported cases of Taipan or probable Taipan bites is shown in Table 17. Details about the local effects of Taipan venom at the site of the bite are scanty, but it would appear that the bites may be painless, and local reaction is usually minimal. However, swelling and bruising and even necrosis have been described in a few cases. The systemic problems of envenomation by Taipans are multiple, and include severe paralysis, severe coagulopathy (namely, the defibrination syndrome) and muscle destruction may occur. Renal failure may potentially occur. Impaired conscious state or loss of consciousness appear to occur frequently with Taipan bites, and convulsions are a common accompaniment of bites by this species. The onset of symptoms may be extremely rapid, and death may occur in less than 60 minutes after the bite, although such a rapid demise is unusual (Sutherland *et al.* 1980).

CASE 11: TAIPAN BITE

A 10 year old boy was bitten on his left middle finger while playing in the garden of his home, in a semi-rural area. First aid was not applied for at least 40 minutes, at which time a pressure/immobilisation bandage was used by ambulance officers. When seen at the regional hospital about 1½ hours after the bite he complained of nausea, and the bitten finger was swollen and black, with two puncture marks visible. There was no evidence of lymphadenopathy, bruising, or paralysis and observations were normal (BP 100/80, pulse 75). He was alert and talking freely. An i.v. line was inserted, clotting studies done, and blood was tested for venom (VDK).

At 4 ½ hours post bite he commenced vomiting, and axillary lymphadenopathy was noted. The results of clotting tests became

available at this time and showed a marked coagulopathy (FDP 1280 mg/l; platelets normal, 295 × 10^9/l).

In view of both clinical deterioration and the coagulopathy he was given one ampoule of polyvalent AV i.v. slowly, diluted, preceded by adrenaline and antihistamine, commencing approximately 5½ hours post bite. About an hour later he developed a red macular rash on his abdomen and legs, and was given hydrocortisone. This settled. Coagulation studies were repeated at 5 hours post bite (i.e. just after completion of AV) and were similar to the previous figures. A further ampoule of polyvalent AV was given at 7 hours post bite. Clotting studies at 10 hours post bite showed no improvement, and a thrombocytopenia was now evident (19 × 10^9/l).

Further advice was sought, and it was recommended that a VDK assay of the bite site be performed. This was weakly positive for Taipan venom. A further one ampoule of polyvalent AV and one ampoule of Taipan AV were given i.v. at 13 hours post bite. The patient was subjectively improved and clotting studies at 15 hours post bite showed substantial resolution of the coagulopathy, with reduced FDPs (80 mg/l) and increased platelets (287 × 10^9/l). At no stage was there evidence of paralysis. Creatine phosphokinase was not measured. The child made an uneventful recovery after the last dose of AV, and the swelling and discolouration of the bitten finger resolved over 24 hours.

This case illustrates the importance of using the bite site to sample for the VDK, and the principle that adequate correct AV will reverse a venom induced coagulopathy in most cases.

Copperheads (*Austrelaps* species)

The taxonomy of Copperheads in Australia is still the subject of revision, and it is likely there will be three species defined. Copperheads have small fangs but can deliver a moderate amount of toxic venom. Bites by Copperheads have been poorly documented in the past and therefore it is difficult to state with certainty the effects that might be expected following bites by these snakes. In the author's experience bites by the

Table 17: Summary of some reported cases of Taipan bites (*Oxyuranus* spp.).

Reference	Age of victim	Sex	Impaired conscious state	Convulsions	Paralysis	Coagulopathy	Muscle destruction	Renal failure	Local tissue injury	Outcome
FLECKER (1940)	49	M	+	–	–	?	–	–	Wound scarified	Survived
	35	M	+	+	?	?	?	?	?	Fatal
	39	M	?	?	+	?	?	?	?	Fatal
FLECKER (1944)	Adult	M	+	+	?	?	?	?	?	Fatal
	20	F	+	+	?	+	–	–	?	Fatal
	Adult	M	+	+	+	?	–	–	–	Fatal
REID & FLECKER (1950)	19	M	+	–	+	?	–	–	?	Survived
BENN (1951)	20	M	+	+	+	?	–	–	Oedema, Bruising, Necrosis	Fatal
SUTHERLAND *et al.* (1980)	4	M	+	?	?	?	?	?	?	Fatal
BRIDGEN & SUTHERLAND (1981)	39	M	?	–	+	+	+	+	–	Survived
TRINCA (1969)	Adult	M	+	?	+	+	?+	–	Oedema, Bruising	Survived
MIRTSCHIN *et al.* (1984)	37	M	–	–	–	?	–	–	–	Survived
CAMPBELL (1967) 6 cases	Adults	5M 1F	1+	–	3+	2+	–	–	–	Survived
LESTER (1957)	10	M	+	–	+	+	–	–	?–	Survived

Adelaide Hills variety of Copperhead have not been associated with systemic envenomation, and the bites have been painless, with no local reaction (Fig. 32). There has been no evidence of either paralysis or coagulopathy. However, Sutherland (1983) quotes a case of Copperhead bite (Victorian variety) associated with impaired conscious state and paralysis. Muscle destruction with myoglobinuria was not recorded in this case, although this might be expected in at least some cases of Copperhead bite. Certainly neurotoxic paralysis is a potential complication. As there are definite similarities between Copperheads and Tiger Snakes, it might be expected that some bites will result in a significant reaction at the bite site, with local pain, swelling and bruising, and possibly even necrosis.

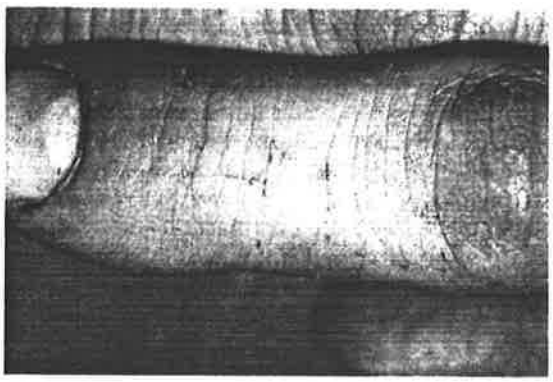


Fig. 32. The index finger of a 24 year old man bitten by an Adelaide Hills Copperhead (*Austrelaps* sp.). The teeth entry marks are small but visible, and there is no other local reaction. No systemic envenomation occurred.

Rough Scaled Snake (*Tropidechis carinatus*)

This species is also related to the Tiger Snakes, but has only small fangs, and a low venom yield. Its venom is less toxic than many other Australian dangerous snakes, but serious and potentially fatal bites can occur and have been documented by Trinca *et al.* (1971) and more recently by Patten *et al.* (1985). The latter case demonstrated evidence of severe paralysis, severe muscle destruction, a mild coagulopathy, minimal local tissue reaction at the bite site, and apparent renal failure. There was loss of consciousness, and at one stage cardiac arrest occurred, but convulsions were not noted. Bites by this species must clearly be treated as potentially very dangerous.

Other Species

A number of other species of Australian venomous snakes have been variously reported to cause injuries to man.

SMALL EYED SNAKE (*Cryptophis nigrescens*)

This small snake has caused at least one fatality due to renal failure associated with massive muscle destruction (Furtado and Lester 1968). There was also a coagulation disorder present in this case. However, the victim was a reptile keeper who had been bitten by this species on several previous occasions without harm, and the venom of this species is certainly less toxic than the recognised dangerous snakes (Broad *et al.* 1979).

BLUE-BELLIED OR SPOTTED BLACK SNAKE (*Pseudechis guttatus*)

As this species is a member of the Black Snake group, bites may result in at least mild envenomation. It is probable that envenomation will most closely follow that seen by the Red-bellied Black Snake, but bites should be treated with caution (Sutherland 1983).

COLLETT SNAKE (*Pseudechis colletti*)

This species is also a member of the Black Snake group, and the venom might be expected to cause significant muscle destruction in a snake bite victim. Thus bites may show similarities to bites of the Mulga Snake, or perhaps the Red-bellied Black Snake.

GENUS *Hoplocephalus*

There are three species in this genus, and they are all rarely encountered except by herpetologists. It is possible that bites by these species may cause paralysis or coagulopathy, and they should certainly be treated with caution (Flecker 1952; Sutherland 1983).

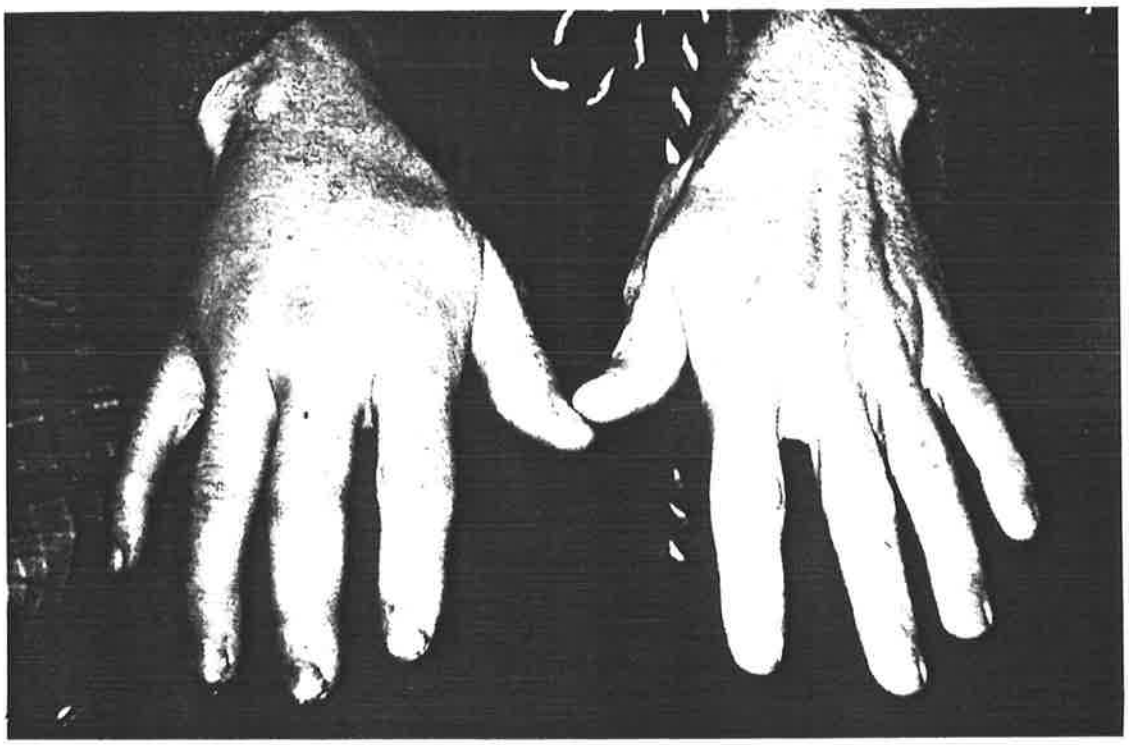
YELLOW-FACED WHIP SNAKE (*Demansia psammophis*)

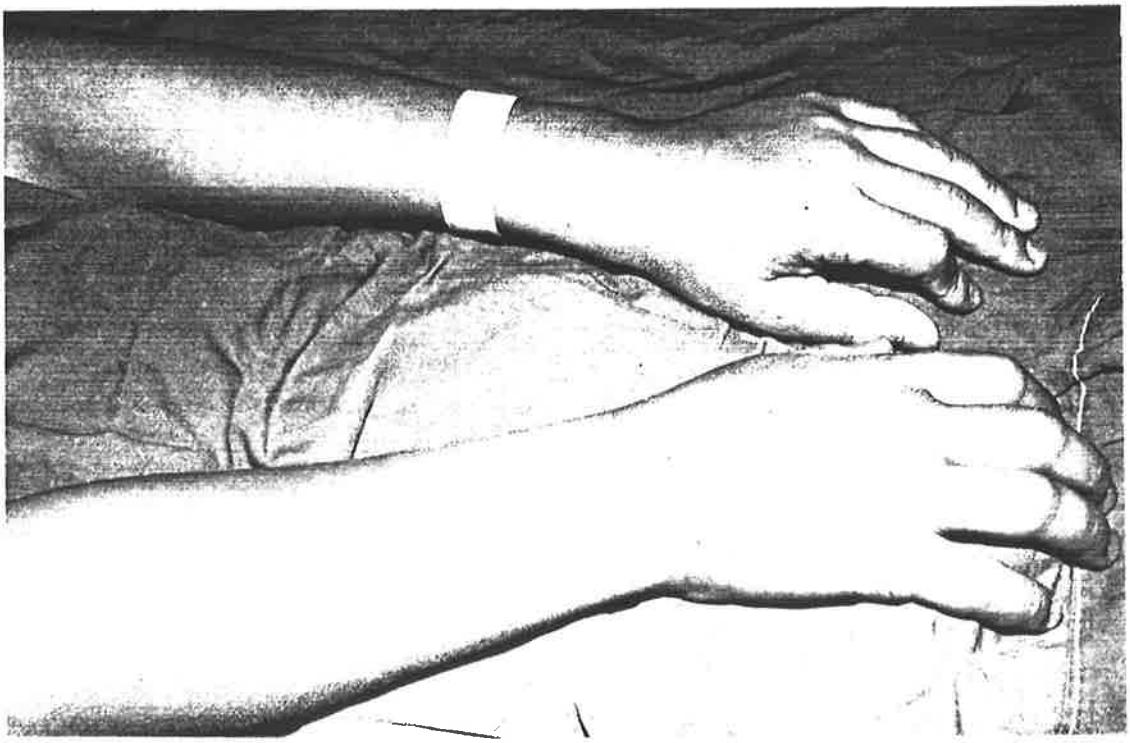
This is a relatively common snake across Australia. Bites cause local pain and swelling, with a very mild systemic illness (Figs 33 and 34). It is responsible for many bites involving children in northeastern Australia.

A more extensive list and details may be found in White (1981) and Sutherland (1983).

◄ Fig. 33. The hands of a 24 year old amateur herpetologist showing obvious swelling of the right hand following a bite by a Yellow-faced Whip Snake (*Demansia psammophis*).

► Fig. 34. Marked swelling in the hand and distal forearm of a 9 year old boy allegedly bitten by a Whip Snake (?*Demansia psammophis*).





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ELAPID SNAKES: MANAGEMENT OF BITES

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◄ Skull of Taipan, *Oxyuranus scutellatus*.

Controversy and the treatment of snake bite seem inextricably linked. Over the years there has been a vast array of treatments suggested, both for first aid and definitive medical management, and the Australian medical literature has abounded with arguments between protagonists of one theory or another. Australia is by no means unique in this regard, and snake bite and its cures seem to have occupied a prominent part in the public imaginations. Even the most recent work on first aid in Australia by Sutherland and colleagues (Sutherland *et al.* 1979), expounding methods which are now generally well accepted, has attracted some spirited comments in the medical literature over the last few years. Campbell (1966a,b) and more recently Sutherland (1983) have reviewed some of the old medical remedies for snake bite including the use of strychnine, and ammonia! We can be thankful that such dangerous 'treatments' have now passed into the realm of history.

The advent of antivenom, commencing with Tiger Snake antivenom produced by the Commonwealth Serum Laboratories (C.S.L.), Melbourne in 1930, ushered in the modern era of snake bite management in Australia. Since this time there has been a steady decline in the death rate from snake bite in Australia (Sutherland 1983; White this volume). More recently the development of a simple, safe, and effective method of first aid by Sutherland and colleagues (Sutherland *et al.* 1979) and the introduction of sophisticated methods of detecting snake venom in victims by the same team (Coulter *et al.* 1974; Sutherland *et al.* 1975) have further improved the outlook for those unfortunate enough to be bitten by an Australian venomous snake.

In discussing the treatment of snake bite in Australia, the principal aims of treatment must be remembered. For Australia, these aims for the treatment of snake bite may be summarised in order of application as: prevention of venom reaching the systemic circulation; neutralisation of any circulating venom; correction of venom induced abnormalities; and maintenance of vital functions of life (White 1981).

With the correct application of these treatment aims, in combination with the informed use of modern treatment methods, death from snake bite in Australia should be rare.

FIRST AID FOR SNAKE BITE IN AUSTRALIA

The principal purpose of first aid for snake bite is to delay the onset of serious snake bite problems, at least until the victim can reach appropriate medical care where such problems can be successfully managed. In the process such first aid must also cause minimum harm to the victim.

The question of minimum harm may initially sound peculiar in relationship to first aid, but if we consider some previous methods of first aid in vogue throughout Australia and many other parts of the world as well, much harm could and was done to the poor victim of a supposed snake bite in the name of first aid. The dangers of scarification or cutting or excising of the snake bite wound are obvious, and some of the awful tragedies caused by the amputation of the bitten area following supposed snake bite have been resurrected in graphic detail by Sutherland (1983). Such methods of first aid cannot be justified, and when one considers that even for cases of snake bite by truly venomous species, many bites will be ineffectual, and result in no harm to the victim, then scarification, excision, and most especially amputation, can be seen as first aid only for the desperately foolish.

Even the much touted tourniquet, or ligature, which completely stops blood flow to and from the bitten area, has been shown to be dangerous and to have even caused the loss of limbs on occasion (Russell 1983) and cannot now be recommended as a form of first aid.

In a classic paper in 1941, Barnes and Trueta demonstrated that Black Tiger Snake venom movement from the bite site was largely in lymph, via the lymphatics, which are small vessels draining from the periphery of the body through the lymph nodes, and finally to the blood circulation in the chest. These small easily obstructed channels, have lymph flow through them (towards the blood stream) greatly enhanced by the movement of surrounding muscles, which act as pumps forcing the lymph centrally. Thus movement of the bitten limb will only increase movement of the venom from the bite site towards the blood stream, where it can do most harm.

Drawing on this knowledge, and using recently developed techniques for venom detection in the blood stream, Sutherland and colleagues developed an experimental model with monkeys to study the flow of venom from the bite site to the blood stream and throughout the victim (Sutherland *et al.* 1979, 1981). They conclusively demonstrated that most venom is transported via the lymphatic system from the bite site and not via direct absorption by capillaries into the blood stream. Thus some simple first aid method reducing flow in the lymphatic vessels should retard the absorption of venom, and this formed the basis of their classic paper (Sutherland *et al.* 1979).

These studies initially using Tiger Snake (*Notechis scutatus*) venom, showed that firm crepe or elastic bandage applied over the bite site, then the rest of the limb, and combined with immobilisation of the limb, effectively prevented virtually all venom from reaching the systemic circulation. Release of this bandage was followed by the rapid onset of systemic envenomation. They estimated that the pressure of the bandage was around 55 mm of mercury, but that such a bandage did not appear to cause circulation problems for the limb as a whole, and could be left in place for several hours without fear of damaging the limb.

In view of this work, the following has now been adopted as accepted first aid for snake bite in Australia by the National Health and Medical Research Council (Edmondson 1979).

Action

1. Immediately apply a broad firm bandage to cover the bitten area. In the case of a limb as much as possible of the limb should be bound. The bandage should be bound as tightly as for a sprained ankle.

2. Immobilise affected limb with some form of splint; leave the bandage and splint on until medical care is reached.

3. Bring transport to victim if possible; do not permit him to move around more than is necessary.

4. If the snake can be killed safely bring it with the victim.

5. Do not wash the bite site.

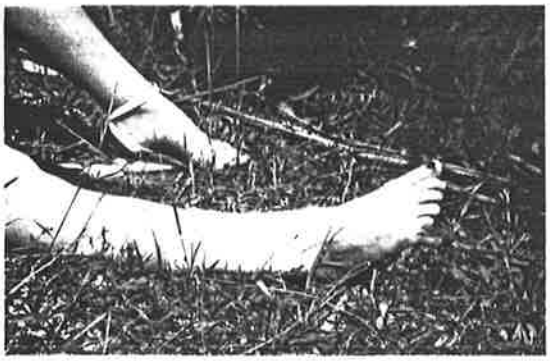
NOTE

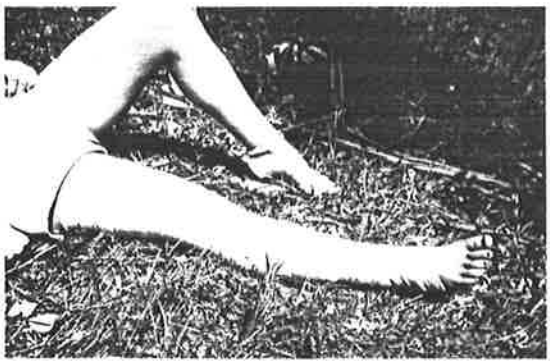
a. Do not apply an arterial tourniquet.

b. Do not cut or excise the bitten area.

c. The principles outlined are for snake bite only. Other types of envenomation such as spider bite frequently require alternative methods of treatment.

The fifth positive action above has been





added, because of the development of snake venom detection kits. As these kits give best results from venom left on the skin surface, it is important to emphasise that the snake bite wound NOT be washed or cleaned in any way.

The application of a pressure/immobilisation bandage is illustrated in Figs 1-4. If the victim is wearing jeans or slacks, and is bitten on the lower limb, it may be most practical to bind the bandage over clothing rather than risk increasing venom movement by removal of clothing.

At least two cases of snake bite have been reported where this pressure immobilisation method of first aid was used, in both cases successfully. Pearn *et al.* (1981) described a case of Brown Snake (*Pseudonaja textilis*) bite to a reptile keeper; who applied two Esmarch's bandages to the bitten limb, excepting the area of bite on the thumb, immediately following the accident. Two hours after the bite, with the bandages still in place, the patient was symptomatically well, and assays for venom and coagulation disturbance were negative. The bandage was then released, and within 5 minutes the patient had developed a bursting headache and progressively developed further evidence of significant systemic envenomation. Repeat studies at this time detected circulating Brown Snake venom, and evidence of a coagulopathy. Antivenom was administered, and the patient made a complete recovery.

Murrell (1981) described a Tiger Snake (*Notechis scutatus*) bite to a herpetologist, with subsequent application of two crepe bandages to the bitten finger and the rest of the arm up to the elbow, and splinting. While the bandage was in place, there was no evidence of systemic

Figs 1-4. The application of pressure/immobilization first aid in the treatment of snake bite.

1. The patient is laid down if possible, and a broad elastic bandage or similar (pantyhose, torn strips of clothing) is firmly applied over the bite site at the same pressure used for a sprained ankle.

2. This bandage is then extended to involve as much of the bitten limb as possible, being applied firmly, but not so tightly that blood circulation to the whole limb is impaired.

3. The limb is positively immobilized using further bandage and any available implement as a splint.

4. In the case of a bite to the hand or arm the same form of bandage is applied, and the arm immobilized with a splint and sling.

envenomation, but on removal of the bandage, systemic envenomation ensued. This was successfully treated with antivenom. In this case, however, there was a ring on the bitten finger, and pain in the swollen finger distal to this ring occasioned the removal of first the bandage and then the ring, with subsequent development of envenomation. Thus there is some doubt in this case whether it was the ring or the bandage which was most important in delaying central movement of venom.

Harvey *et al.* (1982) described a case of Black Tiger Snake (*Notechis ater humphreysi*) bite in a herpetologist, with the immediate application of appropriate first aid. He developed symptomatic evidence of systemic envenomation within one hour of the bite, and while the bandage was still in place. In this case the bandage consisted of strips of a dismembered cotton t-shirt initially, followed by a wide crepe bandage encasing the whole arm. Antivenom was administered approximately one hour after the bite, but the bandage was maintained in position for over two hours, and this patient developed a small area of necrosis in the region of the bite.

Balmain and McClelland (1982) described a case of snake bite in a 3 year old boy, where the only compression bandage available was pantyhose. This rather unorthodox bandage was applied some 20 minutes after the actual bite, and the subsequent pattern of symptoms of envenomation, which were mild but initially occurred while the bandage was in place, leave some doubt about its effectiveness. Subsequent testing showed this bite to be due to a Mulga Snake (*Pseudechis australis*). Sutherland (1982b) commented on this case, and suggested that potentially pantyhose may be quite an effective compression bandage for the first aid treatment of snakebite. These rather positive comments on the pressure immobilization method have not gone entirely unchallenged. Anker *et al.* (1982a,b) have suggested that these cases do not prove the effectiveness of this first aid method and that their own investigations of the method using a 'mock venom' (radio-labelled sodium iodide) showed that such a bandage would not effectively immobilize venom. This was in contrast to the experimental evidence presented by Sutherland *et al.* (1979, 1981). Anker *et al.* suggested that a firm pad over the site of the bite, with a bandage to hold it in place, exerting a pressure on the bite site of approximately 70 mm of mercury, was a more effective way of immobilising venom. This proposed change in first aid has been criticised as introducing further changes when uniformity would be more helpful (Fisher 1982) and some elements of their experimental technique have also been criticised (Pearn *et al.* 1982). This controversy remains partially unresolved, but it appears to be the general opinion of medical authorities throughout Australia, including this author, that the original method proposed by Sutherland, and adopted by the National Health and Medical Research Council of Australia, should remain the official recommended form of first aid for snake bite in Australia. It is safe, relatively simple to apply, and is fairly easily understood.

Sutherland (1983) has discussed the use of first aid in snake bite in detail. One initially unforeseen problem with pressure immobilisation first aid, is the continued use of this first aid on a snake bite victim long after they have reached a medical treatment facility. If the snake venom has some potential for local tissue destruction, as is the case with Tiger Snake (*Notechis scutatus*) venom, then such prolonged immobilisation of the venom might increase the chance of significant local tissue damage, and even necrosis. This appears to have occurred on at least two reported occasions (Frost 1981; Harvey *et al.* 1982). It is therefore now recommended that after the patient has reached a medical treatment facility and initial precautions have been taken, such as the insertion of an intravenous line, and preparations made for infusion of antivenom should this become necessary, then the bandage should be removed. Only if the patient is already showing signs of severe envenomation should the bandage be left in place at the medical treatment facility. The rationale in this situation is that the bandage may help to prevent further rapid movement of venom into the circulation, and so avoid making an already bad situation worse. It seems rather pointless to have a symptomless patient suffering from possible snake bite sitting in hospital with first aid still in place, thus potentially delaying the development of symptoms of snake bite, and so delaying a decision about the appropriate choice of treatment.

A common problem asked of lecturers on the management of snake bite is what to do if you are bitten by a snake in a remote area, and you are alone, have no way of calling for help, and have no bandages. This and slightly less drastic problems of snake bite in remote areas have been dealt with by Sutherland (1979a, 1983). The snake bite victim in remote areas should be aware that in many cases of snake bite insufficient venom is injected to cause death, and for those few who are very seriously bitten, death is rarely a rapid event, and many hours may be available for transport to medical care. The correct application of the pressure immobilisation bandage in this situation should substantially delay venom absorption, and further increase the victim's chances of reaching medical care in time for effective treatment to be given. For the victim without bandages, torn up strips of clothing, or as mentioned earlier, pantyhose, may be effectively substituted. Where at all possible the victim should refrain from any activity which moves the muscles of the bitten limb. Thus a person bitten while alone, where the bite is to a foot or other part of the lower limb, would be best advised to bandage and immobilise that limb with a splint as previously described, and then use a branch or similar object as a crutch so that help may be sought without unduly moving the bandaged limb.

THE MEDICAL TREATMENT OF SNAKE BITE

The correct medical treatment of snake bite where there is evidence of significant systemic envenomation is the prompt administration of an appropriate antivenom in appropriate quantity. All aspects of medical treatment of snake bite can be related back to this simple statement. From this three basic questions arise (White 1986).

1. When to use antivenom?
2. Which type of antivenom?
3. How much antivenom?

The following sections deal with the answers to these questions in detail, but they may be briefly summarised as:

1. Antivenom should be given if, and only if, there is evidence of significant systemic envenomation, and then it should be given at the earliest opportunity.

2. Wherever possible a monovalent antivenom should be used in preference to polyvalent antivenom, but the identity of the snake must be known either by positive identification of the snake, positive venom identification, or by the pattern of clinical signs and herpetological knowledge, as determined by a toxinologist experienced in this field.

3. Enough antivenom must be given to neutralise all circulating venom, and in a major bite this may be several times the normally recommended dose.

Immediate Assessment of the Patient on Presentation

Accurate initial assessment of a patient presenting with a history of definite or possible snake bite should ensure the successful outcome of treatment in virtually every case. All snake bites should be treated as a potential emergency, and all staff treating a snake bite should be aware of the urgency of the situation.

A history of the circumstances of the bite should be sought, either directly from the patient, or failing that from a witness. Note the time the bite occurred, the geographic location of the patient at the time of a bite, a description of the length and colour of the snake if this was observed, whether the bite was a single strike, or multiple bites, and whether the snake hung on and chewed for a period of time; what sort of first aid was applied and when, and whether the patient has had significant medical problems in the past, and particularly if they have been bitten by a snake before, or have received antivenom before, or have a history of atopy. The type and progression of symptoms and signs should be noted, together with their time of onset and duration. In particular ascertain if the patient has experienced headache, nausea or vomiting, abdominal pains, pain at the bite site or draining lymph nodes, difficulty with speech, or blurred vision. If loss of consciousness or convulsions have occurred this is highly significant, and indicative of systemic envenomation. A more detailed list of symptoms and signs may be found in the preceding paper.

While the history is being determined, observe the patient for signs of early paralysis such as drooping upper eyelids, lack of facial expression, difficulty with speech, and undue irritability. Check the lymph nodes draining the bitten area, for evidence of tenderness or enlargement. Gently check voluntary muscle

power, and seek evidence of muscle movement pain. A full general examination should be performed, although in a patient showing obvious evidence of systemic envenomation it is important not to delay treatment any longer than absolutely necessary. In the obviously seriously envenomated patient the essential parts of a history and examination can be conducted at the same time, while preparations for treatment are commenced.

The snake bite wound must be examined, but if the history or examination suggests that a significant bite has occurred, or if there is already evidence of systemic envenomation, then it is unwise to remove the bandage before an intravenous line has been inserted. It may be possible however to cut away a portion of the bandage over the actual bite site. This should be carefully examined noting the pattern of bite marks, and in particular if there is evidence that more than one bite has occurred. Also note if there is any sign of bleeding from the bite wounds, or associated oozing or swelling. The bandage applied immediately over the bite site should be saved, and swabs should be taken from the bite site at this time to allow detection of snake venom using the C.S.L. venom detection kit. The necessary swab stick and solution are provided in the venom detection kit, and the swab should be firmly rubbed over the wound several times to ensure that any venom on the skin is collected. Further details about the use of the venom detection kit will be given later.

On insertion of an intravenous line, or from a separate site, but in either case before any antivenom is given, blood must be taken for appropriate investigations. These are discussed separately.

After this phase of initial assessment is completed the pressure immobilisation bandage or other form of first aid should be removed, in all circumstances *except* when the patient already has clearly established major systemic envenomation, in which case the bandage may be left in place until antivenom has been given, and then removed.

If the patient on presentation is showing no symptoms or signs of envenomation, and there are good reasons to believe that envenomation is unlikely to develop, either because there is considerable doubt about a snake bite having occurred, or because several hours have elapsed since the time of the possible bite, without evidence of problems (particularly if no first aid was applied) then it may be acceptable to proceed with the history and examination as outlined, but delay the insertion of an intravenous line.

In all cases a urine sample should be sought from the patient, the volume measured, and the colour recorded.

The patient should be fasted, or at the most allowed only clear fluids orally. All probable or definite snake bite victims should be admitted for constant care over a minimum 18–24 hour period from the time of the bite, and in all cases where there is a suggestion of systemic envenomation, it is preferable to have the patient in an intensive care facility.

Investigations

The key investigations on blood samples are those designed to detect a coagulopathy. If no laboratory facilities are available, the only such test available to the medical practitioner is the whole blood clotting time, which unfortunately is not entirely reliable. It is generally preferable to use a glass test tube rather than a plastic specimen tube to measure whole blood clotting time, and if there is no evidence of clot formation after 10 minutes in a glass tube, and certainly after 20 minutes, this may be taken as reasonable evidence of a coagulopathy. However, whenever more sophisticated investigations are available, the author believes they should be used.

As a useful base line the prothrombin time (P.T.; also prothrombin ratio, P.R.) and activated partial thromboplastin time (A.P.T.T.) are useful guidelines. Most laboratories will also be able to detect fibrin degradation products (F.D.P.), and probably determine fibrinogen level as well. The blood film should also be examined, and a platelet count performed. If any of these show evidence of a coagulopathy, then more detailed coagulation studies may be performed if laboratory facilities are available. In the management of coagulopathy following snake bite the critical parameters are prothrombin time, APTT, fibrinogen level, FDP level and platelet count.

Although blood transfusion is very rarely required as treatment for snake bite, it is worth taking a sample for the blood group and hold as a precaution. If no venom detection kit is

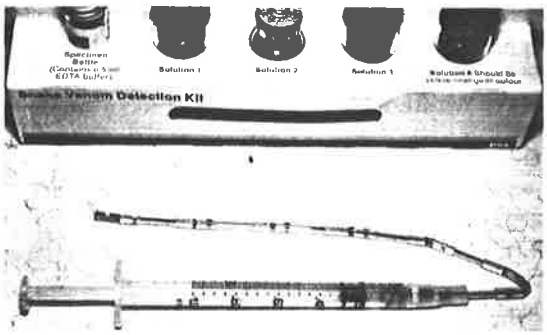


Fig. 5. The Commonwealth Serum Laboratories' Venom Detection Kit (VDK) disassembled. All required solutions are provided in the packet and clearly labelled. The syringe and attached capillary tubes are the hub of the kit.

available, it is worth freezing a small sample of plasma for possible use in venom detection studies at a laboratory so equipped.

Routine electrolyte studies should be performed, as should studies of basic renal function, liver enzymes, and creatine kinase (evidence of muscle destruction).

Urine should be tested for evidence of proteinuria and haemoglobinuria, and where possible for myoglobinurina as well. A sample may be frozen for future venom analysis should this prove necessary.

The Venom Detection Kit

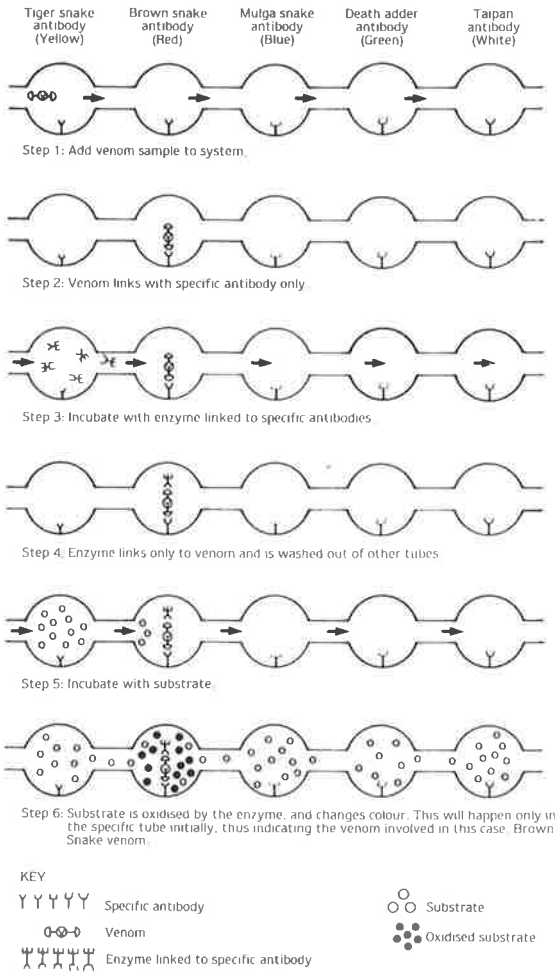
The first methods of venom detection in Australia used a radio-immuno-assay which was sensitive to nanogram quantities of venom, and has been successfully used to identify venom in clinical cases of snake bite, and at autopsy (Sutherland *et al.* 1975). Coulter *et al.* reported in 1978 that the technique could detect 0.4 nanogram per ml of crude venom, and that the presynaptic neurotoxin, Notexin, from the Tiger Snake (*Notechis scutatus*) could be detected at concentrations as low as 0.1 nanogram per ml. For detection of venom using this assay, it was recommended that samples be taken before antivenom administration, and that 2 mls of serum, and 2 mls of urine, both frozen, should be sent to CSL for testing, with prior notification to CSL that specimens were being despatched (Sutherland and Coulter 1977).

However, following the development by Theakston *et al.* (1977) of a micro ELISA (enzyme linked immunosorbant assay) for detecting and assaying snake venom and venom antibody, Coulter *et al.* (1980) developed an ELISA based venom detection kit for Australia, which would distinguish between each of the 5 main venom groups, representing the 5 different varieties of monovalent antivenom available. The first test kits were made available for assessment in 1979 (Sutherland 1979b), and the following year a simplified kit was made available for general distribution. This venom detection kit (VDK) may detect as little as 5-10 nanograms of venom in the sample, and takes approximately 30 minutes to perform (Figure 5). The development of this new improved kit which contain all equipment and reagents required for the complete test, has been described in detail elsewhere (Chandler and Hurrell 1982).

The principles of the kit are relatively simple. Each of the 5 colour coded tubes has a specific antibody linked to it (Table 1). The sample containing venom is washed through all the tubes and the venom links with the antibody in the tube specific for that venom (Fig. 6). Approximately 10 minutes is required for this incubation. The sample is washed out, and the tubes are then filled with an enzyme conjugate which is also linked to specific antibody. This is

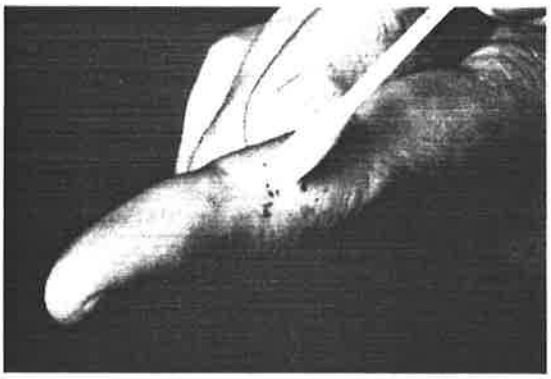
Table 1: Specificities of C.S.L. ELISA snake venom detection kits (V.D.K.).

Tube (specific antibody)	Species Giving Positive Reaction
1. Yellow	Tiger Snake *(Notechis sp.)* Red-bellied Black Snake *(Pseudechis porphyriacus)* Copperhead *(Austrelaps sp.)* Rough-Scaled Snake *(Tropidechis carinatus)*
2. Red	Brown Snake Western Brown Snake (or Gwardar) Dugite *(Pseudonaja spp.)*
3. Blue	Mulga Snake *(Pseudechis australis)* Papuan Black Snake *(Pseudechis papuanus)* Red-bellied Black Snake *(Pseudechis porphyriacus)* Copperhead *(Austrelaps sp.)*
4. Green	Death Adder *(Acanthophis sp.)*
5. White	Taipan *(Oxyuranus sp.)* Small-scaled Snake *(Oxyuranus sp.)* Rough-scaled Snake *(Tropidechis carinatus)*



▲ Fig. 6. Schematic of operation of C.S.L. ELISA snake venom identification kit.

▼ Fig. 7. Use of a swab soaked in the solution contained in the specimen bottle of the VDK, to detect venom at the bite site by rubbing firmly over all bite marks and associated skin.



incubated for a further 10 minutes during which time the enzyme antibody conjugate will bind to any bound venom. Following a further washing, the substrate solution is drawn into the tubes, and this in turn will react with any enzyme conjugate which has been bound. This will result in a colour change in that tube only initially, though if left long enough the colour may change in adjacent tubes, and finally vitually all the tubes in the system. For a result to be positive, the control tube should rapidly change colour on addition of the substrate, and the first tube to change colour definitely following this will indicate the venom involved (and therefore the appropriate monovalent antivenom to use).

The best samples for venom detection are obtained from swabs of the bite site, where venom left on the skin surface will be detected. If the wound has been washed the chance of detecting venom is significantly reduced, although sufficient venom may still be present to get a positive result. Bandages or clothing immediately adjacent to the bite site may also have venom soaked into them, and should therefore be kept in case they are needed as a last resort for testing. The swab stick and sample solution are contained within the kit, and the swab stick, moistened in the sample solution, should be rubbed firmly over and around the bite site to ensure that any venom is collected (Fig. 7).

Urine may also give a positive result on venom detection if the patient has established systemic envenomation, as a significant proportion of venom is excreted via the kidneys. If, however, there is no evidence of systemic envenomation then it is pointless to test the urine. On occasion venom may be successfully detected in blood samples using the VDK, but blood has proved far less reliable than samples from the bite site or urine (Hurrell and Chandler 1982).

Hurrell and Chandler (1982) have reviewed the first two years' use of the VDK, and their figures are summarised in Table 2. 130 cases were reported, of which 38 showed evidence of envenomation, and venom was successfully detected in 32 of these. In 5 of the 6 cases where venom testing was negative, but the patient was envemonated, the sample tested was blood. These represented 5 of the 8 kit failures reported. This report was criticised (Sutherland 1982a) for not presenting all

Table 2: Results of V.D.K. usage, 1980–1982 (after Hurrell and Chandler 1982).

	V.D.K. Result		
	Positive	Negative	Invalid
Definite Symptoms	32	6	5
Doubtful Symptoms	5	12	—
No Symptoms	7	58	1

available cases, and questions were raised about the incidence of faults in these kits. Furthermore it was pointed out that during the period of the survey no less than 385 cases of snake bite where antivenom was used were also reported to CSL. Given that only 38 of the 130 cases showed evidence of envenomation requiring antivenom, it would appear that as few as 10% or even less of cases of snake bite in Australia with envenomation had a VDK investigation performed to determine which antivenom would be appropriate. This problem has recently been reviewed (White 1986). Perusal of antivenom sales by CSL, based on information kindly provided by CSL, shows that to date there has been no significant change in antivenom usage in regard to the ratio of monovalent to polyvalent antivenom used (Table 3) despite the introduction of the VDK. Total sales of each antivenom type in Australia are given in Table 4. In the opinion of this author these figures document an unfortunate under-utilisation of the VDK, which is a very useful tool in the management of snake bite. It is to be hoped that more centres dealing with snake bite will make routine use of the VDK and reduce the use of polyvalent antivenom.

One final point about the VDK must be stressed. The detection of venom at the bite site indicates only two things: (a) the type of snake involved in the snake bite, and therefore the appropriate monovalent antivenom required to neutralise that venom, (b) positive evidence that a snake was involved in the injury. It does NOT determine whether envenomation has occurred, and the decision to give antivenom or not must be based on clinical and/or laboratory evidence of systemic envenomation, and NOT the presence of a positive VDK from a bite site sample.

Table 3: Relationship of ratio of monovalent AV to polyvalent AV sales to distribution of Venom Detection Kits (based on information kindly supplied by C.S.L.).

	Pre V.D.K. (79–80)	Post V.D.K. (83–84)
Total monovalent AV sales	3,803 (85%)	3,630 (85%)
Total polyvalent AV sales	685 (15%)	626 (15%)

Deciding Whether Systemic Envenomation has Occurred

In some cases it will be obvious from the patient's general condition and history that envenomation has occurred, and antivenom treatment is indicated. For instance the patient with evidence of paralysis clearly has systemic envenomation. Similarly the patient who is obviously irritable or unconscious, is most likely to have systemic envenomation. The patient with general signs and symptoms of envenomation such as headache and abdominal pain has at least some degree of systemic envenomation (but beware of an acute anxiety state).

Table 4: Antivenom sales in Australia (based on information kindly supplied by the Commonwealth Serum Laboratories).

	Number of ampoules sold					Cost per ampoule*
	1979–1980	1980–1981	1981–1982	1982–1983	1983–1984	
Tiger Snake AV	2,019	2,116	2,499	2,098	2,108	$ 60.00
Brown Snake AV	1,048	1,286	1,680	2,358	986	$ 35.00
Black Snake AV	281	420	459	141	275	$407.30
Death Adder AV	310	351	222	282	187	$350.90
Taipan AV	145	145	230	204	74	$529.40
Polyvalent AV	685	753	742	585	626	$556.10
Snake Venom Detection Kits	—	—	655	667	890	$ 85.00

*Based on subsidised cost to hospitals, as of 20/12/1985. Actual cost if purchased privately is significantly higher.

Some patients may have had a brief period of headache before reaching medical help, and on presentation feel subjectively well. If several hours have already elapsed since the bite occurred it is probable that they will not develop significant systemic envenomation, although they should still be admitted for observation. Tests of coagulation may give an early clue, and obviously a coagulopathy is indicative of systemic envenomation. Indeed, the presence of coagulopathy may be a very sensitive early test for envenomation, as it may precede major symptoms and signs of envenomation. Evidence of venom spread to the draining lymph nodes is certainly suggestive that systemic envenomation is imminent, if not already established. A more detailed assessment of all symptoms and signs is given in the preceding paper.

Sometimes patients may present within half an hour of being bitten, and especially if correct first aid has been applied, many of these patients will not have developed systemic envenomation. Thus tests of coagulation may be normal at this time, so after the first aid is removed they should be repeated an hour or so later, and if in doubt a further two hours after that. Obviously if the patient develops symptoms or signs of systemic envenomation before that then coagulation tests should be repeated immediately. Regular examination of the urine for haemoglobinuria and proteinuria will also be helpful during a prolonged assessment period waiting for evidence of systemic envenomation to manifest.

A recent report emphasises the need for special caution in managing a snake bite in a woman with advanced pregnancy (Sutherland *et al.* 1982). A patient with advanced pregnancy placed in a supine position is at risk of developing the supine hypotensive syndrome due to compression of the inferior vena cava by the pregnant uterus. This may lead to fatal circulatory collapse as occurred in the reported case. In this situation it is better to nurse the patient on her side. Indeed, purely to avoid inhalation of vomitus it is preferable to nurse any snake bite patient with systemic envenomation on the side rather than on the back.

Antivenom

Antivenom is reserved for those cases of snake bite with definite evidence of systemic envenomation. If there is evidence of significant systemic envenomation then antivenom should be given without delay. (Trinca 1963; Sutherland 1975; Sullivan 1979; Sutherland 1974; Campbell 1967; White 1981; Sutherland 1983). The aim of antivenom is to neutralise all circulating venom and all venom yet to reach the circulation.

Snake antivenoms in Australia are manufactured by the Commonwealth Serum Laboratories, Melbourne, and are produced by hyperimmunising horses. This of course means that they are horse serum, and in fact contain approximately 17% horse serum proteins (Sutherland 1977b). They are therefore capable of inducing significant allergic reactions, the extent of which will depend on the volume of antivenom given, the speed with which it is given, whether the patient has been previously exposed to horse serum, and whether the patient has a natural tendency to allergic reactions.

The first snake antivenom in Australia was specific for Tiger Snake venom and was released in 1930. Antivenoms are now available for all major venomous snakes in Australia, divided into 5 venom groups each with a specific monovalent antivenom. There is also a polyvalent antivenom which covers all groups. These antivenoms and the snake species they cover and approximate volume per vial are given in Table 5. Each vial contains enough antivenom to neutralise the average yield of venom milked from the snake species in question. Therefore the volume of Brown Snake antivenom is quite small, while the volume of Black Snake antivenom, designed to neutralise bites by the Mulga Snake, is quite large. This is reflected in the relative number of units of antivenom for each type of antivenom. One unit of antivenom will neutralise in vitro 0.01 milligram of dry snake venom (Sutherland 1983).

The quantity of antivenom per vial can only be taken as an approximate guide to the appropriate dosage of antivenom, as many factors determine the quantity of venom injected into the victim. These will include the size of the snake, how many times it has bitten, and, of course, the species of snake involved. The size of the patient is not relevant, as the snake is unlikely to distinguish between children and adults in the quantity of venom it injects, and it is the quantity of venom injected which

Table 5: Antivenoms available to Australian elapid venoms, produced by C.S.L.

Antivenom (AV)	Species effective for	Units per vial	Average volume per vial (mls)
Brown Snake AV	Brown Snake *Pseudonaja textilis* Western Brown Snake (or Gwardar) *Pseudonaja nuchalis* Dugite *Pseudonaja affinis*	1000u	4
Tiger Snake AV	Tiger Snake *Notechis scutatus* Black Tiger Snake *Notechis ater* Copperhead *Austrelaps sp.* Rough-scaled Snake *Tropidechis carinatus* Red-bellied Black Snake *Pseudechis porphyriacus* Collett's Snake *Pseudechis colletti* Blue-bellied Black Snake *Pseudechis guttatus*	3000u	6.8
Death Adder AV	Death Adder *Acanthophis antarcticus* Desert Death Adder *Acanthophis pyrrhus* Northern Death Adder *Acanthophis praelongus*	6000u	20
Taipan AV	Taipan *Oxyuranus scutellatus* Inland Taipan (Small-scaled or Fierce Snake) *Oxyuranus microlepidotus*	12000u	40
Black Snake AV	Red-bellied Black Snake *Pseudechis porphyriacus* Mulga Snake *Pseudechis australis* Collett's Snake *Pseudechis colletti* Blue-bellied Black Snake *Pseudechis guttatus*	18000u	35
Polyvalent AV	Equivalent to: Brown Snake AV Tiger Snake AV Death Adder AV Taipan AV Black Snake AV	 1000u 3000u 6000u 12000u 18000u	48

determines the quantity of antivenom required. For a single uncomplicated bite one vial of appropriate antivenom is often sufficient. However, for multiple bites, or more severe bites, several times the minimum dose may be required, and there are reports of up to 10 times the normal dose being required (Campbell 1967). Recommended minimum doses of antivenom are given in Table 6.

Antivenom must only be given where there is clear evidence of systemic envenomation, in which situation it may be life saving. It is costly and potentially dangerous, and it should never be used if the patient does not have significant systemic envenomation. Unfortunately in the past there has been a tendency for medical officers unused to the treatment of snake bite to exercise poor judgement on the presentation of a case, and to administer antivenom despite the absence of envenomation (White 1983a).

Obviously it is of critical importance to decide what type of antivenom to give, and as this is a major and sometimes difficult decision it will be dealt with separately below.

Before antivenom is given a good intravenous line must be established. Resuscitation facilities and staff able to use them should be immediately on hand, and adrenalin 1 in 10,000 should be drawn up and ready for use. Sutherland (1975, 1983) recommends that a

Table 6: Recommended minimum doses of antivenom (intravenous). Note that many times this dose may be needed. Child's dose the same as for adults (after White 1981, Sutherland 1983).

Snake	Appropriate Antivenom	Minimum Dose
Brown Snake Western Brown Snake (Gwardar) Dugite	Brown Snake AV	1000u
Tiger Snake Copperhead Rough-scaled Snake	Tiger Snake AV	3000u
Black Tiger Snake	Tiger Snake AV	6000u
Chappell Island Black Tiger Snake	Tiger Snake AV	12000u
Red-bellied Black Snake Blue-bellied Black Snake Collett's Snake	Tiger Snake AV OR Black Snake AV	3000u 6000u
Mulga Snake	Black Snake AV	18000u
Death Adder Desert Death Adder Northern Death Adder	Death Adder AV	6000u
Taipan Inland Taipan (Small-scaled or Fierce Snake)	Taipan AV	12000u

non-sedating antihistamine (given IV) and subcutaneous adrenalin be given before the administration of antivenom. It is presumed that this will reduce the chance of unwanted side effects as a result of antivenom administration. In the author's experience, however, the sedating effect of commonly available antihistamines can be a nuisance in assessing the development of further envenomation after antivenom has been administered, particularly in children. The use of subcutaneous adrenalin prophylactically before antivenom administration is not a universally accepted practice, and is not used routinely by the author or his colleagues in anaesthetics and intensive care. However, if there is a history of previous administration of antivenom, or a history of allergy or atopy, then prophylactic subcutaneous adrenalin may be a reasonable precaution. At various times intravenous steroids have also been suggested, and Sutherland (1983) recommends the administration of intravenous hydrocortisone if the patient has a history of allergy or previous exposure to antivenom.

When antivenom is given it should always be intravenously, and should preferably be diluted approximately 1 in 10 in the intravenous line carrier solution (e.g. Hartmann's solution, or normal saline). This should be infused over 20–30 minutes or more, although if life threatening systemic envenomation is present it is reasonable to infuse the antivenom much more rapidly.

In other countries, particularly in North America, it is common practice to skin test with antivenom subcutaneously before giving the therapeutic dose (Russell 1983). Some doubts have been cast on this practice however (Warrell *et al.* 1984), and it is now definitely not recommended for the treatment of Australian snake bite (Sutherland 1983).

It should not be assumed that the first dose of antivenom will be sufficient. It is not uncommon to see an initial improvement following the first dose of antivenom followed by further deterioration half an hour to two hours later, and one should always be prepared to give further doses until there is no further evidence of systemic envenomation. As some of the venom may take a considerable time to move from the bite site to the general circulation it is possible for a continual trickle of venom to occur over several hours, reinforcing the need for careful re-assessment of the patient at regular intervals following antivenom administration, even if the initial response to the antivenom appears to have been good.

Antivenom therapy occasionally fails to

improve the patient's condition. Where there is established paralysis due to presynaptic neurotoxins antivenom may not reach these toxins, and may not reverse the paralysis. However, the usual reasons for failed treatment are:

INCORRECT ANTIVENOM

The wrong monovalent antivenom has been used. This indicates an incorrect assessment of the snake involved, and often it will then be appropriate to use polyvalent antivenom.

INADEQUATE ANTIVENOM

If insufficient antivenom is given, it cannot be expected to remedy the situation, and in this situation further antivenom should be given promptly.

OUT OF DATE ANTIVENOM

Antivenom is normally kept refrigerated, not frozen, and has a shelf life of at least 3 years. Usually the hospital pharmacist is charged with the duty of ensuring that antivenom stocks are appropriate, adequate in quantity, and not out of date.

ANTIVENOM GIVEN BY THE WRONG ROUTE

Unlike some other antivenoms, particularly that for the Red-back Spider, which can be given intramuscularly, snake antivenoms should always be used intravenously as their rate of absorption from muscle is too slow.

ANTIVENOM GIVEN TOO LATE

It is uncertain if it is ever too late to give antivenom, but certainly the greater the delay between envenomation and treatment, the greater the chance of an unfavourable outcome (Sutherland 1977b). Favourable effects of antivenom have been observed up to 60 hours after envenomation (Patten *et al.* 1985).

THE CHOICE OF ANTIVENOM TYPE

Polyvalent antivenom, which covers all species of snake in Australia, is expensive, and more importantly contains a large volume of horse serum. It is therefore much more likely to cause unwanted side effects including potentially life threatening complications such as anaphylaxis (Sutherland and Lovering 1979). The unwanted side effects of antivenom will be more fully discussed in the following section, but it is sufficient to note here that the small volume Brown Snake and Tiger Snake monovalent antivenoms have a much lower incidence of untoward side effects than polyvalent antivenom. Obviously, then, wherever possible monovalent antivenoms should be used in preference to polyvalent.

To use monovalent antivenom, however, it is necessary to determine the type of snake involved in the snake bite incident. The first obvious way of doing this is to rely on the description of the snake by the victim or a witness. However, in only 75% or less of cases of snake bite is the snake actually seen at all, and in many of these it is seen only fleetingly (Munro and Pearn 1978). Worse still, many of Australia's dangerous snakes may be very similar in appearance, and sometimes even in colouration. Thus a brown-coloured snake may be a true Brown Snake, or one of the Taipans, or a Mulga Snake, or even a Copperhead, or brown-unbanded variety of Tiger Snake. Within any one species of snake there may be a wide range of colour and pattern seen, and this is particularly true of the Brown Snakes.

These difficulties are reflected in a survey of the ability of Australians to identify venomous snakes (Morrison *et al.* 1983). This survey conducted at the University of Queensland on an Open Day, sampled Australians from all walks of life and tested their ability to identify well preserved specimens of dead snakes representing the major species found in Queensland. This information is summarised in Tables 7 and 8. It can be seen that the very best group at identifying snakes correctly were medical practitioners, and they were wrong 75% of the time. The average of correct identifications for all groups was only 19%. Some snakes were more easily identified than others, and predictably the Death Adder, which has a very distinctive shape, was correctly identified more often than any other species. Clearly then it would be unwise to rely on the identification of the snake by the victim. It is relatively unusual for the snake to be killed and

Table 7: Ability of Australians to correctly identify well presented, preserved, dead snakes (after Morrison *et al.* 1983).

Subgroup	Mean correct answers for 10 different snakes
Medical Practitioners	25%
Medical Students	25%
Primary Schoolchildren	24%
High School Graduates	20%
Secondary Schoolchildren	19%
Tertiary Graduates in biological sciences	16%
AVERAGE	19%

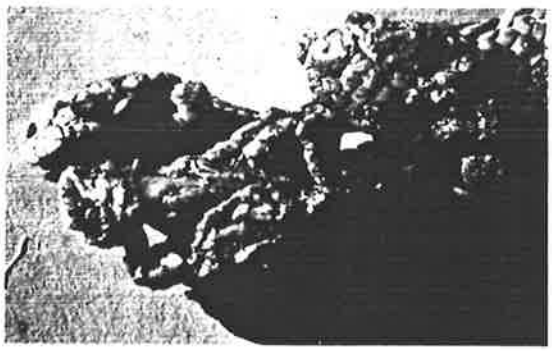
Table 8: Ability of Australians to correctly identify particular species of snake, from well preserved dead specimens (after Morrison *et al.* 1983).

Snake	Correct Identification	
	Male (59%) Observers	Female (41%) Observers
Death Adder *(Acanthophis antarcticus)*	67%	47%
Taipan *(Oxyuranus scutellatus)*	33%	20%
Brown Snake *(Pseudonaja textilis)*	24%	20%
Tiger Snake *(Notechis scutatus)*	22%	13%
Rough-scaled Snake *(Tropidechis carinatus)*	12%	0
Mean Correct Identification	22%	15%

brought in with the victim, but even in good condition there is a significant chance it may be mis-identified by a medical practitioner, and this chance is greatly increased given the usual mangled appearance of dead snakes presented for identification (Fig. 8). Museum or university reptile taxonomists can almost always provide accurate identifications from specimens or good descriptions and, if such experts can be consulted, administration of appropriate monovalent antivenom can proceed. If not, however, advice from other 'expert' herpetologists should be treated with caution. Identification of the snake by description or from the specimen by people untrained in this specialized area is not a practical way of determining which monovalent antivenom to use.

A second alternative is to identify the snake from its venom. This of course is the principle behind the CSL venom detection kits, whose use has already been described. These kits will correctly identify which of the five monovalent antivenoms is appropriate, and should only take 30–40 minutes to do so. In an emergency they may be run more quickly, though this will reduce their sensitivity. However, this does not cope with a situation where all the venom has been zealously washed away from the bite site by an ill-informed first aider, and furthermore the venom detection kit must be available to those who are treating the snake bite. Unfortunately the distribution of venom detection kits has not matched the distribution of medical practitioners giving antivenom (Sutherland 1982a; White 1986). When it is available, however, the venom detection kit is an excellent way of determining appropriate antivenom.

Fig. 8. The extremely mangled head region of a snake presented together with its snake bite victim. Painstaking examination of this specimen revealed that it was a Common Brown Snake (*Pseudonaja textilis*).



There is a third alternative which has recently been discussed (White *et al.* 1985; White 1986). By combining taxonomic and toxinological knowledge about snakes and snake bite an experienced toxinologist may be able to make a 'best guess' decision on the snake most likely to be involved in a snake bite incident. This may result in the selection of a single appropriate monovalent antivenom, or more usually the use of two complementary monovalent antivenoms. The most usual combination is that of Brown Snake antivenom and Tiger Snake antivenom, although the exact mix will depend on the situation.

The herpetological knowledge may allow the toxinologist to identify the geographic area in which the patient was bitten as containing only a limited range of dangerous venomous snakes. If these species are covered by only one or two monovalent antivenoms, then obviously there

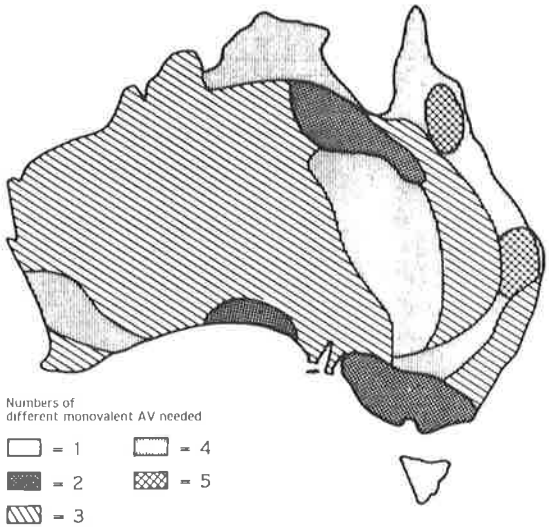


Fig. 9. Antivenom map of Australia.

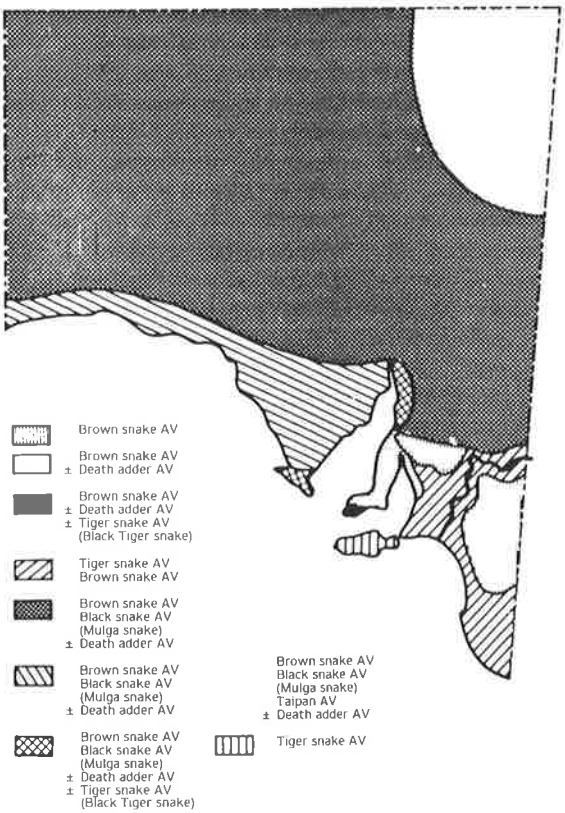


Fig. 10. A more detailed antivenom map than Fig. 9, but referring only to South Australia.

will be no need to use polyvalent antivenom. If three or more antivenoms will be needed to cover all possibilities the polyvalent antivenom will often be more appropriate. Fig. 9 divides Australia into antivenom zones, determined by the distribution of venomous snakes. Each zone corresponds to the number of venom types found within that region. Thus Tasmania is a zone 1, that is only one venom type (Tiger Snake venom type) is found, and therefore only one monovalent antivenom is required. At the other end of the extreme some regions have 4 or even 5 different venom varieties within them, and here polyvalent antivenom is necessary if the snake cannot be identified using the VDK. Looking at the distribution State by State it is possible to draw up a far more detailed map (Fig. 10) as shown for South Australia. This rather complicated map may be simplified as shown in Fig. 11. This system is currently being evaluated by the author in South Australia.

As is apparent from the information presented in the preceding paper there are some real clinical differences between some groups of venomous snakes in terms of the clinical picture of envenomation they may cause. This particularly applies to the appearance of the local bite site, and to a lesser extent the pattern of systemic envenomation. As this pattern is further refined, considerably assisted by the confirmation of the snake involved using a VDK, so the ability to distinguish between bites of different species is improved. As an example, bites by the Brown Snakes (*Pseudonaja* spp.) usually cause little or no local pain, swelling, or other local reaction. In contrast bites by Tiger Snakes (*Notechis* spp.) usually cause local pain, some swelling, and bruising or necrosis. Bites by Mulga Snakes (*Pseudechis australis*) usually cause marked local swelling of the bitten limb. Similarly there are differences in the pattern of systemic effects of the venom. Combining this knowledge with the herpetological knowledge often makes it possible to predict the type of snake involved in a snake bite incident. It must be stressed that this is a relatively new way of determining the most appropriate antivenom, and development is far from complete. Details of appropriate decision criteria used in this method for every area of Australia are obviously well beyond the scope of this discussion. It remains to be shown whether this method once fully

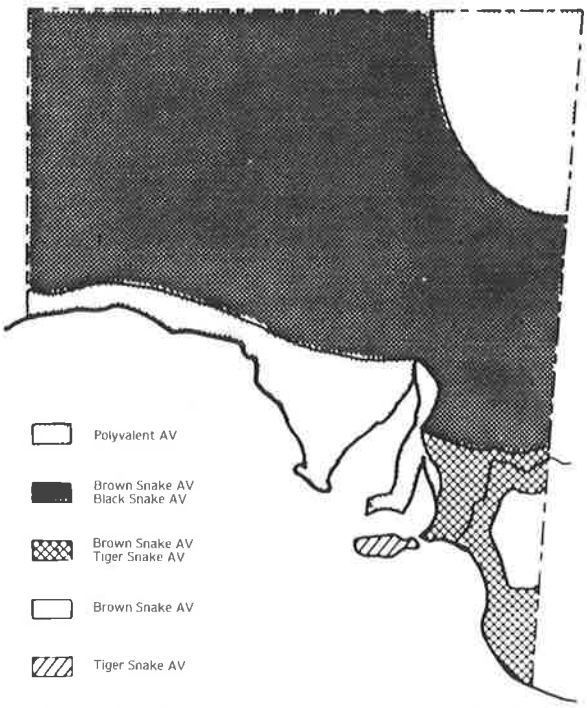


Fig. 11. A simplification of Fig. 10 more clearly showing which antivenoms are appropriate.

developed will be adequately reliable for all areas of Australia. The author firmly believes that the development of this technique is desirable, but that its development and application throughout Australia will require the concentration of experience in treating snake bites in the hands of just a few committed toxinologists with both herpetological and medical skills. Each such toxinologist should cover a large hinterland, and should be involved to a variable extent in the management of every case of snake bite occurring within that hinterland.

Untoward Reactions to the Administration of Antivenom

Antivenom is undoubtedly life saving in the treatment of severe snake bite, but is nevertheless potentially dangerous, and should never be used without appropriate indication. Occasionally severe reactions to antivenom do occur, and they are more likely to be seen in individuals who have had previous doses of antivenom. This will often apply to amateur reptile keepers, amongst whom there is a widely held belief that antivenoms are more dangerous than the venom. This belief is groundless. As reported in the preceding paper, a survey of recent snake bite fatalities revealed two cases where an anaphylactic reaction following the administration of antivenom was thought to have been the cause of death. However, over this period several thousand people would have received antivenom, and it cannot be stated with certainty that those two deaths apparently due to antivenom would not have occurred anyway as a result of envenomation if antivenom had been withheld.

Sutherland (1977a) examined the anticomplementary activity of Australian snake antivenoms, and concluded that they possessed significant anticomplementary activity, and that this could potentially cause significant problems in a patient infused with antivenom even for the first time. It could explain why an anaphylactic reaction occasionally occurs in this situation. Obviously such reactions would not be predicted by previous skin sensitivity testing for subcutaneous test doses. However, the role of anticomplementary activity in reactions to antivenom is still unclear (Warrell *et al.* 1984).

There have been several series reporting complications of antivenom therapy. Campbell (1967, 1969) has reported complications following the use of antivenom in Papua New Guinea. In a series of 61 patients, 28 (46%) had some form of adverse reaction. However, in only 2 (3%) did true anaphylactic shock develop. These figures are summarised in Table 9. More recently Sutherland and Lovering (1979) analysed 181 cases of snake bite reported to the Commonwealth Serum Laboratories over a 12 month period. They noted that polyvalent antivenom had the highest incidence of complications. A summary of their findings is provided in Table 10. Unfortunately the number of cases using some of the monovalent antivenoms was small, making the statistical significance of the findings uncertain. Nevertheless a considerable number of cases involved the use of polyvalent antivenom, and considering that the average quantity of antivenom infused in this latter group was nearly 70 mls, a 22% incidence of untoward reactions is hardly suprising. In comparison the incidence of untoward reactions using a combination of Tiger Snake and Brown Snake antivenoms was only 5.5%. The overall incidence of complications for all antivenoms was 13%,

Table 9: Complications of antivenom therapy in Papua New Guinea (after Campbell 1969).

Complication	Number with Complication (n = 61)	Percentage
Reaction involving skin — urticaria, itching or oedema	15	25%
Febrile reaction alone — rigor, shivering, fever	8	13%
General reaction	3	5%
Anaphylaxis	2	3%
TOTAL	28	46%

Table 10: Incidence of untoward reactions following the use of snake antivenoms, over a one year period (after Sutherland and Lovering 1979).

Antivenom Used	Number of cases	Number of patients who suffered reactions		% of cases with reaction	Average quantity of AV used (mls)
		Severe	Mild		
Tiger Snake AV	16	1	1	12.5%	10.2
Brown Snake AV	20	—	1	5%	4.2
Combination of above two AV	36	2	—	5.5%	13.8
Death Adder AV	3	—	—	—	30
Black Snake AV	12	—	—	—	17.3
Taipan AV	—	—	—	—	—
Polyvalent AV	86	9	10	22.1%	69.6
Polyvalent plus other AV	12	—	—	—	71.7

which is considerably less than in Campbell's series. Immediate reactions to antivenoms included sudden hypotension, rash and bronchospasm, urticaria, collapse, hyperthermia, sweating, headache, colicky abdominal pain, and vomiting. Delayed reactions included urticaria, arthralgia, polylymphadenopathy. Immediate reactions occurred only when the combination of premedication and slow infusion of diluted antivenom was not used.

One of the most important delayed problems following antivenom administration is the development of serum sickness. Serum sickness is more likely to occur if a large volume of antivenom has been infused, such as occurs if polyvalent antivenom or multiple doses of antivenom are given. Especially in this situation it is probably worthwhile informing the patient of the problems associated with serum sickness, as they occur several days after antivenom has been given, and the patient may not associate the symptoms of serum sickness with the previous treatment for snake bite (Sutherland 1983).

Serum sickness is an immune complex or type 3 hypersensitivity disease. It classically develops 8 days after exposure to the foreign antigen, in this case antivenom, though it may occur at any time from 3 to 4 days up to 14 days after antivenom is given. The severity is quite variable, and in most cases the disease is relatively mild and self limiting. Common symptoms include fever, generalised lymphadenopathy, urticaria, and joint pains. In more severe cases there may be deposition of immune complexes in the kidneys leading to kidney damage which is usually mild. Measured serum complement may be low, and there may be a transient albuminuria. Occasionally there is oedema of the face, and headache, nausea, vomiting, abdominal pain, diarrhoea, cardiac arrhythmias, and pericarditis may all occur. Rarely there may be neurological disorders such as a unilateral mononeuritis with weakness and sensory deficit in the area affected. It is usually self limited, and resolves within one to three weeks. In the more serious case a considerably longer recovery period may be expected.

Treatments available include antihistamines, anti-inflammatory agents such as aspirin, and in more severe cases the use of corticosteroids. The foregoing is not a comprehensive treatise on serum sickness and its management, more details of which may be sought in general medical textbooks.

Non-specific Management of Snake Bite

Local pain at the bite site is only encountered in some cases, following bites by certain species such as the Tiger Snake, but generalised pain and especially abdominal pain may be a problem in many cases. Frequently the administration of appropriate antivenom will remedy this problem, although the antivenom itself may occasionally cause abdominal pain. Pain relief in this situation is difficult, and morphine is contra-indicated (Sutherland 1974). Diazepam and paraldehyde have been suggested (Sutherland 1975). Campbell suggests that no sedation should be given to snake bite victims, and certainly not long acting drugs like paraldehyde (Campbell, pers. comm., 1982).

There are no firm guidelines for the treatment of pulmonary oedema in snake bite, although the prompt neutralisation of circulating venom with appropriate antivenom is obviously important. Diuretics may also be helpful. Because of the potential for vomiting and aspiration pneumonia, oral fluids should be withheld if possible, and fluid requirements met via intravenous therapy. Patients should also be nursed on their side and if unconscious or semiconscious or paralysed, secretions should be sucked out as necessary.

Infection following snake bite is not a significant problem, but as the wounds are penetrating the usual precautions to prevent tetanus infection should be followed. Antibiotics are rarely needed.

The need for meticulous observation of cases of snake bite should be impressed on all attending staff, and in particular nursing staff should be made aware of special observations which apply to a case of snake bite. In particular the early signs of neurotoxic paralysis should be discussed, and the patient should be assessed frequently, including waking the patient. All observations should be completely documented. Resuscitation facilities should be readily available, as well as staff able to use them.

Fluid input and output should be carefully monitored, and the patient's weight should be recorded on admission. This will become particularly important should renal failure develop. The need for repeated tests, particularly of coagulation, and also of serum electrolytes and, where appropriate, renal function has already been noted.

Management of Neuromuscular Paralysis

Neuromuscular paralysis due to potent neurotoxins in some Australian snake venoms may be a prominent feature of snake bite, and the progression of symptoms and signs has already been discussed in the previous paper. The only specific treatment for neurotoxin induced paralysis is neutralisation of the venom with antivenom, and the sooner this is given the better. As discussed earlier, those venoms containing presynaptic neurotoxins may cause a paralysis which once established cannot readily be reversed by the administration of antivenom. In the majority of cases, where antivenom has been administered long before the development of complete paralysis, the patient will show evidence of paralysis arrested at the stage reached at the time of antivenom administration. As there is a considerable latent period between binding of presynaptic neurotoxins and their final paralytic action, it is possible that despite adequate appropriate antivenom therapy, there may be a small progression in the degree of paralysis following antivenom treatment, but this should be self limiting. If the paralysis continues to progress it suggests that either insufficient antivenom has been given, or possibly incorrect antivenom has been used. If there is severe neurotoxic paralysis consider using several times the recommended minimum dose of antivenom.

Bites by the Taipan, Inland Taipan, Tiger Snakes, Brown Snakes, and the Rough Scaled Snake may all fail to show reversal of paralysis despite antivenom treatment. The situation for Copperhead bites is unclear. Death Adder bites with paralysis will probably show reversal of the paralysis after sufficient antivenom has been given. Bites by members of the Black Snake group including the Mulga Snake are unlikely to cause true paralysis.

If severe paralysis is established then ventilation will probably have to be maintained by artificial means. In the past this has often necessitated a tracheostomy, though modern methods of nasotracheal intubation have largely replaced tracheostomy in this situation. It should be remembered that if there is a significant coagulopathy then continued oozing around a tracheostomy wound may prove a real problem (Campbell 1964a, b), and this is a further argument against the use of tracheostomy. The

expected sequence of recovery from paralysis was discussed in the preceding paper.

Management of Coagulopathy

A detailed discussion of coagulopathy following snake bite was given in the previous paper. The usual problem is the defibrination syndrome, and the primary treatment of this in snake bite is neutralisation of all circulating venom with appropriate antivenom. In some cases this may require multiple doses of antivenom, either to cope with the large volume of venom injected, or to mop up residual venom trickling into the circulation from the lymphatic system. In the author's experience after all circulating venom is neutralised, normal homeostatic mechanisms usually restore adequate clotting function within one to three hours. However, complete return to normal of all factors may take several days. In particular the prothrombin time may remain prolonged for several days without clinical or laboratory evidence of an ongoing coagulopathy. Heparin does not appear to have any place in the management of snake bite induced defibrination. (Warrell *et al.* 1976; Sutherland 1983). In cases of severe coagulopathy following snake bite fresh frozen plasma and cryoprecipitate infusions have been suggested (Sutherland 1983); however, in the author's experience adequate antivenom has been the only required treatment, a view supported by Campbell (Campbell, pers. comm. 1982). Reid *et al.* (1963) found fibrinogen unhelpful in the treatment of defibrination due to Malayan Pit Viper snake bite. Herrmann *et al.* (1972) have successfully treated defibrination syndrome following bites by the Brown Snake group using antivenom alone. The author has reported similar experience with bites by Brown Snakes and Tiger Snakes (White 1981, 1983b; White *et al.* 1984).

Rarely a true disseminated intravascular coagulation (DIC) may be present, and in this event there will be an associated thrombocytopenia which is not seen in the defibrination syndrome. In this situation, which is usually seen in association with renal failure, it may be necessary to consider the use of platelets and fresh blood. The role of heparin in this situation is uncertain. In one case of renal failure following snake bite, associated with a DIC, heparin was used without clear benefit, but subsequent administration of large doses of appropriate monovalent antivenom was followed with resolution of the coagulopathy (White and Fassett 1983). This temporal association between antivenom therapy and resolution of the coagulopathy may of course be coincidental.

The Management of Myolysis

As with other problems associated with snake bite the only really appropriate treatment is the administration of appropriate antivenom. It is unclear what other treatment can be given for severe muscle destruction, but obviously it will be important to maintain a high fluid load and close surveillance of kidney function. In the recovery phase dietary adjustment such as a high protein diet may prove helpful.

The Management of Renal Failure

It is clearly beyond the scope of this discussion to list the treatment of renal failure, and details of this should be sought from specialised texts on this subject. It should be noted, however, that renal failure may occur following snake bite, either in association with delayed treatment and a coagulopathy such as seen in bites by Brown Snake (*Pseudonaja* spp.) group, or in association with major muscle destruction as may be seen after bites by Mulga Snakes (*Pseudechis australis*), Tiger Snakes (*Notechis* spp.), and Taipans (*Oxyuranus* spp.). The majority of cases of snake bite induced renal failure, though they may require dialysis for extended periods, show eventual resolution and return to normal or near normal kidney function. However, occasionally the patient may succumb as a result of renal failure. The advice of physicians specialised in the management of renal failure should always be sought when renal failure following snake bite occurs.

The Management of the Local Tissue Injury

Only rarely is there sufficient local tissue injury to warrant any form of medical treatment, and in most cases this will be limited to ensuring the wound is clean and free of infection, thus allowing normal healing processes to occur.

When there is gross oedema in association with the bite, the bitten limb should be elevated to try to minimise impairment of blood circulation. It is extremely unlikely that such oedema would ever become severe enough to threaten the vascular flow to the limb. Should this situation ever arise the experience of

toxinologists in treating Viper bite overseas should be borne in mind. Escharotomy should not be contemplated unless there is clear evidence by compartmental pressure measurements of a true compartment syndrome.

In those exceptional cases where a large area of skin damage occurs it may be necessary to debride the wound and cover with a skin graft, but this is best left until it is clear that the major life threatening problems of snake bite are completely controlled and the extent of damage is well defined, usually some days later. To the best of the author's knowledge there is only one report of an injury sufficient to require amputation (White this volume, Aspects of Envenomation).

Immunisation Against Snake Venom

Mass immunisation against snake venom has been used overseas with some success (Sawai 1979). This is appropriate in areas where there are large concentrations of both people and venomous snakes, with a consequently high incidence of snake bite. This is not the case in Australia. However, there is one report of immunisation against an Australian snake venom to help protect a reptile keeper subject to occasional snake bites, after he had developed an allergy to horse serum (Wiener 1961). The man, aged 47, was actively immunised with Tiger Snake venom. This involved 20 injections over a period of 13 months commencing with an initial dose of 0.002 mg of venom, with a final dose of 25 mg of venom. The maximum level of circulating antivenom recorded was 5.2 units per ml and at 4 months the level was 2 units per ml. This patient was subsequently bitten by a Tiger Snake and although he developed local oedema, no systemic problems ensued. As there was a subsequent rise in antibody titre, Wiener thought that there had been effective envenomation, and that the immunisation was successful.

Allergy to Snake Venom

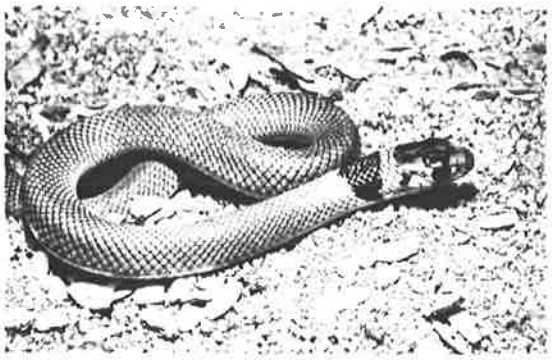
Reptile keepers who suffer multiple episodes of snake bite run the risk of developing an allergy to the snake venom, akin to the situation of allergy to bee venom. Should this occur they may develop an acute and life threatening anaphylactic reaction to even a tiny quantity of venom if rebitten. This danger is unfortunately poorly appreciated by amateur reptile keepers, who all too often consider being bitten by a venomous snake a badge of honour. The author has seen one case of Red-bellied Black Snake bite in a 15 year old with development of an acute allergic reaction involving mild bronchospasm, hypotension, and generalised urticaria. Fortunately this reaction was self limiting, and by the time of presentation at the hospital, emergency treatment was not required.

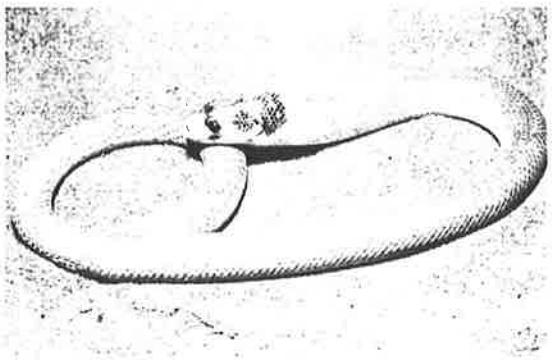
Sutherland (1983) has described two other cases of allergy to snake venom. Both were in herpetologists, and both involved Tiger Snakes. In one case the reaction was severe but non-fatal, and in the other, a 47 year old part time showman with a history of asthma, the bite was followed with collapse within a very short period, and death despite attempts at resuscitation. This case was one of those included in the survey of recent snake bite deaths in the preceding paper.

Because of the complexity and toxicity of snake venoms Sutherland (1983) has suggested that there is no place for desensitisation. He recommends that in addition to the standard treatment for anaphylaxis such as intravenous adrenalin, and cardiopulmonary resuscitation, antivenom should be infused as rapidly as possible as it may play the role of a blocking antibody as well as reducing any toxic effects of the venom. Undoubtedly the most effective treatment after it has become clear that a herpetologist is allergic to a snake venom, is the absolute avoidance of these reptiles.

Autopsy After Fatal Snake Bite

Snake bite is relatively rare, and death from snake bite in Australia is definitely rare. There is still much to learn about the action of snake venoms on man, and while every snake bite fatality is a tragedy, the opportunity to learn more about the effect of snake venoms should not be lost. Thus any fatality definitely or possibly due to a snake bite should result in a detailed autopsy, and where the findings are at all unusual every attempt should be made to publish them in the medical literature. In particular, at autopsy the pathologist should look at the appearance of the bite site, and examine it for evidence of local tissue damage, and take small samples from this region and draining lymph nodes for venom measurement. Blood and urine samples should also be taken and frozen for later venom measurement if this proves





▲▲ Fig. 12. A juvenile Common Brown Snake (*Pseudonaja textilis*) showing the typical black mark on the back of the head, followed by a pale band on the adjacent neck, followed by a further black band. Juvenile specimens of this species may also exhibit numerous bands or speckles along most or all of the body.

▲ Fig. 13. The Black-headed Scaly Foot Legless Lizard (*Pygopus nigriceps*). This lizard's colouration and markings are superficially similar to those of the juvenile Brown Snake, for which it is sometimes mistaken.

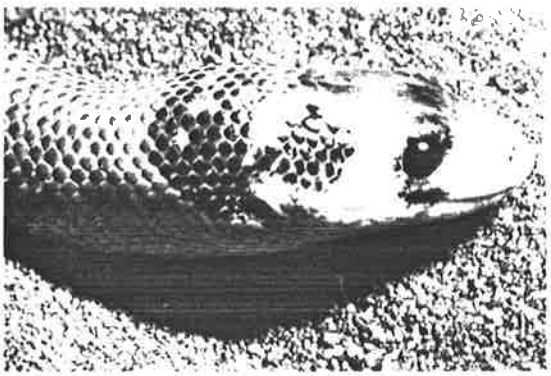
► Fig. 14. *Pygopus nigriceps*. Note the external ear opening just posterior to the final angle of the jaw, typical of a legless lizard, but never seen in a true snake.

practical. Biopsies of selected muscle groups should be performed and examined for evidence of muscle damage. Evidence of coagulopathy induced haemorrhage should be sought in all major organs, and particularly the brain. The kidneys should be examined in detail searching for evidence of macroscopic or microscopic damage. If the urine is dark in colour it should be checked for myoglobin. Haemolysis may be evident from haemolytic staining of the aorta and cardiac chambers, although this may also be seen as a consequence of early putrefaction during the hot Australian summer (White and Pounder 1984).

When a patient has died from unknown causes amongst which snake bite is a possibility, pathologists should maintain a high index of suspicion for this diagnosis as sometimes the changes may be subtle. It may not be possible to identify a bite site. This does not preclude the diagnosis of snake bite however. If there is doubt about the diagnosis every attempt should be made to obtain samples for venom determination. In a badly decomposed specimen it has been suggested that the vitreous humour of the eye may be useful as a sample for venom detection (Sutherland 1983). Other samples which may be of value in detecting snake venom include blood, urine, tissues around possible bite sites, draining lymph nodes, a swab taken from the bite site before excision, and any clothing or bandage over possible bite sites. More detailed discussion of this subject may be found elsewhere (White and Pounder 1984; Sutherland 1983; Plueckhahn 1983).

Pseudo-Snake Bite

Patients sometimes present with a history of possible snake bite, but on further investigation it is revealed that the organism involved was in all probability not a venomous snake. Medical



practitioners managing cases of suspected snake bite should be aware of this problem, although if the rule that antivenom is only administered to patients showing evidence of systemic envenomation is followed, then antivenom will never be inappropriately used.

There are a number of non-elapid snakes in Australia, most of which are non-venomous and those most likely to cause snake bite are pythons, and colubrid snakes. This latter group includes snakes such as the Green Tree Snake (*Dendrelaphis punctulatus*), and the Brown Tree

Snake (*Boiga irregularis*). The tree snakes do not have fangs at the front of the mouth but otherwise the pattern of teeth is similar to that of elapid snakes. The Green Tree Snake lacks fangs and the Brown Tree Snake has small fangs at the rear of the mouth. A bite by one of these snakes would leave a similar pattern of marks to that seen after an elapid bite, except no anteriorly placed fang marks would be seen. Neither is dangerous to man. They are arboreal snakes, and are long and slender in appearance as befits their lifestyle. Pythons have a formidable array of backward curving teeth in the upper and lower jaws, and are likely to leave a very complex pattern of teeth marks in the victim. The number of punctures seen may be quite high, and in excess of those likely to be encountered following any venomous snake bite. As the teeth are long and may penetrate deeply, the wound is often painful, and secondary infection will frequently occur. The patient will not develop systemic envenomation, however, as pythons are non-venomous.

Some Australian 'legless lizards' (family Pygopodidae) have a similar appearance to venomous snakes, and one of the largest species, *Pygopus nigriceps*, has a colour pattern very similar to that of a juvenile Brown Snake (Figs 12–14). This species of legless lizard may also rear up and bite in a fashion reminiscent of a Brown Snake, albeit a very small one. Even a large legless lizard is unlikely to cause a significant local tissue injury, and as all are non-venomous, systemic envenomation will never ensue. Nevertheless, particularly for a child, a bite from one of these legless lizards may be misinterpreted as a snake bite. The author has seen one case of legless lizard bite in a child. It occurred at school. The lizard had been previously captured by one of the students, and brought to school for display. While showing off in triumph his capture, the child was bitten by the legless lizard, and this was misinterpreted by teaching staff as a snake bite. The hapless

Fig. 15. A mangled dead specimen of legless lizard (*Delma* sps) which bit a child, with a consequent diagnosis of snake bite, until the hapless lizard was correctly identified.

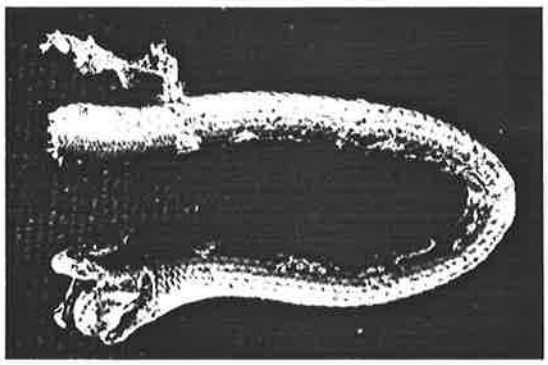
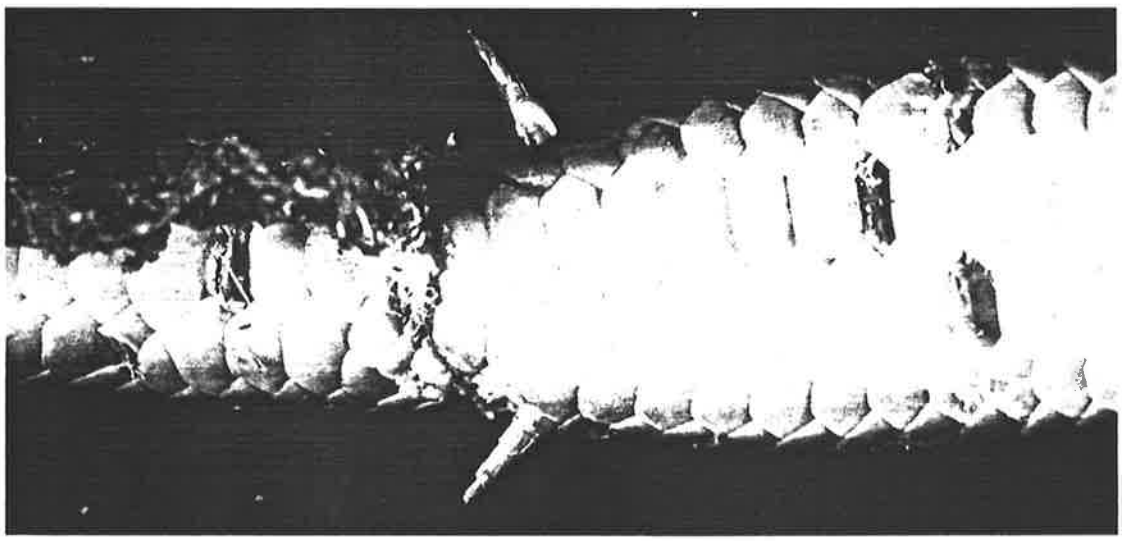
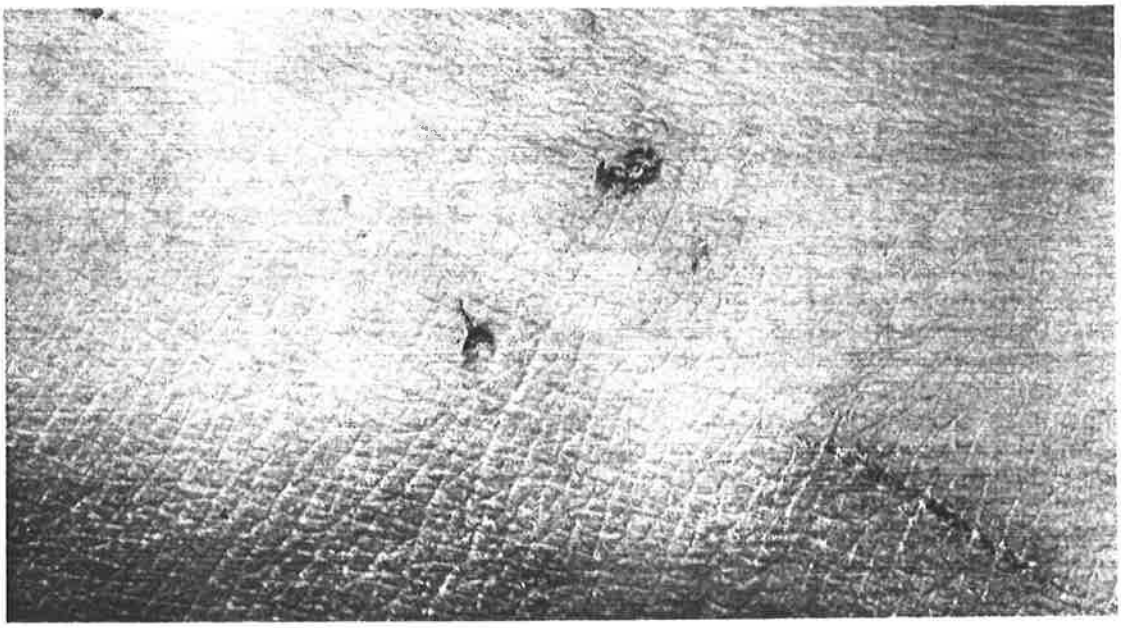


Fig. 16. The same specimen as in Fig. 15, showing the vent region at the termination of the body, and commencement of the tail, with the two vestigial hind limbs of flaps moved into the erect position. Such flaps are typical of legless lizards, and are never found on venomous snakes. Note also the underbelly scales on the body which are paired. In a venomous snake a single large scale will run the entire width of the underbody.





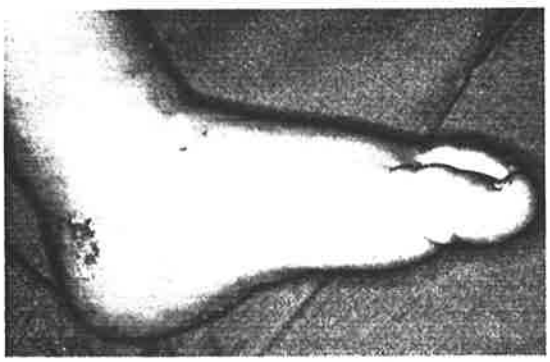
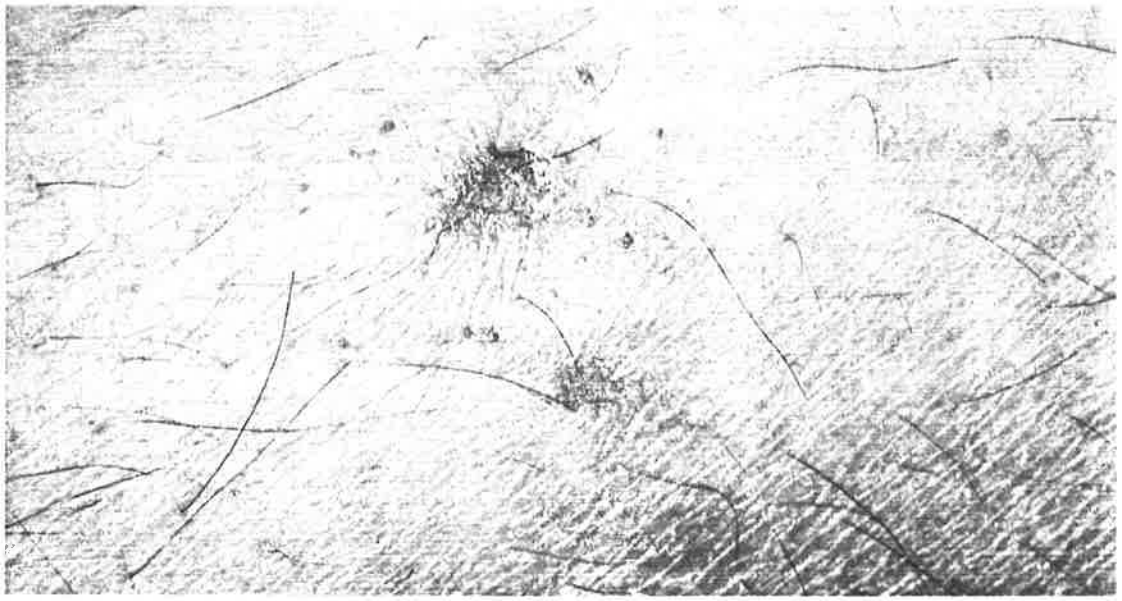


Fig. 17. The foot of a 2 year old boy bitten by an unseen assailant while crawling through long grass in the Adelaide Hills area. The child developed no evidence of envenomation, and no venom was detected at the bite site.

Fig. 18. Same case as in Fig. 17. Note the two obvious puncture marks, with very mild and circumscribed local erythema. There is no evidence of teeth scratch marks or entry marks from teeth of the lower jaw of a snake. The position on the foot is such that a snake would have difficulty biting in this position without other teeth making contact with the foot.

lizard was promptly killed, and presented with the child to a hospital casualty department. None of the medical staff in the casualty department was able to distinguish the legless lizard from a snake, and the usual measures for investigating and managing a snake bite were instituted. It was only some 2 hours later when the 'snake' was identified correctly as a legless lizard that the true situation was apparent. The child, of course, was then promptly discharged. The offending 'snake' is illustrated in Fig. 15. A particular clue to detecting legless lizards masquerading as snakes, is the presence of vestigial hind limbs just anterior to the vent, which appear laterally as flaps (Fig. 16). Their presence gives rise to the other common name for these lizards, namely flap-footed lizards. These flaps are the only external vestige of limbs left in these animals. Unlike snakes the tail is very long, and may often be two or more times the length of the body. The head shape is slightly different from that of a snake, there are external ear openings towards the back of the head, and the tongue is fleshy rather than the fine forked tongue found in snakes (Fig. 14).

Every year the author sees a number of cases of bites by organisms unseen, in which a tentative, diagnosis of snake bite has been made because the local wound consists of two distinct fang puncture marks. Some typical cases are illustrated in Figs 17–22. It can be seen that the common feature of these cases is the presence of two quite distinct puncture marks, separated by a variable distance of 5–10 mm, with no other teeth marks present, and no other significant local reaction. These teeth marks are often in such a position that, had a snake been responsible, it is hard to imagine how the fangs



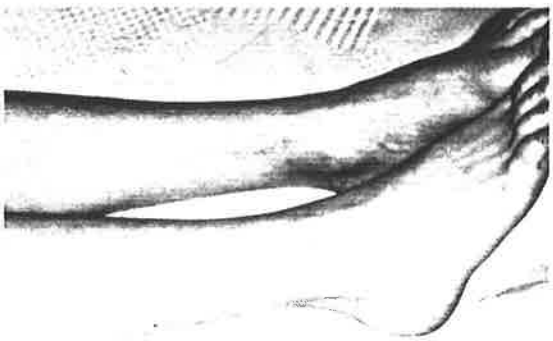


Fig. 19. An apparent bite just above the ankle of a boy aged about 8. The organism involved was not definitely seen, and apart from mild local pain of short duration no other symptoms or signs developed, and no venom was detected at the bite site.

Fig. 20. Same case as in Fig. 19. Note two distinct marks consistent with stings or fang entry marks, without evidence of other teeth marks.

could have entered the skin so definitely without other teeth, particularly in the lower jaw, also marking contact with the skin. In all of these cases no snake venom has been detected from the region of the bite, and there has been no evidence of envenomation, either in terms of symptoms or signs, or laboratory investigations such as detection of coagulopathy. These patients usually report being bitten by something while in the garden or a bush area, and on looking down to investigate the source of pain nothing is obvious. No snake is seen. Pain is usually not severe, and lasts only a very brief time. No other symptoms or problems develop.

The author suspects that these 'snake bites' are not due to snakes at all, but possibly some arthropod. The prime candidate is probably one of the trapdoor spiders (Mygalomorphae). All have large, long fangs that could be expected to produce an injury similar to that seen in these cases (White 1984) (Figs 23–24). Unfortunately, no cases with this lesion, where the spider is the proven culprit, have been personally seen by the author, although he is aware of at least one case where the lesion was as described, and where the spider was seen to bite, was collected and identified as a female Trapdoor Spider (*Aganippe subtristis*). It is the author's practice to treat all such cases, however, with considerable caution, including use of a VDK to try to detect snake venom at the bite site. If in any doubt coagulation studies are also performed. If these investigations are negative, and the patient presents with a history as described, with no development of either symptoms or signs, then they are usually discharged 6-8 hours after the bite. For medical practitioners inexperienced in assessing the local bite appearance, however, it would probably be safer to treat these cases as potential snake bite, and observe them for the usual 18–24 hour period.

New Directions in the Management of Snake Bite

In the foreseeable future it is likely that antivenom will remain the mainstay of treatment of snake bite in Australia. The future mechanism of production of that antivenom, however, is less certain. New techniques in biotechnology, including the use of monoclonal antibodies, and more sophisticated techniques for refining antivenom, may allow the development of more potent antivenoms, of smaller volume, and with a much lower incidence of unwanted side effects. In North America Russell *et al.* (1985) have described new techniques for further refining standard antivenom, and have successfully applied this to antivenom used in North America. As Australian antivenoms are already quite highly refined, it remains to be seen whether the North American techniques will be appropriate in the Australian situation. Monoclonal antibody technology might allow production of extremely specific antivenoms, and their very degree of specificity might in itself be a problem, as they might not have a wide enough spectrum of activity to cover the diversity of minor variations in venom found within any one species. A further possibility (Mebs and Kornalik 1984) involves the detection of critical venom components found in various subforms in most venoms, and which share common antigenic sites. This might allow the preparation of antivenom based on antibodies to these critical sites, and so allow a more effective and less risky polyvalent antivenom. However, research in this field is still at an early stage.

Fig. 21. The foot of a 5 year old girl bitten by an animal unseen on the underside of the left foot. No envenomation developed, and no venom was detected from the bite site.

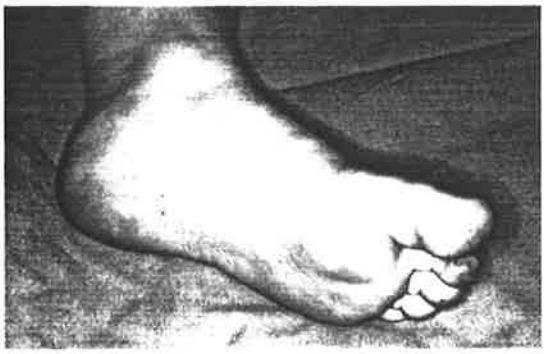
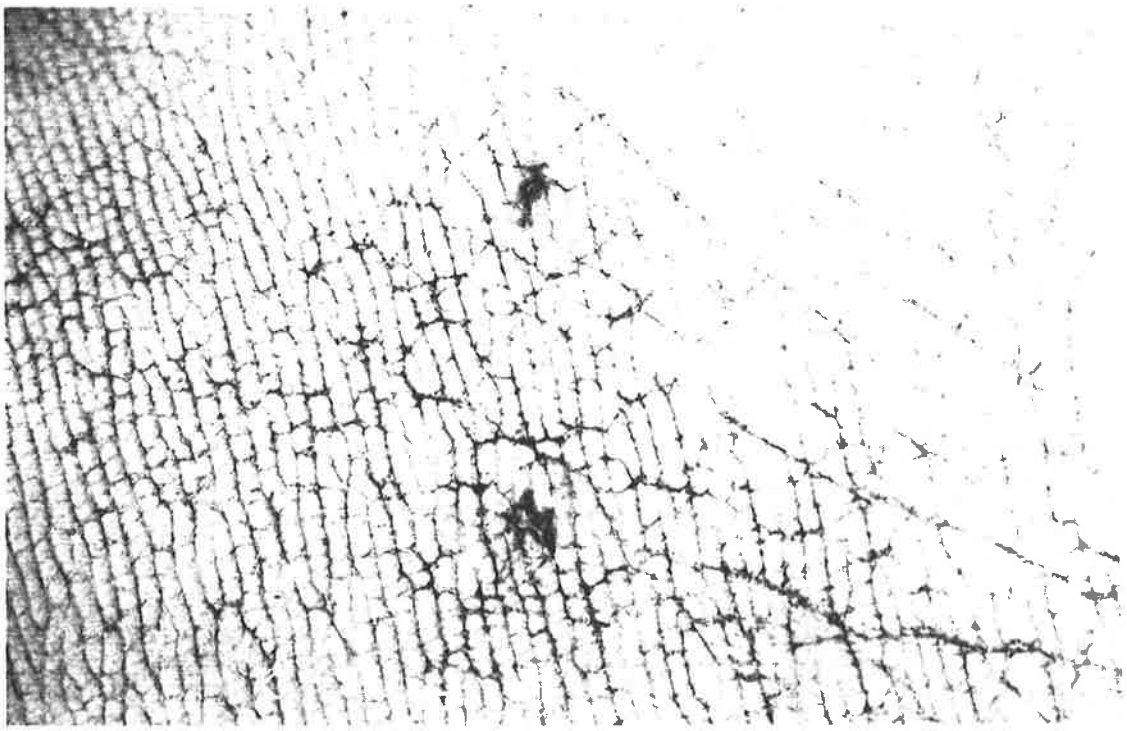


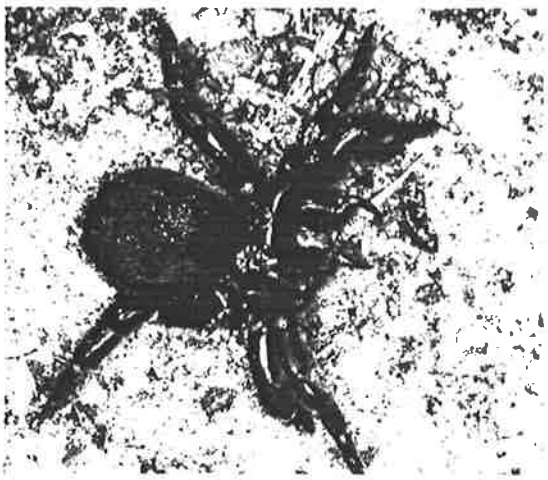
Fig. 22. Same case as in Fig. 21. Note two distinct entry marks, with absence of local reaction or other teeth entry marks.



Whatever developments the future may bring, it is already true that Australians bitten by venomous snakes have an excellent chance of surviving as a result of modern methods of first aid and treatment already well established.

▼ Fig. 23. A mature female Adelaide Trapdoor Spider (*Aganippe subtristus*).

▼▼ Fig. 24. *Aganippe subtristus* in close-up, showing the large and powerful fangs well separated prior to attack.



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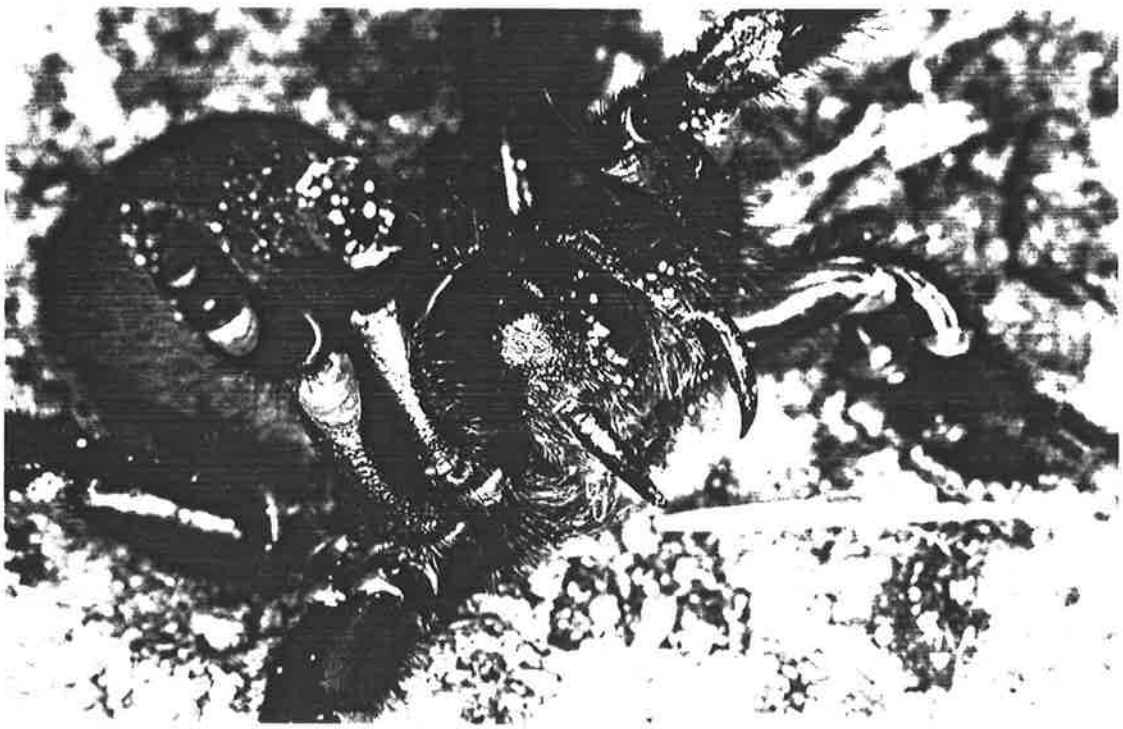

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A REVIEW OF 105 CASES OF SUSPECTED SNAKEBITE IN SOUTH AUSTRALIA

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INTRODUCTION

While snake-bites in Australia may represent less than 2% of cases of bites and stings notified to Poisons Centres in Australia[1], it is undoubtedly an important cause of envenomation, and is probably responsible for the majority of deaths due to envenomation on mainland Australia. This excludes envenomation caused by marine organisms. It has been estimated that between 1,000 and 3,000 people are bitten by snakes each year in Australia, and of these at least 200, and possibly as many as 500, receive antivenom treatment. It has been estimated that on average there are four to five deaths per year in Australia from snake-bite, although the most recent statistics suggest this level may have dropped to one to two fatalities per year[2,3].

Despite the considerable number of case reports of snake-bite in the medical literature, there have been relatively few analyses of large series of snake-bites. This may reflect several factors including the relative paucity of cases in most major centres equipped to undertake such studies, and the difficulties in confirming the diagnosis, and the tendency for the already small number of available cases to be further diluted due to management by a variety of practitioners, without the routine involvement of one or two experts in each regional centre.

The following report details experience with 105 cases of suspected snakebite presenting in South Australia.

METHOD

A retrospective review of cases of suspected snake-bite admitted to three major teaching hospitals in Adelaide, South Australia, namely the Adelaide Children's Hospital, the Royal Adelaide Hospital, and Flinders Medical Centre, were combined with the author's own list of cases personally dealt with over a ten year period. A number of the cases identified in the retrospective review of hospital case records were also those seen by the author. All cases where a discharge diagnosis of snake-bite was made were included.

Each case was ascribed to one of six categories based on information available. These categories were:

1. Positive venom identification.
2. Positive specimen identification.
3. Good patient description of snake in a geographical area known to have a

restricted snake fauna.

4. Good patient description of a snake in a geographical area known to have a diverse snake fauna.
5. Poor description of the snake.
6. Cases where the history and examination in retrospect left doubt about the diagnosis of snake-bite.

Cases in categories 1 to 3 allowed assignment of that case to a particular species group of snake.

The records for each case were reviewed to determine the following factors:

1. Cause of bite - accidental or illegitimate (bite to a snake keeper or handler, or while attempting to collect the snake).
2. Local reactions to the bite - pain, swelling, cellulitis, necrosis.
3. General symptoms of envenomation - headache, nausea, vomiting, abdominal pain, central nervous system disturbance including dizziness, drowsiness, loss of consciousness, or convulsion.
4. Coagulation disturbance - laboratory evidence.
5. Neurotoxicity - objective signs of paralysis.
6. Myolysis - symptoms of muscle movement pain, raised creatine kinase levels.
7. Renal failure - laboratory evidence of impaired renal function.

RESULTS

Of the 105 cases of suspected snake-bite, 87 cases met the criteria for definite snake-bite, where a snake was seen to bite, there was local evidence of fang entry, or where the patient had symptoms and signs suggestive of snake-bite and snake venom was identified. In the remaining 18 cases there was no evidence of systemic envenomation, and despite the local appearance of fang marks or possible fang marks no snake was seen to bite the victim.

Thirty-eight of the 87 cases had definite evidence of systemic envenomation and a further 4 cases had symptoms and signs which may have been due to mild systemic envenomation. Thirty-nine of the 87 cases received antivenom treatment, and no cases of anaphylaxis to the antivenom were recorded. None of the cases was fatal. Thirty-three of the 87 cases were bites to reptile handlers or keepers, or persons trying to collect live venomous snakes.

Bites by particular species of snakes or species groups were as follows:

Brown Snakes, genus *Pseudonaja* - 27 cases plus 11 probable cases
Tiger Snakes, genus *Notechis* - 13 cases
Death Adders, genus *Acanthophis* - 7 cases
Mulga Snakes, *Pseudechis australis* - 6 cases
Red-bellied Black Snakes, *Pseudechis porphyriacus* - 10 cases
Copperheads, genus *Austrelaps* - 2 cases
11 cases uncertain

Brown Snake Bites

Possible species involved are *Pseudonaja textilis, P. nuchalis, and P. affinis.* Of the 27 definite cases and 11 probable cases, 8 were illegitimate bites. Thirteen cases (34%) had evidence of systemic envenomation. Six cases had a coagulopathy. In 4 cases this was defibrination with normal platelet count, and in 2 cases (both associated with acute renal failure) there was a true disseminated intravascular coagulation with platelet consumption. Two cases showed evidence of mild neurotoxicity, neither requiring assisted ventilation. No cases showed evidence of myolysis. Two cases developed acute renal failure, both with delayed antivenom treatment, and both responding after a period of haemodialysis. Overall local reactions to Brown Snake bites were minimal, pain being recorded in only two cases, swelling in one case, and cellulitis in a further case. Headache occured in 13 of the 38 cases, nausea and/or vomiting in 6 cases, abdominal pain in 5 cases, dizziness or drowsiness in 10 cases, and there was one case of loss of consciousness with convulsions.

Tiger Snake Bites

Nearly all cases involved *Notechis scutatus*, the only other species being *N. ater.* Of 13 cases 7 were illegitimate bites. Ten cases (77%) had evidence of systemic envenomation. Six cases showed coagulopathy, which in all instances was a defibrination syndrome. Three cases showed neurotoxicity, none requiring assisted ventilation. Two cases had evidence of mild myolysis with raised creatine phosphokinase levels, but apparently no muscle movement pain. No cases with renal failure were seen. Local pain at the bite site was a common feature following Tiger Snake bites, with 8 of the 13 cases reporting significant local pain, 8 cases associated with local swelling, and 2 cases developed local necrosis. In one case this required subsequent skin grafting, but this case was also associated with prolonged use of an inappropriate arterial tourniquet. Headache was reported in 3 of the 13 cases, nausea and vomiting in 5, abdominal pain in one, loss of consciousness in 2 cases, and one case had convulsions.

Death Adder bites

These were all ascribed to the one species, *Acanthophis antarcticus.* Of the 7 cases, all were illegitimate. Two of the 7 had definite evidence of systemic envenomation, and a further case had doubtful evidence. In none of these cases was coagulopathy documented, nor myolysis or renal failure. Two cases had evidence of neurotoxicity, not requiring assisted ventilation. Local pain was reported in 5 of the 7 cases, local swelling in 3 cases, and local cellulitis in 3 cases. Headache was reported in one case, nausea and vomiting in 2 cases, and dizziness and drowsiness in one case. There were no cases of loss of consciousness or convulsions.

Mulga Snake bites

All bites due to *Pseudechis australis.* Of the 6 cases, 4 were illegitimate. Five had evidence of systemic envenomation, and in 3 of these there was definite evidence of coagulopathy, and an unsubstantiated report of coagulopathy in a fourth. Neurotoxicity was not seen in any case, nor was renal failure, but 2 cases had evidence of myolysis with raised creatine phosphokinase levels. Local pain was seen

in 5 of the 6 cases, local swelling in all 6 cases, usually quite extensive swelling, and cellulitis or necrosis was reported in 2 cases, one of them requiring amputation of the bitten digit. Headache was reported in 3 cases, nausea and vomiting in 5 cases, abdominal pain in 3 cases, dizziness and drowsiness in 2 cases, and brief loss of consciousness in one case. No convulsions were recorded.

Red-bellied Black Snake bites

These were all due to *Pseudechis porphyriacus*. Of the 10 definite bites, 4 were illegitimate. Five cases had evidence of systemic envenomation, based principally on systemic symptoms. One case had evidence of coagulopathy, but no cases developed neurotoxicity, myolysis, or renal failure. In 2 of the 5 cases there was an apparent allergic component, and in one of these, an individual who had been bitten many times previously by venomous snakes including *P. porphyriacus*, the patient presented shortly after the bite, with cardiac arrest, before antivenom was given. Pain was noted in 6 of the 10 cases, swelling in 6 of the 10 cases, usually quite extensive, and cellulitis or necrosis was seen in 3 cases. In no cases was headache reported, but in 4 cases there was nausea or vomiting, abdominal pain in 3 cases, and dizziness or drowsiness in 3 cases.

Adelaide Hills Copperhead bites

This species, genus *Austrelaps*, has yet to receive scientific decription and naming. Of the 2 definite cases and 2 probable cases, one was an illegitimate bite. Only one case showed possible mild systemic envenomation with mild local pain, limited local oedema, and nausea. This resolved without antivenom treatment.

DISCUSSION

While the advent of immunological testing for snake venom using the Commonwealth Serum Laboratories Venom Detection Kit has increased the number of cases where at least the genus of snake involved can be determined, in many regions of South Australia the snake fauna is quite restricted, and a toxinologist with a good working knowledge of the snake fauna, its appearance and variation in colour forms, its distribution and habits, can in many cases narrow down the potential snakes involved to just one or two species groups. In the past in Australia there has been a tendency to suggest that there is little difference in the symptoms and signs of snake-bite between the various groups of venomous snakes. The author's personal experience, as shown in the above study, is that there are some definite defining patterns for each species group. The key factors locally are significant local pain, significant local swelling, and systemically, coagulopathy, neurotoxicity, myolysis. A summary of overall findings to date for each of the 5 major groups of snakes causing snake-bite in South Australia is listed in Table I.

SUMMARY OF EFFECTS

	Local			General				
	Pain	Swelling	Cellulitis	Coagulopathy	Neurotoxicity	Myolysis	Renal	Drowsiness
Brown Snakes	-	-	-	+	±	-	+	+
Tiger Snakes	+	+	+	+	+	+	-	±
Death Adders	+	±	±	-	+	-	-	-
Mulga Snakes	+	++	+	±	-	+	-	+
Black Snakes	+	++	+	±	-	-	-	±

It can be seen there are definite differences between the Brown Snake group, Tiger Snake group, and Death Adders. Similarly there are differences between the Mulga Snakes and Black Snakes as a single group and the other three groups. Differentiation between Mulga Snake bites and Red-bellied Black Snake bites is usually easy in that their geographic ranges and habitat are quite different, the appearance of the snake is strikingly different, and the extent and seriousness of systemic envenomation is usually different, with Red-bellied Black Snake bite usually associated with only mild envenomation.

The number of cases currently accumulated for each group is still small, and thus while this information is useful to toxinologists in determining the most appropriate antivenom treatment for a given case of snake-bite, the author does not advocate its adoption universally within Australia as a method of determining the type of snake involved in the case of snake-bite, and therefore antivenom treatment.

The more extensive and correct use of the Venom Detection Kit to determine the type of snake involved in a snake-bite incident will assist in defining and determining the accuracy of the diagnostic symptomopathology listed in Table I.

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The five-ringed brown snake, *Pseudonaja modesta* (Gunther): report of a bite and comments on its venom

Julian White, Vaughan Williams and Johann H. Passehl

ABSTRACT The first case report of systemic envenomation by the five-ringed brown snake, *Pseudonaja modesta*, is presented. The patient had mild general symptoms of envenomation only, with no coagulopathy or evidence of paralysis. Data on *P. modesta* venom is presented, which shows an absence of a coagulant component in this venom. The venom gives a positive reaction to brown snake venom in the CSL Venom Detection Kit.

(Med J Aust 1987; 147: 603-605)

Members of the brown-snake genus *Pseudonaja* are collectively one of the most important causes of snake-bite in Australia.[1,2] The taxonomy of this genus is under review by a number of workers,[3,4] but of the currently- recognized species, all have been reported as potentially dangerous to humans except one, *P. modesta*. This latter species, which is commonly known as the five-ringed brown snake or collared brown snake, is the smallest member of the genus, with an average adult total length of 0.45 m (Figure 1).[5]

Various authorities have stated that *P. modesta* is harmless to humans,[5] or unlikely to cause significant injury.[1] No studies have been published of its venom. Recent chromosomal analysis has suggested the validity of its inclusion within the genus *Pseudonaja*, and its close relationship to *P. inframacula* and *P. guttata*,[4] both of which are considered to be dangerous species.[1]

We here report the first medically-documented bite by *P. modesta*, with proven systemic envenomation, and some subsequent studies of the venom of this species.

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No reprints will be available.

Clinical record

On December 14, 1986 at approximately 5.30 p.m. an 11-year-old girl was bitten on the dorsum of her left foot by a brown-coloured snake approximately 0.5 m long. The snake was killed by her brother and the head and neck brought, with the patient, to the local hospital (Booleroo Centre District Hospital) at 6.00 p.m. where she was seen by one of the authors (J.H.P.). Treatment on presentation was bed-rest and cleansing of the site with normal saline. (Washing the site is not recommended for snake-bite in Australia, as it may remove venom from the skin surface, thus reducing the chance of venom identification by means of the Venom Detection Kit [Commonwealth Serum Laboratories, Melbourne]. There is no evidence that venom on the skin surface is dangerous to the patient.)

The only initial symptom was nausea, which persisted for about six hours

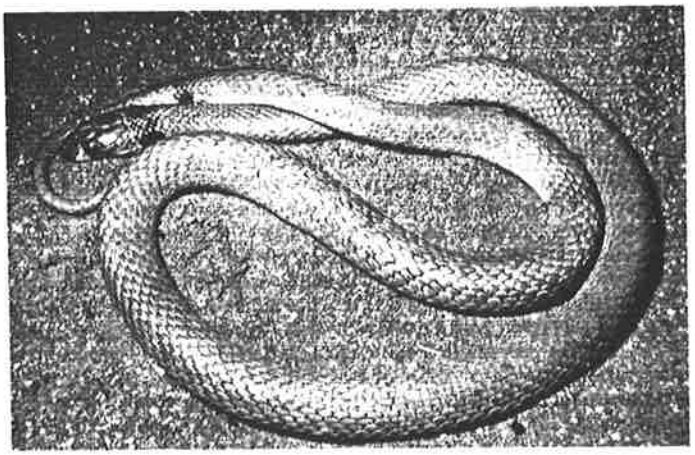

FIGURE 1: The five-ringed or collared brown snake, Pseudonaja modesta.

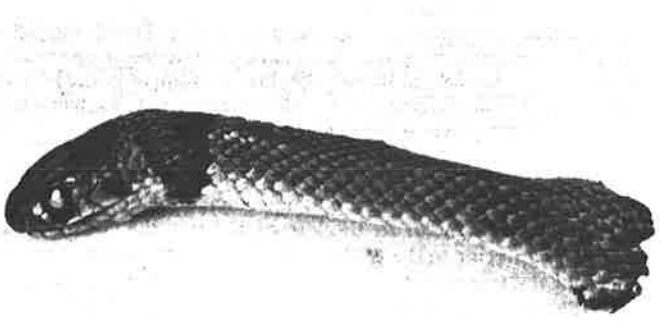

FIGURE 2: The head and neck of the P. modesta that was responsible for the snakebite described in this report.

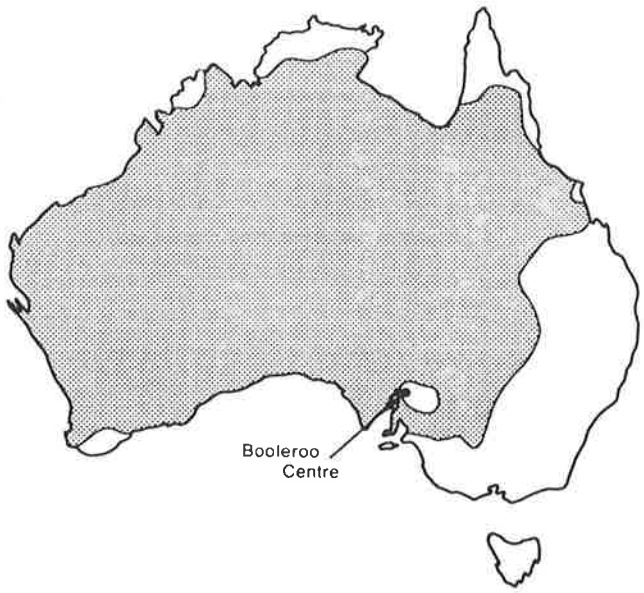

FIGURE 3: The computer-predicted distribution of P. modesta in Australia (shaded).[7]

without vomiting. This was followed by mild headache which occurred a half-an-hour after the bite and lasted for 12 hours, and abdominal pain, which also occurred a half-an-hour after the bite and lasted for 16 hours. The bite site showed two small puncture wounds but no initial local reaction. There was no evidence of paralysis and specifically, ptosis, diplopia, and dysarthria were not present. Muscle movement pain could not be elicited. No bleeding tendency was noted. Only limited laboratory facilities for coagulation studies were available; therefore, only the prothrombin ratio and the activated partial thromboplastin time tests were performed at one hour, two hours, and 19 hours after the bite: all gave normal results.

Serum and urine were tested separately for snake venom at two hours after the bite by means of a current CSL Venom Detection Kit. No venom was detected in serum, but a strongly-positive result for brown-snake venom was recorded for the urine. At 2.5 hours after the bite, the patient described mild "burning" at the site of the bite and a patch of local erythema of approximately 1.5 cm in diameter was observed. Lymphadenopathy or tenderness were not noted in the left groin at any stage.

In view of the minor systemic symptoms, and the lack of paralysis or coagulopathy, a decision was made to withhold antivenom and to observe the patient. By 16 hours after the bite all her symptoms had disappeared. However, some pain at the site of the bite and mild abdominal discomfort recurred when the patient was mobilized and went to have a shower. These symptoms subsided within 2.5 hours, and at 26 hours after the bite, the patient was discharged fit and well. Superficial desquamation was noted over the area of previous erythema surrounding the site of the bite three days later but no other sequelae were evident.

The remains of the snake (Figure 2) were identified as of *P. modesta* by one of us (J.H.P.) by means of a monograph on snake-bite in South Australia.[6] This identification was confirmed subsequently in Adelaide (by J.W.). The finding of this specimen of *P. modesta* records a new distribution area for this species, but not a surprising one, as the snake was caught in a region that is close to the distribution range which has been predicted by climatic analysis by computer (Figure 3).[7] The status of the adjacent area, which has been predicted to be unsuitable for this species, remains unknown.

Methods

Venom from *P. modesta* (single specimen, registration no. SAM 24656) and *P. textilis* (pooled from several specimens) was obtained from the Herpetology Department of the South Australian Museum, which has a collection of frozen whole venoms. The venoms had been collected directly into capillary tubes and frozen in liquid nitrogen to maintain the integrity of the venom constituents during transport from the field collection site to the laboratory. The venoms were kept at −76 °C before testing, when they were thawed and protein estimations were performed by means of the Bio-Rad protein estimation kit (Bio-Rad, Richmond, California).

The venoms were then adjusted to 100 μg of protein/mL in 0.05 M sodium acetate/0.15 M sodium chloride, pH 5.8. Twenty micrograms of protein were loaded onto an LKB, TSK G3000 SW 8 mm × 300 mm (gel filtration) column for both *P. modesta* and *P. textilis*. To elute the proteins, 0.05 M sodium acetate/0.15 M sodium chloride (pH 5.8) was used at a flow rate of 0.5 mL/min. Detection was at 280 nm. Fractions of 0.5 mL were collected into plastic tubes which were capped and refrigerated at 2 °C–6 °C before testing for coagulant activity (within 24 hours). Chromatography conditions were controlled by the LKB Gti Ultrochrom system.

The whole venom and collected fractions were tested for coagulant activity by the addition of 50 μL of whole venom or fraction to 100 μL of pooled, citrated normal human plasma (pooled from 10 healthy adult volunteers) and then timing the appearance of a clot by means of a manual tilt-tube method in a 37 °C water-bath.

Results

No coagulant activity was detected in the whole venom or venom fractions of *P. modesta*, which is in contrast with other *Pseudonaja* venoms. In similar experiments, *P. textilis* venom at low concentrations rapidly produces clotting of normal plasmas.[15,16]

Gel-filtration chromatography of *P. modesta* (whole venom) produced an elution profile that was substantially different from that of *P. textilis* (Figures 4 and 5). The coagulant-containing fractions of *P. textilis* (shaded) have no equivalent in *P. modesta*. The only peak equivalent is the largest peak and "shoulder" peaks

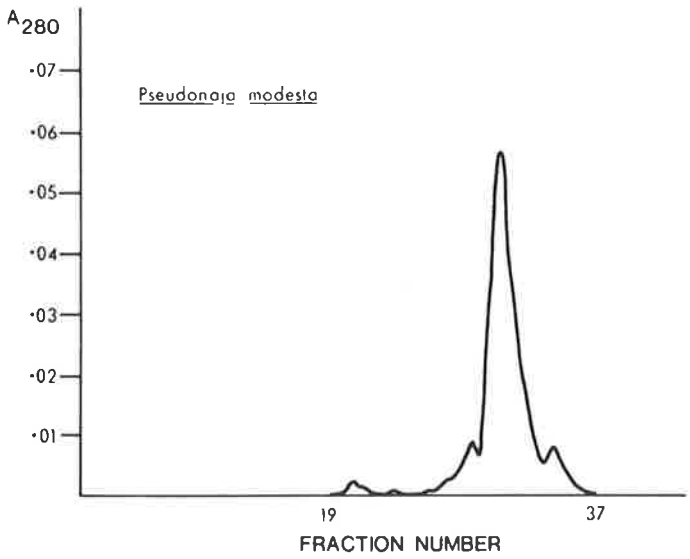

FIGURE 4: Chromatography of P. modesta whole venom. Twenty micrograms of protein were loaded onto a TSK G3000 SW gel filtration column. Fractions of 0.5 mL were collected by elution of the venom with 0.15 M sodium chloride/0.5 M sodium acetate (pH 5.8) at a flow rate of 0.5 mL/min.

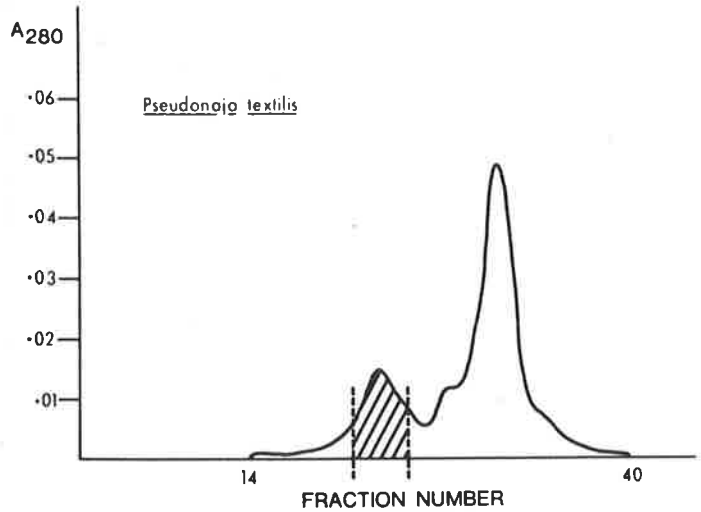

FIGURE 5: Chromatography of P. textilis *whole venom. Conditions as in Figure 4 for* P. modesta. *Shaded area represents the coagulant activity.*

of this component, which suggests a similarity in the major toxin component, although this was not tested.

Discussion

Coagulopathy is a common accompaniment of systemic envenomation by *Pseudonaja* spp. and consequent defibrination may be severe.[2,6,10-13] There is definite evidence that such a coagulopathy potentially may be fatal due to cerebral haemorrhage,[14-16] although death due to other types of haemorrhage has not been recorded. All *Pseudonaja* venoms which have been studied previously have shown coagulant activity *in vitro* and a coagulopathy *in vivo*,[8,9,17] and all are from species which are likely to be lethal in humans.

The clinical record that is reported here documents a *P. modesta* bite which was associated with mild, general systemic symptoms of envenomation and proven systemic circulating venom (by excretion in urine), but no evidence of any coagulation abnormality. This implies that bites by this species may not cause coagulopathy, possibly due to the absence of a coagulant component in the venom.

The studies of whole *P. modesta* venom confirm this proposal; the whole venom was unable to clot substrate plasma. Furthermore, the coagulant-associated peak that was seen in the elution profile of *P. textilis* venom is absent in the profile of *P. modesta* venom.

Therefore, the available evidence demonstrates that *P. modesta* bites will not be associated with coagulopathy, and thus clotting studies will not be helpful in the determination of the extent of systemic envenomation, or the titration of antivenom treatment; this is in direct contrast to bites by other *Pseudonaja* spp.

While our single patient showed only mild problems in spite of circulating venom, and did not require antivenom, it cannot be assumed that all subsequent cases of bites by *P. modesta* will be mild. Caution should prevail, with observation of the patient in a hospital setting for approximately 24 hours. Antivenom should be withheld unless the patient shows clear evidence of major systemic envenomation, in which case monovalent CSL Brown Snake Antivenom appears to be the most appropriate antivenom.

Acknowledgements

The authors acknowledge gratefully Dr Kevin Cheney, Director, Department of Haematology, Adelaide Children's Hospital, for the use of departmental facilities in this research, and for helpful advice. Dr Terry D. Schwaner kindly provided the *P. modesta* venom. Dr Isobel E. Speed provided valuable advice on this paper. Dr Peter Clements provided advice on aspects of protein fractionation. The Photography Department, Adelaide Children's Hospital, helped to prepare some of the illustrations. Mrs Olivia Lammas typed the manuscript.

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Severe envenomation following multiple bites by a common brown snake, *Pseudonaja textilis*

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Pseudonaja textilis snakebite

Severe envenomation following multiple bites by a Common Brown Snake (*Pseudonaja textilis)*

ABSTRACT

A case report of envenomation by a common brown snake, *Pseudonaja textilis*, in a 3 year 4 month old boy is presented. He suffered an apparent *grand mal* fit within ten minutes of the bite, with spontaneous recovery, and subsequent development of a severe coagulopathy of the defibrination type, requiring 5 ampoules of brown snake monovalent antivenom (CSL) for reversal. The association of envenomation by snakes and convulsions is discussed, as is the management of severe defibrination due to envenomation.

KEY WORDS

Snakebite; Coagulopathy; Convulsions; Brown Snake

Severe envenomation following multiple bites by a Common Brown Snake (*Pseudonaja textilis)*

INTRODUCTION

The Common Brown Snake (*Pseudonaja textilis*) is the most commonly encountered dangerous snake in South Australia, being responsible for more snakebites than any other species, especially in Adelaide and environs.[1] The following case report documents a patient with severe envenomation, defibrination and early development of a *grand mal* fit.

CASE REPORTS

On 7/11/1987 a 3 year 4 month old boy was bitten by an 86 cm snake, subsequently identified (by JW) as *Pseudonaja textilis*. The incident occurred at a caravan park in Elizabeth, a satellite town of Adelaide. The snake was discovered by a group of children in a park, who annoyed it, whereupon it lunged at our patient, biting him on the right ankle, and wrapping its body around his right leg. It was observed to bite at least twice. It then moved up his shorts before falling off and attacking a cat. The snake was then killed by an adult called to the scene. The actual bite occurred at 1205. Within minutes a local compressive bandage was applied over the bite site and the child transported to a 24 hour clinic, arriving at about 1210. At this time the child was awake, but drowsy. The following description of events at the clinic was given by the child's mother.

The bandage was removed by a doctor, and the child started crying, started "shaking all over", commenced spasmodic movements, became pale, clammy, went limp, with the face becoming a grey colour, then blue, the rest of the body remaining white. The jaw was clenched tight, the tongue "swollen". The child was incontinent of urine and faeces. An ambulance was called (1212), an oxygen mask applied, and the child's chest "pushed hard" once over the sternum, whereupon a breath was taken and "froth" exuded from the mouth.

The ambulance arrived at 1217. The ambulance officers noted the child was drowsy, opening eyes to voice, not verbalising, obeying commands, breathing normally, clammy skin, flushed skin colour, not fitting. The child was transported to the local hospital, arriving at 1232. The child was there noted to be awake, not drowsy, but sweaty, rash on hands and leg, face

pale, pulse 130 regular, BP 140/90, no palpable right inguinal nodes, no neurological deficit, pupils equal and reactive to light.

An intravenous line was inserted, and blood taken for venom detection using the Commonwealth Serum Laboratories (CSL) Venom Detection Kit (VDK). This gave a negative result. The child remained awake, but drowsy, and progressively more irritable, with pulse, BP and respiratory rate all stable. During this period the child vomited twice, and also complained of abdominal pain. Further advice was sought at 1430. It was then recommended that blood be taken for coagulation studies, 15 mg IV phenergan given (the child had a past history of asthma), and then one ampoule of Brown Snake antivenom given IV over 15 minutes, diluted about 1:10 in 4% Dextrose in Saline solution.

One of the authors (JW) first saw the patient at 1500, at which time antivenom had been added to the IV line, but the line was blocked. The local bandage over the bitten leg, having been re-applied by the ambulance crew, was still in place. The child was very irritable, intermittently screaming and struggling. A new IV line was promptly inserted in the left foot and antivenom administration commenced. The results of coagulation tests became available at this time (Table 1), confirming a marked coagulopathy. The site of the removed IV line in the right wrist continued to ooze significantly, and indeed did so for at least a further 3 hours. As soon as the antivenom infusion ceased, the child was transferred to a large teaching hospital for further treatment, arriving at 1630, whereupon a further two ampoules of Brown Snake antivenom were given IV, diluted about 1:10 in Normal Saline, infused over 20 minutes. From the time of first antivenom infusion until this time the child was alternately screaming and struggling or inert (though rousable).

There was no evidence of paralysis, and specifically no ptosis, gaze paralysis, or weakness of limb muscles was detectable. The bandage on the right leg was removed after completion of the second antivenom infusion, at 1650. Several bite and scratch marks were evident over the right ankle posteriorly (Figure 1), but no associated oedema, erythema, or bruising was evident. The coagulation tests demonstrated a continuing coagulopathy, still severe 2 hours after the second antivenom infusion (Table 1), the child still being intermittently irritable and drowsy. A further 2 ampoules of antivenom were given, as before, commencing at 2000. By 2230 the child was much less irritable, and coagulation tests showed considerable improvement. Further antivenom was therefore withheld, and coagulation tests repeated at 0005 on 8/11/1987. At this time the child was awake, not distressed, and happily talking to his parents. The coagulation tests showed further resolution of the coagulopathy, and no further antivenom was given.

The child made an uneventful recovery, and was discharged on 9/11/1987. Subsequent enquiry revealed that the child suffered frequent nightmares about snakes and became very agitated on seeing any long thin object even vaguely resembling a snake.

DISCUSSION

This case illustrates several important aspects of severe snakebite in Australia. Firstly, the initial episode of collapse, which occurred within a few minutes of the bite, appears to have been associated with a *grand mal* convulsion, in this case well described by the child's mother. This response to snakebite in children has been noted before for bites by *Pseudonaja* sps[2,3], *Notechis* sps[4,5], *Oxyuranus* sps[6,7,8], *Pseudechis* sps[9], but rarely in such detail. Nevertheless it is clear that, particularly in children following significant snakebite, one of the earliest responses to systemic envenomation is collapse, which may be followed by fitting, from which the child recovers before antivenom is administered. This collapse may occur within ten minutes of the bite. The component or components of Australian elapid venoms responsible for this presentation have not been adequately demonstrated and the mechanism remains obscure.

The onset of collapse immediately following removal of the first aid by the first doctor consulted may well be coincidental, as the child was already drowsy prior to its removal. This case does not provide firm evidence for or against the effectiveness of the currently accepted first aid for snakebite in Australia. However the authors strongly support this first aid method, as developed by Sutherland and colleagues.[10]

The removal of first aid by the first doctor seen, when he had no facilities to treat snakebite, and in particular no antivenom, was unfortunate. First aid should only be removed when the patient is in a hospital with resources to treat snakebite, and those resources have been mobilised, and in particular appropriate antivenom is ready to hand if needed.

Severe envenomation following multiple bites by a Common Brown Snake (*Pseudonaja textilis)*

The initial delay in appropriate investigations and treatment at the first (peripheral) hospital was unfortunate. In retrospect the history gave clear indication of systemic envenomation. Coagulation tests should have been performed at the outset, and then a first dose of antivenom given. Using the VDK to test blood for snake venom in this situation is unrewarding and probably inappropriate. The main role of the VDK is to detect snake venoms on the skin over the bite site, or in urine (only if patient is systemically envenomed), and to thus indicate the most appropriate monovalent antivenom for treatment. In our experience blood has proved an unreliable test sample for the VDK. In this case the use of the VDK was unnecessary as the dead snake was available for identification. It should be noted that a positive VDK result from a bite site skin swab in the absence of evidence, clinical or laboratory, of systemic envenomation, is not an indication for antivenom therapy.

The severity of the coagulopathy seen after significant envenomation by *Pseudonaja* sps is well demonstrated by this case. There is a marked defibrination, but no significant effect on platelet count. Fibrin degradation products are markedly elevated, both measured by standard techniques for FDP and by the recently available XDP technique. The positive result with XDP in snakebite defibrination has not been documented previously. This technique uses a monoclonal antibody to the D-dimer Fragment, only produced as a breakdown product of crosslinked fibrin. This suggests that the snake's venom procoagulant, which activates prothrombin, results in formation of crosslinked fibrin strands before these are broken down, rather than the breakdown of fibrinogen directly to FDPs.

The use of coagulation test results to determine response to antivenom therapy is well demonstrated by this case. Clinical improvement and resolution of the coagulopathy were well correlated. An envenomed child

still irritable despite antiveom therapy is a good indication that further antivenom is required. The use of five ampoules of antivenom in this case is certainly indicative of the large quantity of venom that may be injected, especially with multiple bites, but does not represent the maximum amount of antivenom that has been used. The ability of antivenom alone to reverse the coagulopathy is also demonstrated, without the need for replacement therapy with fresh frozen plasma.

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ACKNOWLEDGEMENTS

The authors thank Dr Kevin Cheney for support and for reviewing the manuscript, the Photography Department, Adelaide Children's Hospital, for preparing the illustration, and Mrs Olivia Lammas for typing the manuscript.

ILLUSTRATION

Figure 1 Right ankle of child bitten by 86 cm brown snake, showing multiple bite and scratch marks, without significant local oedema or bruising.

Table 1 Results of coagulation studies in relation to antivenom administration

Date	7/11/87	7/11/87	7/11/87	7/11/87	7/11/87	7/11/87	7/11/87	8/11/87	8/11/87	8/11/87	9/11/87
Time	1430	1500	1630	1730	1900	2000	2230	0005	0930	1700	0930
Time after bite (hrs-mins)	2-25	2-55	4-25	5-25	6-55	7-55	9-25	10-55	18-25	27-55	43-55
Antivenom administration*	-	1 vial	2 vials	-	-	2 vials	-	-	-	-	-
Whole blood clotting time (min)	-	-	-	∞	13.5	-	6.5	5	-	6	4
APTT (secs)	>200	-	-	>10 min	135	-	58.4	47	32.5	33	31
Prothrombin ratio	>11.5	-	-	>10	>10	-	2.1	1.7	1.25	1.2	1.0
Fibrinogen g/l	<1.8	-	-	0.02	0.02	-	0.45	0.45	0.86	1.49	2.31
FDP ug/ml	>40	-	-	16,000	6,000	-	2,000	1,000	120	120	15
XDP ng/ml	-	-	-	>64,000	32,000	-	-	-	-	-	-
Platelet Count 10^9/l	-	-	-	242	235	-	259	210	196	-	276
Haemoglobin g/l	-	-	-	132	130	-	128	111	111	-	123

*CSL Monovalent brown snake antivenom, 1000 units per vial

ΓΙΟΝ IN VENOM CONSTITUENTS WITHIN A SINGLE ΛTED POPULATION OF PENINSULA TIGER SNAKE (*NOTECHIS ATER NIGER*)

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(*Accepted for publication* 29 *April* 1987)

AMS and J. WHITE. Variation in venom constituents within a single isolated population of ı tiger snake (*Notechis ater niger*). *Toxicon* **25**, 1240–1243, 1987. — Venom from seven ls in a homogeneous isolated population of *Notechis ater niger* on Roxby Island, off the ıstralia coast, was subjected to gel filtration liquid chromatography and polyacrylamide ophoresis to determine the extent of homogeneity of the venom constituent profile. The ion chromatograms from six of the specimens were easily equated, while the seventh ı slight variation. Sodium dodecyl sulphate-polyacrylamide gel electrophoresis of the n a native and reduced state showed no detectable difference between six of the seven ls, the remaining individual showing minor changes only in the native venom.

ırch frequently requires quantities of whole venom in excess of 100 mg to antial yields of fractions under study. To this end, pooled venoms are gain the required quantities. Problems have been encountered using pooled ı respect to consistent results, as various factors must be taken into ı. Firstly, are the individuals included in the pool from the same geographic ɔndly, are they from a single population within a locality? This problem may ıntered in a situation of dedicated venom supply, wherein the individuals may ıtive of the general population of that species, since genetic variability will ɔms of differing constitution within a single litter (CHIPPAUX *et al.*, 1982; ORNALIK, 1984). To study this problem we selected seven individuals of er snake (*Notechis ater niger*) from Roxby Island (a small 92 hectare island ı Australia coast) as representatives of an isolated ecologically homogeneous nd attempted to delineate any variation in their venom constituent profile ıtography and electrophoresis.

ıs separately collected from seven snakes (single milking) into capillary n in liquid nitrogen and maintained in the laboratory at −76°C. On thawing, were adjusted to a concentration of 1 mg of protein per ml by dilution in l/0.05 M Na acetate, pH 5.8. Protein estimates were performed using Bio- ı kit (Bio-Rad Laboratories, Richmond, CA). The venoms were

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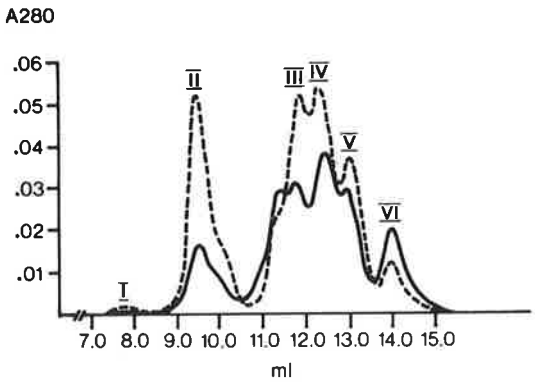

FIG. 1. GEL FILTRATION CHROMATOGRAMS.
veraged peak heights of chromatograms obtained from venoms 1 – 6. —— Chromatogram obtained from venom 7.

raphed on an LKB TSK G3000 SW column using an LKB Gti Ultrochrom em; 200 µl of a 1 mg/ml concentration solution were loaded on the column. vas 0.5 ml/min. The eluent was 0.15 M NaCl/0.05 M Na acetate, pH 5.8. SDS- performed essentially according to the method of LAEMMLI (1970), using a g gel and a 15% separating gel. Specimens were applied in the native state and eduction with β-mercaptoethanol.

ie seven venoms gave a chromatogram that gave perfect correlation of peaks imposed. Figure 1 shows the chromatogram obtained from averaging the peak the six venoms. Peaks II and III had distinctive shoulders, suggesting the of at least two further peaks partly concealed. Figure 1 also shows the ram obtained from the seventh specimen. Seven peaks can be seen; the six s are aligned and the seventh peak coincides with the shoulder of peak III. This at the appearance of the chromatogram of specimen seven may be related more ein content of each peak rather than to the appearance of a new peak. SDS- the seven venoms in an unreduced form shows a single band change for umber seven (Fig. 2a). When the venoms were reduced with β-mercaptoethanol ces were noted between the banding patterns from any of the snakes (Fig. 2b). enoms can pose problems for researchers, particularly in isolating minor toxins be peculiar to only some of the members of that pool, and then in varying Purchase of another pooled venom from the same species may be lacking the interest, as those animals to which the toxin is peculiar are not included. collected from individuals for the pool from differing geographic locations the venom content, while the quantity of any particular component may vary dividuals within a group. Substantial variation in toxicity of venoms from a cies has been demonstrated for elapid snakes from different parts of a range (IRWIN *et al.*, 1970). Our study examined seven individuals of an pulation, situated on an island off the South Australia Coast. This population tudied ecologically and been shown to be a small homogeneous population R, 1985). We found that the venom from six of the seven snakes contained the nponents, as determined by gel filtration chromatography and gel resis. The difference noted in the individuals related to the amount of each t present. Quite large differences were noted in peak heights of the major

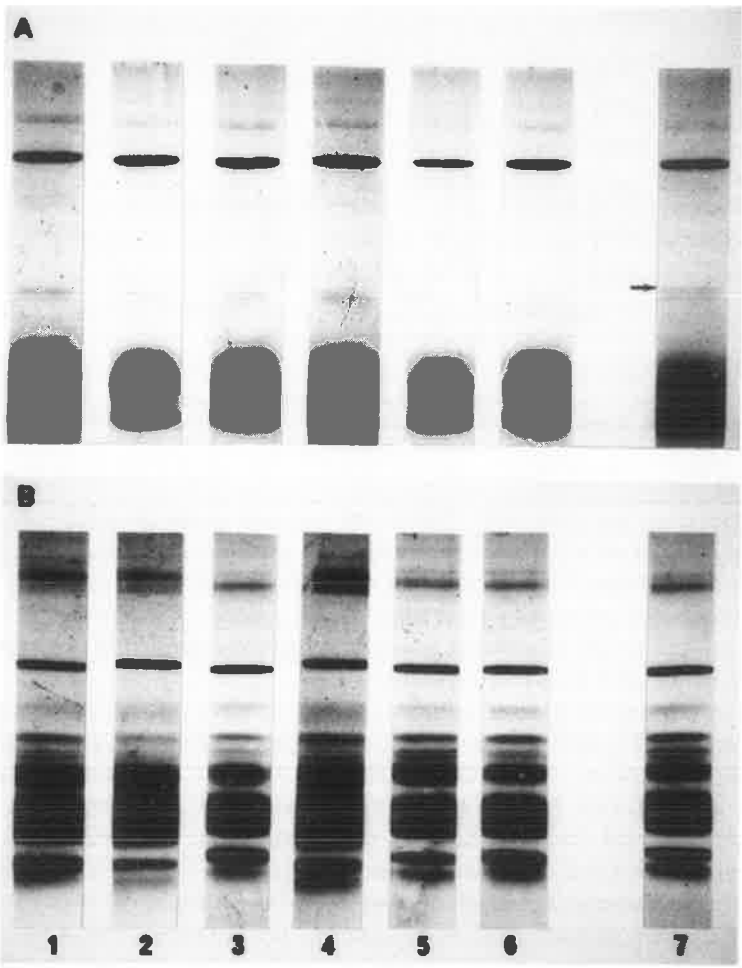

FIG. 2. SDS-PAGE OF NATIVE AND REDUCED VENOMS.
(A) Transposition of band in venom 7 in the native venom series is indicated by an arrow
(B) Homogeneity of banding in the reduced venom series is seen between all seven venoms.

peaks, indicating that the variation in amounts of each component present within a population could result in masking of an attribute under study. This may also e differences in toxicity, with varying concentrations of a key lethal componen seventh individual under study produced a slightly different gel filtration chromat and SDS-PAGE banding in the unreduced form. The difference obtained chromatogram may reflect the amount of protein present in each pea superimposition of the chromatograms showed a close affinity between the obtained, but of differing heights. SDS-PAGE in the reduced form produced id banding and suggests that the differences noted in the native form relate to only a variation in the venom component under discussion, which after reduction is ne This difference argues in favour of examining venoms in a native and reduced stat comparing their constituents. In an isolated population, such as that studied, there appear to be a homogeneous venom produced, ideal for venom research. A furthe

ormed to compare the venoms of a sub-species of *Notechis* from isolated reas using the techniques employed in this study.

ents — The authors thank Dr K. CHENEY for his support of the study, Dr T. SCHWANER of the n Museum for his interest in the work and for the supply of venom samples, Medical Illustration Children's Hospital for preparation of figures and Mrs O. LAMMAS for typing the manuscript.

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EFFECTS OF A MYOTOXIC PHOSPHOLIPASE A_2 ISOLA FROM *BOTHROPS ASPER* VENOM ON SKELETAL MUS SARCOPLASMIC RETICULUM

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(*Accepted for publication* 29 *April* 1987)

J. M. GUTIÉRREZ, G. ROJAS, B. LOMONTE, J. A. GENÉ and L. CERDAS. Effects of a myot phospholipase A_2 isolated from *Bothrops asper* venom on skeletal muscle sarcoplasmic reticul *Toxicon* **25**, 1244–1248, 1987. — The myotoxin from *B. asper* snake venom inhibited Ca-ATI activity of rabbit sarcoplasmic reticulum after incubation *in vitro*. Inhibition was non-compet and albumin enhanced the effect of the toxin. Furthermore, *B. asper* myotoxin hydrol sarcoplasmic reticulum phospholipids and induced a dose-dependent release of horsera peroxidase that had been trapped in sarcoplasmic reticulum vesicles. Binding studies indicated myotoxin does not bind to any particular protein of this membrane, suggesting that the t might interact with phospholipids. Inhibition of Ca-ATPase is probably a consequence o alteration in sarcoplasmic reticulum phospholipids.

A MYOTOXIC phospholipase A_2 has been isolated from *Bothrops aspe* (GUTIÉRREZ *et al.*, 1984*a*). Pathogenesis of myonecrosis induced by this toxin investigated and it was concluded that the plasma membrane is the first cellular to be affected (GUTIÉRREZ *et al.*, 1984*a,b*; BRENES *et al.*, 1987) and that the r pathologic changes occur after this initial sarcolemmal disruption. Othe membranes, such as those of sarcoplasmic reticulum, seem to be secondarily since damaged muscle cells contain many small vesicles in their cellular space, pr a consequence of internal membrane alteration (GUTIÉRREZ *et al., 1984b*). Disr sarcoplasmic reticulum may contribute to muscle cell damage, since this organel key role in calcium homeostasis (CARAFOLI, 1982). It is not known, however, if myotoxin affects sarcoplasmic reticulum directly or if this intracellular me damaged secondarily to the activation of cellular phospholipases. In this comm we report on the action of *B. asper* myotoxin on rabbit sarcoplasmic reticulum

Crude *B. asper* venom was a pool obtained from adult specimens collect Atlantic region of Costa Rica. The myotoxin was isolated according to the m GUTIÉRREZ *et al.* (1986). Homogeneity was demonstrated by polyacrylamid electrophoresis (REISFELD *et al.*, 1962). Sarcoplasmic reticulum was isolated fr skeletal muscle according to the method of NAKAMURA *et al.* (1976). Ca-ATPas

VARIATION IN VENOM PROTEINS FROM ISOLATED POPULATIONS OF TIGER SNAKES (*NOTECHIS* ELAPIDAE) IN SOUTHERN AUSTRALIA

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V. Williams, J. White, I.D. Schwaner, A. Sparrow. Variation in venom proteins from isolated populations of tiger snakes (*Notechis*: Elapidae) in Southern Australia. *Toxicon* _____, _____ _____, 19___.

ABSTRACT

Pooled venom of peninsula tiger snakes (*Notechis ater niger*) from eleven insular populations and one mainland area, and from a single population of the mainland tiger snake (*N.scutatus*) were subjected to SDS-PAGE and gel filtration chromatography. At least 20 proteins were resolved from the SDS-PAGE, some of which were common to all populations, but many others were highly variable. Elution profiles produced through gel filtration showed a clustering of some populations with like profiles, while others had distinctive patterns. Similarities and dissimilarities between each population venom profile were appraised. Variation in the venom patterns was independent of prey type or local ecology. Through quantitative statistical analysis of the SDS-PAGE banding patterns, grouping of populations was dependent on their relative geographic position and the time of isolation of each population from one another and the mainland population.

INTRODUCTION

The evolution of venom in snakes is incompletely understood, although relationships between species can be reflected in major toxic components of their venoms (KOCHVA, 1987). Intraspecific variation in venom components has been studied for several species (JIMENEZ-PORRAS, 1964, CHIPPAUX *et al.*, 1982, MEBS and KORNALIK, 1984, GREGORY-DWYER *et al.*, 1986, WILLIAMS and WHITE, 1987); some components are conserved whilst others are variable. This study compares components of whole venom among several insular populations of tiger snakes (*Notechis scutatus* and *N.ater*) in South Australia.

Tiger snake populations in Southern Australia were isolated 6-10,000 years ago by eustatic rises in sea levels (RAWLINSON, 1974). Populations on islands more distant from adjacent mainlands probably were isolated earlier than those more proximal to the (present) mainland. Clusters of small islands (archipelagos) were formed later from larger isolated areas, as sea levels rose. Prior to this last inter-glacial period, sea levels were lower and tiger snakes probably formed a more or less continuous series of interbreeding populations, from Southern Queensland to near Perth, Western Australia, along the now flooded continental shelf (SCHWANER, 1985). SCHWANER (1985, 1986) and SCHWANER and SARRE (in press) suggested that all populations of tiger snakes in Australia are conspecific (one species), and that variation in their colour patterns, scutellation and body proportions is explicable by the recent fragmentation of a broad ranging species into small, isolated areas where subsequent selection and genetic drift

accentuated ontogenetic and geographic variation already present in local populations.

These islands and their snake populations provide a useful model to study changes in venom components after isolation. Inter-island comparisons of physical and biological variables with variation in proteins of whole venom may help to explain the processes that best account for those changes.

MATERIALS AND METHODS

Localities studied

Venom was examined from *N.ater niger* specimens from each of the following localities in South Australia (Figure 1 clockwise from the top): Partney, Reevesby, Roxby and Hareby Islands in the Sir Joseph Banks Group, Spencer Gulf; Islet 475 in Pelican Lagoon, Kangaroo Island; Grindle and Hopkins Islands in the Thorny Passage area of lower Spencer Gulf; Williams Island at the southern tip of Eyre Peninsula; Coffin Bay (mainland) southwestern tip of Eyre Peninsula; Franklin, Goat and St Peter Islands in the Nuyts Archipelago, West Coast of Eyre Peninsula. The pooled venom from *N.scutatus* was collected from specimens near Lake Alexandrina at the mouth of the River Murray. Only adult snakes (>800 mm snout to vent length) were milked for venom, except those from Roxby Island where adults average 700-800 mm in body length (SCHWANER, 1985).

Treatment of whole venom

The venom on collection was frozen in liquid nitrogen and then maintained at -76°C in the laboratory. Upon thawing, the venoms were adjusted to a concentration of 1mg of protein per ml in 0.15 M NaCl/0.05 M NaOAc (pH 5.8), as determined by the Bio-Rad protein estimation kit (Bio-Rad Laboratories, Richmond, CA).

Gel filtration chromatography

Whole venoms (200 ul of a 1 mg/ml solution) were applied to an LKB G 3000 SW column (8 x 300 mm). A flow rate of 0.5 ml/min was used and the eluent was 0.15 M NaCl/0.05 M NaOAc (pH 5.8). Conditions were maintained by the LKB Gti Ultrochrom system.

SDS-PAGE

Electrophoresis of the whole venom samples was performed essentially according to the method of LAEMMLI (1970) with a 4% stacking gel and a 15% separating gel; 25 ul of native venom was loaded into the wells and 140 V applied until the dye marker (bromophenol blue) was within 2 cm of the gel bottom. The gels were stained with a 0.125% Coomassie Blue R250 (Bio-Rad Laboratories).

Data analysis

Due to the partial concealment of some peaks in the gel filtration elution profiles, only SDS-PAGE banding was subjected to full data analysis. The whole venoms were run side-by-side in SDS-PAGE and a total of twenty individual bands were identified from the thirteen populations. Within each population the presence or absence of each of the twenty bands was recorded and each band was taken as a character for that population (Table 1).

A population versus character state matrix was used to ordinate localities using a principal components analysis (Table 2 and Figure 3). A triangular matrix of dissimilarities between all possible pairs of populations was calculated using the "city block" measure (DUNN and EVERITT, 1982). The matrix is then manipulated by the algorithm of principal components to extract a set of orthogonal vectors (or axes) that best explain variation in banding patterns among the populations. When only the first two (or three) principal components vectors are used to represent the data, the populations can be plotted on a graph (Figure 3). The relative positions of the populations in the graphical representation are an approximation of their relative similarity with respect to their banding patterns. That is,

the Euclidean distance between any two populations in the principal components space is an approximation of the Euclidean distance between the corresponding points in the original space (DUNN and EVERITT, 1982). The analysis is phenetic; it ordinates data according to similarity and makes no assumptions about the genealogical relationships of the populations.

A minimum spanning tree (algorithm of FARRIS, 1970) computed from a matrix of pairwise differences in bands between populations (Table 3) was fitted to the localities plotted on the two principal component axes (Figure 3). This method assesses how well ordination of the populations, in terms of their banding pattern similarities, is preserved on a two-dimensional map produced by the principal components analysis described above (DUNN and EVERITT, 1982). The algorithm seeks to construct a tree of minimum overall length that connects a set of straight-line segments joining the populations such that no closed loops occur, each population co-ordinate is visited by at least one line, and there are connections between any pair of points.

A comparison between the pairwise differences in banding patterns among populations (Table 3) and the branch lengths between the corresponding pairs of localities in Figure 3, is known as the cophenetic correlation, and is another measure of how well the original pairwise differences are preserved by the tree. The analysis groups populations by parsimony; that is, a particular set of groupings is based on a rationalisation of the minimal numbers of changes required to arrive at the shortest tree. In this sense the analysis assumes that the groupings are genealogical.

Prey types available to adult snakes at the various localities are depicted in Figure 3. Pairwise differences in banding patterns between populations

(Table 3) were correlated with their respective straight-line distances (in kilometres) and sea depths (in metres) between localities (Table 4).

RESULTS

Gel filtration chromatography

The geographic distribution of the populations examined and the elution profiles obtained from the venoms of the snakes in each area are shown in Figure 1. Appraisal of like-eluting gel filtration peaks grouped the following areas as producing similar profiles: (1) the Sir Joseph Banks group of islands; (2) Grindle and Hopkins Islands in the Thorny Passage area of lower Spencer Gulf; and (3) a group containing Coffin Bay, St Peter and Goat Islands. Franklin Island (in close proximity to St Peter and Goat Islands), Williams Island (in close proximity to Grindle and Hopkins Islands), Kangaroo Island and Lake Alexandrina produced distinctly exclusive elution profiles.

One peak, eluting at the lowest molecular weight, was common to all venoms, while two others were present in all venoms but those from Kangaroo Island and *N.scutatus* at Lake Alexandrina.

SDS-PAGE

Electrophoresis of whole venom resolved a total of 20 protein bands (Figure 2), distributed variously among the thirteen populations sampled (Table 1). Only two bands were common to all populations, although four others were found in populations in Spencer Gulf or at localities on or adjacent to Eyre Peninsula. Groups of populations with the same (or very similar) banding patterns included (1) islands of the Sir Joseph Banks Group; (2) islands in the Thorny Passage; and (3) mainland Coffin Bay and the islands immediately adjacent to Eyre Peninsula (St Peter and Goat). Venoms from Franklin Island, Kangaroo Island and Lake Alexandrina were least similar to

these groups or to each other, paralleling the results obtained by a rudimentary assessment of the gel filtration elution profiles.

Three significant principal components vectors explained 95% of the variation in banding patterns among populations (Table 2). The factor scores for PCI contrasted the three most distant populations (West Franklin Island, Kangaroo Island and Lake Alexandrina, with low scores) from the rest (with high scores). PCII contrasted populations on or adjacent to western and southern Eyre Peninsula, and Lake Alexandrina to the east (low scores), with those of Spencer Gulf and Thorny Passage (high scores). PCIII contrasted Kangaroo Island, Franklin Island and the populations on or near western Eyre Peninsula (low scores) with Lake Alexandrina and the islands in Spencer Gulf and Thorny Passage (high scores). Thus each component ordinated populations into three groups: (1) Spencer Gulf; (2) the southern and western Coast of Eyre Peninsula; and (3) West Franklin Island, Kangaroo Island and Lake Alexandrina, respectively.

A plot of the first two principal components axes (Figure 3) accounted for 86% of the variation among populations in protein banding pattern. West Franklin Island, Lake Alexandrina and Kangaroo Island are well separated from each other and from the other populations, with Kangaroo Island occupying a central position between points representing populations to the north, east and west. A distinct cluster, comprising the islands of Spencer Gulf, can be subdivided into a Sir Joseph Banks Islands group and a group in Thorny Passage. Coffin Bay and islands adjacent to Eyre Peninsula also form a cluster, but Williams Island is a distinct member of that group.

The minimum spanning tree (Figure 3) constructed from the matrix of pairwise differences in number of protein bands has a total length of 21

units. The cophenetic correlation between the original input matrix of pairwise differences (Table 3) and the computed branch lengths between populations (Figure 3) is extremely high ($r = .98$, $n = 24$, $p < .001$). Branch lengths between populations and pairwise, straight-lined distances between points in multi-dimensional (principal components) space (Figure 3) also show close concordance ($r = .93$, $n = 78$, $p < .001$).

Prey available to adult tiger snakes in many localities are limited to a single type (SCHWANER, 1985) which varies among islands. The distributions of prey types among localities (Figure 3) show no correspondence to similarity in components of whole venom among populations.

The correlation between pairwise differences in numbers of protein bands among populations (Table 3) and straight-line distances (in km) between these localities (Table 4) is significant ($p < .01$) but explains only 27 percent of the variance in protein banding patterns. However, a correlation between differences in numbers of bands and minimum depth of seas separating these populations (Table 4) is also significant ($p < .01$) and accounts for 61 percent of the variance between localities.

DISCUSSION

Snake venom is a complex mixture of proteins. SDS-PAGE (and gel filtration chromatography) separates this mixture into bands (peaks) of similar protein molecules, each of which is the product of one or more genes (McFARLAND *et al.*, 1979). The presence or absence of particular proteins in individual samples of whole venom can be attributed to differences in genes encoding these proteins. Furthermore, when the genetic differences are between samples from different localities, these may be explained by various processes that determine gene frequencies in populations (LEWONTIN, 1978).

Low intrapopulational and higher interpopulational variance in gene frequencies is characteristic of genetic drift in conspecific populations (HEDRICK, 1983) and this is the pattern for venom proteins in tiger snakes examined in this study. Genetic drift would not be an important factor in widespread populations due to gene flow and greater numbers of individuals in populations, both of which tend to preserve variation and buffer changes in gene frequencies.

We have previously demonstrated the absence of intrapopulational variation in venom components of tiger snakes from Roxby Island (WILLIAMS and WHITE, 1987) in contrast to within-litter variation noted by CHIPPAUX *et al.* (1982) and MEBS and KORNALIK (1984) with gaboon vipers (*Bitis gabonica*) and eastern diamond back rattlesnakes (*Crotalus adamanteus*). Interpopulational variation in venom components of whole venom from different localities was not related to their prey (Figure 3). A positive correlation would be expected only if all the genes encoding venom components were selected for

killing a particular type of prey. The absence of a correlation, however, does not exclude selection for genes encoding particular venom components.

Collectively, protein components in whole venom of tiger snakes vary according to distance between localities sampled and time of isolation of populations (as implied by the maximum depths of the sea between localities [Table 4]). Intraspecific variation in venom components for the jumping viper *Bothrops nummifera* has also been shown to be associated with geographic location, but independent of prey, climate, season and age (JIMENEZ-PORRAS, 1964).

Distance of separation would appear to be a minor contributing factor to component variation however, as conservation of venom components over 300 km is noted between samples from Coffin Bay and Goat and St Peter Islands. GREGORY-DWYER *et al.* (1986) reported a similar finding for the western diamond back rattlesnake, *Crotalus atrox* and MINTON and WEINSTEIN (1986) also found banding patterns to be conserved in *C. atrox* in all but one region, from Southern Texas into areas of Southern Oklahoma.

The over-riding effect of isolation, however, is to alter gene frequencies and precipitate divergence among populations. In particular the time of that isolation appears to explain the largest amount of variation, and this factor suggests that the genes responsible for these proteins were influenced by genetic drift. Having shown conservation over distance, in contrast to this Franklin Island, which is only 18 km from St Peter and Goat Islands, differs in 12 protein bands (Table 2). However the time of isolation of this island as inferred by maximum sea depth (40 m) (Table 4) is far longer than the length of isolation of St Peter and Goat Islands from the mainland (i.e. Coffin Bay 5 m) or indeed the isolation of the Sir Joseph Banks group of islands from one another (2-10 m) where like-profiles

are obtained. Thus the effect of isolation and consequent genetic drift within these populations accounts for the variation noted in banding patterns.

The anomaly noted by MINTON and WEINSTEIN (1986) in the variability of venom components in *C. atrox* from Southern Texas and the Rio Grande valley may be answered in terms of isolation, analogous to the tiger snakes. Although the populations are in close proximity, a physical barrier may isolate them into separate breeding populations, with resultant divergence of venom components, however no indication was given of this in the above paper.

ACKNOWLEDGEMENTS

The authors thank Dr K. Cheney for his continued support of this work, B. Miller and S. Sarre for assistance in the field and Mrs O. Lammas for typing the manuscript.

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LEGEND FOR FIGURES

Figure 1 Map of South Australia showing the location, and the gel filtration elution profile of each population studied.

Figure 2 SDS-PAGE of the native whole venoms.
Lanes 1 and 14, *N.scutatus* Lake Alexandrina; 2, Roxby I.; 3, Partney I.; 4, Reevesby I.; 5, Hareby I.; 6, Grindle I.; 7, Hopkins I.; 8, Williams I.; 9, (mainland) Coffin Bay; 10, St Peter I.; 11, Goat I.; 12, Franklin I.; 13, Islet 475, Pelican Lagoon, Kangaroo Island.

Figure 3 Diagram of results of principal components and minimum spanning tree analyses, and comparisons of prey types for points representing populations studied. PC I and PC II are the first two principal components axes upon which points A-M are projected by the scores in Table 2. A = Franklin I.; B = Lake Alexandrina; C = Islet 475 (Pelican Lagoon, Kangaroo Island); D = Williams I.; E-F-G = Goat I., St Peter I., Coffin Bay, respectively; H-I-J = Reevesby I., Partney I., Hareby I., respectively; K = Roxby I.; L = Hopkins I.; M = Grindle I. Lines between points (locations) are branches of the minimum spanning tree based on the data in Table 3, and numbers are relative branch lengths.

able 1

DS-PAGE of the whole venom from each of thirteen locations (Figure 2) produced a total f 20 individual protein bands. The protein banding pattern for each locality with espect to these 20 bands was determined. The presence of a band = 1, absence = 0. rincipal component analysis was then applied to this data (Table 2).

	Bands																			
opulations	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
oxby	0	0	0	1	0	1	1	0	0	1	0	1	1	0	1	0	0	0	0	1
artney, Reevesby, areby	0	0	0	1	0	0	1	0	0	1	0	1	1	0	1	0	0	0	0	1
rindle, Hopkins	0	0	0	1	0	0	1	0	0	1	0	1	1	0	1	1	0	0	0	1
illiams	0	0	1	1	0	1	0	0	0	1	0	0	1	0	1	0	0	0	1	1
offin Bay, St Peter, oat	1	0	1	1	1	0	0	0	0	1	0	1	1	0	1	0	0	0	1	1
ranklin	0	0	0	1	0	0	0	1	0	0	1	0	0	1	0	0	0	1	0	1
I	0	0	0	1	0	0	0	0	1	0	0	1	0	0	1	0	0	0	0	1
ake Alexandrina	0	1	0	1	0	0	0	0	0	1	0	0	0	1	0	0	1	0	0	1

able 2

nalysis of the banding patterns obtained from Table 1 by principal components. The rincipal component scores calculated from that data, position points (i.e. opulations) along three orthogonal axes, the first two of which are presented in igure 3. The cumulative variance refers to the amount of variation among banding atterns of the thirteen populations explained by the analysis.

	Scores		
opulations	I	II	III
oxby	.032	.110	.050
artney, Reevesby, areby	.009	.069	.000
rindle, Hopkins	.027	.140	.008
illiams	.074	-.124	.091
offin Bay, St Peter, oat	.194	-.107	-.026
ranklin	-.397	-.103	-.074
I	-.103	.021	-.141
ake Alexandrina	-.270	-.072	.138
um. Variance	.628	.855	.954

able 3

atrix of pairwise differences in protein banding patterns. Each number in the matrix s the difference in bands between the respective populations determined by a ide-by-side comparison of SDS-PAGE results (Table 1, Figure 2). The minimum spanning ree incorporated in Figure 3 was calculated from these dissimilarities.

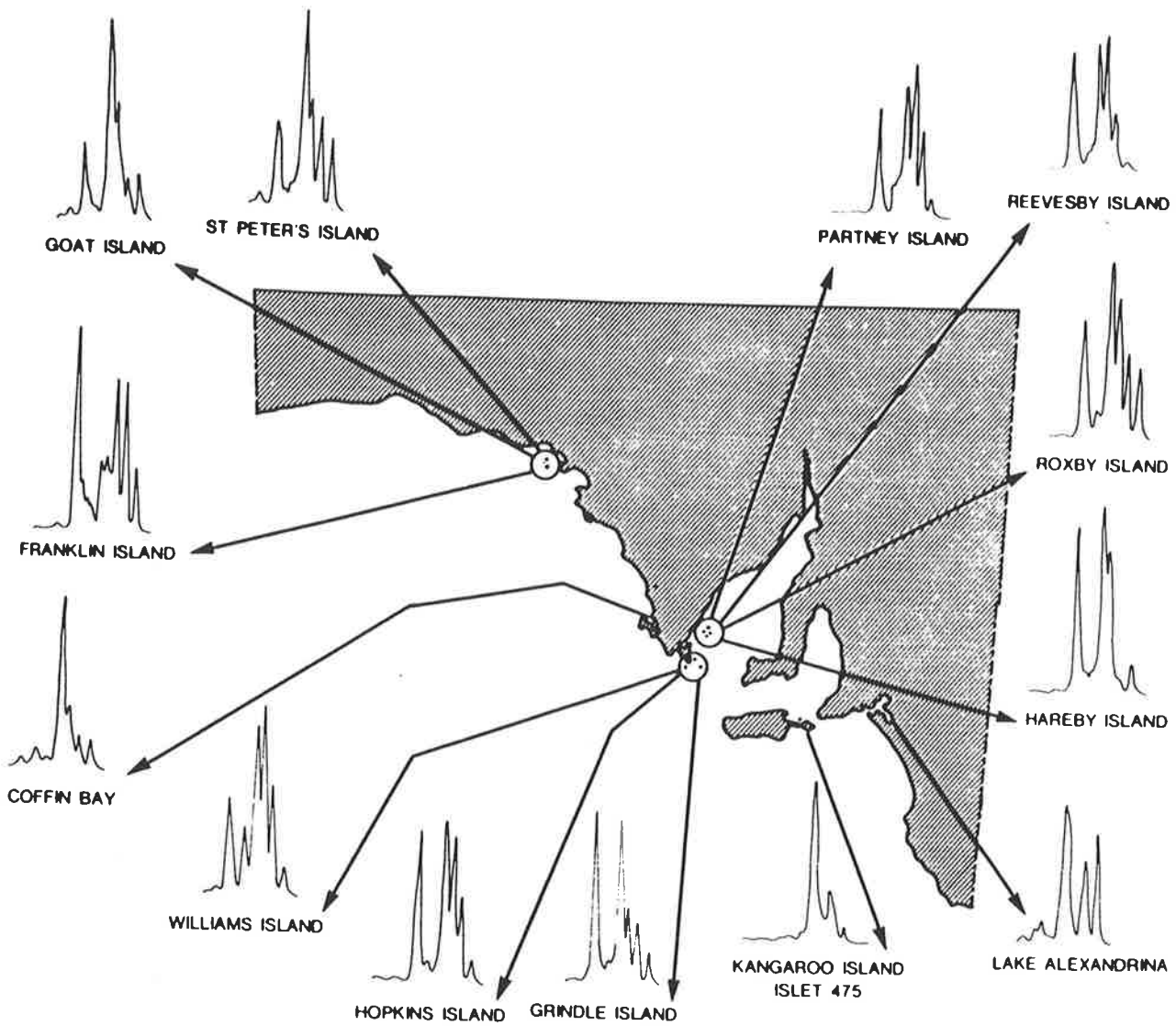
opulations	1	2	3	4	5	6	7	8	9	10	11	12	13
Roxby	-												
Partney	1	-											
Reevesby	1	0	-										
Hareby	1	0	0	-									
Grindle	2	1	1	1	-								
Hopkins	2	1	1	1	0	-							
Williams	4	5	5	5	6	6	-						
Coffin Bay	6	5	5	5	6	6	4	-					
St Peter	6	5	5	5	6	6	4	0	-				
0 Goat	6	5	5	5	6	6	4	0	0	-			
1 Franklin	10	9	9	9	10	10	10	12	12	12	-		
2 KI	5	4	4	4	5	5	7	7	7	7	7	-	
3 Lake Alexandrina	8	7	7	7	8	8	8	10	10	10	6	7	-

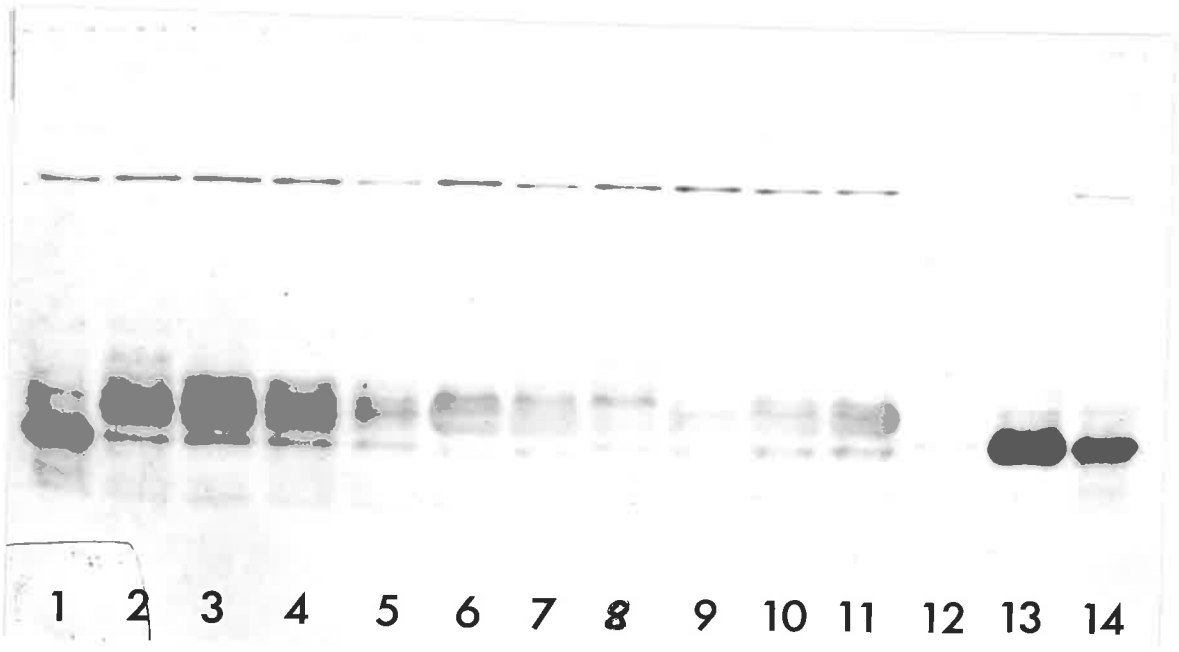
able 4

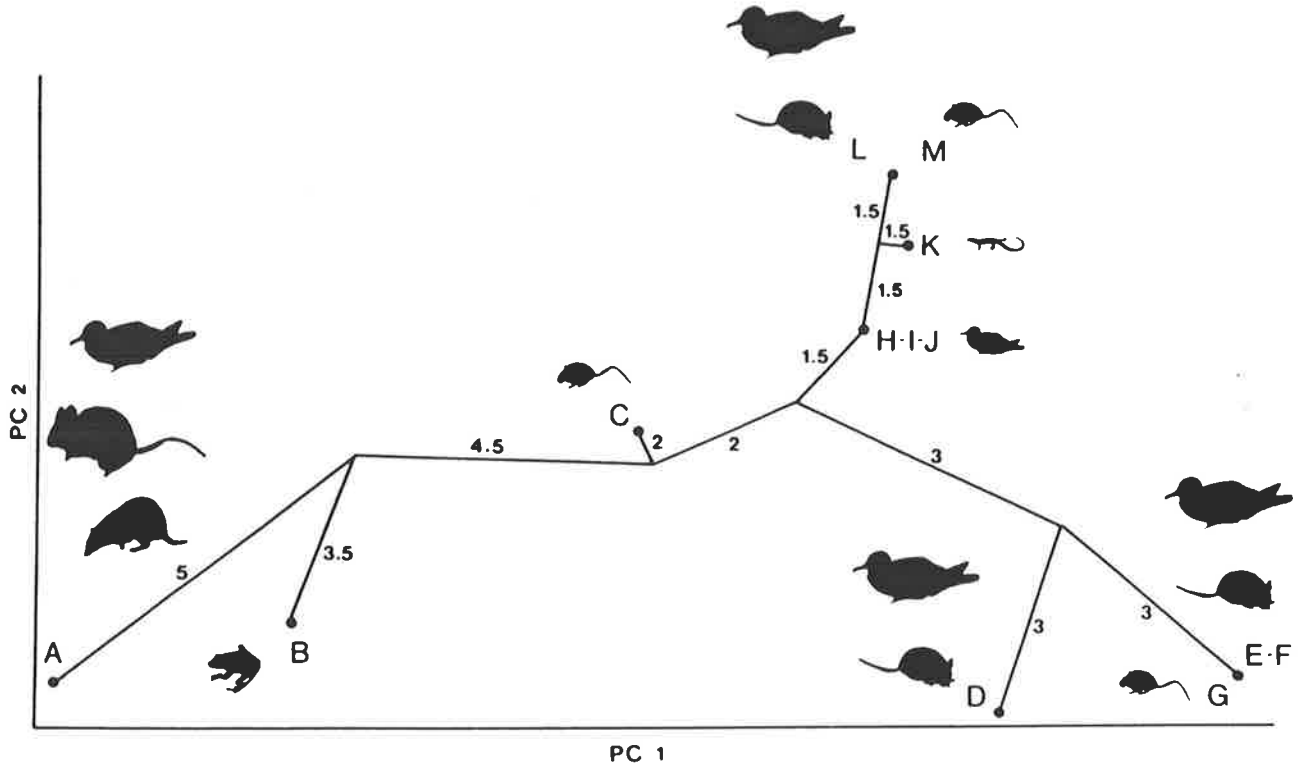
atrices of pairwise, straight line distances (in km) (lower matrix) and maximum sea epths (in m) (upper matrix) between the populations studied.

opulations	1	2	3	4	5	6	7	8	9	10	11	12	13
Roxby	-	10	10	5	17	17	27	27	27	27	40	48	48
Partney	7	-	2	5	17	17	27	27	27	27	40	48	48
Reevesby	5	2	-	5	17	17	27	27	27	27	40	48	48
Hareby	2	6	3	-	17	17	27	27	27	27	40	48	48
Grindle	40	45	45	42	-	20	27	27	27	27	40	48	48
Hopkins	45	51	50	46	7	-	27	27	27	27	40	48	48
Williams	58	58	58	57	15	10	-	27	27	27	40	48	48
Coffin Bay	77	73	73	75	60	67	63	-	5	10	40	48	48
St Peter	350	343	344	346	359	366	365	302	-	5	40	48	48
0 Goat	351	346	347	354	352	369	369	307	3	-	40	48	48
1 Franklin	334	328	329	332	339	349	349	287	18	20	-	48	48
2 KI	122	124	123	121	97	92	92	149	427	429	410	-	37
3 Lake Alexandrina	266	248	258	243	256	251	254	305	554	559	540	81	-

orrelation between pairwise differences in numbers of protein bands and straight line istances (lower matrix) is significant ($p < 0.01$), but explains only 27 per cent of ariance in protein banding pattern, while 61 per cent of the variance is explained by orrelation ($p < 0.01$) between the banding and minimum sea depth separating populations.


GOAT ISLAND
ST PETER'S ISLAND
PARTNEY ISLAND
REEVESBY ISLAND
ROXBY ISLAND
FRANKLIN ISLAND
HAREBY ISLAND
COFFIN BAY
WILLIAMS ISLAND
HOPKINS ISLAND
GRINDLE ISLAND
KANGAROO ISLAND
ISLET 475
LAKE ALEXANDRINA


1 2 3 4 5 6 7 8 9 10 11 12 13 14



STICKNEST RAT
(Leporillus conditor)

SHORT NOSED BANDICOOT
(Isodon obesulus)

MUTTONBIRD
(Puffinus tenuirostris)

BUSH RAT
(Rattus fuscipes)

WHITE FACED STORM PETREL
(Pelagodroma marina)

MOUSE
(Mus musculus)

STRIPED SKINK
(Ctenotus uber)

FROGS

REVIEW OF CLINICAL AND PATHOLOGICAL ASPECTS OF SPIDER-BITE IN AUSTRALIA

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INTRODUCTION

Spider-bite is probably a significant cause of venom injuries world-wide, but detailed statistics on the extent of this problem are sparse. For a clinical toxinologist used to reviewing cases of suspected spider-bite, the lack of statistics on this subject is easy to understand. Few cases of spider-bite are fatal, and indeed many spider-bites may be so trivial that the victim either does not seek medical attention, or is quite adequately treated by their local doctor, thus these cases never reach the major hospitals where most epidemiology statistics are generated. Furthermore in many cases, while a spider may have been responsible for the injury, it is either not seen to bite the victim, or if seen is inadequately identified. Thus many cases of spider-bite are based on a presumption that a spider was responsible for the original injury, without any firm evidence to confirm this, let alone determine which sort of spider was involved.

It has been stated by Russell[1] and others[2] that it is vital to bring some scientific order to reporting of cases of spider-bite, and that in defining a syndrome of envenomation by a particular species of spider, case reports should be those where the spider was seen to bite, was then subseqently positively identified by a person qualified to perform such identification, and that other medical causes of the medical symptoms and signs ensuing were eliminated. This latter point is most important, as a variety of medical diseases may cause local symptoms and signs which may be mis-diagnosed as the effects of a spider-bite. Russell[1,3] and others[4] have pointed out some of the problems that may occur and cause a mis-diagnosis of Brown Recluse Spider-bite. These include erythema chronicum migrans, Stevens-Johnson syndrome, Lyell's syndrome or toxic epidermal necrolysis, erythema nodosum, erythema multiforme, chronic herpes simplex, infected herpes simplex, purpura fulminans, diabetic ulcer, bed-sore. Obviously this list is far from exhaustive, and has since been added to by Russell and others.

In assessing reports of spider-bite several questions must be posed.

1. Was an organism seen?
2. Was it actually seen to bite the victim?
3. Was it positively and correctly identified?
4. Are the local effects due to venom toxicity or to an allergic reaction, or infection, or exacerbated by pre-existing conditions in the patient, such as vascular insufficiency?

5. Are the general effects due to venom toxicity or allergy or anxiety or infection or some other medical condition?

Thus a confirmed case of arachnidism by a particular spider should fulfil the following criteria:

1. The spider must have been seen to bite the victim.
2. The spider must have been identified by an expert.
3. The local and general problems must be shown to be due to venom toxicity by exclusion of infection, allergy, anxiety, and other medical problems.

Unfortunately this excludes the vast majority of cases of probable spider-bite.

EPIDEMIOLOGY OF SPIDER-BITE

Parrish[5] collected extensive statistics on cases of envenomation in the U.S.A. He analysed fatalities ascribed to envenomation in the ten year period 1950-1959. During that time 460 such deaths were recorded, 14% due to spider-bite, 30% due to snake-bite, and 50% due to stings by hymenoptera. In this last group the vast majority were bee stings, with anaphylaxis being the cause of death rather than primary venom toxicity. Of those 14% or 65 cases of fatality following spider-bite the majority took greater than 24 hours to die (37), with only 2 dying in the first 6 hours. The majority of fatalities were either in young children 4 years or less of age (14) or in older adults 50 years or greater (34). Virtually all of these fatalities were apparently due to bites by the North American Black Widow Spider, *Latrodectus* spp.

As in North America now, Australia has a network of poisons information centres set up in the leading children's hospital in each state capital. These centres all collect statistics on both enquiries about and cases of bites and stings. In the 1983/84 period 30.6% of all such reports were due to stings by bees, wasps or hornets, and 29.9% due to bites or stings by spiders, scorpions, or ticks[6]. Within this sub-group spider-bite would account for the vast majority of all reports. During the same period snake-bite accounted for only 1.5% of cases. A further 29% of cases were due to bites or stings by an unknown organism. Thus in Australia it would appear that spider-bite represents the second largest group of presentations for medical advice or treatment following bites and stings. Of course such figures can only be approximate, as there will inevitably be an element of over-diagnosis of spider-bite in some situations, and conversely an under-diagnosis might also occur.

Fatal spider-bite in Australia is a relative rarity. There have been no confirmed fatalities following bites by the Red-back Spider *Latrodectus hasseltii* since antivenom was introduced in 1956[7]. There have been only 13 fatalities ascribed to the Sydney Funnel-web Spider *Atrax robustus* since 1926[7]. No other spiders in Australia have been confirmed as causing fatalities.

SPIDER TAXONOMY

The taxonomy of Australian spiders is far from complete, with numerous species still awaiting description, and many species groups still in the process of re-examination and re-definition. The family and genus designations used in this paper reflect those currently used by the South Australian Museum, which in turn reflects as near as possible a consensus of views of leading spider taxonomists in Australia.

In the following sections an attempt will be made to summarise information on clinical aspects of spider-bite in Australia, dealing with spiders family by family. This information is based on the author's personal experience in treating cases of confirmed or suspected spider-bite and previous major surveys of this field, which contain extensive bibliographies[8,9,10,11,12]. For a detailed review, with extensive bibliography, the work of Southcott[9] is recommended.

Sub-order *Orthognatha* - the "primitive" or mygalomorph spiders

***Hexathelidae* - Funnel-web Spiders**

This family contains the notorious Sydney Funnel-web Spider and its relatives[13]. There are two genera, *Atrax* containing *A. robustus* and two other species, and *Hadronyche* containing considerably more species, including the only other two species thought to be dangerous to man, *H. formidabilis* and *H. infensus*[8]. Little data is available on other species within this group, and it is possible that other species may also be potentially dangerous to man.

The Sydney Funnel-web Spider, *A. robustus* has a fairly narrow distribution range, largely limited to a region in and around Sydney[13]. The male spider is more dangerous than the female, but in the majority of bites no systemic envenomation occurs. However the large fangs and aggressive attack of this spider may result in intense local pain of short duration. It does not appear to cause local necrosis. Systemic envenomation may develop rapidly.

Systemic envenomation has been studied in monkeys and the following problems have been noted[8]:

1. Cardio-vascular - early hypotension, then hypertension, then insidious hypotension
2. Pulmonary oedema
3. Acute metabolic acidosis
4. Intracranial hypertension
5. Hyperthermia
6. Catecholamine excretion
7. Haemoconcentration - fluid shift to GIT
8. Rise in creatine kinase levels

In man systemic envenomation initially causes numbness around the mouth, and spasms of the tongue. This may progress to nausea, vomiting, abdominal pain, profuse sweating, brisk salivation, lacrimation, or severe dyspnoea. Confusion, or even coma, hypertension, and pulmonary oedema may then ensue. There may be severe muscle tremors and raised intracranial pressure has been suspected. In some cases there may then be a decrease in secretions and an apparent lull period before the onset of progressive hypotension culminating in irreversible cardiac arrest. In discussing the use of antivenom in the treatment of Funnel-web Spider-bites Sutherland[8] has suggested 7 criteria for systemic envenomation:

1. Muscle fasciculation remote from bite site
2. Marked salivation or lacrimation
3. Pyeloerection
4. Significant tachycardia

5. Hypotension
6. Dyspnoea
7. Disorientation, confusion, impaired consciousness

In the 13 fatal cases reported, 5 died in the first 2 hours, 1 in the next 4 hours, 2 in the period 6-12 hours, 3 in the period 12-24 hours, and 2 took more than 24 hours to die.

Recommended first aid for Funnel-web Spider-bite is the application of a pressure immobilisation bandage and splint as described by Sutherland. This should be left in place until the patient has reached a facility fully equipped to treat such patients. If systemic envenomation develops antivenom should be used, without pre-treatment with adrenaline. A more detailed discussion of the toxinology, clinical aspects of envenomation, and treatment of Sydney Funnel-web Spider-bite may be found in Sutherland 1983[8].

Dipluridae - **Wishbone and Funnel-web Type Spiders**

The best known of spiders in this group is *Dekana diversicolor*. Literature review suggests that bites by these spiders may cause local pain, oedema, erythema, but systemic problems are unlikely. The author has not seen confirmed cases of bites by these spiders.

Actinopidae - **Mouse Spiders**

The best known of this group are the typical Mouse Spiders such as *Missulena occatoria*. Adult male spiders of this group are frequently encountered as they wander in search of a mate. Literature review suggests that they probably cause mild local pain and possibly swelling only, without a systemic problem.

The author is aware of several bites by this spider, all of which have been associated with brief local pain only. However, Sutherland[8] has studied the venom of these spiders and briefly reports that it is more toxic than that of *Atrax robustus*. He therefore suggests this spider is potentially very dangerous. There are however no clinical cases to support this assertion. However as many cases of *Atrax* bite cause minor problems only, yet fatalities have occurred, some caution must be expressed about the potential danger posed by *Missulena*.

Barychelidae - **Trap-door Spiders**

This group are typified by *Idiomata blackwalli*. Most authors suggest that these spiders will cause mild local pain and swelling only, without systemic problems. The author has not seen any confirmed cases.

Theraphosidae - **Barking Spiders**

The best known of these spiders is *Selenocosmia stirlingi*. This large tarantula-like spider has only rarely been reported as biting man. Bites may apparently cause local pain, oedema, erythema, or it may be initially pain-free. Systemic problems include nausea, vomiting, headache, photophobia, rigors, malaise, and urinary frequency. The author has seen no confirmed cases.

Ctenizidae - **Trap-door Spiders**

The best known of these spiders is *Aganippe raphiduca*, found in Western Australia. Here it is said to cause severe effects, but there are no case reports to confirm this. The author's personal experience is with *A. subtristis*, the Adelaide trap-door spider, with 3 cases where the spider was seen to bite and was subsequently identified either by the author or at the South Australian Museum. All 3 cases were trivial, with either no pain or brief mild pain, an indistinct or single puncture mark, and no systemic problems. These spiders are potentially a frequent cause of spider-bite in Adelaide, as they are common in suburban gardens, and are likely to be dug up from their burrow retreat during the course of ordinary gardening procedures. It is probable that many bites from this spider occur, but are considered trivial by the victim and so not reported.

Sub-order *Labidognatha* - **the "modern" spiders**

Theriidae - **Red-back Spider**

The Red-back Spider, *Latrodectus hasseltii*, is a significant cause of spider-bite in Australia. It is a relatively common species, found in many gardens and homes throughout Australia, including inland arid regions away from man's habitation. Its significance in Australian folk lore, particularly in relation to the outhouse toilet used by many Australian homes until recent times, is typified by the song "The Red-back on the Toilet Seat". This spider is a close relative of the Black Widow spiders of North America and Europe.

There have been two reviews of Red-back Spider-bite[14,15], both based on cases reported to the Commonwealth Serum Laboratories in Melbourne. The reported local effects and general effects of envenomation by this spider are given in Tables I and II. It can be seen that the predominant finding, both locally and systemically, is pain.

Table I - LOCAL EFFECTS

	Wiener 1961 (n = 167)	Sutherland & Trinca 1978 (n = 2144)
Pain	90% (? local or general)	76%
Erythema	20% (+ weals)	33%
Oedema	18%	24%
Heat	-	19%
Pruritis	7%	4%
Lymphangitis	3%	-

Table II - GENERAL EFFECTS

	Wiener 1961 (n = 167)	Sutherland & Trinca 1978 (n = 2144)
Regional lymph node swelling or pain	4%	19%
Generalised pain	90% (?local or general)	39%
Nausea/vomiting	24%	20%
Sweating	38%	15%
Malaise	-	10%
Parasthesiae	7%	10%
Pyrexia	8%	8%
Insomnia	10%	8%
Dizziness	17%	8%
Muscular weakness	11%	-
Muscular spasm/ tremor/rigor	24%	-
Hypertension	2%	3%

Criteria useful for determining systemic envenomation include:

1. Generalised pain
2. Significant sweating, distant to bite site
3. Nausea, vomiting, headache
4. Significant malaise
5. Muscular tremor, spasm, rigor
6. Hypertension

Latrodectism may mimic other conditions, including acute abdomen, and a suggested scheme for differential diagnosis of these two conditions has been suggested[16]. The author has seen at least one case in South Australia where an initial diagnosis of acute appendicitis was made, and only after detailed questioning was a history of spider-bite obtained[17]. Treatment with specific Red-back antivenom effected a rapid and complete cure. In the author's experience a significant number of Red-back bites will not result in either significant local pain or systemic envenomation.

There is no specific first aid recommended for Red-back Spider-bites, and in particular the pressure immobilisation bandage is not recommended. The application of a local ice pack has been suggested as a useful method of reducing local pain. As symptoms of systemic envenomation may take several hours to develop it is recommended that cases be observed for a minimum of 3 hours, and if systemic envenomation does occur it is best treated with Red-back Spider antivenom given IM. If the initial diagnosis was correct a rapid and usually complete resolution of all symptoms will occur, often within 30 minutes. Where a severe bite has occurred there may be initial resolution followed by relapse, and this is an indication for a second dose of antivenom.

The Red-back Spider is well known in Australia, and is sufficiently distinctive in appearance that most victims will describe the spider that bit them with a clarity sufficient to ensure little doubt about the identity of the culprit. In addition the symptoms and signs of latrodectism are quite distinctive, and this makes it possible to accurately diagnose latrodectism in many cases even without a spider presented for formal identification. Confirmation of the diagnosis follows an appropriate response to antivenom treatment. However the author has seen several cases in adults where the pattern of local and systemic symptoms and signs is the same as classically seen in latrodectism, but where the spider was either not seen, or not seen well enough for certain identification, and where trial of antivenom therapy failed to produce any therapeutic response. It therefore seems likely that another organism, possibly a spider, is also capable of causing a syndrome akin to latrodectism.

Several other Theriid spiders may cause minor problems. *Archaearanea tepidariorum* is reported to cause mild local pain, erythema, itchiness, and mild nausea and headache.

Argiopidae - **Orb-weaving Spiders**

This family contains many species, most of them small and unlikely to cause significant injury in man. The Golden Orb-weaver Spiders, *Nephila* spp. are large spiders with large fangs and might therefore cause locally painful bites, although there are no reports of systemic problems. The common Garden Orb-weaver *Eriophora* spp. is reported to cause brief local pain only. In two cases the author has seen where the spider was positively identified, one in a child caused mild brief local pain only with local erythema, and the other in an adult caused local pain and erythema associated with headache and vomiting.

Heteropodidae - **Huntsman Spiders**

This family contains a variety of usually large spiders often found in and around houses, the most common species being in the genera *Delena, Heteropoda, Isopoda* and *Olios*. Most reports suggest bites by these spiders cause local pain only, without necrosis, and only occasionally causing systemic problems such as headache, nausea, and vomiting. In two cases of Huntsman bites seen by the author there was mild, brief local pain only, and no systemic problems. In addition the author has encountered numerous other cases where the spider described was almost certainly a Huntsman, again the bite causing mild brief local pain only.

Salticidae - **Jumping Spiders**

Most Jumping Spiders are small, and some certainly enter houses. Most reports of envenomation centre on *Mopsus mormon* which may cause local pain, oedema, discolouration, and has been suggested by one author[8] as a possible cause of severe skin lesions, though not backed up by case reports. The author has not seen any bites by these spiders.

Lycosidae - **Wolf Spiders**

These are very common ground hunting spiders found throughout Australia, and commonly in gardens in suburban areas. There are apparently no confirmed medical case reports of bites by these spiders, and opinions vary on their possible effects.

Raven suggests they cause local pain only in most cases, and occasionally a mild systemic illness. Sutherland[7,8] however believes they are the probable cause of local necrotic lesions and significant systemic problems in a series of cases seen in Victoria. The author has not seen any confirmed cases of Wolf Spider-bite.

Miturgidae - **Lined Spiders**

There are apparently no documented cases of bites by these spiders, genus *Miturga*, from Australia, although there is a case reported from New Zealand with local pain, oedema, induration, sweating, and joint pains. The author has not seen any confirmed bites by these spiders.

Clubionidae - **Sac Spiders**

The best known spiders in this group are those belonging to the genus *Chiracanthium*. While little information is available on bites by these spiders in Australia, overseas experience suggests they may cause local pain, ulceration, malaise, headache, nausea, and coma. Reported bites from Australia are non-medical reports. The author has not seen any confirmed bites by this species, and indeed these spiders seem uncommon.

Gnaphosidae

The only important member of this family in Australia appears to be the White Tipped Spider, *Lampona cylindrata*. Most authors suggest this spider can cause significant problems including local pain, swelling, local ulceration, blister formation, and in some cases systemic problems including headache, nausea, vomiting, and muscular pain. There is however a lack of carefully documented cases to justify this fearsome reputation for what is a very common spider found inside many houses. One recent case report notes a mild stinging sensation and swelling of short duration as the only effects of a definite bite by this species[18]. In only one case has the author seen a patient allegedly bitten by this spider, where the spider was definitely identified, and in this case there was local erythema only. The father of this patient reported he had been bitten several times by this spider and that the bites caused him no problem.

Dictynidae - **Black House Spiders**

The most important members of this group, in the genus *Badumna*, are commonly found around metropolitan houses and gardens. Most authors suggest that bites result in local pain which can be severe and spread in a manner similar to Latrodectism. Bites may be associated with variable systemic symptoms including sweating, nausea, vomiting, headache, giddiness, and muscular pain. There are two reports, both non-medical, of bites causing local tissue injury. The author has seen five confirmed cases, all associated with local pain, erythema, but no ulcer formation or necrosis. In three cases there were no systemic problems, and in two cases a mild systemic illness with sweating, anxiety, and chest tightness. These latter symptoms could be as easily related to anxiety about the bite as to effects of envenomation.

Loxoscelidae - **Brown Recluse Spiders**

The distribution of these spiders in Australia is still uncertain, and they may well represent an introduced fauna. The most commonly encountered is the Fiddleback Spider, *Loxosceles rufescens*. In some parts of the world *Loxosceles* spiders are known to cause a severe local reaction including local necrosis, and in some cases a severe systemic reaction known as viscerocutaneous loxoscelism associated with a haemolytic anaemia, fever, jaundice, haematuria, and prostration[19]. In South America this latter condition apparently carries a 30% mortality rate. The species responsible is *L. laeta*. Similar, though probably less severe, problems are encountered in North America, where the chief culprit is *L. reclusa*. However as Russell[1,3] and others[2,4] have pointed out the diagnosis of loxoscelism in North America may often be made on shaky evidence, and the condition is probably over-diagnosed. There are no confirmed cases of loxoscelism from Australia and the author has seen no cases where the spider has been caught following a bite.

NECROTISING ARACHNIDISM AND RELATED PROBLEMS

Sutherland has reported a number of cases of puzzling local tissue injury, sometimes associated with a systemic illness, for which no standard medical cause could be found[7,8]. It would appear that some, if not all, of these cases were associated with a bite by an unidentified organism. Sutherland has suggested this is most likely to be a spider, and while no proof of this is currently available, he has suggested that Wolf Spiders may be the most likely culprit. This is based on the finding of Wolf Spiders in abundance in the areas where some victims were bitten. From Sutherland's work there appear to be three clinical types of necrotising arachnidism seen by him in Victoria.

Type I - slowly progressive and relatively painless full thickness skin loss, not associated with systemic problems. Negative microbial cultures from the local wound.

Type II - very painful local lesion associated with redness, skin loss over 24 hours, but not in all cases, and rapid improvement of symptoms. No systemic problems.

Type III - painful local lesion with associated skin discolouration, and full thickness skin necrosis and swelling. Systemic problems include shock, prostration, severe diarrhoea, but the patient remains initially afebrile, with negative blood cultures.

The author has also seen numerous cases of significant local reaction following an apparent bite, in some cases a spider being seen to bite though unfortunately not captured for identification, and in other cases no bite being felt, no organism seen, the patient merely waking up in the morning to discover a local lesion. While in some cases the development of a local cellulitis can ultimately be traced to an infected cut or abrasion, there remain a number of cases associated with an apparent bite or sting where significant local cellulitis, and on occasion persistent ulcer formation, occurs. Many of these cases are associated with systemic problems such as a feeling of malaise, headache, or nausea. This may persist for two to three days. Local cellulitis is often slow to respond to antibiotic therapy, and only rarely is an organism successfully grown from the local wound. The majority of such cases are probably dealt with by general practitioners as cases of local cellulitis of unknown

cause, and usually result in the victim taking several days to weeks off work. In some cases the ulcers have persisted for three months, causing major distress for the patient and significant interference with work. In Adelaide alone such cases may run into the dozens each year, as many GPs the author has discussed this problem with admit to seeing one or two cases in the last twelve months.

It is beyond the scope of this paper to detail each case, and define the pattern of symptoms and signs seen. It is likely that at least some of these injuries are the result of spider-bite, and it may be that several different species of spider are involved. The injuries caused probably result in significant economic dislocation. There is no adequate treatment available for this problem at present. Progress in understanding this problem can only be made if the identity of the offending organism is known. There are two possibilities for achieving this. The first relies on chance, that is, a patient seeing the spider bite them, carefully collecting the spider, and presenting for evaluation, an event which has yet to occur! The second involves selecting a number of possible culprit species, extracting venom, and producing some form of venom detection test to try and identify the spider involved from samples from the wound site. The technical problems of this approach are obvious.

SNAKE-BITE OR SPIDER-BITE?

In reviewing many cases of suspected snake-bite the author has seen one group with fairly distinctive symptoms and signs. In these cases no organism is seen to bite the victim, but they feel a definite bite, and on examining the bitten area discover two distinct puncture marks less than 1 cm apart, associated with minimal and short-lived local pain. There is no local erythema, swelling of significance, no development of other localised reaction, and apart from the two puncture marks no other teeth marks or scratches are seen in the vicinity of the bite or sting. Snake venom is never detectable from the skin surface, the patient remains systemically well, and coagulation testing reveals no evidence of coagulopathy.

As two fang puncture marks are the classic description of snake-bite it is usually assumed by both the victim and treating medical officers that a snake-bite has occurred. In the author's experience snake-bites in Australia do not leave a pattern similar to this, and in view of the evidence it is very doubtful that these cases represent snake-bite. It is more likely they represent a bite or sting by some other organism. The close proximity of two puncture wounds in all cases suggests that the attacking organism has two fangs or stings used simultaneously, rather than an organism with a single sting stinging twice. It is therefore likely that the organism involved is a spider, but the spider must be large, and almost certainly would be an *Orthognath* spider such as one of the Trap-door Spiders. Unfortunately until a patient presents with such an injury, together with the attacking spider, this hypothesis remains unproved. Those cases of Trap-door Spider-bite seen by the author have been associated with mild local pain only, but only a single fang puncture mark was seen. The author is aware of a single case of Trap-door Spider-bite in Adelaide where the spider was definitely identified, and where the patient had two fang puncture marks similar to those described in the aforementioned cases. However, this case was not seen by the author, details are incomplete, and therefore it cannot be used as proof of this association.

SUMMARY

For medical practitioners interested in toxinology in Australia, spider-bite has proved a difficult and often unrewarding area, as only rarely is the spider seen to bite and then reliably identified, in association with detailed medical recording of any symptoms and signs. As a result most information on spider bite in Australia is anecdotal. This paper has summarised available information. It is possible that the development of venom detection tests such as the ELISA based tests for snake venom developed by Theakston and colleagues will be applied to a diagnosis of spider bite, and potentially may dramatically increase the quantity and quality of information on arachnidism available in Australia.

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Bites by the white-tailed spider, *Lampona cylindrata*

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Lampona cylindrata spiderbites

Abstract

Seven cases of definite (6) or highly probable (1) bites by the white-tailed spider, *Lampona cylindrata* are presented. In all cases local pain was of short duration, usually only mild, or itchy, and associated with local redness, with no ulcer formation, and blister formation in only one case (the only uncertain case). No cases developed local skin necrosis. In no cases were significant general symptoms noted, but 2 cases developed immediate anxiety symptoms which rapidly subsided after reassurance.

Key Words

White-tailed spider; spiderbite; *Lampona cylindrata* bites.

Introduction

Spiders are an ever present and important part of Australia's environment. Spiderbite is hardly a rare event, as doctors and others interested in this topic would know, yet published case reports of spiderbite are rare, with the exception of bites by red-back spiders and Funnel-web spiders, the only Australian spiders confirmed as potentially fatal to man.

In the authors experience few people actually see a spider bite, then catch it, have it identified, and report symptoms and problems to medical practitioners experienced in this field.

Recent publicity about spiderbite following a paper in the MJA[1,2] has caused a public perception that bites by the white-tailed spider *Lampona cylindrata* cause major skin necrosis. We here report 7 cases where a white-tailed spider was seen to bite, and the subsequent effects.

Case Reports

Case 1. An 18 year old man was bitten on the right buttock by a spider on a towel. The spider was caught and later identified as *L. cylindrata*. The bite caused a mild stinging sensation locally lasting only a few minutes, and two small prick marks were seen. The area bitten developed a small (approx. 1 cm) lump which lasted about 24 hours. The patient was "shocked and frightened" by the bite, was anxious, and noted slight nausea shortly after the bite, which subsided quickly. No further after effects were noted, and two weeks later there was no evidence of local or general problems.

Case 2. An adult woman was bitten on the left upper arm while in bed by a spider later identified as *L. cylindrata*. The spider was brushed off following the initial stinging sensation. A small red lump (approx. 1 cm) developed at the bite site, which was stinging and itchy for about ten days. In the first hour after the bite the local pain was "severe" but thereafter minor. After ten days the local lump resolved and no further problems developed. At no stage did the patient feel generally unwell; she did not experience either nausea or headaches.

Case 3. A 60 year old man was bitten on the abdomen while in bed. The spider was caught and later identified as *L. cylindrata*. The bite was not noted as painful but rather was itchy for 4 - 5 days, and a red area at the bite site (size uncertain) was noted for 10 days. No general symptoms occurred.

Case 4. A 14 year old girl was bitten on the throat while taking a bath. The spider was caught and later identified as *L. cylindrata*. The bite was not associated with significant pain, but some local swelling developed which resolved by the next day. The patient noted that it was painful to swallow food that night. While 2 puncture marks were seen, no local redness was noted. The patient felt cold in the arms and legs that night, but was otherwise well.

Case 5. An adult woman was bitten on the buttock when sitting down on a stool. The spider was caught and later identified as *L. cylindrata*. The patient noted sharp instant pain, of short duration, and a small area of redness was seen at the bite site. Both pain and redness had disappeared within one hour. Immediately following the bite the patient described anxiety, fearing the spider was a funnel-web, with associated shaking, sweating, cold sensations and aches and pains. All subsided immediately on reassurance that the spider was not a funnel-web spider.

Case 6. A one year old boy was bitten by a spider which was captured by the child's father, and later identified as *L. cylindrata*. Details on this case are scant but it appears that 3 separate red raised lesions were seen around the bite area, but no other lesions were seen or developed, the child was not upset, and no general symptoms occurred. The child's father commented that he had been bitten by the same sort of spider on several occasions, never suffering more than local itchiness.

Case 7. A woman reported a bite to her child by a spider, the description of which was consistent with *L. cylindrata*, though

no spider was available for identification. Details of this case are scant and attempts to gain further information have been unsuccessful. The child was apparently perfectly well generally, but did develop a small blister at the bite site which settled within three hours leaving only a small area of redness.

Discussion

In reporting the effects of spiderbite in man it has been suggested that only when a case report fulfils the following criteria, can it be said with certainty that the reported effects are due to spider venom; (a) A spider was seen to bite the patient, (b) the spider was caught and identified by an expert, (c) the ensuing symptoms, signs, problems were not due to any other cause such as infection, allergy, anxiety, vascular problems etc.[3,4] Unfortunately, only very rarely are such conditions met. In North America problems associated with reporting presumed spider bites speculatively linked with a particular species of spider, usually *Loxosceles reclusa*, have occasioned comment and controversy.[4,5,6,7] This is particularly true of the local necrotic lesions often, and possibly quite erroneously ascribed to *L. reclusa*. In this context it has had medico-legal implications.[6,8]

In the cases reported in this paper, enough of the conditions mentioned above were met to justify their classification as reports of the effect of bites to man by *Lampona cylindrata*. While our series is larger than any previously published, it nevertheless represents only a few cases, and should not be taken as representative of all bites by this spider. It does demonstrate that bites by *L. cylindrata* may often be associated with relatively minor local and/or general effects. The severe skin necrosis reported by Spring[1] and by Sutherland[2,11] which were speculatively blamed on spiderbite, with *L. cylindrata* a prime suspect, do not fit the criteria listed above for defining the effects of spiderbite. The authors are not aware of any case reports of bites by *L. cylindrata* where significant local damage at the bite site was documented.

A review of the literature does not provide much positive evidence on the effects of *L. cylindrata* bites. Sheppard reports a confirmed bite with a

local mild stinging sensation and swelling of short duration as the only effects.[9] Musgrave[10] reports two cases, one in a six year old boy bitten on the big toe, with associated "cold turns, itchiness, temperature, headache, and the vicinity of the bite became discoloured." In the second case, due to *L.fasciata*, an adult bitten on the ankle developed "swelling" lasting for one hour. Sutherland[11] states "*L. cylindrata* is firmly implicated in causing local burning pain followed by a variable illness. Sometimes swelling, discolouration and blistering precedes ulceration. In other cases, an itchy lump may develop which may be irritating for days. Nausea and vomiting sometimes occurs. Because of its ubiquitous nature, this spider must be under suspicion as a cause of massive skin necrosis." More recently Sutherland[2] states ".... certain spiders, such as the white-tailed spider (*Lampona cylindrata)* have been 'caught in the act' and the site of the bite has then ulcerated deeply." Raven and Gallon[12] also suggest that *L. cylindrata* bites cause "local discolouration and ulceration" and systemic symptoms, but their reference is Sutherland. Southcott[13] in his major review of arachnidism quoted only Musgrave's (non medical) reports listed earlier, and also a reference to toxicity of this species by both Mascord[14], and Main[15], who also mentioned local pain following bites.

The comments by Sutherland[2,11] are not supported by case reports, and therefore it is uncertain whether this information should be included in assessing the likely effects of bites to man by *L. cylindrata*.

The evidence of our own cases suggests that the dire consequences of bites by this spider, suggested by Sutherland[2], are only one part of the spectrum, and that in some cases, possibly the majority, bites are trivial. Certainly these spiders do not routinely cause significant local tissue injury, at least in Adelaide, where the spider is common.

Indeed, so common is this spider, that were it the culprit in cases of massive skin necrosis, it would be expected that such cases would have surfaced in Adelaide. While this is not the case, the authors are aware of minor skin lesions with ulcer formation seen in Adelaide, and possibly due to spiderbite. It may be that some or all of these cases are due to *L. cylindrata* bites, but there is presently no evidence to substantiate this.

Acknowledgements: The authors gratefully acknowledge their colleagues who have assisted in collection of case records and allowed access to records, especially Dr. D. Lee (S.A. Museum); Mr. K. Coulthard and staff of the P.I.C. (ACH). The Photography Department, ACH produced the illustration from an original supplied by Julian White. Janet Bradford typed the manuscript.

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Case reports of spider bites in South Australia, excluding bites by the red-back spider.

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Case reports of spider bites in South Australia,

excluding bites by the red-back spider.

Abstract

23 cases of spiderbite, where the spider was positively identified, are presented. In no case was significant local injury such as ulcer formation or necrosis noted. Bites by *Aganippe subtristis* (Adelaide trapdoor spider) (3) caused local pain without general symptoms. Bites by *Badumna insignis* (Black house spider) (5) caused local pain, sometimes severe, usually without general symptoms. Huntsman spiders caused local pain (*Isopeda* ,3) occasionally with general symptoms including vomiting (*Olios*, 1). *Eriophora* (3) and *Phonognatha* (1) caused local pain and redness. A single wolf spider bite caused sharp brief local pain only.

Key Words: Spiderbite, *Aganippe*, *Badumna*, Huntsman, Wolf Spider

Case reports of spider bites in South Australia, excluding bites by the red-back spider.

Introduction

While spiders are an ubiquitous part of our environment, and doubtless many people are bitten each year, mostly with trivial results; published case reports of spiderbite are few. Following an association with the Poisons Information Centre, Adelaide Children's Hospital and the South Australian Museum (SAM), the senior author has been consulted on many occasions about suspected spider bites. On only a few occasions is a spider seen to bite, and then made available for identification. This paper documents those latter cases, including case reports given to SAM, in each of which the spider was sent to the authors for identification. In many cases the authors were not able to personally see the patient at the time of presentation.

Case reports of spider bites in South Australia,

excluding bites by the red-back spider.

Case Reports

Mygalomorph spiders, including trapdoor spiders.

Case 1. *Hadronyche adelaidensis* HEXATHELIDAE

A 44 year old woman was bitten on her left ring finger while gardening, turning over soil, and therefore accidentally excavating the vertical tube nest of this spider. While a single puncture mark was seen, the bite was not described as painful, and no general symptoms developed.

Case 2. *Misgolas andrewsi* IDIOPIDAE

A 25 year old man was bitten on the right index finger while gardening. No definite puncture mark was noted but there was slight redness and swelling, and local pain of short duration. No general symptoms were noted.

Case 3. *Aganippe subtristis* IDIOPIDAE

A 23 year old man was bitten on the left forearm while gardening. No definite puncture marks were seen, and no redness was noted, but the bite was locally painful for a short period. The patient was anxious about the bite, and described short-lived mild nausea, dizziness and dry throat.

Case 4. *Aganippe subtristis*

An adult woman was bitten on the hand while gardening, pulling up roots. Two puncture marks were noted, and the hand was sore for one day. Some induration was noted around the bite site. No general symptoms developed.

Case 5. *Aganippe subtristis*

A 50 year old man was bitten on his right middle finger while gardening, digging up couch grass. He was not aware of a painful bite but noted the spider clinging to his finger, and on inspection there were three small areas of blanching on the finger. No pain or other local problems developed, and there were no general symptoms.

Case 6. Black house spider *(Badumna insignis)* AMAUROBIIDAE

A 24 year old man was bitten on the finger and experienced intense local pain, spreading to the whole hand, then arm, and tenderness in the right axilla. There was no local erythema or oedema noted. The patient was not noted as suffering general symptoms.

Case 7. *B. insignis*

A 27 year old female was bitten on the right hand with consequent local pain and swelling and development of chest pain and shortness of breath which settled over a few hours. No ulceration or necrosis locally was noted.

Case 8. *B. insignis*

A 25 year old man was bitten on the finger tip with consequent moderate to severe local pain lasting about one hour, not associated with other local signs, but the patient was noted as anxious and sweaty.

Case 9. *B. insignis*

A 21 year old man was bitten on the finger with consequent local pain which settled in about one hour. A single prick was noted locally without erythema or swelling. No general symptoms were noted.

Case 10. *B. insignis*

A one year old girl was bitten on the left ankle, with apparently little effect. On arrival at a hospital the child was noted to be generally well and happy, with a small (approx. 1 cm) area of erythema about the bite site. No general symptoms were noted. Local pain, if it occurred was not noted.

Huntsman Spiders

Case 11. *Olios calligaster*

A 28 year old woman was bitten on her right second toe while putting a shoe on (the spider having secreted itself in the shoe). She noted initial pain, not severe, which apparently subsided, but about 30 - 40 minutes later she felt faint and vomited. She was noted to have a BP $^{120}/_{70}$ and pulse 76, regular, at this time, was given IM "Phenergan" and IV "Maxolon", and improved to normal over the next 4 hours.

Case 12. *Isopeda* sp.

A 10 year old boy was bitten on the tip of the left index finger, experiencing an initial stinging sensation, then no further local or general reactions.

Case 13. *Isopeda pessleri*

A 2 year old boy was bitten on the sole of the foot when putting

on gum boots. By the time the child was seen at a hospital there was a small area of local erythema noted, and the child was systemically well, and remained so.

Case 14. *Isopeda pessleri*

A 55 year old woman was bitten on the right middle finger while cleaning a rubbish bin. Local pain developed immediately, lasting 30 minutes, without local swelling or redness. The pain was described as feeling like needles. No general symptoms developed at that time, however, the patient noted that 2 days later she experienced nausea, malaise, and cold hands and feet, which lasted one day. No problems were noted at the bite site at this time.

Orb-weaving Spiders ARANEIDAE

Case 15. *Eriophora* sp (probably *biapicata)*

A 26 year old woman was bitten with subsequent local pain, slight bleeding from wound, erythema, and later headache and vomiting.

Case 16. *Eriophora* sp (probably *biapicata)*

A one year old girl was bitten on the left shoulder, with subsequent mild local pain and erythema, of short duration, and no noted general symptoms.

Case 17. *Eriophora biapicata*

An adult woman was bitten with resultant local pain and erythema (1 cm) lasting 1 - 2 hours, associated with local tingling, but no general symptoms.

Case 18. *Phonognatha graeffei*

A 2 year old child was bitten with no significant local or general

reaction noted although 2 small puncture wounds were seen at the bite site.

Jumping Spiders, SALTICIDAE

Case 19. *Holoplatys* sp.

A 67 year old woman was bitten on the left hand with consequent local stinging, burning, intense itching, local swelling and redness, and general symptoms of headache, and vomiting. Administration of adrenaline (route uncertain) by the LMO caused rapid cessation of all symptoms. The patient had previously had allergic reactions to hopper ant stings (*Myrmecia pilosula)*, and had undergone a course of desensitization for these insects. The reaction to this spider bite was apparently similar to previous reactions to hopper ant stings.

Case 20. *Breda jovialis*

A 9 year old boy was bitten on the right hand, with consequent mild local pain, raised wheal, and localized sweating. No general symptoms were noted.

Case 21. Genus uncertain.

An adult woman was bitten on the leg with immediate sharp pain, described as similar to a bull ant sting, lasting only a short (unspecified) time, but subsequently itchy, with a small area of local redness. No general symptoms were noted.

Wolf Spiders LYCOSIDAE

Case 22. *Lycosa ?godeffroyi*

An adult male was bitten on the right index finger with consequent sharp local pain which subsided in a few minutes. There was no

local swelling, redness, blistering, or ulceration, and no general symptoms were noted.

Combfoot (Theriid) spiders THERIDIIDAE

Case 23. *Achaearanea tepidariorum*

A 25 year old man was bitten on the right wrist, with no local or general reaction noted.

Discussion

The cases reported here represent only a small proportion of the spiderbites about which the authors have been consulted, but in the other cases, the spider was not available for definite identification. Many spiders are not easy to confidently identify from verbal descriptions by non-experts, but in some cases the description is sufficient for identification. The following discussion will therefore draw on the senior author's personal experience with such cases, plus reports in the literature.

Bites by the red-back spider, although accounting for a significant number of all spiderbite enquiries will not be discussed here as they have been well defined elsewhere.[1,2,3]

Mygalomorph (Orthognath) spiderbites do not account for many spiderbite enquiries. In Adelaide most such bites are due to the Adelaide trapdoor spider, *Aganippe substristis*, a common spider frequently dug up from its burrow by gardeners. The three reports listed here were all associated with quite minor problems, especially considering the size of this spiders fangs, often 5mm or more in length (in comparison common brown snake fangs on adult snakes average only 2.8 mm in length). Experience with other probable *A. subtristis* bites suggests that most and probably all bites by this species are relatively minor with mild short-lived local pain only as the usual consequence. The authors are unaware of any cases where this spider was implicated, which resulted in significant local tissue injury.

The closely related *A. raphiduca* from Western Australia is said to cause severe effects[4], although case reports to give substance to this comment are lacking.

The two other mygalomorph spiders with case reports included here are superficially similar in appearance to *A. subtristis*, and the effects of their bites, on the very limited evidence provided here, appear to be minor and local only.

Another commonly encountered and visually distinctive mygalomorph is the male mouse spider *Missulena insigne*, and related species. While the senior author is aware of several probable bites by these spiders, all of which were associated with brief local pain only, in no case was it possible to confirm with a properly identified spider. This is unfortunate as Sutherland has studied the venom of *Missulena* and found it more toxic than even *Atrax robustus* venom. In consequence he has suggested that bites by *Missulena* spp should be treated with great caution[3]. There are no medical reports of significant illness caused by the spider, but a non-medical report is certainly suggestive of serious envenomation, possibly responsive to Funnel-web spider antivenom[5]. A detailed report of this case published in a medical journal should prove most interesting.

Of the many araneomorph (Labidognatha) spiders, several are commonly in contact with man. The black house spider, *Badumna insignis* has enjoyed a reputation as causing significant bites associated with both severe local pain, possibly ulceration, and also systemic effects including sweating, nausea, vomiting, headache, giddiness, and muscular pain[3,5,6,7,8,9]. The five cases noted here were mostly associated with local pain of variable severity, but local ulceration was not seen, nor were general symptoms common or prominent. Experience with other probable cases of *B. insignis* bites suggests that local pain, only occasionally severe, is the most common consequence, and that local ulceration if it does occur, is certainly not common. The authors have no records of cases where

ulceration occurred in association with a probable bite by this spider, but know of one possible case where a local blister was initially noted.

Huntsman spiders, like *B. insignis*, are commonly found round and even in man's dwellings. The common genera are *Delena*, *Heteropoda*, *Isopeda*, and *Olios*. *Heteropoda* and *Olios* are said to cause both severe local pain, and general symptoms including headache and vomiting, at least in some cases[3,4,5,6,7]. The authors' experience has been that most probable huntsman bites have been associated with minor short-lived local pain only, without general symptoms. The four cases documented here mostly fit this pattern. Case 11, with some general symptoms, was bitten by *Olios calligaster*. The contribution of anxiety to the general symptoms in this case is hard to assess.

The garden orb-weaving spiders, *Eriophora* spp, are commonly encountered on summer nights when the incautious person ventures into the garden without a torch. The literature suggests that these spiders cause mild local pain only[4,6,7], which is confirmed by the authors experience with probable cases, and cases 16 and 17. Information collected on case 15 is scant.

Bites from jumping spiders (Salticidae) have not been well documented, but Musgrave[6] quotes a single case in an adult man, bitten by *Mopsus mormon* which resulted in "painful swelling with local discolouration", and Sutherland[3] notes that an un-named salticid in Melbourne produces "a painful lesion which may be uncomfortable for a week." Our three cases confirm that bites from these small spiders may cause local pain and redness, without ulceration and necrosis. However, the range of species is large and so few cases, though useful, can hardly be considered as necessarily representative. Case 19 is unusual and it is unfortunate that further details are not available. It would seem that the patient suffered

an allergic reaction to the bite. The relationship between the spider's venom and hopper ant venom is unknown.

Bites by wolf spiders in Australia are poorly documented, but Main[9] mentions a woman bitten by an unidentified wolf spider who suffered for weeks with a painful weeping sore at the bite site and Sutherland[3] notes there is circumstantial evidence in several cases of local necrosis that a wolf spider was the biting organism. One of these cases also developed nephrotic syndrome. Our single case shows no evidence of these problems, and Raven and Gallon[10] suggest most bites cause no reaction.

There are a number of other spiders commonly encountered by man which may cause injury, but these will not be discussed here, as the authors do not have definite case reports. Information is available in reviews of Australian spider bite[3,4,10,11].

For all spider bites there may be a wide spectrum of effects, both local and general, and the case reports given above should only be taken as indicative of a part of that spectrum. The authors do not infer from these cases that the spider mentioned cannot or does not, under certain circumstances, cause severe local or general problems. However, it would appear that some spiders, such as *Badumna insignis*, which are frequently encountered by man and appear to bite regularly, do not usually cause severe reactions, either local or general, other than pain. Therefore general comments on bites by these spiders, which may give the impression that bites are severe, yet without case reports to support such assertions, should be treated with caution.

Where treatment is necessary for the spiderbites mentioned herein, it must therefore be symptomatic. In most cases reassurance is the most efficacious therapy. It would greatly assist our knowledge of spiderbite

in Australia if all cases where the spider is caught are recorded in detail, the spider identified by an expert, and the information either passed on to individuals with an interest in this field, for publication, or directly published by the treating doctor. Where possible, colour photographs of the bite site should be obtained.

Acknowledgements

The authors wish to thank medical and scientific colleagues and members of the public, who have provided information on which this paper is based. In particular Mr. K. Coulthard and the staff of the Poisons Information Centre, A.C.H.; Dr. D. Lee, S.A.M.; and the staff of the Information Centre, S.A.M., deserve special thanks. The seminal role played by Dr. Ron Southcott in stimulating interest in spiderbite in S.A., and his generous help given to the senior author over the years is most gratefully acknowledged. The Department of Photography, A.C.H., kindly prepared the illustration from original slides by Julian White. Janet Bradford typed the manuscript.

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Illustrations

Records of the Adelaide Children's Hospital, Vol. 3, No. 2, 1983–84

Latrodectism: An Unusual Cause of Abdominal Pain A Cautionary Tale

by Michael Harbord* and Julian White†

INTRODUCTION

Although there are a number of poisonous spiders in various regions of Australia, the Red-back spider (*Latrodectus hasseltii*) is the only spider dangerous to man found throughout the entire country. It is not an aggressive spider but nevertheless Red-back spider bites are reported in over 300 cases annually.[1] Often the spider is identified at the time of the bite, but in other circumstances the diagnosis is made from the combination of clinical features and response to treatment. However, the clinical features of systemic envenomation (Latrodectism) by this spider may mimic other acute disease states, as illustrated by the following case in which the diagnosis was not obvious at the time of referral.

CASE REPORT

On 28 May 1984 a 12-year-old boy was referred from a station in the Northern Territory with a short history of progressive lower abdominal pain and a provisional diagnosis of appendicitis. When seen in the Casualty Department at this hospital his abdominal pain had resolved, but he complained of "shooting" pains in the limbs, generalized weakness, profuse sweating and shivers. He had been anorexic, but there was no history of vomiting or diarrhoea. He had no headache and no visual disturbance.

Further questioning revealed that he could remember being bitten behind his right knee, when lying in his sleeping bag, some 36 hours before presentation. He had not noticed a spider at the time, and went back to sleep, only to be awoken two hours later with pain in the right popliteal fossa, where he could feel a small lump at the site of the bite.

This was followed by pain in the groin, and then by lower abdominal pain which gradually increased in severity and for which he was seen by the local doctor. At that time he had pain in both iliac fossae and examination revealed rigidity of his abdomen.

In view of the examination findings he was referred to hospital, but while in transit his abdominal pain became intermittent, and limb pain, malaise, and soaking sweats were more apparent.

On examination in Casualty he was sweating but had no fever (temp. 36.5°C), and the distal extremities felt cold. The blood pressure was 130/80 with a heart rate of 84, and a regular rhythm. He had marked tremor of arms and legs which persisted with activity. Tendon reflexes were brisk and symmetrical, and there was no loss of sensation. Cranial nerves were normal. There was no significant lymphadenopathy. His abdomen was soft on palpation with no tenderness, and bowel sounds were audible. A small erythematous mark was still present in the right popliteal fossa. The Glucometer reading was 6.3 mmol/L and urinalysis showed traces of protein and ketones.

At this time it was considered that the combination of intermittent pains, malaise, coarse tremor and sweating without fever in a child with a definite history of a bite warranted a therapeutic trial of Red-back spider antivenom. Consequently a single dose of 500 units was given by intra-

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muscular injection 41 hours after the bite, concurrently with promethazine cover.

Within 15 minutes there was marked reduction in tremor and sweating, with a noticeable improvement in wellbeing, and all symptoms completely disappeared over the next 12 hours.

The boy was well enough for discharge the following day.

DISCUSSION

This case illustrates the diagnostic confusion which may result from insufficient history, and the mimicry of other acute disease states by latrodectism. It is conceivable that the child might have been subjected to exploratory abdominal surgery, had not repeated history-taking finally revealed the story of a spider bite.

When a patient presents with sudden onset of malaise, pain, sweating, muscle weakness, and tremors, latrodectism should be included in the differential diagnosis. In children this is especially true, as a history of spider bite may not be obtainable.

In the experience of one of us (J.W.) a significant number of Red-back spider bites do not result in systemic envenomation, so that symptoms in these cases may be minimal, or localized to pain and erythema at the bite site. When systemic envenomation does occur, it may take several hours to develop, the first sign of spread being painful enlargement of draining lymph nodes. The more general symptoms of latrodectism are varied, with sweating, muscle tremor, generalized pain, nausea, and vomiting being particularly prominent.[2,3,4] Abdominal pain may be a dominant feature in children.[5] However, many other symptoms and signs may be associated with latrodectism, including paraesthesiae, pyrexia, dizziness, headache, rash, hypertension, pruritis, dyspnoea, anorexia, and paralysis.[3,4] Nevertheless, sweating, malaise, and muscle weakness and tremors are usually the most prominent symptoms of latrodectism if treatment is delayed.

Minor bites do not require treatment with antivenom, but all cases of systemic envenomation should receive antivenom intramuscularly. Before the advent of antivenom, in 1956, there were a number of case reports documenting prolonged symptoms of latrodectism, ranging from two or three days to two months.[6] The mortality in untreated patients has been estimated at 5%, but no deaths from latrodectism have been recorded in Australia since specific antivenom became available.[2]

Response to treatment with antivenom is often dramatic, and may clinch the diagnosis in uncertain cases. Benefit from antivenom may occur even if treatment is very delayed. In 1961 Southcott[6] reported a case of latrodectism in a 46-year-old soldier who presented with the progressive development of pain, sweating, and agitation following a bite on the scrotum in a country privy. Antivenom was given 80 hours after the bite, with dramatic resolution of symptoms. Similarly, our case showed a dramatic response to antivenom 41 hours after the bite. While in most cases a single dose (500 units) of antivenom will be adequate, a second dose should be considered if there is incomplete resolution of symptoms. Approximately 20% of Red-back spider bites occur in children aged 16 years and under;[1] thus a history of spider bite may not be readily elicited so a high index of suspicion should be maintained, to avoid a missed diagnosis. Although the overall clinical picture of latrodectism is quite distinctive, it may mimic conditions such as torsion of the testis, an acute abdomen, and viral infection. The absence of local signs of a bite does not preclude the diagnosis of latrodectism. The potentially serious consequences of untreated latrodectism, particularly in children, should not be forgotten.

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APPENDIX 1

SOUTH AUSTRALIAN HERPETOLOGY GROUP (INC.)

REPORT OF FIELD TRIP TO BLANCHETOWN AREA, August 24th, 1975

Trip Leader - Julian White

Aim:- To compare the Reptiles of the Murray corridor with those of the Murray plain, and to ascertain the presence of Carpet Pythons in the Blanchetown section of the Murray Corridor.

Report:- A convoy of four vehicles was used, transporting 14 herpetologists to the field area.

The first area searched was the Murray corridor. The section of this 2,000 kilometre wildlife corridor searched was the east bank about 10 kilometres north of Blanchetown, working south for 3-4 kilometres. This bank is bordered by limestone cliffs for most of its length, with very little bank, and bordered by River Gums. The soil is mostly sandy to limestone rocky, with varying amounts of low vegetation cover. In sections the bark widens to include mud flats with thick scrub and reeds. There are many dead trees, both on the bank and in the water, and there is one isolated backwater. The main current of the River runs next to the bank.

The area supports different reptile species from those of the dryer Murray plain on either side. Evidence of two species of tortoise were found, in the form of carcasses. The Murray Short Neck *(Emydura macquarii)* was by far the most common. Local farmers noted that they had only recently seen Long Neck Tortoises *(Chelodina longicollis)* on the banks, with thick weed growth on their carapaces as evidence of recent, submerged hibernation.

The lizard fauna is sparse in species, there being a predominance of small skinks. (See table I). Small skinks of the genus *Morethia* move around amongst the low vegetation, especially where the bank is wider. *Cryptoblepharus* inhabit the dead logs, as do the much larger Water Skinks *(Sphenomorphus quoyii)*. Two species of Gecko were seen, Binoe's Gecko *(Heteronotia binoei)*, and the Marbled Gecko *(Phyllodactylus marmoratus)*.

Tiger Snakes *(Notechis scutatus)* are the most common snakes in this section of the corridor, several being sighted by the Group. One large, male Common Brown Snake *(Pseudonaja textilis)* was caught, and released. In contrast to the two Tiger Snakes captured (and released), it was in excellent condition, and quite corpulent.

Although no Carpet Snakes *(Morelia argus)* were found, part of a slough from this species was discovered, at the base of the cliff. under an overhanging part of the cliff face, about 4 metres above water level, and near some River Gums. This slough was deposited with the Museum.

The only other species found was a Blind Snake *(Typhlina sp.)* found while we were leaving the site. It was located under a large, dead tree stump, in loose sand, about 4 metres above water level. There were termites in the stump.

Two species possibly in the are, but not seen, are the Tree Goanna *(Varanus varius)*, and the Tree Skink *(Egernia striolata)*.

The Group then moved to an area of Triodia and Mallee Scrub, on loose red sand. Here, three species of small skink, including a possibly undescribed species of *Hemiergis*, were found. A pair of Painted Dragons was found in a shallow burrow. The female was gravid. At this stage, time having run out, the Group split up, and returned home.

TABLE I :- Species seen in the Murray Corridor, 8-10 km. north of Blanchetown, on the East bank of the River. (34°15'S by 139°38'N).

Tortoises:-

Emydura macquarii	-	several shells seen.
Chelodina longicollis	-	one shell seen.

Snakes:-

Typhlina sp.	-	
Morelia argus variegata	-	remains of slough found.
Pseudonaja texitilis	-	one live specimen seen.
Notechis scutatus	-	several live specimens seen.

Lizards:-

Cryptoblepharus boutoni	-	several seen on logs.
Morethia obscura	-	numerous.
Menetia greyi	-	numerous.
Sphenomorphus quoyii	-	several seen near water.
Phyllodactylus marmoratus	-	one seen.
Heteronotia binoei	-	several found.

TABLE II :- Species seen on the Murray Plain, 8 km. NE of Blanchetown, on the southern side of the Blanchetown, Kingston Road. (34°17'S by 139°46'E)

Lizards

Morethia obscura
Menetia greyi
Hemiergis sp.
Amphibalurus pictus

TABLE III :- Species collected, name of permit holder, and present disposition. (S.A.M. = South Australian Museum).

ecies	Common Name	No. Collected	Name of Permit Holder	Disposition (Held by)
hlina sp. (bituberculata)	Blind Snake	1	J. White	S.A.M. R14762
ptoblepharus boutoni	Small Skink	1	J. White	S.A.M. R14763
ethia obscura boulengeri	Small Skink	2	J. White	S.A.M. R14765
etia greyi	Small Skink	1	J. White	S.A.M. R14767
iergis sp.	Small Skink	1	J. White	S.A.M. R14766
eronotia binoei	Binoe's Gecko	2	J. White	S.A.M. R14764
hibolurus pictus	Painted Dragon	2	D. Levi	D. Levi

(The pair of Painted Dragons were retained by D. Levi for observations and experiments in breeding biology).

TABLE IV :- Members present on trip.

Julian White (~~President - S.A.H.G.~~) (leader)
Darryl Levi (Treasurer - S.A.H.G.)
John Fowler (Vice-President - S.A.H.G.)
Chris Hughes (Committeeman - S.A.H.G.)
Jenny Levi
Peter Hartley
Simon Ostler
Mark Wells
Ray Maquire
Nick Toy
Paul Worthley
Mark Galliford
Deriek Stow
Harry Ehmann

REPORT OF FIELD TRIP TO COXES SCRUB AREA

SEPTEMBER 21ST, 1975

Trip Leader - Chris Hughes

Aim:- To conduct a short survey on the Herpetofauna of Coxes Scrub Conservation Park and Mount Observation.

Report:- A convoy of 5 vehicles was used to transport 19 Herpetologists to the field area.

Coxes Scrub is an area of a few square km. of almost untouched bushland which has been declared a Conservation Park. It is approximately 18 km. NNW of Goolwa, and Mt Observation is just across the road from it. The vegetation is mainly low Eucalypt scrub and Banksia on white sand with varying degrees of ground cover, but generally fairly dense.

The area supports a variety of reptile species, the most common being *Lerista bouganvilli*, *Hemiergis decresiensis*, and *Tiliqua rugosa*, all of which were observed frequently. The only other species of skink observed was *Lygosoma triliniata* which was collected on an open sand patch near a tree. Only one species of Geckoe was collected. *Pygopus lepidopodus* was collected in the open, and two *Aprasia striolata* were collected under stumps; one was of the striped colour variety and the other a plain brown. There were no Dragons actually caught in the scrub but locals report *Amphibolurus barbatus* from the area. Only one species of snake was found in the scrub, this was the copperhead *Denisonia superba*.

A look at Mount Observation and the area infringing the road turned up two more *Pygopus lepidopodus*, four *Denisonia flagellum*, one Juvenile *A. barbatus* and several *Phyllodactylus marmoratus* as well as three species of frogs; - *Litoria ewingi*, *Lymnodynastes tasmaniensis* and *Pseudophryne bibroni* (a possible range extension).

Locals reported sighting a Red Bellied Black snake *Pseudechis porphyriacus* in the Conservation park but we did not observe any.

Another short stop was made beside the Onkaparinga River but nothing was collected.

TABLE I - Species collected in Coxes Scrub C.P. - Lat. 35° 22's
Long. 138° 44'E

C. Hughes permit holder

Species	Common Name	No.Collected	Disposition
Snakes			
Denisonia superba	Copperhead	1	S.A.M. R14892
Skinks			
Lerista bouganvilli	Bouganvilles Skink	1	S.A.M. R14897
~~*Lygosoma trilineata*~~ → *Leiolopisma trilineata*	Grass Skink	1	S.A.M. R14898
Hemiergis decresiensis	Three-toed Skink	1	S.A.M. R14899
Geckoes			
Phyllodactylus marmoratus	Marbled Gecko	1	S.A.M. R14896
Legless Lizards			
Aprasia striolata	Worm Lizard	2	S.A.M. R14895
Pygopus lepidopodus	Scalyfoot	1	S.A.M. R14894

One Shingleback *Tiliqua rugosa* was seen but not collected.

TABLE II - Species collected on Mt. Observation - Lat. 35° 21'S
Long. 138° 44'E

C. Hughes permit holder

Species	Common Name	No.Collected	Disposition
Snakes			
Denisonia flaggellum	Little Whip Snake	4	1 S.A.M. R1489 2 J. Hill 1 R. Elcock
Legless Lizards			
Pygopus lepidopodus	Scalyfoot	2	2 D. Levi
Dragons			
Amphibolurus barbatus	Bearded Dragon	1	1 P. Hartley

Those specimens retained by members will be used for breeding study, and public education.

TABLE III - Members present on trip.

Chris Hughes (leader)
Julian White (President S.A.H.G.)
John Fowler (Vice President S.A.H.G.)
Darryl Levi (Treasurer S.A.H.G.)
Julia Smith (Librarian S.A.H.G.)
Anne Bushel (Committee member S.A.H.G.)
~~Chris Hughes~~ (Committee member S.A.H.G.)
Alex Bushel
James Bushel
Mark Galliford
Allison Hill
John Hill
Sue Hill
Nick Joy
Simon Astler
James Reeves
Derek Stone
Paul Worthley
Mark Worthley
Richard Elcock

NOVEMBER 16TH, 1975 - TRIP - TO PT. PRIME AND PT. PARHAM

Trip Leader - J. White

Aim:- To continue the S.A.H.G. survey of reptiles of the coastal areas north of Adelaide.

Report:- One vehicle was used to transport seven personel to the field area. The Group first looked at the area of coastal scrub around Pt. Prime. Weather conditions were hot, humid, and overcast. The area consisted of shellgrit soil and low dunes, with medium density low coastal scrub, with considerable dead wood and leaf and seaweed litter.

One of the first finds was a small Brown Snake *(Demansia texitilis)*, which was seen to disappear down a small hole. After capture and identification it was released, as were the numerous Shinglebacks *(Tiliqua rugosa)* and Peron's Skinks *(Hemiergis peroni)* which were found in the area, the later mostly under leaf litter singly, the former usually under bushes and in pairs.

Two species of *Lerista* were collected, and an interesting Striped Skink, probably *Ctenotus uber*. Other lizards collected are listed in table I.

After lunch, the Group moved up the coast to Pt. Parham, and a red sand dune about 1 km. north of the town, where Gould's Goanna are known to be abundant. The dune is surrounded by farmland, and is covered in rabbit warrens and Goanna holes. There was numerous evidence of Goanna activity including an amazing abudance of Goanna tracks. One Gould's Goanna was briefly sighted. However, the interesting find in this area was that of a Little Whip Snake *(Unechis gouldii)*, which is most unusual as this species is usually associated with wetter and hillier country.

All specimens collected were deposited with the S.A. Museum.

TABLE I:- List of species caught or sighted at Pt. Prime
(Lat. 34°31'S ; Long. 138°18'E)

NAME	COMMON NAME	NO. SEEN	NO. COLLECTED	S.A.M. REG. NO.
Demansia texitilis	Brown Snake	2	-	-
Phyllodactylus marmaratus	Marbled Gecko		1	R15035
Tiliqua rugosa	Shingleback	10+	-	-
Ctenotus uber orientalis	Striped Skink	-	2	R15034
Morethia adelaidensis	-	3	1	R15036
Hemiergis peroni	Peron's Skink	10+	-	-
Lerista frosti	-	-	2	R15039
Lerista terdigitata	-	-	1	R15038
Aprasia inaurita	Worm Lizard	-	1	R15037

TABLE II:- List of species caught or sighted at Pt. Parham
(Lat. 34°25'S ; Long. 138°15'E)

NAME	COMMON	NO. SEEN	NO. COLLECTED	S.A.M. REG. NO.
Urechis gouldii	Little Whip Snake		1	R15040
Varanus gouldii	Gould's Goanna	1	-	-
Tiliqua rugosa	Shingle Back	6+	-	-
Monetia greyi	Grey's Skink	2	-	-

TABLE III:- List of members present

Julian White (leader)
John Fowler
Chris Hughes
Mark Galliford
Nick Joy
Derek Stone
Paul Worthley

FIELD TRIP TO GOOLWA - WAITPINGA AREA - December 14th, 1975

Leader - Chris Hughes

Aims:- To survey the Herpetofauna of the coastal sandhills near Goolwa and Waitpinga.

Report:- A convoy of 3 vehicles was used to transport 12 herpetologists to the field area.

The sandhill area approximately 1 km. from Goolwa, is composed of large white dunes covered with low scrub of varying density. The area is bounded on one side by the ocean, and on the other by a large freshwater body.

Shortly before reaching the first stop, an adult Long-Neck Tortoise *(Chelodina longicollis)* was found near the roadside; this was photographed and then released.

In the sandhills the species found were all common, namely the Painted Dragon *(Amphibolurus pictus)*, the Shingleback *(Tiliqua rugosa)*, Peron's Skink *(Hemiergis peroni)* and *Lerista frosti*. A small number of *Hemiergis peroni* were collected as food lizards. One dead juvenile Bearded Dragon *(A. barbatus)* was also found, and a Brown Snake slough.

The sandhills at Waitpinga Beach were also looked at by a small party. These hills were slightly redder in colour than those at Goolwa, with similar vegetation and some areas of limestone outcrops. Among the species collected here were the Little Whip Snake *(Unechis flagellum)* and White's Skink *(Egernia whitei)*.

A brief examination of an area near the mouth of Waitpinga Creek resulted in 7 Black Snakes *(Pseudechis porphyriacus)* being found, although none were collected.

TABLE I - Species found in Goolwa Sandunes

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Chelodina longicollis	Long-neck Tortoise	1	-	-
Amphibolurus pictus	Painted Dragon	6	-	-
Amphibolurus barbatus	Bearded Dragon	1	-	-
Tiliqua rugosa	Shingleback	3	-	-
Lerista frosti	-	10	-	-
Hemiergis peroni	Peron's Skink	30+	15	(food lizards)

TABLE II - Species found at Waitpinga Beach (Lat. 35°31'S; Long. 138°29'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Unechis flagellum	Little Whip Snake	-	1	R15093
Pseudechis porphyriacus	Black Snake	7	-	-
Egernia whitei	White's Skink	-	1	R15094
Menetia greyi	Grey's Skink	-	1	R15095
Lerista frosti	-		1	R15096
Hemiergis peroni	Peron's Skink	10+	-	-
Phyllodactylus marmoratus	Marbled Gecko	1	-	-

TABLE III - Members Present

Chris Hughes (Leader)
Julian White
Darryl Levi
John Fowler
John Hill
Shiela Hill
Allison Hill
Julia Smith
Debbie Leggo
Steve Giddings
Mark Galliford
Nick Joy

BARATTA STATION - S.E. FLINDERS TRIP - OCTOBER 11-13, 1975

Trip Leader:- J. White

Aim:- To assess the herpetofauna of the S.E. Flinders Ranges.

Report:- Two vehicles were used to transport ten personel to the survey area, approximately 320 km. north of Adelaide. the campsite was 3 km. north of Baratta homestead. Weather conditons were variable, with patchy cloud cover, and frequent strong winds.

The Group drove to Orroroo, and from there drove north on the Johnburgh-Belton Road to Baratta. The first specimens were caught at a small creek just south of Belton. Several small skinks were collected (table 1). From here the group moved north-east to Buckalowie Creek, where the rock faces beside the creek were examined. These contained Stokes Skinks *(Egernia stokesii)*, some smaller lizards (table 2) and some Common Bluetongues *(Tiliqua scincoides)*, the latter find being an extension of range. A Bearded Dragon was seen in the creek bed.

From Buckalowie Creek, the Group moved north to Baratta Station, and the campsite, just north of Baratta homestead. Unlike the area to south and west, the northern part of Baratta was only lightly grazed, and still had considerable areas of natural vegetation left, including stands of Spinifex bush. The area was also very hilly, with numerous creeks, and large rock outcrops, proving an ideal habitat for many species of reptile.

The creek next to the campsite had permanent rock pools, with a large yabbie population, and numerous frogs. Two species of frog were observed, the Marbled Frog *(Lymnodynastes tasmaniensis)*, and the small Froglet *(Crinia sp.)*.

The rock faces above the creek harboured several species of reptile (table 3). Stoke's Skinks were common, and one *Egernia margaretae* was seen, although too deeply wedged in the rocks to be collected. Several species of gecko were collected, including the Thick-tailed Gecko *(Phyllurus milii)*.

Beside the creek bed, a Yellow-Faced Whip Snake *(Demansia psammophis)* was collected, as was a Stone Gecko *(Diplodactylus vittatus)*. Shingleback lizards and Bearded Dragons were common.

From the campsite, the group moved north towards Bibliando homestead, through the main stands of rocky hillside, with numerous spinifex bushes. Two species of legless lizard and a Spotted-necked Skink *(Tiliqua branchialis)* were caught under the spinifex: (table 4).

Several more species were collected under some rubbish at Bibliando homestead outhouses, including a Striped Skink *(Ctenotus saxatilis)* (table 5). An unusual dragon lizard was sighted here, but unfortunately, it eluded capture. It was the size of a Painted dragon, with the elongate snout typical of *Diporiphora*, grey in colour, and with a distinct yellow band running down the side of its body, from the rear of the head, to the base of the tail.

The group also visited Holowilena Station, to the west of Baratta, but found it very heavily grazed, and with little or no suitable habitat left for reptiles.

The group returned to Adelaide via Milang and Waukaringa ruins, which lie just south-east of Baratta, on the north western tail of the Olary Ridge. This heavily grazed country is interspersed with flat savanna, now almost devoid of vegetation as a result of overstocking and rabbit plagues.

At Milang ruins, two species were collected; a small Curl Snake *(Denisonia suta)*, and a Desert Banded Skink *(Sphenomorphus richardsoni)*. (table 6).

Further south, the Waukaringa ruins, which were more extensive, were quite rich in reptile fauna, including a different species of *(Ctenotus uber orientalis)* to that from Bibliando, and a Western Bluetongue *Tiliqua occipitalis*, which is the most northerly point this species has been found in S.A., and is a valuable extension of its range. (table 7).

From Waukaringa, the group drove to Yunta, and so back to Adelaide. A total of 26 species of reptile and 2 species of amphibian were seen or collected on the trip. The exact location data, and details of species seen or collected at each location are listed in the following tables.

Although no large snakes were seen on the trip, the manager of Baratta Station listed three species common in the area. These were the Brown Snake *(Demansia texitilis)*, the Black Snake *(Notechis ater)*, and the Mallee Tiger Snake. This latter species was described by the manager as large, robust, with numerous yellowish bands ringing the whole length of the body. He said it was the commonest species in the area, but was only seen during the warm summer months. We do not know what species he was reffering to when talking about this Mallee Tiger Snake, and we will be interested to obtain some specimens on future trips to the general area.

TABLE 1 Species caught 6 km. South of Belton (lat. 32°17'S ; long. 138°44'E)

Species	No. Seen (not collected)	No. Collected	Disposition
Menetia greyi	–	1	SAM R14900
Morethia boulengeri	2	2	SAM (2) R14901
Lerista muelleri	4	3	SAM (2) R14902

TABLE 2 Species caught at Buckalowie Creek (lat. 32°12'S ; long. 138°50'E)

Species	No. Seen (not collected)	No. Collected	Disposition
Heteronotia binoei	2	1	SAM R14903
Tiliqua scincoides	1	1	D. Levi
Egernia stokesii	1	–	
Amphibolurus barbatus	1	–	

TABLE 3 Species caught at Baratta (1) (lat. 31°57'S ; long. 139°5'E)

Species	No. Seen (not collected)	No. Collected	Disposition
Phyllurus milii	1	1	D. Stone
Diplodactylus vittatus	–	1	SAM R14905
Gehyra variegata	–	1	SAM R14904
Heteronotia binoei	3	–	–
Tiliqua rugosa	1	–	–
Egernia stokesii	2	–	–
Egernia margaretae	1	–	–
Tympanocryptus tetraporophora	–	3	SAM (1) R14906
Amphibolurus barbatus	1	–	–
Demansia psammophis	–	1	N.A.

TABLE 4 Species caught at Baratta (2)
(Lat. 31°55'S ; long. 139°5'E)

Species	No. Seen (not collected)	No. Collected	Disposition
Tiliqua branchialis	–	1	SAM R14912
Delma nasuta	.	1	SAM R14913
Delma australis	1	1	SAM R14914

TABLE 5 ~~Species seen at Milang ruins~~
(lat. 32°18'S ; long 139°26'E)

Species	No. Seen (not collected)	No. Collected	Disposition
Morethia boulengeri	5	1	SAM R14911
Lerista muelleri	.	1	SAM R14909
Cryptoblepharus boutonii	1	1	SAM R14910
Ctenotus saxatilis	.	1	SAM R14907
Amphibolurus sp.	1	–	–

TABLE 6 Species seen at Milang ruins
(lat. 32°3'S ; long. 139°10'E)

Species	No. Seen (not collected)	No. Collected	Disposition
Sphenomorphus richardsoni	–	1	m. Gallifard
Denisonia suta	–	1	SAM R14908

TABLE 7 Species seen at Waukaringa ruins
(lat. 32°18'S ; long. 139°26'E)

Species	No. Seen (not collected)	No. Collected	Disposition
Ctenotus uber orientalis	.	3	SAM (3) R14917
Morethia adelaidensis	–	1	SAM R14915
Tiliqua occipitalis	–	1	RNA
Amphibolurus barbatus	2	–	–
Gehyra variegata	–	1	SAM R14916

TABLE 8 Personel present on tripL

Julian White (Leader - S.C.P. No. 221)
Chris Hughes
John Hill
Allison Hill
Shiela Hill
Mark Galliford
Steven Giddings
Derek Stone
Debbie Leggo
Julia Smith

TABLE 9 Species caught on permit and their immediate disposition and species seen but not collected.

Species	Common Name	No. Collected and disposition
Skinks (15 species)		
Menetia greyi	-	SAM (1)
Morethia adelaidensis	-	SAM (3)
Morethia boulengeri	-	SAM (1)
Lerista muelleri	-	SAM (4)
Sphenomorphus richardsoni	Desert-Banded Skink	C. Hughes (1)
Egernia stokesii	Stoke's Skink	none collected
Egernia margaretae	Rock Skink	none collected
Tiliqua scincoides	Common Bluetongue	D. Levi (1)
Tiliqua rugosa	Shingleback	none collected
Tiliqua occipitalis	Western Bluetongue	~~D. Levi~~ (1) RNA
Tiliqua branchialis	Spotted-necked Skink	SAM (1)
Cryptoblepharus boutoni	Bouton's Skink	SAM (1)
Ctenotus saxatilis	Striped Skink	SAM (1)
Ctenotus uber orientalis	Striped Skink	SAM (3)
Geckoes (4 species)		
Heteronotia binoei	Bynoe's Gecko	SAM (1)
Gehyra variegata	Dtella	SAM (2)
Diplodactylus vittatus	Stone Gecko	SAM (1)
Phyllurus milii	Thick-tailed Gecko	D. Stone (1)

Species	Common Name	No. collected and disposition
Dragons (3 species)		
Amphibolurus barbatus	Bearded Dragon	none collected
Amphibolurus sp. (nobbi)	-	none collected
Tympanocriptus tetraparophora	Earless Dragon	SAM (1);
Legless Lizards (2 species)		
Delma nasuta	-	SAM (1)
Delma australis	-	SAM (1)
Snakes (2 species)		
Denisonia suta	Curl Snake	SAM (1)
Demansia psammophis	Yellow-faced Whip Snake	M. Galliford (1)
Frogs (2 species)		
Lymnodynastes tasmaniensis	Marbled Frog	SAM (1)
Crinia sp.	Froglet	SAM (3)

SURVEY OF STATION COUNTRY NORTH OF MORGAN - BUNGUNNIA AND BALAH STATION

NOVEMBER 22-23, 1975 LEADER:- J. WHITE

Aim:- To survey reptile fauna in the sandridge country between the River Murray and the Olary Ridge.

Report:- Three vehicles were used to transport fourteen personel to the Survey area. Due to restrictions of time and inclement weather it was possible only to briefly survey the two stations immediately north of Morgan, on the old mail-road and Radium Hill Power Line road, namely Bungunnia Station and Balah Station. In both cases written permission to survey was obtained from the owners, who also provied us with much valuable information on interesting areas on their respective properties, which undoubtedly increased the success of the survey.

The area itself has mixed habitats, and is used as pastural country, grazing both sheep and cattle. However the owners have ensured that the land is not overstocked, and as a result there are considerable tracts of virtually untouched scrub, and the whole area is very well vegetated. It is a credit to the owners thoughtfullness.

The first station examined was Bungunnia, about 18 km. north of Morgan. We looked at an area of bluebush flat initially, which had numerous Shinglebacks *(Tiliqua rogosa)*, several Beared Dragons *(Amphibolurus barbatus)* and large Earless Dragons *(Tympanocryptus lineata)*. Unfortunately we were unable to capture any of the latter species. When camping in this area on the night of the 22nd, following a thunderstorm, several specimens of Byrne's Gecko *(Diplodactylus byrnei)* were collected.

The second area examined, also on Bungunnia, was a small tract of Mallee-Spinifex-Bluebush country. This habitat, in contrast to the Bluebush flats, was teeming with reptiles, almost certainly because of the very favourable habitat offered by the spinifex. Species seen are listed in Table I. The Military Dragon *(Amphibolurus fordi)* was very common amongst the spinifex, and Burton's Legless Lizard *(Lialis burtonis)* was also an occupant of these bushes, while the Striped Skinks *(Ctenotus regius)* and Painted Dragon *(Amphibolurus pictus)* were more common around the Bluebush. *Amphibolurus nobbi* was associated with dead Mallee.

From this site we moved north to another area of Mallee-Spinifex, without Bluebush, located along the Radium Hill road on Balah Station. Before being forced to retire to the main road due to a thunderstorm, numerous Military Dragons were observed, and a Desert Skink *(Egernia inornata)* and Spiny Gecko *(Diplodactylus ciliaris)* were collected down the same *inorrata* burrow, at the base of a Spinifex bush. This area is well worth revisiting, especially at night.

On the 23rd, the rain having ceased, we again looked at Bungunnia Station, moving further east and north, up the Radium Hill Road, In the diverse habitats seen, several more species were collected. (Tables 4 - 6). In open country, under dead trees, several Desert Banded Skinks *(Sphenomorphus richardsoni)* were seen. In spinifex country, more Military Dragons, Burton's Legless Lizards, and a new species of Ctenotus were seen. In addition, several Whip Snakes *(Demansia psammophis)* were sighted, coiled up together at the base of a mallee tree, amongst the leaf-litter.

In the more open, stony Mallee, bluebush and mixed scrub, another *A. nobbi* was seen, and a specimen of Tree Skink *(Egernia striolata)* collected. Several more of this latter species were also seen.

In summary, an interesting area with considerable tracts of natural scrub, well worth future visits by the Group.

TABLE I - Locality (1) Bungunnia Station Lat. 33°52'S, Long, 139°46'E
Mallee - Spinifex - Bluebush Scrub

SPECIES	COMMON NAME	NO. SEEN	NO. COLLECTED	S.A.M. REG. NO.
Lialis burtonis	Burton's Legless Lizard	-	1	
Ctenotus regius	Striped Skink	2	3	R 15051
Egernia inornata	Desert Skink	-	1	R 15050
Lerista orientalis	-	-	1	R 15041
Gehyra variegata	Dtella Gecko	-	1	R 15042
Amphibolurus fordi	Military Dragon	10+	2	R 15049
Amphibolurus pictus	Painted Dragon	4	1	NRA
Amphibolurus nobbi	-		2	R 15156

TABLE II - Locality (2) Balah Station Lat. 33°39'S, Long. 139°55'E
Mallee - Spinifex Scrub

SPECIES	COMMON NAME	NO. SEEN	NO. COLLECTED	S.A.M. REG. NO.
Egernia inornata	Desert Skink	-	1	R 15049
Diplodactylus ciliaris	Spiny Gecko	-	1	R 15043
Amphibolurus fordi	Military Dragon	10+	-	

TABLE III - Locality (3) Bungunnia Station Lat. 33°52'S, Long. 139°43'E
Bluebush flats

SPECIES	COMMON NAME	NO. SEEN	NO. COLLECTED	S.A.M. REG. NO.
	Byrnes Gecko	-	4	R 15944 A-D

TABLE IV - Locality (4) Bungunnia Station Lat. 33°52'S, Long. 139°49'E
Open plains, sparse tree cover

SPECIES	COMMON NAME	NO. SEEN	NO. COLLECTED	S.A.M. REG. NO
Sphenomorphus richardsoni	Desert Banded Skink	1	1	R 15047

TABLE V - Locality (5) Bungunnia Station Lat. 33°52'S, Long. 139°50'E
Mallee - Spinifex Scrub

SPECIES	COMMON NAME	NO. SEEN	NO. COLLECTED	S.A.M. REG. NO.
Lialis burtonis	Burton's Legless Lizard	3	-	
Ctenotus schomburgkii	Striped Skink	-	1	R 15045
Egernia inornata	Desert Skink		-	
Amphibolurus fordi	Military Dragon		-	
Demansia psammophis	Yellow-faced Whip Snake		-	

TABLE VI - Locality (6) Balah Station Lat. 33°47'S, Long. 139°51'E
Mallee, Bluebush, mixed scrub

SPECIES	COMMON NAME	NO. SEEN	NO. COLLECTED	S.A.M. REG. NO.
Egernia striolata	Tree Skink	6	1	R 15046
Amphibolurus nobbi	-			

TABLE VII - Members Present on Trip

Julian White (Leader - S.C.P. No. 221)
Chris Bourn
Graham Boyce
Peter Elcock
Richard Elcock
John Fowler
Mark Galliford
Peter Hartley
Chris Hughes
Debbie Leggo
Simon Ostler
Julia Smith
Andrew Todd
John Warmington

TABLE VIII - Total List of Species seen or collected

SPECIES	STATUS IN AREA	NO. SEEN	NO. COLLECTED
Pygopodidae:			
Lialis burtonis	Common	3	1
Scincidae:			
Ctenotus regius	Common	2	3
Ctenotus schomburgkii	Uncommon	-	1
Egernia inornata	Common	1	2
Egernia striolata	Common	6	1
Lerista orientalis	Common	1	1
Tiligua rugosa	Common	6+	-
Sphenomorphus richardsoni	Common	1	1
Geckonidae:			
Gehyra variegata	Uncommon	-	1
Diplodactylus ciliaris	Uncommon	-	1
Diplodactylus byrnei	Common	-	4
Agamidae:			
Amphibolurus barbatus	Uncommon	1	-
Amphibolurus nobbi	Uncommon	-	3
Amphibolurus fordi	Abundant	100++	2
Amphibolurus pictus	Uncommon	4	1
Tympanocriptus lineata	Uncommon	3	-
Elapidae:			
Demansia psammophis	Uncommon	3	-
*Pseudechis australis**	Uncommon to rare	1	-

* - dead specimen killed by owner of Balah Station)

REPORT OF FIELD TRIP TO WOOMERA AND EYRE PENINSULA

DECEMBER 27TH 1975 to JANUARY 3RD 1976

Leader:- Julian White

Aim:- To make a preliminary herpetological assessment of the Woomera Prohibited Area.

Report:- Ten herpetologists attended this eight day trip, using three vehicles.

The weather for the whole trip was variable, from warm to very hot, and collecting conditions were poor on most occasions due to the heat, and consequent fatigue of collectors.

The route was Adelaide, Pt. Augusta, Woomera, Arcoona Station, Woomera, Pt. Augusta, Corunna Hills, Siam Station, Nonning Station, Yardea Station, Minipa, Streaky Bay, Sceale Bay, Mt. Wedge, Whyalla, Adelaide.

Extensive use was made of powerful spotlights powered by a small generator. A considerable number of species were seen spot-lighting, especially geckos, but also some skinks and snakes.

On the road up to Woomera from Pt. Augusta several Gouldi Goannas *(Varanus gouldii)* were seen. On Arcoona Station several areas were visited. The sandhill country east of the Station proved unprofitable. A striped Skink *(Ctenotus regius)* and a Crested Dragon *(Amphibolurus cristatus)* were seen. The hot conditions probably inhibited reptile activity - it certainly affected Herpetologist activity, and is contributed to our poor results in this area.

Near the Station, several bearded Dragons *(A. barbatus)* were noted, as well as numerous Binoe's Geckoes *(Heteronotia binoei)*. Around the edge of Arcoona Lake, next to the homestead, a colony of Stoke's Skinks *(Egernia stokesii)* was found, along with numerous *Morethia adelaidensis*. The gibber Desert on Arcoona was populated mainly with Earless Dragons *(Tympanocriptus tetraporophora)*, which were observed sunning themselves in the tops of low bushes.

An attempt was made to reach Roxby Downs Station, but the road was flooded and impassable. The Painted Dragon *(A. pictus)* was seen in this sandhill country.

From Arcoona we returned to Woomera, and examined a large rock outcrop on the edge of the town. Children's Python *(Liasis childreni)* had been recorded from this rock outcrop, and we were fortunate enough to observe a specimen. Also seen were a King Brown Snake and a Yellow Faced Whip Snake. The Desert Banded Skink *(Sphenomorphus richardsoni)* was very numerous in the rock outcrop. Table IV lists the species seen.

This rock outcrop is very close to habitation, and is under considerable collecting and destructive pressure from the local people. Children use it frequently, and from residents of Woomera, we learned that snakes are being illed there. All species of reptile in this outcrop must therefore be considered endangered, but especially the Children's Python. If possible, it would be advisable to declare the outcrop a reserve, and protect it with suitable barricades to keep all but genuine researchers out. Unless this is done, the Children's Python will probably become extinct in this outcrop in under five years.

From Woomera, we moved south, still on Arcoona Station, to look at a rock outcrop at Disputed Creek. The species seen at this extensive rock face are listed in table V, but most notable was the Arcoona Rock Dragon *(A. fioni)*, an interesting variant of the common Eyre Peninsula Rock Dragon.

Our next herpetological stop was Corunna Hills, where we added three species to our list of reptiles from this area. (Table VI) These were the Tree Skink, the Desert Banded Skink, and the Beaked Gecko. Interestingly, few specimens of the species found common on our winter visit were seen.

From Corunna, we moved west towards Yordea Station. At Lake Gilles Tank, two species of snake were found at the bottom of the tank, along with numerous tadpoles and frogs, which the snakes had apparently been eating.

From Lake Gilles Tank, we moved west across Siam Station, where an Earless Dragon *(Tymparocriptus lineata)* was collected, to Nonning Station. Here, we made contact with two Sweedish Herpetologists who were working on the Station.

From Noning, we moved onto Yardea Station, where we made camp. Here several species of reptile were collected, including the fascinating Crested Dragon *(Amphibolurus cristatus)*. This extremely swift footed lizard was observed in moderate numbers on the saltbush flats, where its footprints covered the coarse red sand. Most specimens lived in either hollow logs, which were quite common, or in burrows. These latter resembled goanna burrows, initially, with large wide entrances. However, they had a steep incline and continued for about 60 - 100 cms., being about 30 - 40 cms. beneath ground level at their deepest point, the chamber where the lizard resided. The burrow took one or more sharp turns before reaching the chamber, which was not significantly larger than the burrow in dimensions. There was usually only one lizard per burrow. Other species found on Yardea Station were listed in tables VIII, IX, XI and XII.

From Yardea we moved south, through sandhills and rocky outcrops. Although we did not have time to stop and collect, this area is well worth a revisit.

We then moved across to the West Coast, and examined the Sceale Bay area. This narrow strip of fixed coastal dunes and scrub supports a wide range of vegetation, including *Triodia*. Table XIII lists the species caught in this area. Most notable was the Queen Adelaide Dragon *(Amphibolurus adelaidensis)*, an uncommon species, very similar in form to the Earless Dragons, but with a distinct tympanum, and greyish in colour. Several specimens of this species were observed active amongst the low bushes.

The trip concluded by visiting Whyalla to renew our aquaintance with the Western Herpetology Group and its budding Fauna Park.

Our thanks are due to Mr. J. Oag of Arcoona Station, for his help and interest in us while on his Station, and to Mrs. Hall of Corunna Station, for again making us welcome.

We are very grateful to William Haughton Pty. Ltd., who lent us the Honda Generator which we used to power our spotlights for nocturnal work.

TABLE I - Species seen in Sandhills - Arcoona Station

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Ctenotus regius	1	-	-	-
Amphibolurus cristatus	1	-	-	-

TABLE II - Species seen on edge of Arcoona Lake ($31^{o}1'S \times 137^{o}3'E$)

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Egernia stokesii	3	1	D. Levi	-
Morethia adelaidensis	10	2	S.A.M.	

TABLE III - Species seen on Arcoona Station - Gibber desert ($30^{o}54'S \times 137^{o}1'E$)

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Tympanocriptus tetraporophora	10+	5	2 C. Hughes 3 S.A.M.	
Amphibolurus pictus			S.A.M.	

TABLE IV - Species seen at Woomera rock outcrop ($31^{o}15'S \times 136^{o}50'E$)

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Sphenomorphus richardsoni	6+	1	S.A.M.	
Morethia boulengeri	4+	2	S.A.M.	
Ctenotus robustus	2+	2	-	-
Phyllurus milii	1	-	-	-
Pseudechis australis	1	-	-	-
Demansia psammophis	1	-	-	-
Liasis childreni	1	-	-	-

TABLE V - Species seen at Disputed Creek rock outcrop ($31^{o}21'S \times 136^{o}56'E$)

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Ctenotus uber	2	1	S.A.M.	
Amphibolurus fioni	3	3	1 J. White 1 S.A.M.	
Tympanocriptus tetraporophora	-	1	S.A.M.	

TABLE VI - Species seen at Corunna Hills (32°39'S x 137°8'E)

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Sphenomorphus richardsoni	-	1	S.A.M.	
Egernia striolata	6	1	S.A.M.	
Tiliqua branchialis	1	-	-	-
Tiliqua rugosa	1	-	-	-
Rhynchoedura ornata	-	4	2 S.A.M.	
Pseudechis australis	1	-	-	-

TABLE VII - Species seen in Lake Gilles Tank (32°37'S x 136°52'E)

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Pseudonaja nuchalis	-	1	J. White	-
Suta suta	1	1	M. Galliford	-

TABLE VIII - Species seen on Yardea Station Location 1 (32°22'S x 135°52'E)

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Amphibolurus cristatus	10+	1	D. Levi	-
Ctenotus schomburgkii	-	1	S.A.M.	

TABLE IX - Species seen on Yardea Station Location 2 (32°24'S x 135°50'E)

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Rhynchodeura ornata	-	1	S.A.M.	
Diplodactylus	4	1	S.A.M.	
Unechis brevicauda	-	1	S.A.M.	

TABLE X - Species seen on Siam Station (32°33'S x 136°40'E)

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Tympanocriptus lineata	-	1	S.A.M.	

TABLE XI - Species seen on Yardea Station Location 3 (

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Egernia striolata	-	1	D. Levi	

TABLE XII - Species seen on Yardea Station Location 4 ($32^{o}22'S \times 125^{o}38'E$)

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Varanus gouldii	-	1	C. Hughes	-

TABLE XIII - Species seen at Sceale Bay ($33^{o}00'S \times 124^{o}13'E$)

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Pygopus lepidopodus	1	1	S.A.M.	
Amphibolurus adelaidensis	4	2	S.A.M.	
Amphibolurus fordi	10	2	S.A.M.	
Amphibolurus pictus	2	-	-	-
Ctenotus brooksii	3	1	S.A.M.	
Morethia	-	1	S.A.M.	
Lerista frosti	-	1	S.A.M.	
Hemiergis peroni	3	-	-	-
Diplodactylus vittatus	2	2	R. Forsyth	-

TABLE XIV - Species seen at Streaky Bay ($32^{o}59'S \times 134^{o}12'E$)

SPECIES	SEEN	COLLECTED	DISPOSITION	S.A.M. REG. NOS.
Phyllurus milii	-	1	S.A.M.	
Phyllodactylus marmoratus	-	1	S.A.M.	

TABLE XV - Members Present

Julian White (Leader)
Darryl Levi
Chris Hughes
John Fowler
Simon Ostler
Peter Hartley
Mark Galliford
Nick Joy
Debbie Leggo
Ross Forsyth

TABLE XVI - Lists of species seen on trip

SCIENTIFIC NAME	COMMON NAME	NO. SEEN	NO. COLLECTED
AGAMIDAE (Dragons)			
Amphibolurus barbatus	Bearded Dragon	1	-
Amphibolurus pictus	Painted Dragon	2	1
Amphibolurus cristatus	Bicycle Lizard	10+	1
Amphibolurus fordi	Military Dragon	10+	2
Amphibolurus adelaidensis	Queen Adelaide Dragon	4+	2
Amphibolurus fioni	Arcoona Rock Dragon	3	3
Amphibolurus vidiceps	Inland bearded Dragon	10+	-
Tymparocriptus tineata	Earless Dragon	-	2
Tymparocriptus tetraporophora	Earless Dragon	10+	6
SCINCIDAE (Skinks)			
Sphenomorphus richardsoni	Desert Banded Skink	6+	2
Egernia stokesii	Spiny-tailed Skink	3	1
Egernia striolata	Tree Skink	5	2
Tiliqua branchialis	Mourning Skink	1	-
Tiliqua rugosa	Shingleback	3	-
Tiliqua scincoides	Bluetongue	1	-
Tiliqua occipitalis	Western Bluetongue	2	-
Ctenotus schomburgkii	Striped Skink	-	3
Ctenotus uber	Striped Skink	2	3
Ctenotus robustus	Striped Skink	2+	4
Ctenotus brooksii	Striped Skink	3+	3
Morethia adelaidensis	-	10+	
Morethia boulergeri	-		
Lerista frosti	-	-	1
Hemiergis peroni	Peron's Skink	3	-
VARANIDAE (Goannas)			
Varanus gouldii	Gould's Goanna	5	1
PYGOPODIDAE (Legless Lizards)			
Pygopus lepidopodus	Scalyfoot	1	1
GEKKONIDAE (Geckoes)			
Diplodactylus vittatus	Stone Gecko	2	3
Rhynchoedura ornata	Beaked Gecko	-	5
Phyllurus milii	Barking Gecko	1	1
Phyllodactylus marmoratus	Marbled Gecko	-	1
Gehyra variegata	Dtella	-	1
Heteronatia binoeii	Binoe's Gecko	10+	-
BOIDAE (Pythons)			
Liasis childreni	Children's Python	1	-

ELAPIDAE (Venomous snakes)

Pseudechis australis	King Brown	3	-
Pseudongja nuchalis	Western Brown	3	1
Demansia psammophis	Yellow-faced Whip Snake	2	-
Suta suta	Curl Snake	1	1
Unechis brevicauda	Mitchell's Short-tail snake	-	1

REPORT OF TRIP TO THE AREA IN AND AROUND

SCORPION SPRINGS CONSERVATION PARK

January 24 - 26, 1976

Leader:- J. White

Aim:- To survey the reptiles of this bush area.

Report:- Four vehicles were used to transport thirteen herpetologists to the field areas.

The area looked at is fairly uniform over a large area, and consists of a white sand base, with undulating dunes, all thickly covered with vegetation. Scrub density varies, from areas of low Eucalypt, interspersed with heath and spinifex, to more open scrub, with Banksia and other small shrubs.

Most specimens were caught near the camp site (Table I), or along a nearby track (Table II). Cool overcast weather inhibited reptile activity, and was probably causative in our lack of success with pitfall traps. Our 150 metre trap line caught no reptiles, although two Pigmy Possums *(Cercartetus concinnus)* were caught on the first night.

The two most common reptiles were the Military Dragon *(Amphibolurus fordi)* and the Snake-eyed Skink *(Morethia adelaidensis)*, both of which frequented the low shrubs in the more openly vegetated areas. Both the Jacky Lizards *(A. muricatus)* and the Painted Dragons were found along the edges of the sandy tracks through the scrub. Only one Striped Skink *(Ctenotus brooksi)* was seen, and both the Bearded Dragon and Shingleback were relatively uncommon.

Of the snakes, the two Master's Snakes *(Drysdalia masteri)* were caught crossing the sandy track during the day, while the Mitchell's Short-tail Snake *(Unechis brevicauda)* was found under a rotting stump.

No reptiles were observed active at night. In addition to reptiles, numerous species of insects and spiders were collected and deposited with the S.A. Museum.

TABLE I - Locality I - (34°41'S by 140°47'W)

SPECIES	COMMON NAME	STATUS	NO. COLLECTED	S.A.M. REG. NO
Amphibolurus fordi	Military Dragon	Abundant	1	R1513
Amphibolurus barbatus	Bearded Dragon	Uncommon	-	
Ctenotus brooksi	Striped Skink	Uncommon	1	R1519
Morethia obscura	Snake-eyed Skink	Abundant	1	R1513
Lerista bouganvillii	-	Common	1	R1513
Menetia greyi	-	Uncommon	-	-
Drysdalia masteri	Master's Snake	Common	2	R1519
Pseudonaja taxitilis	Brown Snake	Uncommon	-	-
Unechis brevicauda	Mitchell's Short-tail Snake	Uncommon	1	
Tolma australis	[illegible]	[illegible]	1	R151

TABLE II - Locality II - (35°40'S by 140°46'W)

SPECIES	COMMON NAME	STATUS	NO. COLLECTED	S.A.M. REG. NO
Amphibolurus muricatus	Jacky Lizard	Uncommon	1	RNA
Amphibolurus pictus	Painted Dragon	Common	2	R1514
Amphibolurus fordi	Military Dragon	Common	-	-
Morethia obscura	Snake-eyed Skink	Common	-	-
Tiliqua rugosa	Shingleback	Uncommon	-	-

TABLE III - Members Present

Julian White (Leader)
John Fowler
Mark Galliford
Steve Giddings
John Hill
Shiela Hill
Allison Hill
Paul Huggins
Chris Hughes
Debbie Leggo
Simon Ostler
Paul Roach
Phillip Abbott (A.G.S.A.)

REPORT OF TRIP TO STURT RIVER GORGE

FEBRUARY 8TH 1976

Leader:- J. Fowler

Nine people in three vehicles attended this trip. The Sturt River, which enters the sea at the Patawalonga Glenelg, passes through a narrow gorge between Darlington and the Coromandel Valley. The area visited was around the flood control dam, the Sturt River Conservation Park being just to the west of the area surveyed. Several species of reptile were seen, the most interesting being Cunningham's Skink *(Egernia cunninghami)*, of which there are several colonies in the rock faces of the Groge. As these rock faces are readily accessable, the Cunningham Skinks here are both ideal for detailed study, and also quite vulnerable to human predation.

A brief visit was also made to the Cherry Gardens region, concerntrating on the area immediately north of the Power Station. Here it was hoped to find an interesting variant of the Striped Skink (similar to *Ctenotus uber)*, the true identify of which has yet to be determined. However, none were seen.

TABLE I :- Species seen in Sturt River Gorge (Lat. $35^{o}3'S$; Long. $138^{o}35'E$)

Scientific Name	Common Name
Egernia cunninghami	Cunningham's Skink
Ctenotus robustus	Striped Skink
Lerista bouganvillii	Bouganville's Skink
Hemiergis decresiensis	Three-toed Skink
Sphenomorphus quoyii	Common Water Skink
Underwoodisaurus milii	Barking Gecko
Amphibolurus decresii	Tawny Dragon
Pseudonaja textilis	Brown Snake
Pseudechis porphyriacus	Red-bellied Black Snake

Table II:- Species seen at Cherry Gardens (Lat. $35^{o}5'S$; Long. $138^{o}38'E$)

Scientific Name	Common Name
Egernia whitei	White's Skink
Tiliqua rugosa	Shingleback
Hemiergis decresiensis	Three-toed Skink
Lerista bouganvillii	Bouganville's Skink
Amphibolurus decresii	Tawny Dragon
Phyllodactylus marmoratus	Marbled gecko

Table III:- Members Present

John Fowler (Leader)
Mark Galliford
Steve Giddings
Allison Hill
John Hill
Shiela Hill
Paul Huggins
Julia Smith
Neil Smith

REPORT OF FIELD TRIP TO BALAH STATION

FEBRUARY 21 - 22, 1976

Leader: J. White

Aim:- To survey herpetofauna of this region

Report:- 11 herpetologists were transported to the area in 4 vehicles.

This area was briefly surveyed by the S.A.H.G. in November 1975, when a large range of species were sighted (see previous report). The habitat is mixed with red sand to limestone base, with areas of Bluebush, saltbush, Casurarine association, and areas of Spinifex, mallee, in dune formation.

The main aim of this revisit was to fill in gaps left by the previous survey, especially regarding nocturnal reptiles.

The weather was warm to hot, and dry. Several species were sighted which were not recorded on the previous trip, notably Gould's Goanna *(Varanus gouldii)* and the Beaded Gecko *(Lucacium damaeum)*.

Unfortunately, spotlighting, though extensive; failed to show any of the burrowing nocturnal snakes which are to be expected present in the area. However, it was of interest to see several Desert Skinks *(Egernia inornata)* active at night.

The Beaded Geckos were common at night, but restricted mainly to the sandy areas, especially in association with Spinifex. In contrast, no Byrne's Geckos *(Diplodactylus byrnei)* were seen this time, possibly because they are associated only with Bluebush plains, which is where we found them last time.

Finally, we must again thank the property owners who so readily let us survey their land.

TABLE I - SPECIES CAUGHT AT LOCALITY I - BALAH STATION (Lat. 33°46' Long. 139°45')

Species	Common Name	No. Seen	No. Collected	S.A.M. REG. No.
Tympanocriptus lineata	Earless Dragon	-	1	R 15157

TABLE II - LOCALITY II - BALAH STATION (Lat. 33°45' Long. 139°46')

Species	Common Name	No. Seen	No. Collected	S.A.M. REG. No.
Varanus gouldii	Gould's Goanna	1	-	-

TABLE III - LOCALITY III - BALAH STATION (Lat. 33°39' Long. 139°55')

Species	Common Name	No. Seen	No. Collected	S.A.M. REG. No.
Amphibolurus fordi	Military Dragon	6+	-	-
Amphibolurus barbatus	Bearded Dragon	3	-	-
Tiliqua rugosa	Shingleback	3	-	-
Egernia inornata	Desert Skink	6+	-	-
Egernia striolata	Tree Skink	4+	-	-
Cryptoblepharus boutoni	Briton's Skink	4+	1	R15159
Lerista meulleri	-	2	1	R15160
Lucasium damaeum	Beaded Gecko	5+	1	R15158

TABLE IV - Members Present

Julian White (Leader)
Stuart Pillman
Anne Pillman
John Fowler
Chris Hughes
Julia Smith
Mark Galliford
Simon Ostler
Garry Whisson
Terry Morely
M. Morely

REPORT ON FIELD TRIP TO BLOWHOLE CREEK AREA

MARCH 21ST, 1976

Trip Leader: Chris Hughes

Aim:- To conduct a short survey on the Herpetofauna of this area.

Report:- Four vehicles were used to transport eleven people to Blowhole Creek. The area looked at was approximately 7 km. S.E. of Cape Jervis. (Lat. 138°11'S Long. 35°39'E). The vegetation on the steep hills was very dense, and the dominant areas of interest were the numerous small rocky outcrops. There were two habitats looked at on the rocky hillsides, four species of skinks *Egernia whitii, Leilopisma guichenoti, Hervergis decresiensis* and *Lerista bouganvilli*, were found. One species of Gecko, *Phllodactylus marmoratus* and one species of Dragon, *Amphibolurus decresii* were also found.

The other area was along the banks of the creek. Here several *porphyriacus* were seen; one was examined and released.

TABLE 1 - species encountered Lat. 138°11'S Long. 35°30'E

SPECIES	COMMON NAME	FATE
Pseucechis porphyriacus	Red Bellied Black Snake	Released
Lerista bougainvilli	Bougainvilles Skink	6 collected - food
Ceilopisma guichenoit	Grass Skink	Released
Hemiergis decresiensis	Skink	10 collected - food
Egernia whitii	Whites Skink	Released
Phyllodactylus marmoratus	Marbled gecko	14 collected - food
Amphibolurus decresii	Tawny dragon	Released

TABLE II - members present

Chris Hughes (Leader)
Julian White
John Fowler
Stuart Pilman
Richard Elcock
Mark Galliford
Steve Giddings
Alison Hill
John Hill
Shiela Hill
Derik Stone

REPORT ON TRIP TO UNO RANGE, EYRE PENINSULA

JUNE 12 - 14, 1976

Leader:- C. Hughes

Aim:- To survey the reptile fauna of this area, and compare it to that of Corunna Hills.

Report:- Two vehicles were used to transport seven members to the survey area.

Uno Range is a large isolated rock outcrop situated on the north east of Eyre Peninsula, at the eastern most end of the Gawler Ranges. It is currently used as a pastoral property, being heavily utilized by both sheep and cattle, the former reaching most of the range. In consequence, it has few areas of completely natural scrub.

The range is approximately 10 km in length, this being a NW to SE axis, and varies between 1 - 2 km in width. Although there are several habitat types, three main habitat groups were examined by the Group. These were, the rocky areas of the Range, including covered slopes; the sand plains on the NW edge of the range, where both and saltbush were common, with interspersed mallee and native Quondong; the bluebush - saltbush flats surrounding the range, this being the most heavily grazed area.

On the way to Uno, the Group checked Lake Gilles Government Tank, to rescue any snakes which might have become trapped therein. On this occasion there were four Curl Snakes and one Western Brown trapped at the bottom, all in poor condition. Two of the Curl Snakes died shortly afterwards.

After first apraising Uno Homestead of the Groups activities, the area around and on the range was surveyed. Due to the vagaries of roads and terrain, the actual survey jumped from habitat to habitat, rather than systematically search each habitat in turn.

The first area looked at was the rocky habitat on the western side of the bluff, (Table II) and the sandy saltbush area at the base of the bluff, between rock outcrops. This latter section yielded only a Striped skink and a Shingleback (asterisks, Table II).

Next stop was a sandy area, thick with *Triodia* near the bluff, grading through mallee, to saltbush on the plains. As at Corunna, the *Triodia* yielded *Diplodactylus elderi*, *Tiliqua branchialis*, but, no *Delma*. The mallee section contained evidence of *Amphibolurus cristatus*, though no specimens were seen. (See Table III)

Camp was established in this area, but spotlighting was unseccessful. However, a search of an adjacent rockface in the morning revealed a large male Peninsula Dragon *(Amphibolurus fionii)*.

A further area of *Triodia* and Mallee was examined, and this time two species of legless lizard were found, as well as some species found in the other Triodia area. *Tiliqua branchialis* was particularly common

under the *Triodia*. (See table IV).

Finally the homestead area was examined. This had areas of rubbish, saltbush plain, and some mallee. Quite a wide range of reptiles lived in this area (Table V), and several frogs *(Lymnodynastes tasmaniensis)* were found in abandoned wells. In addition to those species actually seen, the landowner mentioned several species of reptiles he had discovered in the area. From his descriptions, these appeared to be the Crested Dragon *(Amphibolurus cristatus)*, the Bearded Dragon *(Amphibolurus vitticeps)*, Gould's Goanna *(Varanus gouldii)*, Western Brown Snake *(Pseudonaja nuckalis)*, King Brown Snake *(Pseudechis australis)*, Curl Snake *(Suta suta)* and the Desert Banded Snake *(Vermicella bertholdii)*.

TABLE I Species at Lake Gilles Tank (Lat. 32°37'S by Long. 136°53'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Suta suta	Curl Snake	-	4	R.N.A.
Pseudonaja nuchalis	Western Brown Snake	-	1	R.N.A.

TABLE II Species at Stop I - Bluff - rocky (Lat. 32°42'S by Long. 136°43'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Ctenotus robustus	Striped Skink	3	1	R15352
Ctenotus regius	Striped Skink	-	1	R.N.A.
Egernia stokesii	Gidgee Skink	3	-	-
Egernia striolata	Tree Skink	4	-	-
Tiliqua rugosa	Shingleback	2	-	-
Gehyra punctata	Dtella	10	1	R15354
Heteronotia binoei	Bynoe's Gecko	10	-	-
Diplodactylus ciliaris	Spiny-tailed Gecko	-	1	R15361
Underwoodisaurus milii	Thick-tailed Gecko	1	1	R15355
Delma nasuta	Legless Lizard	-	1	R15353

TABLE III Species at Stop II - Sandy, triodia, NW of Bluff (Lat 32°40'S by Long. 136°42'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Egernia inornata	Desert Skink	2	1	R15351
Tiliqua branchialis	Mourning Skink	1	-	-
Lerista muelleri	Burrowing Skink	-	1	R15359
Gehyra punctata	Dtella	5	-	-
Heteronotia bynoei	Byrnoe's Gecko	5	-	-
Diplodactylus elderi	Jewelled Gecko	1	1	R15358

TABLE IV Species at Stop III - Sandy, triodia, mallee, near Uno homestead (Lat. 32°40'S, Long. 136°42'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Tiliqua branchialis	Mourning Skink	5	2	R.N.A.
Menetia greyii	-	1	1	R15356
Delma australis	Legless Lizard	1	1	R.N.A.
Lialis burtonis	Burton's Legless Lizard	2	1	R.N.A.

TABLE V Species at Uno Homestead

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus fionii	Peninsula Dragon	3	1	R15350
Tympanocriptus lineata	Earless Dragon	1	1	R15360
Ctenotus robustus	Striped Skink	5	-	-
Gehyra variegata	Dtella	5	-	-
Heteronotia binoei	Byrnoe's Gecko	5	-	-
Diplodactylus vittatus	Stone Gecko	2	1	R15357
Underwoodisaurus milii	Thick-tailed Gecko	3	-	-

REPORT OF TRIP TO WOODSIDE - ECHUNGA AREA

JULY 25TH, 1976

Leader:- C. Hughes

A group of nine herpetologists attended this day trip to areas in and around the Mount Lofty Ranges.

The first area visited was at Bradbury, a hilly region near the Onkaparinga River. This was a thickly vegetated area with patches of almost inpenetrateable bramble and gorse bushes. The hillsides were studded with interesting rocky patches and outcrops, which harboured several species of reptiles and frogs, most notable being Cunningham's Skink *(Egernia cunninghami)* (See table I).

The second area examined was nearer Adelaide, in the vicinity of Sturt Creek. Here vegetation was less dense than at the previous area, the country being otherwise very similar, with rocky outcrops, around which several species were seen (See table II).

From here, the group moved up a tributary of Sturt Creek where a Brown Snake was discovered, before returning to Adelaide.

TABLE I:- Onkaparinga River - (Lat. 35°04'S Long. 138°45'E)

Species	Common Name	No. Seen
Egernia cunninghami	Cunningham's Skink	1
Hemiergis decresiensis	-	> 10
Lerista bouganvilli	Bouganville's Skink	3
Phyllodactylus marmoratus	Marbled Gecko	> 10
Pseudophryne bibroni	Froglet	> 5
Lymnodynastes tasmaniensis	Spotted Frog	> 5

TABLE II:- Sturt Creek (Lat. 35°02'S Long 138°35'E)

Species	Common Name	No. Seen
Amphibolurus barbatus	Bearded Dragon	1
Egernia cunninghami	Cunningham's Skink	4
Ctenotus robustus	Striped Skink	2
Hemiergis decresiensis	-	5
Phyllodactylus marmoratus	Marbled Gecko	5
Underwoodisaurus milii	Thick-tailed Gecko	1
Delma molleri	Legless Lizard	1
Unechis flagellum	Little Whip Snake	1
Pseudophryne bibroni	Froglet	5

TABLE III - Sturt Creek tributary (Lat. 35°03'S, Long. 138°34'E)

Species	Common Name	No. Seen
Hemiergis decresiensis	-	5
Phyllodactylus marmoratus	Marbled Gecko	5
Pseudonaja texitillis	Brown Snake	1

TABLE IV - Members Present

Chris Hughes (Leader)
Graham Armstrong
Werner Brunn
John Fowler
Mark Galliford
Steve Hughes
Nick Joy
Darryl Levi
Julian White

REPORT ON TRIP TO MANNUM

AUGUST 21 - 22, 1976

Leader:- C. Hughes

Two vehicles carried 6 people to the survey sites.

For most of the day of the 21st the weather was fine and warm for the time of year. Our first stop was about 2 km out of Gumeracha in the Adelaide Hills. Here the Onkaparinga comes close to the road and many rocky and reedy areas are to be found. One black snake *(Pseudechis porplyriaeus)* was collected from the area.

Next we stopped at an area near rocky point and had a look around the swamp and cliffs. One of the most common species encountered on this survey, with the exception of a few smaller skinks, was the long necked tortoise *(Chelodina longicollis)* most of which were found dead along the banks of the river and lagoons where the water level had dropped.

Throughout the first day the areas looked at were basically similar in habitat content mainly limestone cliffs and wooded flats. (Table I)

On the second day we were joined by several other members of the group. Despite the drizzle and unpleasant conditions we managed to turn of a fair variety of reptiles at an igneous outcrop a few km north of Mannum on the east side of the river (Table II).

TABLE I Species on R. Murray, West Bank, near Mannum

Species	Common Name	Status
Chelodina longicollis	Long-neck tortoise	Abundant
Cryptoblepharus boutoni	Bouton's Skink	Common
Morethia sp.	Snake-eyed Skink	Uncommon
Menetia greyi	-	
Tiliqua rugosus	Shingleback	Common
Phyllodactylus marmaratus	Marbled Gecko	Common
Underwoodisaurus milii	Thick-tailed Gecko	Common

TABLE II Species on R. Murray, East Bank, near Mannum

Species	Common Name	Status
Ctenotus robustus	Striped Skink	Uncommon
Hemiergis decresiensis	-	Uncommon
Cryptoblepharus boutoni	Bouton's Skink	Common
Menetia greyi	-	
Tiliqua rugosa	Shingleback	Common
Amphibolurus barbatus	Bearded Dragon	Uncommon
Phyllodactylus marmoratus	Marbled Gecko	Common
Underwoodisaurus milii	Thick-tailed Gecko	Common
Pseudonaja textillis	Brown Snake	Common

TABLE III - Members Present

Chris Hughes (Leader)
Graham Armstrong
Werner Brunn
Richard Elcock
John Fowler
Mark Galliford
Peter Hartley
Steve Hughes
Simon Ostler
Julia Smith
Julian White

REPORT ON TRIP TO AREA SOUTH OF SCORPION WELL CONSERVATION PARK

SEPTEMBER 25 - 26, 1976

Leader:- J. White

Aim:- To demonstrate to Peter Aitken (S.A. M. Curator of Mammals), where Pygmy Possums were collected in January 1976, so that he could take soil and plant samples.

Report:- Two vehicles were used to transport eleven members to the survey area.

Weather conditions were mild, with varied cloud cover, and occasional strong winds, but enough sunlight to encourage reptile activity. The same area visited previously by the S.A.H.G. was reviewed, and the sites of capture of the Pygmy Possums, located soil and plant samples being taken by Peter Aitken. No pitfall traps were taken on this trip, and no Pygmy Possums were found.

Far fewer reptiles were seen on this occasion, and in particular, the Military Dragon, which had been very common in January, was only seen in small numbers. No *Ctenotus brooksi* were seen despite extensive searches. The only species of snake seen was the Master's Snake *(Drysdalia masteri)*, two specimens being collected and retained for breeding purposes.

Although unspectacular herpetologically, the trip did fullfil its aim, to aid the Mammal section of S.A.M.

On the way back to Adelaide, a section of the River Murray was visited. We examined the area of the east bank, half way between Tailem bend and Murray Bridge. This area was pastureland, with a levee, and irrigation ditches, and some riverside trees beyond the levee. Water skinks *(Spheno-morphus quoyii)* were quite numerous on the sides of the levee, while several Tiger Snakes *(Notechis scuttatus)* were seen in the irrigation ditches. A juvenile specimen was collected.

TABLE I Species seen at Scorpion Well area (34° 41'S by 140° 47'W)

Species	Common Name	No. Collected	S.A.M. Reg. No.
Amphibolurus fordi	Military Dragon	2	RNA
Amphibolurus vitticeps	Bearded Dragon	-	-
Amphibolurus pictus	Painted Dragon	2	RNA
Morethia obscura	Snake-eyed Skink	-	-
Ctenotus uber	Striped Skink	1	R 15618
Drysdalia masteri	Master's Snake	2	RNA
Delma australis	Legless Lizard	1	R 15619

TABLE II Species seen at Murray River

Species	Common Name	No. Collected	S.A.M. Reg. No.
Sphenomorphus quoyii	Water Skink	1	RNA
Notechis scuttatus	Tiger Snake	1	RNA

TABLE III Members present

Julian White (Leader)
Tricia Andersen
Graham Armstrong
Sue Armstrong
Chris Bourn
Werner Brunn
John Fowler
Mark Galliford
Andrew Todd
Ian Warmington
Richard Warmington

THOMPSONS BEACH - PORT PARHAM SURVEY

13TH-14TH AND 20TH-21ST NOVEMBER, 1977

TRIP LEADER: Chris Hughes

AIM: To conduct a widespread survey of the Northern beach areas and red sand dune areas of Port Paraham, S.A. Although both visits were initially organized as spotlighting trips, some members stayed overnight and continued the survey during daylight hours.

REPORT: On the evening of Saturday 13th, members met at a pre-arranged site on the Thompson's beach road. Here we split up into two groups; one group looked at the Thompson's beach area and the other looked at the coastal dune area of Port Paraham.

Both areas are basically the same in habitat being medium to coarse white sand dune areas with approximately 60% vegetation cover in most areas. The vegetation consists mainly of low shrubs, bushes and pigface.

An initial look around the areas before dusk uncovered *(Tiliqua rugosa)*, *(Hemiergis peronii)* and *(Morethia adelaidensis)* in both areas is fairly large numbers. At Thompson's beach one *(Egernia multiscutata)* was caught but later escaped.

After dark we took the cars onto tracks and drove slowly along looking for reptiles in the headlight beams. At both areas *(Diplodactytus ciliaris)* were found. (Three of the six were collected since they were pregnant and would be useful for observation purposes). At Port Paraham, *(Phylodactylus marmoratus)* was found and at Thompson's beach one *(Typhlina sp.)* was found on a track. Around 10.00 p.m. most members went home, but five stayed on to continue the survey in the morning.

The next day the searching was mainly confined to the red sand dunes of Port Paraham, as was the next weekends searching. Here the vegetation was mainly 4 - 8 feet Boxthorn bushes, with approx. 30% low grasses and herbage cover and in some areas, Mallee. The species found are listed in table III.

TABLE I - Species seen at Thompson's Beach

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Typhlina sp.	Blind Snake	-	1	
Egernia multiscuttata	Sand Skink	1	-	-
Tiliqua rugosa	Shingleback	5	-	-
Tiliqua occipitalis	Western Bluetongue	-	2	R.N.A.
Lemiergis peroni	Skink	5	-	-
Morethia adelaidensis	Snake-eyed Skink	5	-	-
Diplodactylus cilliaris	Spiny Gecko	3	1	

TABLE II - Species seen at Port Parham

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Pseudonaja textillis	Brown Snake	2	-	-
Typhlina sp.	Blind Snake	-	1	
Tiliqua rugosa	Shingleback	5	-	-
Lemiergis peroni	Peron's Skink	5	-	-
Lerista picturata	Burrowing Skink	3		
Morethia adelaidensis	Snake-eyed Skink	5	-	-
Amphibolurus barbatus	Bearded Dragon	1	-	-
Amphibolurus pictus	Painted Dragon	2	-	-
Diplodactylus ciliaris	Spiny Gecko	1	2	
Phyllodactylus marmoratus	Marbled Gecko	1	-	-
Varanus gouldii	Gould's Goanna	3	-	-

TABLE III - Members Present

Chris Hughes (Leader)
Werner Brunn
John Fowler
Mark Galliford
Steve Hughes
Nick Joy
Darryl Levi
Andrew Mower
Julia Smith
John Templer
Julian White

YORKE PENINSULA SURVEY

27TH & 28TH NOVEMBER, 1976

Trip Leader: C. Hughes

Aim: This was a preliminary survey of the Peninsula. Our main objective was to find areas of natural scrub which could harbour valuable reptile populations, but are not parts of National Parks or Reserves.

Report: Three vehicles transported eight people to the survey sites.

Yorke Peninsula is very heavily grazed and farmed, so little scrub is to be found in the central regions, this however does not seem to have affected the populations of larger snakes, notably *(Pseudonaja textillis textillis)* & *(Pseudonaja textillis inframacula)* which are reportedly very common, at least in the mid to southerly regions. One of each of these snakes were found; the later, a Peninsula Brown Snake was collected. All other specimens were found in the coastal regions which were white sand based with low, medium to dense scrub at Tiddy Widdy Beach (34° 25' S Lat. by 137° 25' E Long.), and 1 - 3 metre scrub on limestone based soil near Pt Julia (34° 40' S Lat. by 137° 49' E Long.), and Daly Heads where we spent the night.

Unfortunately very few reptiles were seen at all on the Peninsula south of Ardrossan; the majority were found at Tiddy Widdy Beach 3 km north of Ardrossan. This was an area of extensive sand duens between farmland and the sea. It measured up to 1 km across in some places and several kilometres long in a N-S direction. The area is at present the site for several new holiday shacks according to information given by locals.

TABLE I - Species encountered at Tiddy Widdy Beach (Lat. 34° 25' S by Long. 137° 52' E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus barbatus	Bearded Dragon	1	-	-
Amphibolurus pictus	Painted Dragon	4	-	-
Hemiergis peroni	Peron's Skink	6	-	-
Morethia adelaidensis	-	1	-	-
Tiliqua rugosa	Shingleback	5	-	-
Varanus gouldii	Gould's Goanna	1	-	-
Acanthophis antarcticus	Death Adder	1	1	-

TABLE II - Species encountered at Pt Julia (Lat. 34° 40'S by Long. 137° 49' E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Phyllodactylus marmoratus	Marbled Gecko	8	-	-

TABLE III - Species encountered in farmland near Warooka. (Lat. 34° 56' S by Long. 137° 25' E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Tiliqua rugosa	Shingleback	5	-	-
Pseudonaja textillis textillis	Common Brown Snake	1	-	-
Pseudonaja t. inframacula	Peninsula Brown Snake	1	1	

TABLE IV - Members Present

Chris Hughes (Leader)
Graham Armstrong
Sue Armstrong
Werner Brunn
Mark Galliford
Andrew Mower
Simon Ostler
John Templer

REPORT OF PINAROO - MOUNT GAMBIER SURVEY

27TH DECEMBER '76 - 1ST JANUARY '77

TRIP LEADER: C. Hughes

Aim: To conduct a sweeping survey of the entire South-east corner of the state, noting habitats and areas worthy of more intense survey.

Report: Two cars provided transport for nine people to the survey sites. The method of sampling employed was several intermittant stops at places of potential herpetological interest, from half an hour to overnight duration.

On the morning of 27th December we headed directly to Pinaroo. The first stop was an area of low Mallee and medium-density scrub on yellow sandy ground approximately 25 km south of Pinaroo, (36°32'S Lat. by 140°46'E Long.). Species observed were *(Amphibolurus fordi) (Phyllodactylus marmoratus) (Tiliqua rugosa) (Ctenotus sp.)* - (seen only); none were collected.

The second area visited was approximately 55 km north of Bordertown (Lat. 36°00'S, Long 140°45'E). The vegetation in this area was mainly tall eucalypts on hard soil; habitats were mainly logs and loose bark fallen from trees.

The species encountered were *(P. marmoratus) (Lerista bougainvilli)* and *(Morethia sp.)* .

The next day we moved on South towards Penola and stopped near Mosquito Creek (37°87'S Lat. 140°50'E Long.). Here the specimen found was a large unbanded *(Notechis scuttatus);* this was collected. From there we moved to a larger swamp (37°09'S Lat. 140°45'E Long.) here three large Copperheads *(Australaps superba)* were found. One was collected.

We then moved south east of Penola towards the Dismal swamp. We stayed on Lynwood park, which has been proclaimed an unofficial wildlife sanctuary. The landowner, however, did not mind us looking around the swamps and camping overnight. The only reptiles seen were tiger snakes of which we found eight specimens despite the inclement weather.

The next day we visited a few more swamps but the only specimens seen were again Tiger snakes and several Green Golden Bell frogs *(Litoria aurea)* along with a number of *(Crinia sp.)* and one dead Tortoise *(Chelodina longicollis).*

We then moved further south and stayed in a sand dune area on the coast near Blackfellows caves; only two specimens were caught here, *(Egernia whitei)* and *(Hemiergis peroni).*

The following day we headed north again towards Bool Lagoon hoping to find some suitable areas outside the Reserve area. Much of the day was spent travelling and around 6.00 p.m. we stopped at an area approx. 10 km South of Bool Lagoon (37°13'S Lat by 140°42'E Long.) Here we found a Blotched Bluetongue *(Tiliqua nigrolutea),* this specimen overheated and died in the car.

Another Copperhead was collected in a nearby swamp. The landowner was very helpful in this area; he spent several hours with us both in the field and at the campsite and managed to turn up a Tortoise *(Chelodina longicollis)* while doing duties around the station (this specimen was released).

The next day was spent travelling as was the last day of the trip, January 1st, 1977.

As many short stops were made, tables list species seen in habitat types, rather than map locations.

TABLE I - Species encountered in swamp habitats

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nc
Notechis scuttatus	Tiger Snake	21	1	R.N.A.
Austrelaps superba	Copperhead Snake	4	-	-
Tiliqua nigrolutea	Blotched Blue tongue	1	-	-
Phyllodactylus marmoratus	Marbled Gecko	5	-	-
Chelodina longicollis	Long-neck Tortoise	3	-	-
litoria aurea	Green and Golden Bell Frog	10	3	R.N.A.
Crinia signifera	Froglet	10	-	-
Lymnodynastes tasmaniensis	Marbled Frog	10	-	-
Lymnodynastes dumeruli	Bull Frog	2	-	-
Lymnodynastes peroni	Peron's Frog	2	1	R.N.A.

TABLE II - Species encountered in Sandhill (Blackfellows Caves)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. N
Egernia whitei	Whites Skink	1	1	R.N.A.
Hemiergis peroni	Peron's Skink	4	-	-

TABLE III - Species encountered in Eucalypt Forrest (Lat $36^{o}00'S$ Long. $140^{o}145'E$)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. N
Phyllodactylus marmoratus	Marbled Gecko	3	-	-
Lerista bouganvillii	Bouganville's Skink	2	-	-
Morethia sp.	Snake eyed Skink	1	-	-

TABLE IV - Species encountered in dense scrub near Pinaroo (Lat $35^{o}32'S$, Long $148^{o}46'E$)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nc
Amphibolurus fordi	Military Dragon	4	-	-
Amphibolurus barbatus	Bearded Dragon	1	-	-
Ctenotus sp.	Striped Skink	5	-	-
Tiluqua rugosa	Shingleback	2	-	-
Phyllodactylus marmaratus	Marbled Gecko	5	-	-

TABLE V - Members Present

Chris Hughes (Leader)
Werner Brunn
Mark Galliford
Peter Hartley
Andrew Mower
Simon Ostler
Andrew Todd
Ian Warmington
John Warmington

REPORT OF TRIP TO MIDDLEBACK RANGES, EYRE PENINSULA

January 29 - 31, 1977

Leader:- J. White

Aim:- To review the reptile fauna of the south Middleback Ranges.

Report:- Thirteen herpetologists were transported to the survey area in five vehicles. The weather was warm to hot during the day, with cloudless sky, and moderate cool breezes, and cool to cold nights.

The Middleback Ranges extend in a broken chain, from Iron Duke in the south to Iron Knob, some 90 km to the north. Most ot the ranges are rich in Iron, and the whole chain is under BHP mining lease, although only the northern end is being mined at present. However, the southern end is being tested for mining, and it is possible that this will also disappear in the future. In addition, the whole area is grazed by sheep and goats.

The southern end of the chain, approximately 70 km south of Whyalla, is relatively untouched, though nevertheless crisscrossed with mining tracks. It was this area, centred around Iron Duke and Iron Duchess, and the flat mallee below, which was of principal interest to the S.A.H.G. The ranges here, contain numerous rock outcrops, capable of sheltering many reptiles, with mixed scrub on the slopes merging into the mallee plains around, with areas of spinifex, bluebush and saltbush. The diversity of habitats should favour high reptile populations, with a great deversity of species.

Although the S.A.H.G. only spent one day, and two nights searching the area, a wide range of lizards were seen, but no snakes. These are listed in table I. However, the Western Herpetology Group has also visited this area on numerous occasions, and the species which they have recorded are listed in table II.

On the rocky slopes, the Peninsula Dragon *(A. fioni)* was very common, while skinks were far less abundant, at least in terms of observations. Predictably, both *Egernia stokesii* and *Egernia striolata* were present amongst the rocks, as was the Striped Skink *(Ctenotus sp.)*.

Reptiles seemed far less abundant on the plains, where the Crested Dragon *(A. cristatus)* was most frequently seen. Surprisingly, no Shinglebacks *(Tiliqua rugosa)* or Bearded Dragons *(Amphibolurus vitticeps)* were seen.

At night, the sandy areas at the base of the range were most populous with reptiles, eight species of geckoes being seen. Most notable of these was the Knob-tail gecko *(Nephrurus stellatus)*, a large gecko which is adapted to burrowing in the sand, coming out at night to forage for food. Also active at night on the sandy plains, though also seen during the day, was the Desert Skink *(Egernia inornata)*, another burrowing lizard.

In common with many isolated rocky ranges in South Australia, the Middleback Ranges harbour a wide variety of reptiles. Many of these are of considerable herpetological interest, such as the Carpet Python, but all are represented elsewhere on Eyre Peninsula. Even the complete destruction of these ranges would not threaten the extinction of a single species of reptile. However, the ranges do define the southern-most limit of several species and so some care in preserving part of this area would seem worthwhile. Discussions with B.H.P. indicate that this will take place anyway, as only part of the ranges are economically viable for mining. Although Iron Duke is listed as the locality of specimens caught, all are represented throughout the range, and no herpetological significance should be placed on Iron Duke itself. The S.A.H.G. gratefully acknowledges the assistance given by B.H.P. in conducting this survey.

TABLE I - Species seen on and around Iron Duke (Lat. 33°16'S by Long. 137°7'E)

SPECIES	COMMON NAME	No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus fionii	Peninsula Dragon	30	4	
Amphibolurus cristatus	Crested Dragon	15	4	
Ctenotus schomburgki	Striped Skink	-	1	
Ctenotus	Striped Skink	2	2	
Tiliqua scincoides	Common Blue tongue	1	-	
Morethia	-	-	1	
Cryptoblepharus boutonii	Snkae-eyed Skink	3	2	
Egernia striolata	Tree Skink	5	1	
Egernia stokesii	Stoke's Skink	5	-	
Egernia inornata	Desert Skink	3	3	
Underwoodisaurus milli	Thick-tailed Gecko	-	1	
Nephrurus stellatus	Knob-tailed fecko	-	1	
Rhynchoedeura ornata	Beaded Gecko	-	1	
Leucasirm damaerm	Beaded Gecko	-	1	
Gehyra variegata	Dtella	15	1	
Heteronotia binoeii	Bynoe's Gecko	10	1	
Phyllodactylus marmoratus	Marbled Gecko	1	-	
Diplodactylus ciliaris	Spiny Gecko	-	1	
Lialis burtonis	Burton's Legless Lizard	1	-	

TABLE II - Additional species recorded from the area by W.H.G.

Diplodactylus elderi	Jewelled Gecko
Tiliqua branchialis	Mourning Skink
Delma nasuta	Legless Lizard
Varanus gouldii	Gould's Goanna
Morelia spilotes	Carpet Python
Acanthophis antarcticus	Death Adder
Demansia Psammophis	Yellow-faced Whip Snake
Pseudonaja nuchalis	Western Brown Snake
Simoselaps fascidatus	Desert Burrowing Snake

TABLE III - Members Present on Trip.

Julian White (Leader)
Tricia Andersen
Graham Armstrong
Sue Armstrong
Werner Brunn
John Fowler
Mark Galliford
Steve Giddings
Chris Huges
Nick Joy
Debbie Leggo
Simon Ostler
Julia Smith

REPORT OF A DAY TRIP TO THE SOUTHERN MT LOFTY RANGES:

FEBRUARY 13TH, 1977

LEADER:- G. Armstrong

Aim: On Sunday 13th February, the Herpetology Group visited three separate areas of the Southern Mt Lofty Ranges to endeavour to obtain a specimen of the Mt Lofty Ranges Copperhead, *Austrelaps sp.* Although our efforts went unrewarded in this aspect we did manage to capture several reptiles for the display at Cleland N.P.

Report: The Group met at Stirling at 10.00 a.m. On the preceeding day the Ranges had received a substantial amount of rain for the time of year and we were eager to search for reptiles drying out in the late morning sun. However, the temperature escalated rapidly and by noon all diurnal reptiles encountered were at optimum activity.

The first area visited was an area of natural woodland on the western slopes of Mt George, approx. 3 km. East of Stirling. It was here where the majority of the reptiles were found. Between 10 a.m. and 12 noon we searched the slopes uncovering reptiles not yet aroused by the sun which was still concentrating its rays on the easterly slopes. Reptiles recorded from this area are listed in table one.

Following lunch we moved south to an area just south of Mylor. Here, two separate habitats were looked at by the member; An area of natural scrub on the eastern side of the road and a pastoral sketch of land, dotted with small artificial dams, on the western side of the road. Reptiles recorded from both habitats are listed in table two.

At 3.30 p.m. we moved to an area of isolated scrub, near Piccadilly Valley, where members had previously recorded sighting a Copperhead. Reptiles encountered here are listed in table three.

At 5.30 p.m. the members decided to call it a day and turned their vehicles for home.

TABLE 1. Mt George (Lat 35°00'S by Long. 138°45'E)

Species	Common Name	No. Seen	No. Collected
Unechis flagellum	Little Whip Snake	-	2 for Cleland display
Egernia whitii	White's Skink	-	2 for Cleland display
Leiolopisma trilineata	3 Lined Skink	-	1 for S.A.M.
Leiolopisma guichenoti	Grass Skink	Numerous	10 for food items
Hemiergis decresiensis	3 toed Skink	Numerous	5 for food items
Phyllodactylus marmoratus	Marbled Gecko	2	-

TABLE 2. Mylor (Lat 35°03'S by Long 138°45'E)

Species	Common Name	No. Seen (Pastoral area)	No. Seen (Natural area
Leiolopisma guichenoti	Grass Skink	Numerous	Numerous
Tiliqua rugosa	Stump-tailed lizard	2	-
Amphibolorus decresi	Tawny Dragon	-	6
Psudechis porphyriacus	Red-bellied Black Snake	1	-

No specimens where collected from this area

TABLE 3. Piccadilly Valley (Lat 34°59'S by Long 138°44'E)

Species	Common Name	No. Seen	No. Collected
Egernia whitii	White's Skink	1	5 for food items
Leiolopisma guichenoti	Grass Skink	Numerous	-

Members Present: Graham Armstrong (Leader)
Werner Brunn
Mark Galliford
Steven Giddings
Andrew Mower
Simon Ostler

Visitor: Hans Wilifred

TRIP REPORT - MURRAY MOUTH COMPLEX

26TH & 27TH FEBRUARY, 1977

Trip Leader: C. Hughes

Ten members attended this two day survey of the more easily accessible and unpreserved areas of the Murray Mouth.

The weather over the weekend varied from cool and overcast to fine and mild over both days with the temperature during the day around 22°-25°C.

A great deal of the area looked at was farmland but areas of reeds and long grass were to be found near the waters edge. In a few areas a few hectares of scrubland had been left so we checked these out also.

Having no access or permits to enable us to collect on the Younghusband Peninsula we moved on to the sandhills near the Goolwa barrages which seemed to be more or less a continuation of the same sort of vegetation and morphology.

Speaking with locals on the Nurrung Peninsula we were told of natural crossbreeds between Tiger Snakes and Copperheads, one of which was supposedly lodged with the South Australian Museum and identified as such. From this area we collected two Tiger Snakes, one of which was exceptionally dark, with even a dark belly and only very thin dorsal stripes. The other, a juvenile was much lighter in colour with very obvious stripes.

TABLE I - Stop I - yellow sand with medium density 1 - 2 metre shrub vegetation. (Lat. 35° 44' S by Long. 139° 20' E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No
Lerista bougainvilli	Bougainville's Skink	2	1	

TABLE II - Banks of Lake Albert, near farm buildings. (Lat. 35° 22' S by Long. 139° 14' E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No
Hemiergis peroni	Peron's Skink	3	2	
Notechis scutatus	Tiger Snake	2	2	

TABLE III - (Lat. 35° 32' S by Long. 138° 45' E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No
Amphibolurus barbatus	Bearded Dragon	3	0	-
Amphibolurus pictus	Painted Dragon	5	1	
Lerista frosti	Burrowing Skink	4	1	
Hemiargis peroni	Peron's Skink	8	1	
Tiliqua rugosa	Shingleback	2	0	-

TABLE IV - Members Present

Chris Hughes (Leader)
Werner Brunn
Mark Galliford
Steve Giddings
Nick Joy
Debbie Zeggo
Simon Ostler
Julia Smith
Andrew Todd
Ian Warmington

NORTHERN FLINDERS - COPLEY SURVEY

8 - 11TH APRIL, 1977

Trip Leader: G. Armstrong

Aim:- To conduct a survey of the Northern Flinders around Mt Serle and Brachina Gorge and compare reptile populations with those found in the flatter North Westerly areas around Copley.

Report:- After a days drive we reached Mt Serle homestead at approximately 4.30 p.m. This was the first major stop, although we had made a few short stops along the way. It was here that those on the trip saw their first Red Barred Dragons *(Amphibolurus vadnappa)*. These proved to be fairly common in the area with upwards of 15 specimens being seen during our stay.

Two exceptional finds were made here. Firstly, on top of Mt Serle proper a pair of Tawny Dragons *(Amphibolurus decresii)* were found. These are very similar to the red barred dragons in form but colouration is quite different. Secondly, and perhaps most importantly, an adult Velvet Gecko *(Oedura marmorata)* was also found on top of Mt Serle. This is believed to be the only reliable specimen of the entire genus to be found in South Australia.

On the afternoon of the 9th April we broke camp and headed north west for the flat and sandy areas north of Lyndhurst near Farina.

We checked the ruin of Farina and found a large but very thin sand Goana *(Varanus flaurufus)* in a well, along with a Bearded Dragon *(A. vitticeps)* in similar condition. (Both specimens were rescued). In nearby sandhills we collected some of the interesting skinks and dragons which we expected to find, including *(A. nuchalis)* and *(Ctenotus brooksi)*.

Although we made a great number of very short stops around both locations, all but a few species were found at the longer stops, so in view of the nature of the trip the more insignificant stops will be omitted from the records.

It is, however, notable that a few species were represented only by dead specimens and sloughs in wells and ruins. These were *(Sphenomorphus richardsoni (Pseudonaja nuchallis)* and *(Pseudechis australis)*.

On the afternoon of 10th April we once again broke camp and moved south to Brachina Gorge in order to make the trip home the next day somewhat shorter.

TABLE I - Reptiles recorded from Mt Serle (Lat. 30° 32'S; Long. 138°53'E).

Species	Common Name	No. Seen	No. Collected	Museum Reg. No.
Diplodactylus intermedius	Spiny tailed Gecko	1	-	-
Gehyra variegata	Dtella	numerous	-	-
Gehyra punctata	Rock Dtella	numerous	-	-
Heteronotia binoei	Prickly Gecko	numerous	-	-
Oeudura marmorata	Velvet Gecko	-	1	R.N.A.
Tympanocryptis lineata	Earless dragon	-	1	R.N.A.
Delma australis		-	1	R15958
Amphibolurus vadnappa	Red-barred dragon	5	6	R15956
Amphibolurus decresii	Tawny dragon	-	2	R15957
Cryptoblepharus boutonii	Snakey-eyed skink	-	1	R15960
Ctenotus robustus	Striped skink	1	1	R15962
Egernia margaretae personata		12	2	R15961
Egernia stokesii	Stoke's skink	10	-	-
Morethia boulengeri		numerous	2	R15959
Trachydosaurus rugosus	Shingleback	2	-	-
Lerista muelleri		15	3	R15964
lerista punctatovittata		-	1	R15963
Demansia psammophis	Yellow faced whip snake	-	1	R.N.A.
Unechis brevicauda	Mitchel's short tailed snake	-	2	R.N.A.
Tiliqua branchialis	Mourning skink	-	2	R15953

TABLE II - Reptiles recorded near Farina (Lat. 30°2'S; Long. 138°16'E).

Species	Common Name	No. Seen	No. Collected	Museum Reg. No
Heteronotia binoei	Prickly Gecko	numerous	-	-
Amphibolurus vitticeps	Inland bearded dragon	-	1	R.N.A.
Amphibolurus pictus	Painted dragon	10	3	R15944
Amphibolurus fordi	Military dragon	numerous	6	R15945
Amphibolurus nuchalis	Central netted dragon	-	2	R.N.A.
Tympariocryptis tetraporophora	Earless dragon	2	1	R15946
Varanus flaviorufus	Sand Goanna	-	1	-
Ctenotus regius	Striped skink	8	2	R15947
Ctenotus brooksi	Striped skink	numerous	4	R15948
Ctenotus strauchii	Striped skink	numerous	2	R15949
Menetia greyi	Grey's skink	-	3	R15950
Lerista orientalis		-	1	R15952
Lerista bipes			2	R16951
Trachyolosaurus rugosus	Shingleback	2	-	-

TABLE III - Members Present

Graham Armstrong (Leader)
Sue Armstrong
Steve Berry
Werner Brunn
John Fowler
Mark Galliford
Chris Hughes
Debbie Leggo
Brian Millar
Simon Ostler

TRIP REPORT OF DAY TRIP TO MURRY BRIDGE AREA

DATE : 24TH. JULY 1977 TRIP LEADER : GRAHAM ARMSTRONG

After meeting at the War Memorial at 8.30a.m. the Group departed in three vehicles. The weather was cold and windy and it was overcast for most of the time. After crossing the river at Murray Bridge we stopped and searched a sand dune region approximately 1 km. from the town.Reptiles observed at this location are recorded in table 1.

Our next stop was at Mannum Waterfalls. This is an isolated rocky area surrounding a permanent creek which is located about 10 km. south of Mannum. Reptiles discovered at this location are recorded on table 2.

Due to the cold weather the trip was only moderately successful. A total of three reptiles were collected for the Musem.

TABLE 1. Species recorded at Murray Bridge.
Habitats looked at. 1. Edge of the river.
2. Limestone cliffs bordering river.
3. Sand dunes behind limestone cliffs.

SPECIES	COMMON NAME	No. SEEN	No. COLLECTED.	S.A.M. Reg. No.	HABITAT
Tympanocriptis lineata	Earless Dragon	-	2		Sand dunes.
Lerista bougainvillii	Bougainville Skink	2	-	-	Sand dunes.
Morethia sp.	-	10	-	-	Limestone c
Sphenomorphous quoyii	Water Skink	1	-	-	River edge.
Notechis scutatus	Mainland Tiger Snake	1	-	-	River edge(

TABLE 2. Species recorded at Mannum Waterfalls.
Habitat ; Rocky outcrops surrounded by well grazed pastoral land.

SPECIES	COMMON NAME	No. SEEN	No. COLLECTED	S.A.M. Reg. No.
Morethia sp.	-	Numerous	-	-
Menetia greyi	Grey's Skink	8	-	-
Hemiergis decresiensis	Three-toed Skink	6	-	-
Gehyra punctata	Rock Dtella	Numerous	1	-
Heteronotia binoei	Prickly Gecko	Numerous	-	-
Underwoodosaurus millii	Barking Gecko	3	-	-

TABLE 3. Members present on trip.

1. Graham Armstrong (Leader)
2. Steve Berri
3. Werner Brunn
4. John Fowler
5. Mark Galliford
6. Andrew Mower
7. Julia Smith
8. Andrew Todd
9. Ian Warmington
10. John Warmington
11. Julian Craig

FIELD TRIP TO INNAMINKA VIA THE STRZELECKI TRACK

SEPTEMBER 3 - 11, 1977

Trip Leader:- J. White

Aim:- To sample the reptile population along the Strzelecki Track, and review the area north of Innaminka for further field work.

Report:- Nine members in two vehicles participated, and two members in an N.P. & W.S. vehicle joined us for part of the trip at Innaminka. In addition we met the W.H.G. at Innaminka.

This trip was beset with technical problems which severely marred our herp efficiency, and reduced our available collecting time considerably. We had planned to use a generator for spotlighting, and pit traps in the sandy areas. These were carried in a hired trailer which broke its springs at the early part of the track. This meant that much of our equipment, including pit traps, was left behind, and our time available reduced, as we had to allow time for repair of the trailer on the way back.

Weather on the first three days was very unfavourable, being completely overcast, with strong cold winds. We went up via the western side of the Flinders Ranges, checking several ruins and wells on the way (tables I & II). We spent the first night at Mt Searle Station, and here found several species (table III), but no more *Odeura marmorata*.

On the second day we made our way further north to Lyndhurst and the start of the Strzelecki Track. The Track, which now services the Moomba Gas Fields, has been extensively upgraded by the Highways Dept., and most is in excellent condition, accessible to two-wheel drive vehicles. The first section of the track is uninteresting gibber, heavily grazed by cattle, with few areas for reptiles. There are occasional rock outcrops and ruins and these were checked (tables IV). It was here that our trailer broke its springs, and we had to abandon it, to be collected and repaired on the way back. Several species were caught around the Homestead on our two visits there (table V). We are extremely grateful to the Manager and his staff, who kindly looked after the trailer, and helped us repair it on the way back. He made the interesting comment that illegal shooters are a problem in this area, despite the distance from Adelaide. Certainly rabbits are very common, as they are all along the Track, reaching very high populations in the sandy areas along the northern sections of the Track.

On the third day we left Murnpeowie and the trailer, with its pit traps, generator etc., and headed north-east along the track. After passing through more featureless gibber, we finally started on the sandy areas. The first sand, around Monte-Collina Channel, was of very pale colour, and formed into numerous small tussocks. Here we found a very pale form of *Heteronotia* (table VI). Further north the sand became darker, and well formed into dunes, with mixed low vegetation, including canegrass. However the cold weather inhibited reptile activity, and few species were seen (table VII). We did not see any reptiles spotlighting. However rabbits were numerous, and during the day time, dingoes were frequently seen.

On the fourth day the weather improved, as we reached Moomba. Here we met Charles Balcharek, who has an interest in herpetology, maintains a small collection, and has helped herps from Adelaide before (most noteably, Mike Tyler). We were also called in at Moomba and were kindly shown round by the plant superintendant.

Moomba, like much of the sandy areas or the northern Track, attracted huge numbers of birds of prey, in some cases groups of as many as a hundred, or even more. Dingoes were also common, but few kangaroos or emus were sited. Bearded dragons *(Amphibolurus vitticeps)* were also very common in this area.

More specimens were collected amongst the cane grass of the dune country (table VIII), and, three of these were all active within one small cane grass tussock. From the dune country we moved further north, passing through country severely affected by stock, before reaching Coopers Creek and Innaminka. Here we joined with Geoff Coombe and Stuart Pillman in an N.P. & W.S. Toyota, and we also met with the W.H.G., at a campsite on the banks of the Cooper.

They had been in the area for some time, and reported several sitings of Cooper's Creek Tortoise *(Emydura kreffti?)*, both near the campsite, and also at Coongie Lakes to the north, where it was very numerous. They had also collected a Red-naped Snake *(Eurina diadema)* in this area. A recent slough from a Children's Python *(Liasis childreni)* was found at the Innaminka dump, and the inhabitants at the Innaminka outpost claimed to have sited live snakes, fitting the description of this python, in this vicinity.

From Innaminka, we moved eastward to the sand dune country, which consists of large deep red sand dunes, covered by cane grass, with occasional areas of spinifex. The weather was warm, and reptiles were active, including a large King _rown *(Pseudechis australis)*, found in the cane grass on the side of a dune. Lizards were common in the cane grass tussocks (table IX). We also visited another section of Coopers Creek, and found a Gilberts Dragon *(Lophognathus gilberti)* active on the ground. It rapidly moved to a large gum tree overhanging the water, where it was eventually caught (table X).

Undoubtedly, if we had more time in the Innaminka area, many more reptiles would have been collected, and we will have to revisit the area to provide a better record for the Museum. However time, and trailer problems forced an early return to Adelaide, and little of significance was collected on the way back.

TABLE I - Mern Merna Ruins, western Flinders (Lat 31°37'S by Long 138°23'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Heteronotia binoei	Binoe's Gecko	-	3	R16078
Ctenotus robustus	Striped Skink	2	1	R16076
Cryptoblepharus	Skink	3	1	R16075
Gehyra punctata	Dtella	-	1	R16079
Morethia boulengeri	Snake-eyed Skink	-	1	R16077

TABLE II - Ruins, western Flinders (Lat 31°33'S by Long 138°39'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Morethia adelaidensis	Snake-eyed Skink	-	1	R16073
Varanus gouldii	Gould's Goanna	1 (dead)	-	-
Heteronotia binoei	Binoe's Gecko	-	1	R16074

TABLE III - Mt Searle, north Flinders (Lat 30°31'S by Long 138°55'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus decresii	Tawny Dragon	-	1	RNA
Amphibolurus vadnappa	Red-barred Dragon	-	2	RNA
Amphibolurus vitticeps	Bearded Dragon	1	-	-
Ctenotus uber	Striped Skink	-	1	R16082
Egernia stokesii	Spiny-tail Skink	-	1	RNA
Tiliqua rugosa	Shingleback	2	-	-
Cryptoblepharus plagiocephalus	Skink	-	1	R16080
Morethia boulengeri	Snake-eyed Skink	2	1	R16081
Heteronotia binoei	Binoe's Gecko	5	-	-
Gehyra variegata	Dtella	5	-	-

TABLE IV - Ruins near Mt Lyndhurst Station, Strzelecki Track (Lat 30°11'S by Long 138°43'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Tympanocriptus tetraporophora	Earless Dragon	2	2	R16068
Morethia adelaidensis	Snake-eyed Skink	-	1	R16083
Menetia greyii	Skink	-	2	R16084
Heteronotia binoei	Binoe's Gecko	-	1	R16085
Amphibolurus pictus	Painted Dragon	2	1	R16067

TABLE V - Marnpeowie Station, Strzelecki Track (Lat 29°35'S by 139°4'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Diplodactylus tessellatus	Tessellated Gecko	-	1	R16070
Underwoodisaurus millii	Barking Gecko	-	1	RNA
Suta suta	Curl Snake	-	1	RNA

TABLE VI - Monte - Collina Channel - Strzelecki Track (Lat 29°30'S by Long 139°56'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus pictus	Painted Dragon	2	1	RNA
Heteronotia binoei	Binoe's Gecko	3	2	R16069

TABLE VII - Sand dunes, Strzelecki Track (Lat 29°00'S by Long 149°7'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Sphenomorphus fasciolatus	Desert-banded Skink	-	1	R16086
Lerista bipes	Burrowing Skink	2	1	R16087
Gehyra variegata	Dtella	-	1	R16088

TABLE VIII - Sand dunes, Strzelecki Track (Lat 28°15'S by Long 140°18'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Ctenotus brooksi	Striped Skink	-	1	R16091
Sphenomorphus fasciolatus	Desert-banded Skink	-	1	R16092
Morethia adelaidensis	Snake-eyed Skink	2	2	R16089
Menetia greyii	Skink	-	1	R16090
Amphibolurus barbatus	Bearded Dragon	1	-	-

TABLE IX - Sand dunes east of Innaminka (Lat 27°45'S by Long 140°57'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Diporiphora winneckii	Two-lined Dragon	3	2	RNA
Ctenotus regius	Striped Skink	-	2	R16071
Ctenotus brooksi	Striped Skink	-	1	R16072
Morethia adelaidensis	Snake-eyed Skink	-	1	R16094
Menetia greyii	Skink	-	2	R16095
Pseudechis australis	King Brown Snake	-	1	

TABLE X - Burkes Dig Tree, Coopers Creek (Lat 27°40'S by Long 141°8'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Lophognathus gilberti	Gilbert's Dragon	-	1	RNA
Morethia boulengeri	Snake-eyed Skink	-	1	R16093

TABLE XI - Innaminka dump (Lat 27°45'S by Long 140°45'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Tympanocriptus tetraporaphora	Earless Dragon	-	1	R15109
Menetia greyii	Skink	-	1	R16096

TABLE XII - Members Present:-

Julian White (Leader)
Graham Armstrong
Steve Berry
Werner Brunn
Geoff Coombe (N.P. & W.S.)
Julian Craig
Mark Galliford
Chris Hughes
Andrew Mower
Stuart Pillman (Environment Div.)
Hans Wallfried

FIELD TRIP TO RENMARK AREA

SEPTEMBER 17-18, 1977

Trip Leader:- J. White

Aim:- To confirm reported finds of the Velvet Gecko *(Oedura marmorata)* in this area, amongst Sheaoak stands.

Report:- Eight members in two vehicles moved straight to Renmark, where we initially visited Joe Bredl at his new reptile park. Joe reported that he had caught five *Oedura* in an area just north of Renmark, amongst Sheaoak stands. This area is just south of the new Danggali Conservation Park. Small stands of Sheaoak are found throughout this area, and it is extensively represented in the Conservation Park. However, most stands are small, and many have been ruined by sheep and goats. There are also areas of mallee with regions of *Triodia*, and it is these areas which have the largest reptile diversity and population.

On this occasion, the Group extensively searched several stands of Sheaoak, and sampled *Triodia* areas and saltbush flats as well, but no *Oedura* were located. Although this does not rule out the presence of this species, this area has been well covered over the last few years, and as no *Oedura*, other than those reported by Joe Bredl, have been found, their status in this area remains highly doubtful.

Some other interesting species were collected and these are listed in the tables following. All are previously predicted inhabitants of this region, and most predominate in the *Triodia* habitats.

Before leaving the area, we briefly visited one of the flood lakes of the Murray. On the western side, amongst large dead gum trees, we observed several Lace Monitors *(Varanus varius)*, including one very large specimen. Five were observed in a period of half an hour, all in a small area. There were no nests near by and the area was markedly degraded by sheep and cattle. All specimens were within 100 metres of the water, and most were on the higher branches of the trees. The lake is home to numerous waterbirds, and presumeably the eggs and young of these form the bulk of the Monitors diet. The high concentration of these Monitors in this area, despite extensive intrusion by man, is most pleasing, but nevertheless, this area may be worthy of protection as a Conservation Park, to ensure the habitat and so survival of these interesting Monitors.

TABLE I - Pipeline road to Canopus H.S. (Lat 33°53'S by Long 140°42'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Ctenotus regius	Striped Skink	-	1	R16106
Morethia boulengeri	Snake-eyed Skink	2	1	R16107
Cryptoblepharus plagiocephalus	Grass Skink	1	1	R16108

TABLE II - Pipeline road to Canopus H.S. - Oak Bore (lat 33°40'S by Long 140°32'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Egernia striolata	Tree Skink	3	1	R16105
Sphenomorphus richardsoni	Desert-Banded Skink	-	1	RNA

TABLE III - Pipline road to Canopus H.S. - *Triodia* (Lat 33°41'S by Long 140°33'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos
Pygopus lepidopodus	Scaly-foot	-	1	RNA
Amphibolurus nobbi	Nobbi Dragon	-	1	R16103
Diplodactylus elderi	Jeweled Gecko	2	2	R16098
Diplodactylus intermedius	Spiny-tail Gecko	-	1	R16097
Lucasium damaeum	Beaded Gecko	12+	2	R16104

TABLE IV - Pipeline road to Canopus H.S. - *Triodia* (Lat 33°45'S by Long 140°37'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos
Amphibolurus fordi	Military Dragon	12+	3	R16102
Ctenotus schomburgkii	Striped Skink	-	1	R16100
Diplodactylus intermedius	Spiny-tail Gecko	10+	1	R16101
Heteronotia binoeii	Binoe's Gecko	10+	-	-
Gehyra variegata	Dtella	10+	-	-

TABLE V - Rotten Lake - Sheaoak stand (Lat 34°00'S by Long 140°43'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos
Amphibolurus pictus	Painted Dragon	-	2	RNA
Lerista punctatovittata	Burrowing Skink	-	2	R16099
Amphibolurus barbatus	Bearded Dragon	1	-	-

TABLE VI - Members Present

Julian White (Leader)
Werner Brunn
John Fowler
Mark Galliford
Peter Hartley
Chris Hughes
Brian Millar
Simon Ostler

BARATTA STATION, SOUTH EAST FLINDERS TRIP

JANUARY 28 - 29 - 30 1978

Trip Leader:- Dr J. White (SCP No. 195)

Aim:- To survey the herpetofauna of this region, and to fill in gaps in knowledge of reptile distribution, suggested by our last trip to Baratta.

Report: The S.A.H.G. last visited this region, centering on Baratta Station, in October 1975. As in 1975, the people on Baratta Station were friendly and helpful. Our party consisted of four vehciles, with fourteen herpetologists, including two members of the South Australian Museum.

Weather for the duration of the trip was warm to hot, with cloudless skies and moderate wind.

We reached Baratta Station, via Ororroo, and moved immediately to our camp site next to a natural spring, along a creek bed. Among the fauna frequenting this rocky waterhole, were numerous Euros, *(Macropus robustus)*. The creek was bounded on one side by rocky slopes with numerous crevices. Amongst these were found small numbers of Stokes skinks and Rock Skinks (Table I).

On the first day we followed the road alongside the creek bed, to areas of *Triodia* and low open scrub. Several species were seen in this area, but most notable were the Pretty Worm Lizard *(Aprasia inaurita)*, and the Nobbi Dragon *(Amphibolurus nobbi coggeri)*, neither of which were collected in 1975. As usual, the Nobbi's were found amongst dead trees and leaf litter, while the *Aprasia* was found in *Triodia* (Tables II, III).

The second day was spent on a long exploratory drive along this same road, up to and beyond Bibliando homestead. The terrain was mixed flat, creek area, and rocky hillsides. No remarkable finds were made (Tables IV, V, VI & VII).

The northern section of this track towards Curnamona Station, traverses flat scrubland which has been overstocked, and was singularly improductive from our point of view.

Spotlighting was used on both nights, but no reptiles were seen, either on the tracks, or amongst the *Triodia* on footsearch, the only exception being a single Dtella *(Gehyra variegata)*, active on a tree.

The last day was spent travelling back to Adelaide. Waukauringa ruins were again checked on the way, and two emaciated Brown Snakes were found down the well. These were rescued.

We have consistently found reptiles trapped in these old abandoned wells and buried stone tanks. They act as a reptile sampling device for us, but must be responsible for many reptilian deaths, and render otherwise favourable ruins, distinctly hazardous to reptile occupants.

Overall, the trip was interesting, but yielded no spectacular finds, although filling in the range for the Nobbi Dragon was most useful.

The whole section visited, from northern Baratta, through Bibliando, is in relatively good condition. Every effort should be made to preserve it, as it is now. It would probably be suitable for consideration as a Conservation Park, but this would not seem urgent, as its terrain, isolation, and sensible landholders (Baratta Station) already offer it considerable protection.

TABLE 1 Baratta Station; Campsite and rocky outcrops along creek.
(Lat. 31°57'S by Long. 139°5'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Egernia margaretae	Flinder's Rock Skink	3	1	RNA
Egernia stokesii	Stoke's Skink	2	-	-
Morethia boulengeri	Snake-eyed Skink	1	-	-
Leiolopisma quickenoti	Grass Skink	1	-	-
Heteronotia binoei	Binoe's Gecko	4	-	-
Underwoodisaurus milii	Barking Gecko	1	-	-
Demansia psammophis	Yellow-faced Whip Snake	-	1	RNA

TABLE II Baratta Station; Spinifex scrub near campsite.
(Lat. 31°57'S by Long. 139°5'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Aprasia pseudopulchella	Worm Lizard	-	1	R16648
Ctenotus robustus	Striped Skink	1	-	-
Heteronotia binoei	Binoe's Gecko	3	-	-
Demansia psammophis	Yellow-faced Whip Snake	1	-	-

TABLE III Baratta Station; Mixed Mallee and spinifex scrub.
(Lat. 31°56'S by Long 139°5'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus nobii	Nobbi Dragon	-	2	RNA

TABLE IV Bibliando Station; rubbish around old homestead.
(Lat. 31°52'S by Long 139°6'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Ctenotus robustus	Striped Skink	-	1	R16665
Sphenomorphus richardsoni	Desert-banded Skink	-	1	RNA
Heteronotia binoei	Binoe's Gecko	5	-	-

TABLE V Bibliando Station; spinifex scrub. (Lat. 31°40'S by Long. 139°7'E)

Species	Common Name	No. Seen	No. Collected	Reg. No.
Delma australis	Legless Lizard	-	1	R16650
Delma nasuta	Legless Lizard	-	1	R16649

TABLE VI Bibliando Station; rocky outcrops along creek.
(Lat. 31°49'S by Long. 139°7'E)

Species	Common Name	No. Seen	No. Collected	Reg. No.
Demansia psammophis	Yellow-faced Whip Snake	-	1	RNA

TABLE VII Bibliando Station; rocky outcrops on northernmost section.

Species	Common Name	No. Seen	No. Collected	Reg. No.
Egernia stokesii	Stoke's Skink	4	1	RNA

TABLE VIII Members Present.

Julian White (Leader)
Beryl Morris (S.A.M.)
Tony Sadler (c/- S.A.M.)
Steve Berry
Jordie Duffy
Steve Giddings
Gavin Grigg
Brian Millar
Simon Ostler
Adrian Pemberton
Julia Smith
Andrew Todd
Ian Warmington
John Warmington

TRIP TO BILLIAT CONSERVATION PARK

MARCH 4 - 5, 1978

Leader:- J. White

Permit Nos.; SCP No. 195; RSCP No. 194

Aim:- To collect a representative sample of the reptile fauna of this park.

Report:- Eleven herpetologists in three vehicles visited the area. We initially looked at the north-west corner of the Park, an area used by I.M.V.S. for population dynamics studies. Dr Peter Baverstock (I.M.V.S) and three of his colleagues joined us for the trip. They set up pit-trap lines in the area, and collected independently from us.

The whole area was dry, with thick mallee scrub on well compacted deep yellow sand, and areas of spinifex interspersed between the trees. The area has received little rain in the last two years, and this is reflected in the catch rates. I.M.V.S. Pit-trap catch rates were in the order of 30-40 lizards a day several years ago, following good seasons. However on this trip, the daily catch rate was around 2 - 3 lizards, for an identical trap set up (in the same area).

Generally, we found lizards in small numbers only. Most common were the Military Dragons *(Amphibolurus fordi)*. A list of reptiles seen in the Park is given in the tables section. The only new find, was a Wind Snake *(Typhina australis)* which was found under an old tree stump. Desert Skinks *(Egernia inornata)* were found. All adults collected were in complex burrow systems under small bushes. There was a main entrance, with foot marks present, and 4 - 5 hidden "pop-holes". Invariably, the lizard would retreat to one of the latter, and at the last minute, escape to a second burrow complex, usually 1 metre distant. This would leave fewer pop-holes, and the lizard would retreat to the base of the burrow complex, where it was caught on excavation. Juvenile Desert Skinks appeared to have only single burrow complexes.

After examining Billiat, the Group moved northward to Loxton and the River Murray, searching ruins on the way. We attempted to find suitable habitat for Lace Monitors along the southern bank of the river; but few suitable spots were reachable, most gates being locked, and no Lace Monitors were seen.

TABLE I List of Reptiles seen in Billiat Conservation Park
(asterisk denotes those recorded by I.M.V.S. on previous trip)

Species	Common Name	
Egernia innornata	Desert Skink	
Tiliqua scincoides	Bluetongue	*
Tiliqua rugosa	Shingleback	
Tiliqua occipitalis	Western Bluetongue	*
Ctenotus brooksi	Striped Skink	
Ctenotus brachyonyx	Striped Skink	*
Menetia greyii	Grass Skink	
Morethia obscura	Snake-eyed Skink	*
Lerista bouganvilli	Bouganvilles Skink	
Lialis burtonis	Burtons Legless Lizard	*
Aprasia pulckella	Worm Lizard	*
Delma australis	Legless Lizard	
Diplodactylus damaeus	Beaded Gecko	
Diplodactylus vittatus	Stone Gecko	*
Phyllurus milii	Barking Gecko	*
Amphibolurus vitticeps	Bearded Dragon	
Amphibolurus muricatus	Jacky Lizard	
Amphibolurus nobbi	Nobbi Dragon	*
Amphibolurus fordi	Military Dragon	
Amphibolurus pictus	Painted Dragon	
Varanus gouldii	Goulds Goanna	*
Pseudonaja textilis	Brown Snake	*
Typhlina australis	Blind Snake	

TABLE II Locations of specimens collected on trip

(a) Camp-site ($34^{o}55'$ x $140^{o}20'$) Thick mallee scrub with pale sand

Species	Common Name	No. Collected	S.A.M. Reg. Nos.
Amphibolurus pictus	Painted Dragon	1	R16680
Amphibolurus muricatus	Jacky Lizard	1	R16681
Amphibolurus fordi	Military Dragon	1	R16679
Ctenotus brooksi	Striped Skink	1	R16682
Lerista bouganvilli	Bouganvilles Skink	1	R16677
Menetia greyi	Grass Skink	1	R16676
Delma australis	Legless Lizard	1	R16678

(b) On track ($34^{o}54'$ x $140^{o}20'$) Mallee and pale sand

Species	Common Name	No. Collected	S.A.M. Reg. Nos
Amphibolurus fordi	Military Dragon	1	R16675
Egernia inornata	Desert Skink	2	R16673
Diplodactylus damaeus	Beaded Gecko	1	R16674

TABLE II Locations of specimens collected on trip.

(c) On track (34°53' x 140°20') Open malle.

Species	Common Name	No. Collected	S.A.M. Reg. Nos.
Egernia inornata	Desert Skink	1	R16670
Typhlina australis	Blind Snake	1	R16672
Amphibolurus pictus	Paint Dragon	1	R16671

(d) Dunes and ruins (34°51' x 140°18')

Species	Common Name	No. Collected	S.A.M. Reg. Nos.
Tiliqua rugosa	Shingleback	2 seen, not collected	
Amphibolurus vitticeps	Bearded Dragon	1 seen, not collected	

(e) Ruins near Wanbie (34°45' x 140°20')

Species	Common Name	No. Collected	S.A.M. Reg. Nos.
Lerista punctatovittata	Sand Skink	1	R16683

TABLE III Members Present

Julian White (Leader)
Steve Berry
Werner Brunn
Jordie Duffy
John Fowler
Mark Galliford
Allison Hill
John Hill
Shiela Hill
Andrew Mower
Simon Ostler

TRIP TO MULGATHING STATION AND SURROUNDS

Easter, March 23 - 29, 1978

Leader: Dr J. White

Aim:- The S.A.H.G. last visited this area at Easter 1973, when a wide range of reptile life was encountered. This trip was designed to consolidate and extend the data collected on the previous trip.

Report:- The trip was attended by fifteen herpetologists, in four vehicles. (table 24). Two vehicles, containing seven personel attended only for four days, the remaining two vehicles, with eight personel, staying the full six days.

The group left Adelaide in early evening of 23/3/78, and made camp overnight near Hesso railway crossing (Table 1). The following morning the group moved northwards to Pimba, making several herp stops en-route (Table 2). From Pimba the group moved westwards via Kingoonya and Tarcoola, to reach Mulgathing Station that evening. Several other herp stops were made along the way (Tables 3, 4, 5). Camp was made at Mulgathing rocks, just west of the homestead complex, and spotlighting was undertaken that night (Table 6).

Mulgathing Station, and Commonwealth Hill Station immediately to the north, are very large pastoral properties run by managers, the owners being absentee. The relatively untouched Great Victoria Desert, forms the western boundry of these properties.

Both stations offer varied habitats, with areas of gibber plain, saltbush plain, Mulga scrub, isolated mulga stands, casuarine scrub, bluebush plain, red sandune country with mixed scrub, and some areas of granite outcrops. These latter are concentrated around the main outcrop at Mulgathing rocks, which stands out from the surrounding gibber and saltbush plain.

Grazing of both properties is apparently cycled between the numerous huge paddocks, and those currently grazed, show marked degradation of natural habitat, although not severe enough to destroy the habitat. The currently ungrazed paddocks are in good condition. It should be remembered that this whole area has had several dry seasons in succession, and in view of this, the land appears to be in good condition. Only in a very few areas did we find reptile life abolished by grazing. In summary, although grazing has affected the habitat, it has not, at least on our brief examination, drastically affected reptile survival.

A number of reptile species were collected on Mulgathing Station, both by daylight and nocturnal search techniques. No trap lines were laid. Reptiles were evident in all the mentioned habitats, but judging by the number of tracks, the sandy habitats were favoured. It is interesting to note that in addition to reptile tracks, Hopping Mice (*Notomys*) tracks were very common in the sand-dune areas, as were burrows and droppings. However, despite several burrow excavations, no specimens were located. This area might repay a visit by the mammal club. Of the various species of reptiles collected (Tables 7, 8, 9, 10, 11, 12, 13, 14), the most notable was Gillen's Goanna (*Varanus gilleni*). One dead specimen of the species was collected by us here in 1973, amongst a mulga stand. However, on this trip, we had much more success. In a similar mulga stand, also near the rocks, a live *gilleni* was found under the bark of a dead tree. Extensive search in this area failed to reveal any more specimens. However, later in

the trip, at a further mulga stand, five *gilleni* were collected in a space of fifteen minutes (two of which were released). This suggests to us that *gilleni* were common to very common in their preferred habitat, and that the reason that few have been collected in the past, is that herpetologists have been unsure of exactly where to look (see appendix II).

Of the other reptiles collected on Mulgathing, the Netted Dragon (*Amphibolurus inermis*) is worthy of mention. In 1973 we collected *Amphibolurus reticulatus* from this area, but no specimens were seen this time. The two species are very similar, being in the same species group, and the *inermis* at Mulgathing were morphologically distinct from *inermis* collected along the Pimba-Kingoonya road. They had a shorter tail for body length, and a larger head with more pronounced nasal bossing. However our sample was too small to draw any firm conclusions. Certainly further study in this area would be worthwhile.

After spending a full day at Mulgathing Rocks, searching the surrounding areas, and paying one visit to Charcot Bore to the west, the group moved on to Commonwealth Hill Station on the third day. However we only spent a brief time there (table 15) before moving south again, stopping en-route (table 16), and incidentally collecting the *gilleni*.

Half the group having departed for Adelaide at this stage, the remainder of the group visited portions of Wilgena Station, between Tarcoola and Kingoonya. Wilgena Hill, which is surrounded by red sand country, is an interesting habitat. Its rocky upper slopes are covered with spinifex and the occasional large shrub or small tree. Among the reptiles collected here (table 17), were the Occelated Skink (*Cteuotus pantherinus occelifer*), a specimen of *Amphibolurus fioni* was seen but not collected. This habitat appears delicate, and an effort should be made to ensure its survival. Fencing around the base of the hill would be worthwhile, to keep stock away.

The group moved south-east to a southern section of Wilgena, where sand-dune country intercepts mixed scrub and bluebush plains. Nospectacular finds were made in this area (tables 18, 19, 20).

The following day the group moved eastwards, towards Adelaide, camping overnight at Uno Bluff before returning to Adelaide the following day. The Group hopes to stage a full trip to Uno Bluff later this year, and our short visit on this occasion certainly suggests that this would be worthwhile. Among the reptiles caught here, was a Scalyfoot (*Pygopus nigriceps*) which is an interesting find from this area (tables 21, 22, 23). The areas of spinifex on the slopes of the Bluff offer a completely different habitat to that of the dune country and bluebush flats which surround the Bluff.

Overall, the trip was most successful, although few major range extensions were made. Throughout the trip, spotlighting was most successful.

My thanks go to all who attended the trip and worked so hard to make it a success. The Group is very grateful to the Managers, Owners, and Staff of Mulgathing, Commonwealth Hill, Wilgena, and Hesso Stations for allowing us to visit their properties. We are also grateful to Mr Lucarotti, and the W.R.E. Woomera, for permission to enter the prohibited area, on Mulgathing and Commonwealth Hill Stations.

TABLE 1 Hesso Railway Crossing - Bluebush and mulga scrub.

(Lat. 32°3'S by Long. 137°27'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Nephrurus laevis	Knob-tailed Gecko	-	1	R.N.A.
Pseudonaja modesta	Collared Brown Snake	-	1	R.N.A.

TABLE 2 Gibber plain near Island Lagoon - Port Augusta - Pimba road.

(Lat. 31°24'S by Long. 136°57'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Diplodactylus tesselatus	Tesselated Gecko	-	1	R16720
Tympanocriptus tetraporophora	Earless Dragon	2	2	R16719
Heteronotia binoeii	Binoe's Gecko	-	1	R16718

TABLE 3 Sandy scrub on Pimba-Kingoonya road.

(Lat. 31°11'S by Long. 136°5'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Amphibolurus pictus	Painted Dragon	-	1	R16721
Amphibolurus inermis	Netted Dragon	-	1	R16722
Tymparocriptus tetraporophora	Earless Dragon	-	1	R16723

TABLE 4 Sandy scrub on Pimba-Kingoonya road.

(Lat. 31°12'S by Long. 136°1'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Amphibolurus inermis	Netted Dragon	-	1	R.N.A.

TABLE 5 Sandy scrub on Pimba-Kingoonya road.

(Lat. 31°10'S by Long. 136°21'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Ctenotus reguis	Striped Skink	-	1	R16724

TABLE 6 Mulgathing Station - spotlighting along Charcot-Bore road.

(Approx. Lat. 30°15'S by Long. 133°50'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Underwoodisaurus milii	Barking-Gecko	2	2	R16727
Rynchoedura ornata	Beaked Gecko	4	2	R16726
Diplodactylus damaeus	Beaded Gecko	3	3	R16725

TABLE 7 Mulgathing Station - Charcot Bore road, daylight.

(Approx. Lat 30°15'S by Long. 133°45'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Ctenotus regius	Striped Skink	1	1	R16731
Lerista desertorum	Burrowing Skink	-	1	R16732
Lerista labialis	Burrowing Skink	-	2	R16734
Menetia greyi	Grey's Skink	-	1	R16733
Morethia boulengeri	Snake-eyed Skink	-	1	R16735

TABLE 8 Mulgathing Station - Charcot Bore

(Approx. Lat. 30°13'S by Long. 133°40'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Amphibolurus inermis	Netted Dragon	6	2	-
Lerista bipes	Burrowing Skink	-	2	R16742

TABLE 9 Mulgathing Station - Granite outcrop near Rocks

(Lat. 30°15'S by Long. 133°58'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Varanus gouldii	Gould's Goanna	-	1	R.N.A.

TABLE 10 Mulgathing Station - Mulgathing Rocks

(Lat. 30°14'S by Long. 133°58'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Ctenotus regius	Striped Skink	-	1	R16740
Ctenotus schomburgkii	Striped Skink	-	1	R16741
Tympanocriptus lineata	Earless Dragon	2	1	R16736
Tympanocriptus lineata?	Earless Dragon	1	1	R16737
Gehyra variegata	Dtella	3	1	R16739
Heteronotia binoeii	Binoe's Gecko	2	1	R16738
Amphibolurus inermis	Netted Dragon	4	1	-

TABLE 11 Mulgathing Station - Mulga Stand near Rocks

(Lat. 30°13'S by Long. 133°56'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Varanus gilleni	Gillen's Goanna	-	1	R.N.A
Ctenotus schomburgkii	Striped Skink	-	1	R16730
Tympanocriptus lineata	Earless Dragon	-	1	R16729

TABLE 12 Mulgathing Station - Mulga stand near Rocks.

(Lat. 30°13'S by Long. 133°58'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Ctenotus schomburgkii	Striped Skink	-	1	R16693

TABLE 13 Mulgathing Station - Spotlighting along Charcot Bore road.

(Lat. 30°15'S by Long. 133°50'E Approx.)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Underwoodisaurus milii	Barking Gecko	3	1	R.N.A.
Nephrurus laevis	Knobtail Gecko	-	1	R16728
Diplodactylus damaeus	Beaded Gecko	6	-	-
Rynchoedura ornata	Beaked Gecko	5	-	-

TABLE 14 Mulgathing Station - Muckanipie Ruins.

(Lat. 30°10'S by Long. 134°6'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Morethia adelaidensis	Snake Eyed Skink	-	1	R16746

TABLE 15 Commonwealth Hill Station - Claypan paddoch

(Lat. 29°54'S by Long. 134°15'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Amphibolurus inermis	Netted Dragon	-	1	R16747
Ctenotus leonhardii	Striped Skink	-	1	R16748
Tympanocriptus lineata	Earless Dragon	-	2	R16749

TABLE 16 Mulgathing Station - Mulga Scrub

(Lat. 30°8'S by Long. 134°12'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Varnaus gilleni	Gillen's Goanna	2	3	R16745
Amphibolurus isolepis	Millitary Dragon	-	2	R16744
Gehyra variogata	Dtella	-	2	R16743

TABLE 17 Wilgena Hill, Wilgena Station

(Lat. 30°43'S by Long. 134°44'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Tympanocriptus lineata	Earless Dragon	-	1	R16753
Delma nasuta	Legless Lizard	1	1	R16755
Ctenotus pantherinus occelifer	Occelated Skink	1	2	R16754
Lerista muelleri	Burrowing Skink	-	1	R16750
Diplodactylus ciliaris intermedius	Spiny-tail Gecko	-	2	R16752
Lerista bipes	Burrowing Skink	-	1	R16751

TABLE 18 Wilgena Station, south east section - ruins and well

(Lat. 30°57'S by Long. 134°57'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Cryptoblepharus plagiocephalus	Grass Skink	-	1	R16758
Gehyra variegata	Dtella	-	1	R16759
Morethia boulengeri	Snake-eyed Skink	-	1	R16757

TABLE 19 Wilgena Station, south east section - sand dunes.

(Lat. 30°55'S by Long. 134°58'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Amphibolurus cristatus	Crested Dragon	-	1	R16756

TABLE 20 Wilgena Station, south east section - spotlighting.

(Lat. 30°54'S by Long. 134°57'E Approx.)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Nephrurus laevissimus	Knobtail Gecko	-	4	R16761
Rynchoedura ornata	Beaked Gecko	6	1	R16760

TABLE 21 Hesso Station - bluebush flat near Uno Bluff, spotlighting.

(Approx. Lat. 32°7'S by Long. 137°29'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Nephrurus laevis	Knobtail Gecko	-	1	R.N.A.
Pygopus nigriceps	Scalyfoot	-	1	R16768

TABLE 22 Hesso Station, spotlighting near Uno Bluff (dunes).

(Approx. Lat. 32°7'S by Long. 137°33'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Rynchoedura arnata	Beaked Gecko	12	1	R16763
Diplodactylus damaeus	Beaded Gecko	20	1	R16762

TABLE 23 Hesso Station, Uno Bluff slopes and station ruins.

(Lat. 32°7'S by Long 137°34'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. Nos.
Delma australis	Legless Lizard	-	1	R16765
Tympanocriptus tetraporophora	Earless Dragon	-	1	R16766
Lerista bipes	Burrowing Skink	-	1	R16767
Neobatrachus pictus	Burrowing Frog	-	1	R16764

TABLE 24 Members Present:-

Dr Julian White (Leader)
Steve Berry
Kevin Brown
Werner Brunn
Jordie Duffie
John Fowler
Mark Galliford
Steve Giddings
Darryl Levi
Brian Miller
Andrew Mower
Adrian Pemberton
Beryl Morris (S.A.M.)
Tony Saddler (S.A.M.)
Julia Smith

TABLE 25 List of Species seen and abundance, in preferred habitat.

Species	Status	No. Collected (all areas)
Amphibolurus inermis	Common	6
Amphibolurus vitticeps	Common	0
Amphibolurus pictus	Common	1
Amphibolurus cristatus	Common	1
Amphibolurus isolepis	Common	2
Tympanocriptus lineata	Abundant	6
Tympanocriptus tetraporophora	Common	3
Moloch horridus	Uncommon	0
Tiligua rugosa	Common	3
Ctenotus schomburgkii	Common	3
Ctenotus regius	Abundant	3
Ctenotus leonhardii	Common	1
Ctenotus pantherinus occelifer	Common	2
Lerista muelleri	Common	1
Lerista bipes	Common	3
Lerista labialis	Common	1
Lerista desertorum	Uncommon	1
Morethia boulengeri	Common	2
Morethia adelaidensis	Common	1
Cryptoblepharus plagiocephalus	Common	1
Menetia greyi	Common	1
Nephrurus laevis	Common	3
Nephrurus laevissimus	Common	4
Diplodactylus damaeus	Abundant	4
Diplodactylus ciliaris intermedius	Common	2
Diplodactylus tesselatus	Common	1
Rhynchoedura ornata	Abundant	4
Heteronotia binoei	Abundant	2
Gehyra variegata	Abundant	3
Underwoodisaurus milii	Common	3
Delma nasuta	Common	1
Delma australis	Common	1
Pygopus nigriceps	Uncommon	1
Varanus gilleni	Abundant	5
Varanus gouldi	Common	1
Pseudonaja moderta	Common	1

FIELD TRIP TO ANDAMOOKA RANGES

MAY 13, 14, 15 1978

Leader:- Dr J. White

Aim:- To do a preliminary survey of this area, and if time permitted, to search for Lake Eyre Dragons (*Amphibolurus naculouis*), in the hope of extending their range southwards on Lake Torrens.

Report:- The trip was a preliminary survey only, and so attempted to cover a large area in a short time. Two vehicles and five people participated, but only two of the personel were herpetologists.

The previous two weeks had seen heavy rain and cold weather in the area, and for much of the trip the weather was cold and overcast, so inhibiting reptile activity.

We first visited South Gap Station, in the southern Andamooka Ranges, which are low rounded gibber hills, with little vegetation, and very few rock outcrops. To reach the ranges, we passed through areas of low red dunes, but these were heavily grazed and unsuitable for much reptile life, as were the southern ranges. We examined an area of isolated sand dunes, on the edge of Lake Torrens (Sandy Point), and here found several species of reptiles (tables I & II). We also searched the edge of Lake Torrens in this area, but could find no evidence of Lake Eyre Dragons, although Wolf Spiders were common (*Lycosa* sp.).

From this area we moved along the edge of the Lake, without encountering more reptiles. We then moved north to Pernatty Station, and then to Yeltacowie Station (Abandoned). Time did not permit us to examine this area in detail, but numerous dunes were observed, many of which were covered with canegrass, between which many diverse animal tracks were seen. A small animal, possibly *Diporiphora* sp. was seen in the canegrass, but eluded capture.

We moved westwards around the top of Pernatty Lagoon down to Mt Gunson Station, and hence back to Adealide. Several interesting rock outcrops and dunes were encountered in this area.

TABLE I Sandy Point - Sand dunes (Lat. 31°48'S x Long. 137°43'E)

SPECIES	COMMON NAME	NO. COLLECTED	S.A.M. REG. NO.
Amphibolurus inermis	Netted Dragon	1	R16812

TABLE II Sandy Point - Sand dunes (Lat. 31°47'S x Long. 137°41'E)

SPECIES	COMMON NAME	NO. COLLECTED	S.A.M. REG. NO.
Amphibolurus pictus	Painted Dragon	1	R.N.A.
Ctenotus regius	Striped Skink	1	R16810
Menetia greyi		1	R16811

TABLE III Yeltacowie Ruins (Lat. 31°20'S x Long. 137°21'E)

SPECIES	COMMON NAME	NO. COLLECTED	S.A.M. REG. NO.
Lerista bipes	Burrowing Skink	1	R16809
Heteronotia binoeii	Binoes Gecko	1	R16808

TABLE IV - Trip Members

Julian White (Leader)
Julia Smith
John Pym
Pete Smith
Keith Martin

URO BLUFF - RANGE TRIP

JUNE 3 - 4, 1978

Trip Leader:- M. Galliford

SCP No. 226

Aim:- To survey the Herpetofauna of this range and surrounding bushland.

Report:- The S.A.H.G. had briefly visited this area at Easter of this year, March 24 - 27th.,but decided it should require a full trip so as to see the abundance of reptiles around the range.

Nine herpetologists in three vehicles made up the party and with permission from Kootaberra Station we were allowed to camp on the owners property. The trip was to cover the whole long weekend but because of poor weather on the second day we had to leave the day earlier.

Weather during the trip was variable, from warm and sunny to cold, overcast and drizzly, the latter meant bad reptile collecting.

Hesso was an overnight stop for some members, being the entrance to the normally locked Uro Bluff area, and these members met the other two vehicles the next morning. After obtaining the key to get into the area we headed for the Uro Bluff Homestead, now abandoned,and these ruins were given a thourough look over before heading to the eastern side of the range which resulted in a steep climb up the rocky slopes of the bluff. The rocky slopes were thinly covered with stunted trees and small shrubs and grasses while the plain below was mainly gibber on red sand covered by dominant Bluebush. The Bluff itself rises to a height of 887' (feet) and the actual range on which the Bluff sits is 8km. long in a North - South direction.

Reptiles caught around the Homestead and eastern side of the range may be seen in Tables I and II.

That night spotlighting was undertaken and after along period of nothing we turned up one Beaded Gecko (Lucasium damaeum) which was found along the track back to Hesso Railway - siding.

The next day we drove to the top of the bluff and some of the party walked down a steep gorge on the western side of the range, across the gibber plain below,through some sand dunes and back to camp. The country up the gorge was mainly the same as the eastern side of the range except that the rocky outcrops were more of a sandstone material which had been eroded by the wind to form some remarkable formations where we hoped to find Children's Pythons (Liasis childreni) but none were found although they are probably there without a doubt. The lower slopes of the range were covered almost entirely with spinifex (Triodia sp) and this habitat proved quite successful with two species of Legless Lizard (Delma australis) and (Delma nasuta) being found under the majority of bushes looked under.

The sand dunes , which were covered mainly by Acacia and Mallee scrub, and where our camp was situated, yeilded no spectacular finds although evidence in the form of snake sloughs and goanna diggings were the only finds except one Sand Swimming Skink (Lerista sp.).

We then abandoned the rest of the trip because of the poor weather and eventually headed back home to Adelaide.

To sum it up, because we had visited this area in winter and because of the unfortunate weather conditions we had made no valuable finds although quite a large proportion of the reptile occupants of Uro Bluff were found.
Some species of lizard which we more than likely expected to find , such as the Stokes Skink (Egernia stokesii) an inhabitant of rock crevices, Jewelled Gecko (Diplodactylus elderi) and Burtons Legless Lizard (Lialis burtonis) both inhabitants of spinifex (Triodia sp.),were interestingly enough not found.
To really get an idea of reptile numbers around the Uro Bluff area a trip would need to be made in spring around the months of September - October when reptile activity is at a maximum.
Acknowledgements would like to be made to the drivers of the vehicles present on the trip, to the South Australian Museum for their much needed generous loan of folding stools and to the owners of the Homesteads involved with giving us permission of which without the trip could not have been held.

I. List of Reptiles caught on previous trip:- March 24 - 27th (Easter) 1978.
Hesso - Uro Range:- Stop over.

Species	Common Name
Diplodactylus intermedius	Spiny tailed Gecko
Heteronotia binoei	Binoe's Gecko
Lucasium damaeum	Beaded Gecko
Nephrurus levis	Knob tailed Gecko
Rynchoedura ornata	Beaked Gecko
Delma australis	Spinifex Legless Lizard
Pygopus nigriceps	Black headed Scaly foot
Morethia	Snake eyed Skink
Pseudonaja modesta	Collared Brown Snake

Reptiles caught on this trip:- June 3 - 4th 1978.

II. TABLE I. Uro Range Homestead (Ruin); scattered rubbish on Bluebush plain.
(Lat. 32°7'S by Long. 137°33'E).

Species	Common Name	No. seen	No. Collected	S.A.M. Reg. No.
Heteronotia binoei	Binoes Gecko	5	-	-
Trachydosaurus rugosa	Sleepy Lizard	1	-	-
Morethia	Snake eyed Skink	1	-	-
Unechis gouldii	Gould's Snake	-	1	
Demansia psammophis	Yellow faced Whip Snake	-	1	
Pseudonaja nuchalis	Western Brown Snake	-	1	

TABLE II. Uro Range; small isolated rocky outcrop, hardly no vegetation.
(Lat.32°7'S by Long. 137°35'E).

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Ctenotus robustus	Striped Skink	-	1	
Morethia	Snake eyed Skink	-	1	
Cryptoblepharus boutonii	Bouton's Snake eyed Skink	-	1	

TABLE III. Uro Bluff (Eastern Side); steep rocky slopes, thinly vegetated.
(Lat. 32°6'S. by Long.137°36'E).

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus fionni	Peninsula Dragon	6	2	
Ctenotus pantherinus ocellifer	Ocellated Skink	3	2	
Hemiegis		3	3	
Delma australis	Legless Lizard	5	1	
Delma nasuta	Legless Lizard	5	2	
Heteronotia binoei	Binoes Gecko	7	-	-

TABLE IV. Uro Bluff (Eastern Side); dominant Bluebush covered gibber plain, (Lat. 32°6'S by Long. 137°36'E). - transversed with Triodia sp.

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus vitticeps	Bearded Dragon	1	-	-
Cryptoblepharus boutonii	Snake eyed Skink	3	1	
Ctenotus robustus	Striped Skink	1	-	-
Gehyra variegata	Dtella	5	2	
Diplodactylus intermedius	Spiny tailed Gecko	1	2	

TABLE V. Hesso - Uro Bluff track; red soil with Bluebush and mulga trees, (Lat.32°8'S by Long.137°31'E). - spotlighting a-long track.

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Lucasium damaeum	Beaded Gecko	1	-	-

TABLE VI. Uro Bluff (Western Side); Triodia sp. and steep, rocky outcrops. (Lat. 32°6'S by Long. 137°34'E).

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Ctenotus robustus	Striped Skink	1	-	-
Ctenotus pantherinus ocellifer	Ocellated Skink	1	-	-
Delma australis	Legless Lizard	5	-	-
Heteronotia binoei	Binoes Gecko	5	-	-

TABLE VII. Uro Bluff (Western Side); gibber plain covered by Bluebush. (Lat. 32°6'S by Long. 137°34'E).

Species	Common Name	No. Seen	Collected	S.A.M. Reg. No.
Diplodactylus tessalatus	Tessalated Gecko	-	1	
Diplodactylus intermedius	Spiny tailed Gecko	1	1	

TABLE VIII. Camp - site; situated in red sand dunes North of Uro Bluff H.S. (Lat. 32°6'S by Long. 137°33'E).

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Lerista	Sand Swimming Skink	-	1	

TABLE IX. Members Present.

Mark Galliford (Leader)
Graham Armstrong
Sue Armstrong
Steve Berry
Werner Brunn
Chris Harvey
Andrew Mower
Adrian Pemberton
Julian White

TRIP REPORT: MT REMARKABLE NATIONAL PARK

OCTOBER 7TH - 9TH 1978

Trip Leader; Graham Armstrong

By the early afternoon of Satufday 7th October seventeen people in six vehicles had arrived and set up camp in the camping grounds of the Mambray Creek area of Mt Remarkable National Park.

Due to the short time spent in the Park and the distances which had to be covered on foot, only a small area of the Park could be surveyed. Nearly all specimens recorded were from around the creeks and hills in close proximity to the campsite. Although some members of the party spent a day hiking to the Battery, only one species. i.e. the Yellow-faced Whip Snake (*Demansia psammophis*), was seen which was not recorded from the area surrounding the campsite.

In view of these factors species for which only one or two specimens were seen and identified were considered as "not common" in the Park. Species for which five or more specimens were seen were considered as common, and species for which twenty or more specimens were regarded as very common.

TABLE 3 - Habitat preference and abundance of reptiles

Scientific Name	Habitat Preference	Abundance
Amphibolurus vitticeps	Low shrub and grassland on way into Park	Common
Amphibolurus decressi	Rock outcrops on hillsides	Common
Ctenotus robustus	Grassy areas along creeks, also seen in spinifex	Very Common
Cryptoblepharus plagiocephalus	Active on trunks of Eucalypts	Common
Egernia margaretae personata	Rocky outcrops, more common close to creeks	Very Common
Egernia striolata	Rock outcrops, more common further up hills and trees	Very Common
Hemiergis decresiensis	Beneath log	Not Common
Menetia greyi	Rocky hillsides	Not Common
Morethia boulongeri	Everywhere	Common
Tiliqua scincoides	Amongst rocks	Not Common
Tiliqua rugosa	Low shrub & grassland	Common
Delma molleri	Under rocks near creeks	Not Common
Diplodactylus ciliaris intermedius	Under rocks and bark of Casurina	Common
Gehyra variegata	Rock crevices and under bark of trees	Very Common
Heteronotia binoei	Under rocks and debris	Very Common
Underwoodisaurus millii	Beneath rocks and debris	Very Common
Varanus varius	Active amongst Eucalypt trees	Not Common
Demansia psammophis	Active on "Battery"	Not Common
Pseudophyne bibroni	Beneath rock by creek	Not Common

TABLE 1 - People Present on Trip

1. Graham Armstrong (Trip Leader)
2. Sue Armstrong
3. David Armstrong (Non-member)
4. Gavin Bedford
5. Werner Brunn
6. Julian Craig
7. Jordy Duffy
8. Mark Galliford
9. Chris Harvey
10. Doug Holly
11. Tricia Holly
12. Mario Lombardi
13. Brian Miller
14. Andrew Mower
15. Bary Pane and family
16. Julia Smith
17. Julian White

TABLE 2 - Reptiles and Amphibians seen in Mt Remarkable National Park

Scientific Name	Common Name	Approx. No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus vitticeps	Inland bearded dragon	5	-	-
Amphibolurus decresii	Tawny dragon	3	1	R16956
Ctenotus robustus	Striped Skink	20	2	R16957A R16957B
Cryptoblepharus plagiocephalus	Bouton's Skink	12	2	R16960A R16960B
Egernia margaretae personatas	Brown rock skink	30	1	R16955
Egernia striolata	Striated Skink	30	2	R16958A R16958B
Hemiergis decresiensis	Three-toed Skink	1	1	R16961
Menetia greyi	Grey's Skink	5	-	-
Morethia boulengeri	Snake-eyed Skink	15	1	R16962
Tiliqua scincoides	Common Blue-tongue skink	1	-	-
Tiliqua rugosa	Shingleback Skink	12	-	-
Delma molleri	Legless Lizard	2	1	R16954
Diplodoctylus ciliaris intermedius	Spiny-tailed gecko	6	2	R16964 R16963
Gehyra variegata	Tree Dtella	20	2	R16959A R16959B
Heteronotia binoei	Bynoe's gecko	20	2	R16953A R16953B
Underwoodasaurus millii	Thick-tailed gecko	2	-	-
Demansia psammophis	Yellow-faced Whip Snake	1	-	-
Varanus varius	Lace Monitor	4	-	-
Pseudophryne bibroni	Toadlet	1	-	-

REPORT OF S.A.H.G. HERPETOFAUNAL SURVEY OF INNES NATIONAL PARK

25TH & 26TH NOVEMBER 1978

Trip Leader: Graham Armstrong

Twenty one people met at Stenhouse Bay at 8.00 a.m. on Saturday 25th November 1978. Upon the arrival of Mr R. Jenkins (N.P. & W.S.) the group proceeded to the Shell Beach campground and set up camp. Mr Jenkins then personally revealed the location of the pit trap lines throughout the Park. However due to the shortage of trap line not all were used. The remainder of the day, well into the night and the next morning were spent searching for reptiles.

By midday Sunday the group had broken camp and started back to Adelaide. However several members stopped at Tiddy Widdy Beach, north of Ardrossen, and spent a few hours surveying the beach front and associated dunes. Reptiles seen here are also included at the end of the report in Table 5.

While surveying Innes National Park several methods of collection were used:-

1) Individuals and small groups searched the bush around Shell Beach and Brown's Beach on foot.

2) Vehicles travelling along the various tracks were used to spot reptiles on the edge of the tracks or immediate scrub.

3) Same method as 2, but at night when spotlights were used to detect reptiles crossing the tracks.

4) Pit trap lines.

Tabulated results of these methods are shown in Table 1.

In the short time spent at the Park 11 species were found in the Park and its adjoining areas. These are recorded in Table 2. This represents quite a successful survey, considering the difficulties in detecting reptiles in the type of coastal bushland being surveyed, and was the result of the numbers of members involved, the varied methods of collection used, and the very helpful co-operation of the National Parks and Wildlife Service staff.

Besides the 11 species recorded from the Park a further two species were seen on other areas of Yorke Peninsula which probably occur within Innes National Park. These are the Common Death Adder (*Acanthophis antarcticus*) seen in the coastal dunes at Tiddy Widdy Beach and the Western Bluetongue Skink (*Tiliqua occipitalis*) found on the roadside south of Warooka (see Table 3).

Table No. 1 (Results of Methods used to locate reptiles in Innes National Park)

Method 1 - Searching bush on foot

Species	No. Seen	No. Seen & Captured	No. retained for S.A.M.
Phyllodactyllus marmoratus	3	3	1
Pygopus lepidopodus	1	1	-
Hemiergis peronii	6	5	5
Morethia obscura	6	3	3
Tiliqua rugosus	3	3	1
Demansia psammophis	1	1	1
Pseudonaja textilis inframacula	2	-	-

Method 2 - Spotting side of road from vehicles during day

Species	No. Seen	No. Seen & Captured	No. retained for S.A.M.
Tiliqua rugosus	4	-	-
Amphibolurus pictus	4	1	1
Demansia psammophis	1	-	-

Method 3 - Spotting road from vehicles at night using spotlights

Species	No. Seen	No. Seen & Captured	No. retained for S.A.M.
Diplodactylus vittatus	2	2	1
Phyllodactylus marmoratus	1	1	1
Tiliqua rugosus	1	1	-

Method 4 - Pit trap lines

Species	No. Seen	No. Seen & Captured	No. retained for S.A.M.
Lerista frosti	1	1	1

This method also proved successful in catching a Pygmy Possum.
The *Lerista* was caught in pit trap line N4/6 and the Pygmy Possum in N4/3.

This table does not include a *Pygopus lepidopodus* supplied by N.P. & W.S. Ranger Mr L. Doherty.

Table No. 2 Reptiles recorded from Innes National Park and adjoining areas.

Species	Common Name	No. Seen	No. Collected
Diplodactylus vittatus	Stone Gecko	2	1
S.A.M. REG. NO. R16977	MAP REFERENCE (060,758)		
Phyllodactylus marmoratus	Marbled Gecko	4	2
S.A.M. REG. NO. R16971	MAP REFERENCE (036,710): (039,700)		
NO. R16976	(060,738):		
Underwoodisaurus millii	Thick tailed Gecko	1	1
S.A.M. REG. NO. R16973	MAP REFERENCE (038,747)		
Pygopus lepidopodus	Common Scaly-foot	2	1
S.A.M. REG. NO. R16967	MAP REFERENCE (943,766): (050,723)		
Hemiergis peronii	Peron's Skink	6	5
S.A.M. REG. NO. R16968A&B	MAP REFERENCE (020,749): (943,766)		
NO. R16969A&B	(039,700)		
NO. R16974			

Species	Common Name	No. Seen	No. Collected
Lerista frosti		1	1
S.A.M. REG. NO. R16980	MAP REFERENCE (994,681)		
Morethia obscura	Snake-eyed Skink	6	3
S.A.M. REG. NO. R16970A,B&C	MAP REFERENCE (039,700)		
Tiliqua rugosus	Shingleback Skink	8	1
S.A.M. REG. NO. R16972	MAP REFERENCE - All over Park		
Amphibolurus pictus	Painted dragon	4	1
S.A.M. REG. NO. R16975	MAP REFERENCE (040,728):(912,760)		
Demansia psammophis	Yellow faced Whip Snake	2	1
S.A.M. REG. NO. R16978	MAP REFERENCE (945,715):(948,735)		
Pseudonaja textilis inframacula	Peninsula Brown Snake	2	-
S.A.M. REG. NO. -	MAP REFERENCE (049,721)		

Table No. 3 - Other reptiles recorded during survey

Species	Common Name	No. Seen	No. Collected
Tiliqua occipitalis	Western Blue-tongue Skink	1	1
S.A.M. REG. NO. R16979	LOCALITY - 11 road KM South of Warooka on road to Stenhouse Bay.		

Table No. 4 - People present on trip

Members: Graham Armstrong (Trip Leader)
Susan Armstrong
Gavin Bedford
Steven Berri
Werner Brunn
Julian Craig
Steve Doyle
Jordi Duffy
Mark Galliford
Keith Gilbertson
Doug Holly
Brian Miller
Andrew Mower
Julian White
Corrie Van der Hoek

Non-Members: Dave Armstrong
Ahmen Griffin
Steve Haniford

Table No. 5 - Reptiles recorded from Tiddy Widdy Beach

Species	Common Name	No. Seen	No. Collected
Phyllodactylus marmoratus	Marbled gecko	1	-
Hemiergis peronii	Peron's Skink	1	-
Lerista frosti		2	2
S.A.M. REG. NO. R16981A, R16981B			
Lerista labialis		2	2
S.A.M. REG. NO. R16982A, R16982B			
Tiliqua rugosa	Shingleback Skink	2	-
Amphibolurus pictus	Painted Dragon	1	-
Varanus gouldii	Gould's Sand Goanna	1	-
Acanthophis Antarticus	Common Death Adder	1	-
Pseudonaja textilis	Common Brown Snake	1	-

There were also a species of Blind Snake and a species of legless lizard of the genus *Aprasia* captured but these were released before a positive identification could be made.

TRIP REPORT OF CANUNDA NATIONAL PARK

JANUARY 27TH - 29TH 1979

Trip Leader: B. Miller

Aim: The South Australian Herpertology Group had never visited this coastal area of the South East before and the trip was designed as a herpetological survey of the region. All specimens collected were lodged with the South Australian Museum.

Report: The trip was attended by thirteen S.A.H.G. members in five vehicles. Several personell left Adelaide a day earlier and the parties met at Millicent on the Saturday morning from where we made our way to the ranger's house on the outskirts of Canunda National Park. After this visit a campsite was made inside the reserve in close proximity to Bevilaqua Ford.

Canunda National Park lies approximately to the south west of Millicent and includes some 35 kms of natural coastal scrublands. A few varieties of habitats were surveyed. These included:

a) Large sand dunes with little vegetation and intermittent areas of eroded limestone rock outcrops. These areas were nearly always close to the foreshore and extended approximately ½ km inland. Reptiles collected from this area are shown on Table I.

b) Coastal scrublands that were comprised of low lying natural bushes and trees. This habitat made up the majority of the vegetative type within the park. These areas extended ½ km from the coastline to some 5 kms inland. The scrub type was typical of coastal form and an abundant amount of leaf litter and dead foliage cover was noted. Reptiles from this area are shown on Table II.

c) The third and final type of habitat consisted of mainly swamp lands surrounded by grass tussocks and saturated ground. A number of drainage systems ran through the park into lakes and around these immediate areas large pockets of water were encountered. Reptiles from this area are shown in Table III.

Three four wheel drives were present on the trip and these again proved worthwhile in conveying personell to areas that would have proved inaccessable to conventional vehicles.

After leaving the park on Sunday morning a visit was made to the Dismal Swamp area which lies approximately 10 kms North East of Glencoe. This area consisted of numerous small sections of swamp land surrounded by large areas of *Pinus radiata* plantations. Reptiles from this area are shown on table IV.

Unfortunately the coastal areas of this region seem somewhat devoid of reptile fauna. Although fine weather was encountered throughout most of the trip the area proved hard to survey and provided little encouragement.

The scenery around the coastline was most noteable with numerous breathtaking views of cliff faces.

Additional Notes:-

One interesting note was that of the rangers who informed us of a Sand Goanna that had been seen in the area. This would probably be *Varanus gouldii rosenbergi*, a somewhat melanistic form of the Common Sand Goanna (*Varanus gouldii gouldii*). If this goanna proves to inhabit the area the range extension for this subspecies would be greatly increased. Along with this information he told us of local farmers sighting the Common Death Adder (*Acanthopis antarticus*) within the vicinity. This would also prove to be a range extention and hopefully these two reptiles can be secured on a future trip as the Museum lacks records of these species from this area.

A common Brown Snake (*Pseudonaja textilis textilis*) soon after capture was found to have regurgitated 5 semi-adult rats. The snake measured 1½ metres in length and had obviously taken these animals from a nest raid.

Finally the S.A.H.G. would like to take this opportunity to thank the National Parks & Wildlife Service for their Co-operation whilst undertaking this survey.

TABLE I - Coastal Sand Dunes (Lat. 37°36' South x Long 140°13'East)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Hemiergis peronii	Peron's Skink	4	1	R17015
Trachydosarus rugosus	Shingle Back	2	-	-

TABLE II - Coastal scrubland comprising of low lying scrub and trees (Lat. 37°35' South x Long. 140°10' East)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Tiliqua nigrolutea	Blotched Bluetongue	2	1	R17014
Aprasia striolata	Worm Lizard	1	1	R17017
Lerista bouganvillii	Boulgainvilles Skink	3	1	R17018
Psuedonaja textilis textilis	Common Brown Snake	2	1	R17016

TABLE III - Swamp lands and marshy areas (Lat. 37°41'South x Long. 140°16' East)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Leiolopisma entrecasteauxii	?	2	2	R17019 A-B
Trachydosarus rugosus	Shingle Back	2	-	-
Austrelaps superba	Copperhead	1	-	-

TABLE IV - Dissmal Swamp area

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Notechis scutatus	Tiger Snake D.O.R.	1	-	-
Austrelaps superba	Copperhead	2	-	-
Egernia whiteii	White's Skink D.O.R.	2	-	-
Trachydosarus rugosus	Shingle Back	1	-	-

TABLE V - People present on trip

Brian Miller (Trip Leader)
Gavin Bedford
Julian Craig
Jordy Duffy
Stephen Giddings
Chris Harvey
Mario Lombardi
Andrew Mower
Brian Roberts
Martin Shoemaker
Julia Smith
Corey VanderHoek
Julian White

REPORT OF SOUTH AUSTRALIAN HERPETOLOGY GROUP TRIP

TO CORUNNA HILLS

February 24 - 25, 1979

Leader:- Dr Julian White (Permit No. 265)

Fourteen people in three vehicles participated on the trip. Our original plan was to revisit Uno Bluff, on Hesso Station, but heavy rains forced us to change direction. When we arrived at Corunna, it was still completely overcast. They had had 70 mm of rain in the previous 24 hours. However, the clouds cleared, and for most of the weekend we had sunny, but cool conditions.

Corunna Hills, on Corunna Station, have been surveyed by the S.A.H.G. on two previous occasions. (See previous trip reports). The extensive gorges, rock formations, and spinifex slopes provide rich microhabitats for reptiles.

We saw or collected species of reptiles, all of which had been recorded before from Corunna. Those specimens collected, although duplications of present Museum records, were nevertheless required by the Museum for research and educational purposes.

One species of frog was recorded on the trip, namely the Central Burrowing Frog, *Neobatrachus centralis*. This species has not been recorded from Corunna before, although there are records from adjacent areas. Numbers of this frog were calling, both diurnally and nocturnally, around the margins of temporary pools. Although several specimens were seen in amplexus, no spawn was seen. Eight specimens were collected, as Museum records, and for research purposes.

In addition to native fauna, a number of goats were seen on the property, although there was little evidence of severe damage to the hills vegetation.

The S.A.H.G. gratefully acknowledges the help of the owners of Corunna Station, in allowing us free access to their property.

TABLE I Reptiles recorded at Corunna Hills, on this trip.

(Lat. 32°42'S by Long. 137°6'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus fionii	Peninsula Dragon	10+	2	RNA
Amphibolurus vitticeps	Bearded Dragon	1	1	RNA
Tympanocriptus lineata	Earless Dragon	-	1	RNA
Egernia stokesii	Stoke's Skink	4	-	-
Egernia striolata	Tree Skink	3	2	RNA
Tiliqua branchialis	Spotted-necked Skink	4	2	RNA
Tiliqua scincoides	Common Bluetongue	-	1	RNA
Tiliqua rugosa	Shingleback	-	(4)	(Ed. Dept)
Ctenotus robustus	Striped Skink	3	1	R17099
Ctenotus pantherinus occellifer	Occellated Skink	-	1	RNA
Cryptoblepharus boutonii	Bouton's Skink	1	-	-
Morethia	Snake-eyed Skink	1	-	-
Delma nasuta	Legless Lizard	3	1	RNA
Delma australia	Legless Lizard	2	-	-
Diplodactylus elderi	Jewelled Gecko	4	2	RNA
Diplodactylus vittatus	Stone Gecko	-	1	RNA
Heteronotia binoeii	Bunoe's Gecko	10+	-	-

TABLE II Members Present

Julian White (Leader)
Dave Armstrong
Werner Brunn
Jordy Duffy
Stephen Giddings
Chris Harvey
Brian Miller
Andrew Mower
Brian Roberts
Julia Smith

Craig Chenoweth)
Steven Delean) Primary school students, from Seaview Downs Primary
Stephen Service) School Herpetology Group.
Robert Thompson)

REPORT OF FIELD TRIP TO MIDDLEBACK RANGES

March 24th 1979

Leader:- Dr J. White (Permit No. 265)

A number of herpetologists attending the Second Convention of the Australasian Affiliation of Herpetological Societies, in Whyalla, March 16-19, 1979, visited the Middleback Ranges on 24/3/79 as a Convention field trip. The area examined was the southern section of the ranges, comprising the Iron Duke range. A number of reptiles were seen in this region, especially in the sandy areas around the base of the range. As several permits were operating on this trip, this report will only document those reptiles caught on permit No. 265. These were a pair of Knob-tailed Geckoes (*Nephrurus stellatus*), to be used by Mr Mark Galliford (S.A.H.G.) for breeding research, and a Thorny Devil (*Moloch horridus*), which was lodged with S.A.M. The geckoes were caught while spotlighting, weather conditions being cool to cold. A total of eleven specimens of knob-tailed Gecko were observed during one hours spotlighting at the southern tip of the ranges, in soft sandy country. Numerous other geckoes were also encountered while spotlighting.

The Thorny Devil was found late in the afternoon, in a sandy area, next to a *Triodia* bush. It was clearly active, but not feeding. Of the other reptiles encountered diurnally, two necessitate special mention. Several specimens of a medium sized skink, similar to the Tree Skink (*Egernia striolata*) were found in rock crevices. These appear to be at least a new subspecies of *E. striolata*, but most probably a new species. Two specimens have been lodged at S.A.M. (Not on Permit 265). The other notable find, was a glimpsed goanna like animal, similar in size and description to *Varanus tristis*. Unfortunately this animal eluded capture, and so its identity cannot be confirmed. All other reptiles seen have been recorded from the area previously.

TABLE I - Reptiles seen near Iron Duke 24/3/79
(approx. 33°17'S Lat. x 137°7'E Long.)

Scientific Name	Common Name
Moloch horridus	Thorny Devil
Amphibolurus vitticeps	Bearded Dragon
Amphibolurus cristatus	Crested Dragon
Amphibolurus fionii	Peninsula Rock Dragon
Amphibolurus fordi	Military Dragon
Egernia sp. (?*striolata*)	Tree Skink
Egernia stokesii	Spiny-tailed Skink
Egernia inornata	Desert Skink
Tiliqua occipitalis	Western Bluetongue
Tiliqua rugosa	Shingleback
Tiliqua scincoides	Common Bluetongue
Ctenotus robustus	Striped Skink
Hemiergis	Elongate Triodia Skink
Varnus tristis	Rock Goanna
Nephrurus stellatus	Knob-tailed Gecko
Luscasium damaeum	Beaded Gecko
Rhynchoedura ornata	Beaked Gecko
Diplodactylus intermedius	Spiny-tailed Gecko
Heteronotia binoeii	Binoe's Gecko
Pseudechis australia	Mulga Snake
Pseudonaja nuchalis	Western Brown Snake

TABLE II - Herpetologists present on field trip.

Dave Armstrong (S.A.H.G.)
Graham Armstrong (S.A.H.G.)
Brenton Arnold (S.A. N.P. & W.S.)
Steve Berry (S.A.H.G.)
Steve Doyle (S.A.H.G.)
Harry Ehmann (A.A.H.S.)
Steve Giddings (S.A.H.G.)
Keith Gilbertson (S.A.H.G.)
Graeme Gow (N.T.M.)
Chris Harvey (S.A.H.G.)
Fred Hersey (N.S.W. N.P. & W.S.)
Peter Hudson (W.H.G.)
Grant Husband (A.H.S.)
Darryl Levi (S.A.H.G.)
Peter Mirtschin (W.H.G.)
Dave Morafka (Uni of Arizona)
Andrew Mower (S.A.H.G.)
Harald Nygren (W.H.G.)
Tony Robinson (S.A. N.P. & W.S.)
Martin Schumaker (S.A.H.G.)
Tony Sheargold (A.H.S.)
Julia Smith (S.A.H.G.)
Julian White (S.A.H.G.)

REPORT OF FIELD TRIP TO BLANCHETOWN AREA

MARCH 24TH 1979

Leader:- Dr J. White (Permit No. 265)

To complete some photography on 24/3/79, three S.A.H.G. members, Dr Julian White, Ms Corie Van-der-Hoek, and Mr Steve Berry, visited an area of sand and *Triodia*, with stands of *Mallee* scrub, approximately 10 km north of Blanchetwon, along the Blanchetown to Waikerie road. The area was on the southern side of the road, no more than 200 metres from the bitumen. All *Triodia* bushes were healthy.

Military Dragons (*Amphibolurus fordi*) were very common, active around the edges of the *Triodia*. They were observed eating small ants exclusively. Adult to juvenile ratio was approximately 1:1. On dismantlement of *Triodia* bushes, three species of reptiles were found. The smallest was a *Hemiergis* sp., light brown in colour, pentadactylate, the juveniles having a distinct red tinge to the tail. The second species was a striped skink (*Ctenotus regius*). The third species also appeared to be a striped skink, which keyed out to *Ctenotus piankai*. If this is indeed its species, then the known range of *piankai* has been extended by nearly 1 500 kms. Specimens of all three species from *Triodia* have been lodged with S.A.M.

Table I - Specimen details

Scientific Name	Common Name	S.A.M. Reg. No.
Ctenotus regius	Striped Skink	R17160
Ctenotus sp. (?)	Striped Skink	R17159
Hemiergis millewae	-	R17161

REPORT OF S.A.H.G. TRIP TO ANNA CREEK, LAKE EYRE, AND MOKARI

April 12th-21st 1979

Leader:- Dr J. White (Permit No. 265)

Aim:- Our original aim was to survey the Anna Creek region and edge of Lake Eyre and then move on to survey the Everard Ranges. However the latter locality was not open, so instead the group visited Mokari. All work on the trip can only be considered as initial survey, as far more time will be required in the area looked at.

Report:- The trip commenced with an advance party (D. Holly and S. Giddings) leaving 2 days early to lay pit-trap lines on Anna Creek Station. This they did amongst a low red dune complex, covered with canegrass. No *Triodia* was in evidence, and the area was clearly grazed by cattle. The rest of the group arrived over the next three days. Reptiles caught in this area are noted in table 1, and those caught at nearby Anna Creek homestead and dump, noted in table 2. There were no outstanding finds. On the 14th, a day trip to the western margins of Lake Eyre was held, ending up at the top end of Belt Bay, near Cooinchina Creek. Few reptiles were seen on this road, mostly gibber plain and hills, with some adjacent sandy areas. Time did not permit survey of these dunes. The Lake itself was dry as far as observable. Numerous *Amphibolurus maculosus* holes were seen and excavated, but no specimens were found. Lizards seen and collected on and on the margins of Lake Eyre are listed in table 4. The Earless Dragon was caught on light gibber, on the Lake surface, some 200 metres from shore. The Binoe's Gecko was found under rocks at the shoreline.

The following day was spent checking the Anna Creek dump and sand hills, before moving on towards Oodnadatta. The Anna Creek pitfalls proved moderately successful, especially in catching Striped Skinks and Burrowing Skinks. Dragons seemed able to avoid falling into the pits, their tracks neatly circling the pit margins.

On the road to Oodnadatta several stops were made. The notable ones are listed in tables 5, 6 & 7. Camp near the Peake, was in gibber margined dunes, and here a native marsupial was collected (*Sminthopsis crassicaudata*). At Algebuckina, on The Neals River, were numerous permanent and semi permanent waterholes, teaming with several species of Rainbow Fish, and Desert Goby. The trees along the water-hole margins were populated with Long-Snouted Dragons (table 7) in moderate numbers. These were subsequently seen at other creeks along the William Creek - Oodnadatta road. No Gibber Dragons were sited along the road, though this is a major section of their distribution.

At Oodnadatta, following local advice, it was decided to abandon the trip to the Everard Ranges, and instead the group headed for Mokari, on the eastern side of the Simpson Desert, just below the Northern Territory border. As a long distance, over often abysmal tracks, had to be covered in a short space of time, regretably little time was available for collecting along the way. Tables 8 - 14 cover reptiles observed on this section of the trip.

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Our route lay via Macumba Station, which is reached by a long drive over lower gibber hills and plains. No stops were made here, and consequently no reptiles seen. From Macumba the road wove northwards through lush cattle country, before entering a large sand dune section, the south western tip of the Simpson desert. Here the dunes, approximately 13m high, are long parallel, north south oriented, with thick canegrass cover and occasional spinifex. The interdunal areas are wide flat gibber plains. The group camped in this area just south of Oolgawa Waterhole. Inclement weather stopped extensive searching but an interesting bandless *Sphenomorphus richardsoni* like skink was seen on the dune crest. Unfortunately it eluded capture. On the interdunal areas, Netted Dragons were captured, down shallow burrows (table 8). No low shrubs were available for perching, as described by Heatwole (1970, Ecolg. Monog. 40: 425-57), suggesting that the thermoregulatory behaviour he described is not universally used by this species group.

From Oolgawa, the road wove through a section of gibber plains, creeks, and low rocky gibber hills, some with small rock outcrops. The most prominent of these were searched (table 9), and among other reptiles seen, a type of Red Barred Dragon was collected. This may prove to be a new subspecies on further investigation. It is certainly a significant range extension. A juvenile Perentie was also observed active on the hill slopes adjacent to the Red Barred Dragons, which preferred the smaller rocks, approximately half way up the hill slopes. None were seen on the upper half of the slopes.

Several other stops were made amongst the gibber (tables 10, 11), the only notable find being a very large Perentie, which allowed approach to within 1 metre. It had scars on its back consistent with an attack by an unnatural predator (man).

After the gibber the road wound through several claypans, before entering the north south red dunes of the Simpson Desert. Soon after entering the dunes, Purni Bore was reached. Here a pitfall line was laid amongst the Spinifex (table 12). However, few reptiles, or reptile burrows were in evidence, and the pit traps were not productive, except for *Lerista*.

From Purni Bore, the Group moved eastwards across the dunes to Mokari camp, some 60 kms into the desert. Here, sparse spinifex and canegrass, with low shrubs and trees covered red and interdunal areas and dune slopes. Reptile burrows and tracks abounded everywhere (table 14). Desert Skinks were very common, with an estimated density of 2-4 specimens per square metre. They appeared to be feeding chiefly on large ants. Among the interesting tracks seen, were those of small goannas, weaving amongst the Spinifex. These tracks were almost certainly made by the Desert Pygmy Goanna, though no actual specimens were found.

This area around Mokari, and the road to Mokari are very interesting and will repay more intensive study in the future. From Mokari the Group returned to Adelaide, along the same route. No startling finds were made on the return journey.

The S.A.H.G. wishes to acknowledge the help and cooperation of the following people:- Dick Nunn, Anna Creek Station, Mr Pecanek, Oodnadatta Stores, Adam & Lyn, Oodnadatta Workshop.

Table 1 - Reptiles caught at Anna Creek Camp Sandunes
(Lat 28°54'S x Long. 136°09'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Tympanocriptis intima	Earless Dragon	-	2	R17297
Amphibolurus pictus	Painted Dragon	3	2	R17302
Ctenotus brooksi	Striped Skink	-	2	R17300
Ctenotus regius	Striped Skink	-	2	R17299
Lucasium damaeum	Beaded Gecko	-	1	R17301
Heteronotia binoeii	Binoes Gecko	-	1	R17298

Table 2 - Reptiles caught at Anna Creek Dump
(Lat 28°54'S x Long 136°10'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Lerista bipes	Burrowing Skink	3	3	R17305
Diplodactylus tesselatus	Tesselated Gecko	1	-	-
Ctenotus regius	Striped Skink	-	2	R17304
Pseudonaja nuchalis	Western Brown Snake	1	-	-
Gehyra variegata	Dtella	-	1	R17303

Table 3 - Reptiles caught on road to Lake Eyre - gibber hills
(Lat. 28°47'S x Long. 136°49'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Morethia adelaidensis	Snake-eyed Skink	-	1	R17310
Lerista meuleri	Burrowing Skink	-	1	R17306

Table 4 - Reptiles caught on margins of Lake
(Lat. 28°46'S x Long. 136°53'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus pictus	Painted Dragon	-	1	R17309
Tympanocriptis intima	Earless Dragon	3	1	R17307
Heteronotia binoeii	Binoes Gecko	-	1	R17308

Table 5 - Reptiles seen on road to Oodnadatta - hilly stop
(Lat. 28°35'S x Long. 135°53'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Egernia stokesii	Spiny-tailed Skink	2	-	-

Table 6 - Reptiles and mammals seen at Peake Camp - gibber
(Lat. 28°05'S x Long. 135°50'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Lucasium damaeum	Beaded Gecko	4	-	-
Sminthopsis crassicaudata	Fat-tailed Dunnart	-	1	R.N.A.

Table 7 - Reptiles caught at Neales River crossing
(Lat. 27°54'S x Long. 135°48'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Lophognathus longirostris	Longsnouted Dragon	4	1	R17311

Table 8 - Reptiles caught near Oolgawa Waterhole sand dunes (Lat. 26°49'S x Long. 135°53'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus inermis	Netted Dragon	1	2	R.N.A.

Table 9 - Reptiles caught in rocky hills - road to Mokari (approximate position Lat. 26°45'S x Long. 135°48'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Amphibolurus pictus	Painted Dragon	3	1	R17313
Amphibolurus vadnappa	Red Barred Dragon	-	6	R.N.A.
Heteronotia binoeii	Binoes Gecko	-	1	R17314
Ctenotus brooksii	Striped Skink	6	2	R17315
Varanus giganteus	Perentie	1	-	-

Table 10 - Reptiles caught on Gibber - road to Mokari (approximate position Lat. 26°41'S x Long. 135°40'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Tympanocriptis intima	Earless Dragon	-	1	R17312
Ctenotus broksii	Striped Skink	1	-	-
Varanus giganteus	Perentie	1	-	-

Table 11 - Reptiles caught on gibber - road to Mokari (approximate position Lat. 26°36'E x Long. 135°51'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Gehyra variegata	Dtella	-	2	R17316

Table 12 - Reptiles caught at Purni Bore (Lat. 26°17'S x Long. 136°06'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Egernia inornata	Desert Skink	2	1	R17318
Lerista bipes	Burrowing Skink	2	1	R17317

Table 13 - Reptiles caught on road to Mokari - Simpson Desert (Lat. 26°14'S x Long. 136°20'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Egernia inornata	Desert Skink	-	1	R17319

Table 14 - Reptiles caught at Mokari camp (Lat 26°19'S x Long. 136°27'E)

Species	Common Name	No. Seen	No. Collected	S.A.M. Reg. No.
Egernia inornata	Desert Skink	10	3	R17320
Lerista bipes	-	-	1	R17322
Notoscincus ornatus	-	-	1	R17324
Ctenotus regius	Striped Skink	3	2	R17321
Ctenotus brooksi	Striped Skink	-	1	R17323
Varanus gouldii	Gould's Goanna	1	-	-
Varanus eremius	Desert Pygmy Possum	- tracks only - not positively Ident.		
Nephrurus laevis	Knob-tailed Gecko	-	1	R.N.A.

Table 15 - Members present

Julian White (Leader)
Gavin Bedford
Steve Berry
Werner Brunn
Julian Craig
Steve Doyle
Jordi Duffy
Steve Giddings
Rod Gill
Ronda Gill
Doug Holly
Mario Lombardi
Beryl Morris
Tony Sadler
Julia Smith

Table 16 - Reptiles kept alive by S.A.H.G. members for study purposes.

Species	Common Name	Member	No. Kept	Permit No.
Nephrurus laevis	Knob-tailed Gecko	M. Galliford	1	01348-2
Amphibolurus vadnappa	Red-Barred Dragon	J. Duffy	4	12778-1
Amphibolurus inermis	Netted Dragon	R. Gill	2	12580-2

REPTILES OF THE NINETY MILE DESERT
(prepared by Dr. Julian White, South Australian Herpetology Group Inc.)

This report is a compilation of data collected on a number of trips by the South Australian Herpetology Group Inc., and participants in the N.C.S. Survey in October 1977.

From these trips, a total of 22 species of reptiles have been recorded from the Ninety Mile Desert. These are listed in table 1, which includesthe authors opinions about the relative abundance of each species. Table 2 contains detailed listing of each capture locality including South Australian Museum record numbers.

A number of the species collected were significant range extensions. The wet coastal form of sand goanna, Varanus gouldii rosenbergii is usually found in areas close to the coast, or on islands, especially Kangaroo Island. Prior to these surveys, it had never been recorded so far inland. However the habitat encountered in the Ninety Mile Desert is substantially similar to known habitats of this species in the Coorong region and parts of the lower Mount Lofty Ranges (eg. Coxes Scrub C.P.).

Several of the small skinks collected have provided range extensions. Leiolopisma delicata previously only recorded in the lower south east of S.A., has had the northern extent of it's range extended considerably. Similarly, Lerista frosti has had it's southern range extended.

The small dragons, Amphibolurus fordi and Amphibolurus pictus , are near the southernmost limit of their range, in the Ninety Mile Desert, as is the large skink Tiliqua occipitalis.

Collection methods employed included pitfall and mammal trap lines, spotlighting, selective dismantling of vegetation, and foot searches. Pitfalls captured several species including Ctenotus robustus, Amphibolurus muricatus, Drysdalia mastersii, and Typhlina australis. Mammal trap lines captured Pseudonaja textilis and Trachydosaurus rugosus Spotlighting was singularly unsucessful, although this was partly attributable to unsuitable weather during the survey periods. The most productive capture method was foot search and sellected dismantling of vegetation (especially Triodea).

Most of the reptiles presently inhabiting the Ninety Mile Desert are dependent on natural habitat for survival, and are unlikely to successfully adapt to survival on cleared land. Exceptions to this include the Brown Snake, Pseudonaja textilis, and the Shingleback Skink Trachydosaurus rugosus, both of which have adapted elsewhere even to the extremes of an urban envioronment.

Most of the reptiles of the region are small by reptilian standards. The vast majority utilize the soft sandy soil, leaf litter, and low shrubs and bushes, especially Triodea. Some are found only in association with Triodea, eg. Delma australis and Amphibolurus fordi. This latter species is one of the most abundant reptiles in the area. Several require leaf litter eg. Lerista bougainvillii and Lerista frosti. Some require trees with suitable bark, such as some large Eucalypts. eg Phyllodactylus marmoratus. All of these species would suffer drastic falls in population following land clearing.

Because of this and the fact that the area appears to be an intermediate zone between south-eastern, and northern herpetofauna, it's retention in a natural state is vital.

TABLE 1 ; List of reptiles recorded from the Ninety Mile Desert.

Scientific name	Common name	Status	Habitat
(a) DRAGONS (family Agamidae)			
Amphibolurus vitticeps	Bearded Dragon	Common	General
Amphibolurus pictus	Painted Dragon	Common	Mixed scrub
Amphibolurus fordi	Military Dragon	Abundant	Triodea scrub
Amphibolurus muricatus	Jacky Lizard	Common	Mixed scrub
(b) GOANNAS (family Varanidae)			
Varanus gouldii rosenbergii	Rosenbergs Goanna	Uncommon	Mixed scrub
(c) GECKOS (family Gekkonidae)			
Phyllodactylus marmoratus	Marbled Gecko	Common	Eucalypt stands
(d) LEGLESS LIZARDS (family Pygopodidae)			
Delma australis	Spinifex Legless L.	Common	Triodea scrub
(e) SKINKS (family Scicidae)			
Trachydosaurus rugosus	Shingleback	Common	General
Tiliqua occipitalis	Western Bluetongue	Uncommon	Mixed scrub
Ctenotus robustus	Striped Skink	Common	" "
Ctenotus brooksii	" "	Uncommon	" "
Ctenotus Uber	" "	"	" "
Morethia obscura	Snake-eyed Skink	Abundant	" "
Hemiergis peroni	Perons Skink	Uncommon	" "
Menetia greyii	Greys Skink	Common	Eucalypt stand
Lerista bougainvillii	Bougainvilles Skink	Abundant	General
Lerista frosti	Sand Skink	Uncommon	Mixed scrub
Leiolopisma delicata	Grass Skink	Uncommon	" "
(f) BLIND SNAKES (family Typhlopidae)			
Typhlina australis	Blind Snake	Uncommon	Sandy areas
(g) ELAPID SNAKES (family Elapidae)			
Pseudonaja textilis	Brown Snake	Common	General
Drysdalia mastersii	Master's Snake	Common	Mixed scrub
Unechis brevicauda	Mitchells Snake	Uncommon	" "

TABLE 2 ; Collection localities and specimens.

Scientific name	Number collected (S=seen,identified But not collected.)	S.A.Museum Registration NOS. (RNA= Record not currently available.)
(a) lat. $34^{\circ}41'$ X Long. $140^{\circ}47'$		
Amphibolurus vitticeps	S	-
Amphibolurus fordi	1	R15137
Amphibolurus pictus	S	-
Delma australis	2	R15195, R15619
Ctenotus brooksi	1	R15193
Ctenotus uber	1	R15618
Morethia obscura	1	R15139
Menetia greyi	S	-
Lerista bougainvillii	1	R15138
Drysdalia mastersii	1	R15194
Pseudonaja textilis	S	-
Unechis brevicauda	1	RNA

(b) lat. 35°40' X long. 140°46'			
Amphibolurus muricatus	1	RNA	
Amphibolurus pictus	2	R15140	A&B
Amphibolurus fordi	S	-	
Morethia obscura	S	-	
Trachydosaurus rugosus	S	-	
(c) lat. 35°43' X long. 140°24'			
Amphibolurus vitticeps	1	RNA	
Amphibolurus fordi	1	R16169	
Amphibolurus pictus	1	R16168	
(d) lat. 35°39' X long. 140°45'			
Varanus gouldii rosenbergii	1	R16160	
Delma australis	1	R16176	
Lerista bougainvillii	1	R16175	
(e) lat. 35°40' X long. 140°25'			
Amphibolurus vitticeps	1	R16167	
Amphibolurus fordi	1	R16166	
Amphibolurus pictus	2	R16165	A&B
Morethia obscura	1	R16163	
Ctenotus robustus	2	R16164	A&B
(f) lat. 35°45' X long. 140°20'			
Amphibolurus muricatus	1	R16171	
Drysdalia mastersii	1	R16170	
(g) lat. 35°45' X long. 140°48'			
Phyllodactylus marmoratus	1	R16172	
Morethia obscura	1	R16173	
Menetia greyi	2	R16174	A&B
(h) lat. 35°39' X long. 140°22'			
Typhlina australis	1	R16182	
Drysdalia mastersii	1	R16186	
(i) lat. 35°38' X long. 140°23'			
Trachydosaurus rugosus	2	R16184	A&B
Pseudonaja textilis	1	R16183	
(j) lat. 35°37' X long. 140°35'			
Leiolopisma delicata	1	R16177	
(k) lat. 35°37' X long. 140°23'			
Phyllodactylus marmoratus	2	R16180	A&B
Lerista frosti	1	R16178	
Drysdalia mastersii	1	R16179	
(l) lat. 35°37' X long. 140°24'			
Amphibolurus fordi	1	R16181	
(m) lat. 35°45' X long. 140°21'			
Hemiergis peroni	1	R16185	
Tiliqua occipitalis	1	RNA	
Drysdalia mastersii	1	R16187	

OLYMPIC DAM PROJECT
ENVIRONMENTAL STUDIES

BASELINE REPORT AND IMPACT ASSESSMENT

HERPETOLOGY

Dr Julian White

February 1982

CONTENTS

1 INTRODUCTION

1.1 BACKGROUND

Kinhill Stearns-Roger in late 1980 required a survey of reptile fauna at and in the vicinity of Olympic Dam, as part of baseline studies for an Environmental Impact Statement (EIS). An initial pilot fauna survey by Fatchen and Reid (1980) identified likely and definite reptiles species in the area, and possible impacts of mining development. It is the purpose of this report to expand on this survey, and provide as complete an assessment of the diversity and abundance of reptile fauna of the areas as possible within survey limitations. In addition, likely impacts of the Project on this diversity and abundance are discussed.

1.2 IMPORTANCE OF REPTILES

Australia has a large and diverse reptile fauna population, which has only been studied in detail in recent years. Their physiology allows them to utilize arid Australian habitats successfully, and in consequence they are the most populous and diverse terrestrial vertebrate group in Australian arid habitats, such as that at Olympic Dam. However, the remoteness of, and difficulties of studying, such habitats have resulted in a relative paucity of information about their distribution, abundance, and ecology. Most information on distribution is based on limited surveys, and many areas have barely been examined, so that Museum records cannot give an accurate indication of distribution. Information on reptile distribution in South Australia is limited, the main recent sources all being based on South Australian Museum spirit records (Houston 1978, White 1981) and general knowledge (Cogger 1979). Ecological studies are also limited, although some useful works such as that of Pianka (1972), and Heatwole (1976) exist.

In this relative vacuum of information even simple surveys provide significant extensions of knowledge. Given these factors, and the limitations of time and resources available for an EIS, many conclusions and recommendations of this report are extrapolations from limited data, enhanced by consultant experience in the field of herpetology, particularly within South Australia.

The sheer diversity of reptile fauna encountered in this survey points to the vital role of reptiles in the overall ecology of the Olympic Dam area. Experience with a snakebite incident in the exploratory phase of mining activities illustrates just one of the interactions between man and industry, and the reptile fauna, which makes an assessment of reptile fauna an important aspect of an EIS.

1.3 PLAN OF REPORT

This report covers three main subjects. First, the reptile fauna is discussed in detail, with species lists, and some basic ecological data. Second, the environmental impact of the mining Project is discussed in relation to the reptile fauna. Last, mitigation measures are examined, with recommendations to minimize the effect of mining on reptiles, and reptiles on mining and development.

2 REPTILE FAUNA

2.1 SITE

The country surrounding Olympic Dam is dominated by sections of fixed parallel sand dunes, and intervening swales, claypans, and sections of flat gibber plain. Within the dune and swale sections a variety of vegetation associations exist. These provide a variety of habitats for reptiles allowing a diversity of reptile species.

2.2 EXISTING INFORMATION AND LIMITATIONS

Very little survey work on reptiles in the general area including Olympic Dam had been carried out prior to the EIS studies. There are few records from this area in the South Australian Museum. Based on these records and extrapolating from records in similar habitat near the study area, a preliminary list of reptile species that may be present can be constructed (Table 2.1).

Fatchen and Reid (1980) made a further annotated species list based on personal experience in the area, and reports from mining workers at Olympic Dam (Table 2.2).

However, it can be expected that in such an arid environment, many reptile species will be cryptic or nocturnal, and these species would not be effectively sampled by the methods of Fatchen and Reid, a point they acknowledged in their report.

A further assessment of potential reptile species in the area can be gained by utilizing the experience of a senior field experienced herpetologist, as embodied in the distribution maps of Cogger's (1979) major work on Australian reptiles, although at least some of his maps have proved inaccurate. This yields a list of reptiles which may be present (Table 2.3).

2.3 PRESENT SURVEY

Because of the inadequacy of existing data, an extensive programme of sampling the reptile fauna was planned, with sampling during all four seasons, involving multiple trips to the site. This sampling was designed to answer the following questions:

- What reptile species exist in the Olympic Dam mining area?
- What reptile species exist in similar habitats outside the mining area?
- What is the approximate and relative abundance of these reptile species?
- What are the main food items of these reptiles?

The answers to these questions would then provide the data base for:

- Assessing the importance of the reptile fauna of the mining area, and their uniqueness if any.
- An ongoing assessment of the potential and actual impact of mining on the reptile fauna.

Details of methods will be presented in following sections of this report. Personnel used in the survey were all under the guidance of the principal consultant. The majority of

Table 2.1 **Reptiles in the Olympic Dam area, based on records of the South Australian Museum**

Gekkonidae	**Status**
Rhynchoedura ornata	3
Phyllurus milii	3
Nephrurus laevis	3
Heteronotia binoei	2
Gehyra variegata	1
Diplodactylus vittatus	2
Diplodactylus tessellatus	2
Diplodactylus stenodactylus	3
Diplodactylus ciliaris	3
Diplodactylus byrnei	3
Lucasium damaeum	2
Pygopodidae	
Pygopus lepidopodus	3
Lialis burtonis	3
Delma australis	3
Pygopus nigriceps	1
Varanidae	
Varanus gouldii	3
Agamidae	
Tympanocryptis tetraporophora	2
Tympanocryptis intima	1
Diporiphora winneckei	3
Amphibolurus vitticeps	3
Amphibolurus vadnappa	3
Amphibolurus pictus	2
Amphibolurus maculosus	2
Amphibolurus maculatus	2
Amphibolurus inermis	2
Amphibolurus gibba	3
Amphibolurus fordi	2
Amphibolurus fionni	3

Scincidae	**Status**
Tiliqua rugosa	3
Eremiascincus richardsoni	2
Morethia boulengeri	2
Morethia adelaidensis	3
Menetia greyii	3
Lerista muelleri	3
Lerista bougainvillii	2
Lerista bipes	2
Lerista labialis	1
Egernia stokesii	3
Ctenotus uber orientalis	2
Ctenotus strauchii	1
Ctenotus robustus	3
Ctenotus regius	2
Ctenotus leae	2
Ctenotus brooksi	2
Cryptoblepharus boutonii	3
Typhlopidae	
Typhlina australis	3
Typhlina bituberculata	3
Typhlina endotera	2
Boidae	
Liasis childreni	3
Elapidae	
Demansia psammophis	3
Pseudechis australis	3
Pseudonaja modesta	2
Pseudonaja nuchalis	1
Simoselaps bertholdi	1

Key: 1 = recorded at or near Olympic Dam
2 = recorded within 30 minutes latitude and longitude of Olympic Dam
3 = recorded within 1 degree latitude and longitude of Olympic Dam

Table 2.2 **Reptiles encountered or reported during 1980 Olympic Dam survey (Fatchen and Reid)**

Gekkonidae	**Varanidae**	**Agamidae**	**Scincidae**
Gehyra variegata	Varanus gouldii	Amphibolurus fordi	Egernia inornata
Heteronotia binoei		Amphibolurus vitticeps	Tiliqua rugosa
Lucasium damaeum			
Diplodactylus ciliaris			

Table 2.3 **List of reptile species which may be in Olympic Dam area, based on Cogger (1979)**

Gekkonidae
Diplodactylus byrnei
Diplodactylus ciliaris
Diplodactylus conspicillatus
Diplodactylus elderi
Diplodactylus stenodactylus
Diplodactylus tessellatus
Diplodactylus vittatus
Gehyra variegata
Heteronotia binoei
Lucasium damaeum
Nephrurus levis
Oedura marmorata
Rhynchoedura ornata
Phyllurus milii

Pygopodidae
Delma australis
Delma nasuta
Delma tincta
Lialis burtonis
Pygopus nigriceps

Varanidae
Varanus eremius
Varanus giganteus
Varanus gilleni
Varanus gouldii
Varanus tristis

Agamidae
Amphibolurus cristatus
Amphibolurus fordi
Amphibolurus isolepis
Amphibolurus maculosus
Amphibolurus minor
Amphibolurus nuchalis
Amphibolurus pictus
Amphibolurus reticulatus
Amphibolurus vitticeps
Diporiphora winneckei
Lophognathus longirostris
Moloch horridus
Tympanocryptis intima
Tympanocryptis lineata
Tympanocryptis tetraporophora

Scincidae
Cryptoblepharus boutonii (plagiocephalus)
Ctenotus atlas
Ctenotus brooksi
Ctenotus leae
Ctenotus leonhardii
Ctenotus pantherinus
Ctenotus regius
Ctenotus robustus
Ctenotus schomburgkii
Ctenotus strauchii
Ctenotus uber
Hemiergis millewae
Egernia inornata
Egernia stokesii
Egernia striolata
Lerista bougainvillii
Lerista desertorum
Lerista frosti
Lerista labialis
Lerista muelleri
Lerista punctatovittata
Menetia greyii
Morethia adelaidensis
Morethia boulengeri
Eremiascincus richardsoni
Eremiascincus fasciolatus
Tiliqua branchialis
Tiliqua occipitalis
Tiliqua rugosa

Typhcopidae
Typhlina australis
Typhlina bituberculata
Typhlina endotera

Boidae
Liasis childreni
Python spilotes variegata

Elapidae
Acanthophis pyrrhus
Demansia psammophis
Furina diadema
Neelaps bimaculatus
Pseudechis australis
Pseudonaja modesta
Pseudonaja nuchalis
Simoselaps bertholdi
Simoselaps fasciolatus
Simoselaps semifasciatus
Suta suta
Unechis gouldii
Unechis monachus
Vermicella annulata

experienced field herpetologists in South Australia are 'amateurs', from whom much of the field personnel were drawn. All of these were from the South Australian Herpetology Group Inc. a group of amateur and professional herpetologists closely linked with the South Australian Museum, and with a recent history of active field work in South Australia, especially in arid areas.

2.4 BASELINE STUDIES

The purpose of these studies was to answer the first two questions of Section 2.3.

2.4.1 Methods

Four survey trips were made. The first trip was 17 to 20 April, 1981. Eight herpetologists assisted the consultant on this initial trip, with a total of 220 diurnal man/field hours and 100 nocturnal man/field hours. Representative sites were preselected from aerial photographs and maps, and confirmed by ground survey. All habitat types within the mining area and periphery were surveyed by on foot search. Nocturnal surveys were performed both by vehicle based spotlighting, and on foot spotlighting across dunes, where vehicle access was impractical.

The principal sampling method was pit trapping using standard pit traplines, with metal pits sunk into soil and joined by a 200 mm high inset mesh fence supported by metal strainers. Inter-pit distance was variable, being dependent on terrain and habitat, but was nowhere more than 10 m between pitfalls. Locations of traplines were based on least disturbed examples of good reptile habitat at the periphery of the main exploration area (Diagram 1). Three lines were laid, one extensive over swale and dune (line 1), and two short over open dune (line 4), and wooded dune (line 5). There were approximately 1,500 trap hours on the April survey. Following completion of this trip, pit traps were capped and fences removed.

The second visit was from 18 to 21 September, 1981, with the principal consultant and three other herpetologists, one of whom was Dr T. Schwanner, Curator of Reptiles, South Australian Museum. Sampling methods were identical with those used in April. Traplines 1 and 4 were reset. Line 5 was not used, and a new swale trapline (line 6) was set. Approximately 110 diurnal man/field hours and 36 nocturnal man/field hours were expended, and about 1,200 trap hours over this visit.

The third visit was from 23 to 26 October, 1981, with the principal consultant and three other herpetologists. This visit coincided with the second part of the mammal survey, as planned, and sampling was based on the very extensive series of mammal traplines, the structure of which is virtually identical with reptile traplines. This mammal survey extended for three weeks, and had been in progress for one week at the time of the reptile survey. All reptiles caught in mammal traplines were retained by the principal consultant (mammals), Mr Peter Aitken, Curator of Mammals, South Australian Museum. These were then transferred to the reptile consultant. A large mass of reptiles was accumulated. Processing of these, plus assisting in checking mammal lines, and laying new lines, prevented re-laying of existing reptile lines. The locations of mammal lines (Diagram 1, localities 11-21) are well outside the Operations Area, and allow comparative sampling between prime examples of habitat off-site and on-site sampling. Several thousand trap hours of sampling were obtained using these lines. In addition, approximately 30 nocturnal field/hours were expended.

The fourth visit was from 16 to 23 December, 1981, and two herpetologists participated. This visit was designed to sample summer reptile fauna, collect baseline data on abundance, and collect sufficient freshly killed reptiles for stomach content analysis.

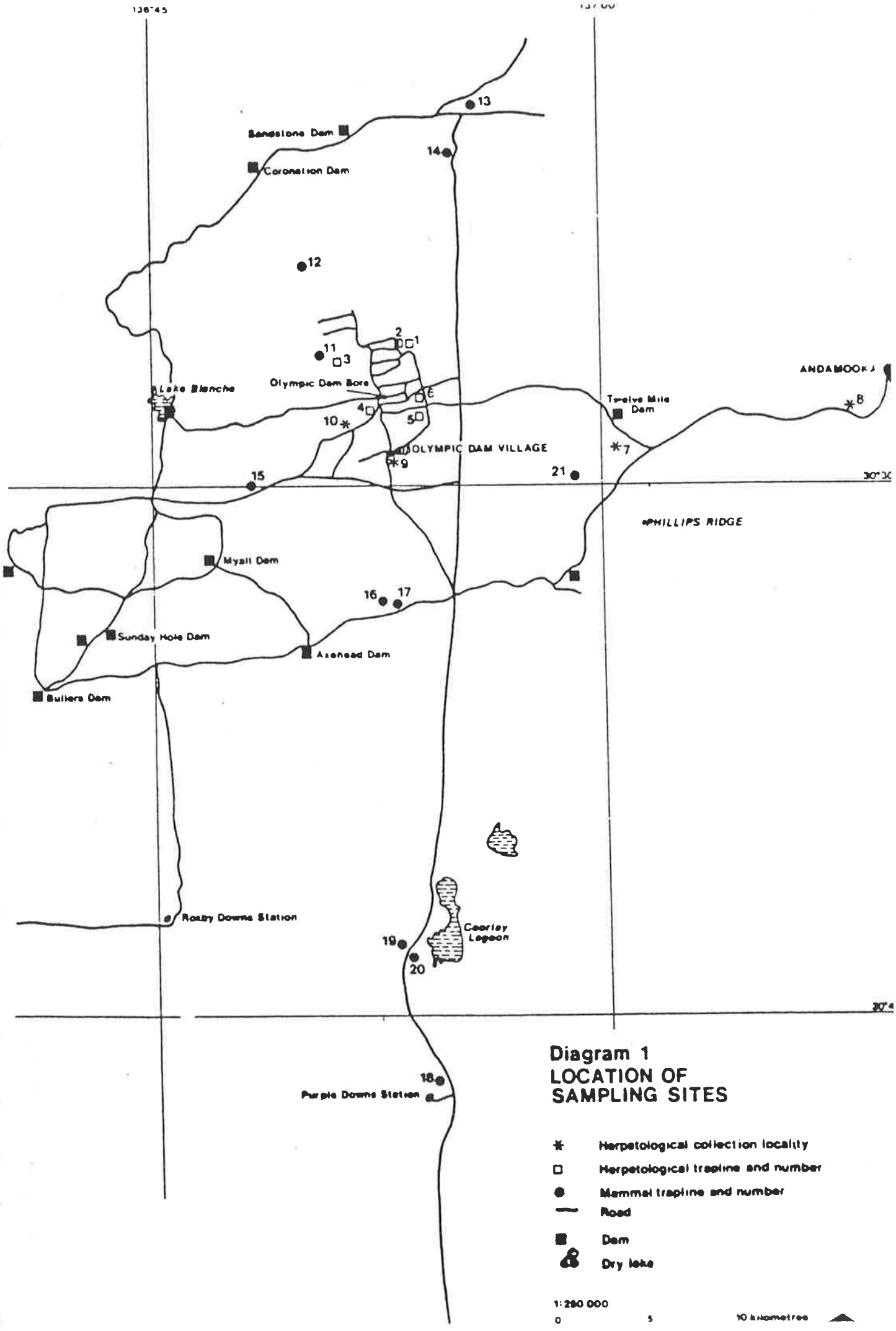

Diagram 1
LOCATION OF SAMPLING SITES

- Herpetological collection locality
- Herpetological trapline and number
- Mammal trapline and number
- Road
- Dam
- Dry lake

1:250 000

0 5 10 kilometres

Three traplines were laid (lines 1 to 3), and sampled over 7 days. In addition, spotlighting was carried out. Approximately 40 nocturnal field/man hours and approximately 80 diurnal field/man hours were expended, in addition to time spent laying and checking traplines.

On all trips, reptiles collected were deposited with the South Australian Museum. All were registered; some were used for tissue sampling, and some were maintained alive for research (Tiliqua spp.).

A brief summary of vegetation associations for each trapline is given in Table 2.4.

Table 2.4 Principal vegetation of traplines

	Line 1	Line 2	Line 3
Swale	Sclerolaena sp. Callitris sp. Maireana sp.	Sclerolaena sp. Callitris sp. Maireana sedifolia	Atriplex vesicaria Dodonaea sp.
Dune	Dodonaea sp. Acacia sp.	Dodonaea sp. Acacia sp. Hakea leucoptera	Dodonaea sp. Acacia sp.

2.4.2 Results

Reptiles collected on each trip are listed in Table 2.5. A complete list of all reptiles registered to date in the South Australian Museum is provided in Appendix A. Table 2.6 lists those reptiles known or likely to occur in the survey area, specifying those found in the mining site, or areas of similar habitat outside this area (control areas). For Table 2.6 locality numbers are based on Diagram 1, with numbers referring to localities on this map, and G indicating numerous finds over a wide area, not codifiable within the distinct numbered localities. Further details of locality may be found by examining South Australian Museum records and South Australian Herpetological Group data sheets.

The survey discovered a surprisingly large diversity of reptile fauna all with a wide and secure distribution. After review of the South Australian Museum records and Cogger (1979) (Table 2.1 to 2.3), and examination of available habitats, it is clear that 63 species of reptile could be expected in the region immediately to the west of Lake Torrens, including Olympic Dam. Several of those would be on the borders of their range and may well not be present. Forty-five species were actually found, a very high percentage of expected fauna, considering the survey time available.

Most species were found in significant numbers at a variety of habitats in the Olympic Dam area, and control areas. Most occupied a variety of habitats, although analysis of trapline results revealed the expected habitat preferences of most species. This will be examined further in Section 2.5.

None of the species found or expected are classified as rare, threatened, or endangered (Jenkins 1979). One type of Ctenotus skink was found outside the Olympic Dam area, which may prove to be a new species. According to Dr Schwanner (pers. comm.) there are specimens of this skink from other areas of the State.

Only two species of reptiles were found in the Olympic Dam area, and not in control sites. Both of these, Diplodactylus stenodactylus and Ctenotus leonhardii are represented in South Australia beyond the control area.

Table 2.5 Reptiles of Olympic Dam Area

Species	Localities			
	April 1981	September 1981	October 1981	December 1981
Gekkonidae				
Rhynchoedura ornata	G 1	G 1, 6	G 12	1, 2, 3
Lucasium damaeum	G 1	G 1, 4	G 12	1, 2
Gehrya variegata	G 9, 10	-	-	-
Diplodactylus stenodactylus	1	G 1, 6	G	1, 2, 3
Diplodactylus conspicillatus	-	1	G 11, 12	1, 2
Diplodactylus ciliaris	-	-	12	-
Diplodactylus tessellatus	-	-	G 11, 14	-
Diplodactylus vittatus	-	-	-	-
Diplodactylus byrnei	-	-	-	-
Diplodactylus intermedius	-	-	-	2
Nephrurus levis	G 10	-	14	2
Heteronotia binoei	G 7, 9, 10	9	G	20
Phyllurus milii	-	-	20	-
Pygopodidae				
Pygopus nigriceps	-	1	11, 14, 18	-
Pygopus lepidopodus	-	-	-	-
Lialis burtonis	-	-	14	-
Delma australis	-	-	-	-
Varanidae				
Varanus gouldii	-	-	G	G
Varanus gilleni	-	-	20	-
Agamidae				
Tympanocryptis lineata	G	-	12	-
Tympanocryptis tetraporophora	7, 10, 11	-	18	7
Tympanocryptis intima	7, 10	-	G	-
Amphibolurus vitticeps	G	G	G	3
Amphibolurus intermis	G 10	G 6	-	12
Amphibolurus pictus	G	-	-	-
Amphibolurus fordi	G 4	G 4	-	1, 2
Moloch horridus	-	-	-	-
Diporiphora winneckei	-	-	-	-
Scincidae				
Tiliqua rugosa	G	-	G	-
Tiliqua occipitalis	5	-	G	-
Lerista labialis	G 1, 4	G 1, 4	4, 12, 14, 16, 17	1, 2, 3
Lerista xanthura	-	-	14	-
Lerista frosti	-	-	18	-
Lerista desertorum	-	-	17	-
Lerista muelleri	-	-	-	-
Lerista bougainvillii	-	-	-	-
Lerista bipes	-	-	-	-
Ctenotus regius	G 1	G 1, 6	14, 17	1
Ctenotus schomburgkii	G 1	G 1, 4	11, 12, 14	1
Ctenotus atlas	G 1	1	14, 18	2
Ctenotus strauchii	G 1	-	13, 18	1
Ctenotus brooksi	-	1	12, 14, 18	1
Ctenotus uber	-	-	18	-
Ctenotus leonhardii	-	-	-	2
Ctenotus sp.	-	-	13	-
Ctenotus leae	-	-	-	-
Menetia greyii	G	G 1	G 11-14, 17, 18	-
Morethia adelaidensis	G	-	13, 14, 18	7
Morethia boulengeri	-	-	-	-
Erimiascincus richardsoni	-	-	12, 14, 19	1, 3
Cryptoblepharus boutonii	-	-	-	-
Typhlopidae				
Typhlina endotera	-	-	G 14, -, 16	1, 3
Typhlina australis	-	-	-	-
Typhlina bituberculata	-	-	-	-
Elapidae				
Pseudonaja nuchalis	-	-	G 14, 19	-
Pseudonaja modesta	-	-	G 12, 16, 18	G
Pseudechis australis	-	-	G	G
Simoselaps bertholdi	-	1, 4	4, 12, 16	2, 5
Simoselaps fasciolatus	-	1	4, 12	3, 5
Suta suta	8	-	-	-
Demansia psammophis	-	-	-	-
Unechis gouldi	-	-	-	-
Unechis monachus	-	-	-	-
Vermicella annulata	-	-	-	-

Table 2.6 Reptiles of Olympic Dam area

Species	Common name	Mining site	Control areas	Not found but may be present
Gekkonidae				
Rhynchoedura ornata	Beaked Gecko	+	+	
Lucasium damaeum	Beaded Gecko	+	+	
Gehyra variegata	Dtella	+	+	
Diplodactylus stenodactylus		+	-	
Diplodactylus conspicillatus	Fat-tailed Gecko	+	+	
Diplodactylus ciliaris	Spiny-tailed Gecko	+	+	
Diplodactylus tessellatus	Tessellated Gecko	+	+	
Diplodactylus vittatus	Stone Gecko	-	-	+
Diplodactylus byrnei		-	-	+
Nephrurus levis	Knob-tailed Gecko	+	+	
Heteronotia binoei	Binoe's Gecko	+	+	
Phyllurus milii	Thick-tailed Gecko	-	+	
Pygopodidae				
Pygopus nigriceps	Black-headed Scalyfoot	+	+	
Pygopus lepidopodus	Common Scalyfoot	-	-	+
Lialis burtonis	Burtons Legless Lizard	-	+	
Delma australis		-	-	+
Varanidae				
Varanus gouldii	Gould's Goanna	+	+	
Varanus gilleni	Gillen's Goanna	-	+	
Agamidae				
Tympanocryptis lineata	Earless Dragon	+	+	
Tympanocryptis tetraporophora	Earless Dragon		+	+
Tympanocryptis intima	Earless Dragon	+	+	
Amphibolurus vitticeps	Bearded Dragon	+	+	
Amphibolurus inermis	Netted Dragon	+	+	
Amphibolurus pictus	Painted Dragon	+	+	
Amphibolurus fordi	Military Dragon	+	+	
Moloch horridus	Thorney Devil	-	-	+
Diporiphora winneckei	Two-lined Dragon	-	-	+
Scincidae				
Tiliqua rugosa	Sleepy Lizard	+	+	
Tiliqua occipitalis	Western Bluetongue	+	+	
Lerista labialis		+	+	
Lerista xanthura		-	+	
Lerista frosti		-	+	
Lerista desertorum		-	+	
Lerista muelleri		-	-	+
Lerista bougainvillii	Bougainvilles Skink	-	-	+
Lerista bipes		-	-	+
Ctenotus regius	Striped Skink	+	+	
Ctenotus schomburgkii	Striped Skink	+	+	
Ctenotus atlas	Striped Skink	+	+	
Ctenotus strauchii	Striped Skink	+	+	
Ctenotus brooksi	Striped Skink	+	+	
Ctenotus uber	Striped Skink	-	+	
Ctenotus leonhardii	Striped Skink	+	-	
Ctenotus sp.	Striped Skink	-	+	
Ctenotus leae	Striped Skink	-	-	+
Menetia greyii	Greys Skink	+	+	
Morethia adelaidensis	Snake-eyed Skink	+	+	
Morethia boulengeri	Snake-eyed Skink	-	-	+
Erimiascincus richardsoni	Desert Banded Skink	+	+	
Cryptoblepharus boutonii		-	-	+
Typhlopidae				
Typhlina endotera	Blind Snake	+	+	
Typhlina australis	Blind Snake	-	-	+
Typhlina bituberculata	Blind Snake	-	-	+
Elapidae				
Pseudonaja nuchalis	Western Brown Snake	+	+	
Pseudonaja modesta	Collared Brown Snake	+	+	
Pseudechis australis	Mulga Snake	+	+	
Simoselaps bertholdi	Desert Banded Snake	+	+	
Simoselaps fasciolatus	Narrow-Banded Snake	+	+	
Suta suta	Curl Snake	-	+	
Demansia psammophis	Yellow-faced Whip Snake	-	-	+
Unechis gouldi	Black-headed Snake	-	-	+
Unechis monachus	Hooded Snake	-	-	+
Vermicella annulata	Bandy-Bandy	-	-	+

In addition to information from the main survey, a sample of five cat stomachs in October, revealed three with Typhlina endotera present, two with apparently several specimens.

2.4.3 Discussion

It is clear from the above that the Olympic Dam area contains a considerable diversity of reptile faunas, but not as diverse as in surrounding control areas.

There is no evidence of species unique to the area. All species present are recorded from other South Australian localities distant from Olympic Dam.

Although not quantifiable, the firm impression is gained that reptile species diversity and abundance is detrimentally affected by alteration of habitat by introduced animals such as rabbits, cattle and sheep.

Most reptiles are largely confined to specific habitats. However, larger species, especially large venomous snakes, and Tiliqua rugosa, T. occipitalis, and Varanus gouldii range over all habitat types, including human habitation areas.

2.5 POPULATION STUDIES

The purpose of these studies was to assess abundance of reptiles in specific habitats. However, time available for this was severely curtailed, and results are, therefore, an indication rather than a direct measure of abundance.

2.5.1 Methods

Three pitfall traplines (as previously discussed) were placed to cover dune and swale habitat in each line. Line locations are on Diagram 1 (localities 1, 2, 3, corresponding to line numbers). Line 1 was the original line laid in April 1981. Lines 2 and 3 were new lines.

Details of each line are shown in Diagrams 2 to 4. Vegetation association analysis is given in Table 2.4.

Line 1 was laid for 7 days and 6 nights, with seventeen pots. Line 2 was laid for 6 days and 5 nights, with fourteen pots. Line 3 was laid for 5 days and 4 nights, with twenty pots.

All lines were checked several times each day, starting just after dawn, and finishing near dusk. All specimens of reptiles in pitfalls were collected, with notation of pit number, and then killed and preserved in 10% formalin solution, for later analysis of stomach contents (Section 2.6).

2.5.2 Results and discussion

All lines collected reptiles successfully, with 149 reptiles caught by the three lines combined. Catches for each species per line, divided into swale and dune segments, is summarized in Table 2.7. Catches per pot for each line are shown in Tables 2.8 to 2.10. Catches for each successive day per line are shown in Tables 2.11 to 2.13.

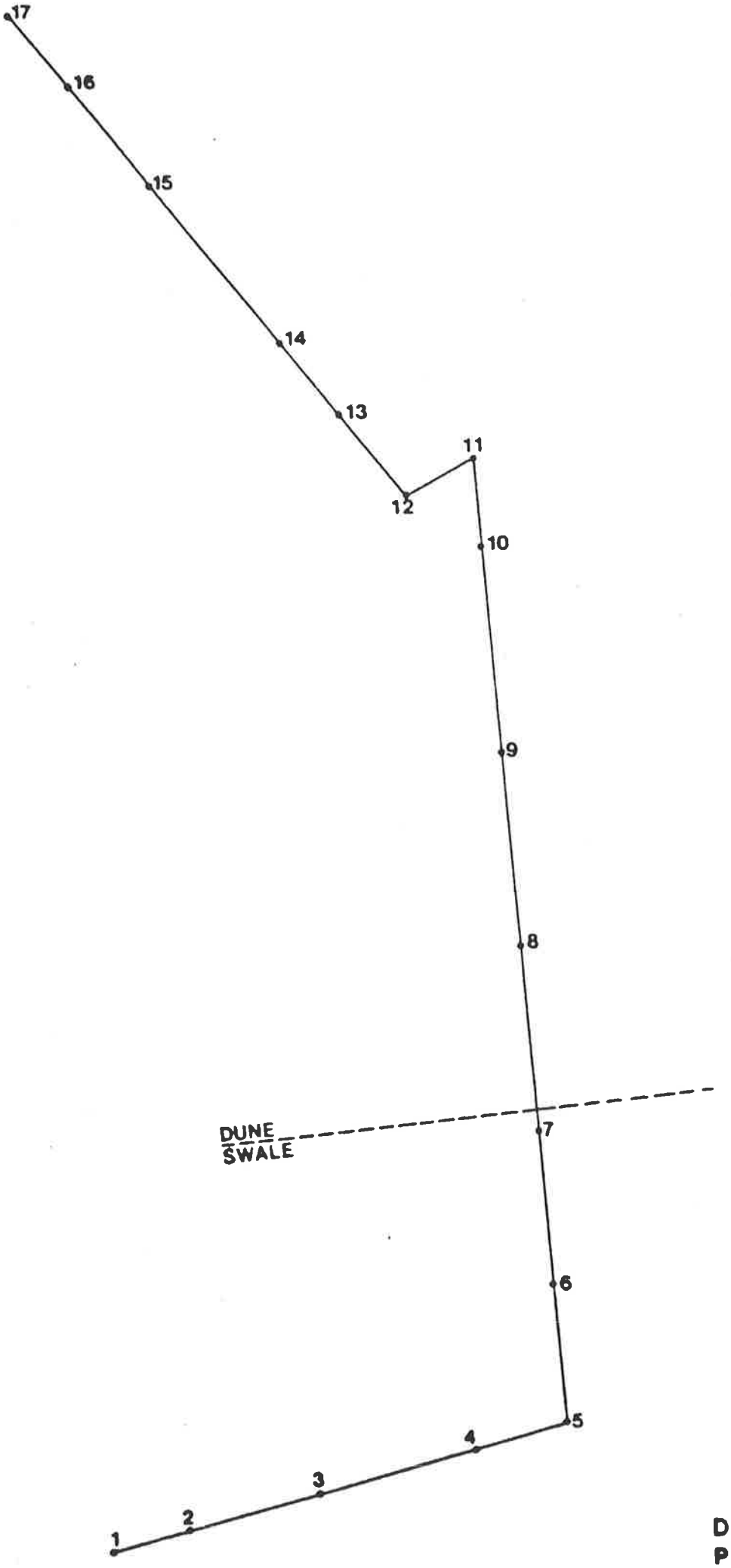

Diagram 2
PITFALL LINE 1

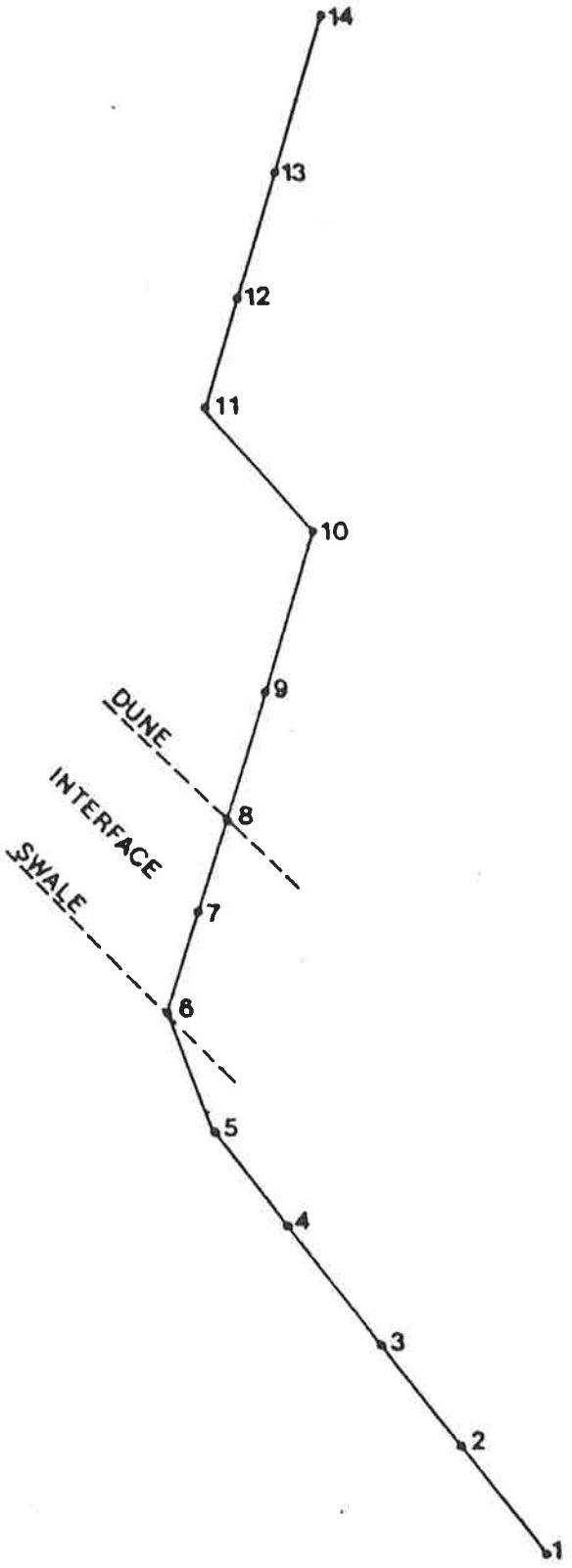

Diagram 3
PITFALL LINE 2

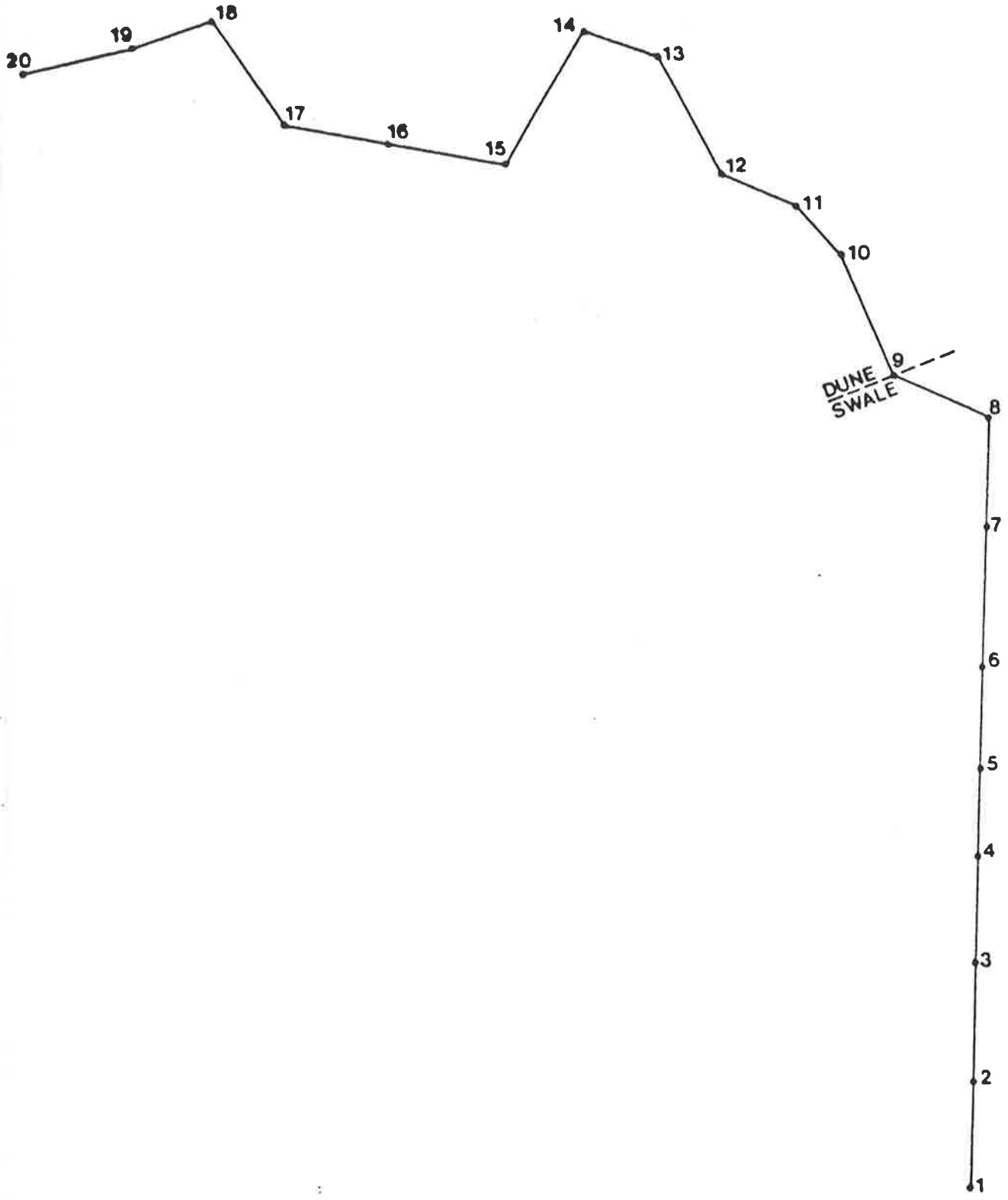

Diagram 4
PITFALL LINE 3

Table 2.7 Summary of trapline data, December 1981

Species	Line 1		Line 2		Line 3		Total
	Swale	Dune	Swale	Dune	Swale	Dune	
Rhynchoedura ornata	9	2	18	-	6	-	35
Lucasium damaeum	-	3	-	-	1	1	5
Diplodactylus stenodactylus	6	1	7	-	3	-	17
Diplodactylus conspicillatus	-	1	7	-	5	-	13
Diplodactylus ciliaris	-	1	-	-	-	-	1
Nephrurus levis	-	1	1	2	-	-	4
Amphibolurus vitticeps (jev)	-	-	-	-	-	1	1
Amphibolurus fordi	-	1	-	-	1	4	6
Ctenotus regius	2	-	6	-	2	-	10
Ctenotus brooksi (+ strauchi)	2	1	1	-	5	1	10
Lerista labialis	3	10	4	6	-	11	34
Erimiascincus richardsoni	-	-	-	1	-	1	2
Typhina endotera	1	1	1	-	-	2	5
Simoselaps betholdi	-	1	-	-	-	-	1
Simoselaps fasciolatus	-	1	-	-	-	-	1
Habitat Totals	23	24	45	9	23	21	
Line Totals	47		54		44		145
Time	7 Days		6 Days		5 Days		

Table 2.8 Trapline data; Collection data per pot (7 days)

		Swale habitat							Dune habitat									
Species	Pot No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
Diplodactylus ciliaris																1		
Simoselaps bertholdi															1			
Simoselaps fasciolatus															1			
Amphibolurus fordi															1			
Lucasuim damaeum												1			2			
Nephrurus levis												1						
Diplodactylus conspicillatus										1								
Lerista labialis							1	2	2	1	1	2		1			2	1
Ctenotus brooksi (+ stauchi)					1		1			1								
Typhlina endotera				1						1								
Ctenotus regius			1			1												
Diplodactylus stenodactylus		2	1	1	2						1							
Rhynchoedura ornata		3		1	3	1	1			1	1							

Table 2.9 Trapline 2; Collection data per pot (6 days)

		Swale habitat									Dune habitat				
Species	Pot No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Nephrurus levis							1						1	1	
Erimiascincus richardsoni															1
Diplodactylus conspicillatus	1	1	1	1	3										
Lerista labialis								1		3	3	1			2
Ctenotus brooksi (+ strauchi)				1											
Typhlina endotera									1						
Ctenotus regius		1	1	2	1				1						
Diplodactylus stenodactylus		2	2	1	2										
Rhynchoedura ornata	3	1	2	2	2	1	2	4	1						

Table 2.10 Trapline 3; Collection data per pot (5 days)

		Swale habitat									Dune habitat									
Species	Pot No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Amphibolurus fordi							1							1		2	1				
Erimiascincus richardsoni													1								
Lucasium damaeum										1										1	
Diplodactylus conspicillatus			1				1	1	1	1											
Lerista labialis											2			1	3	2	1	1	1		
Amphibolurus vitticeps											1										
Ctenotus brooksi (+ strauchi)		1		1	1				1	1			1								
Typhlina endotera															1			1			
Ctenotus regius		1					1														
Diplodactylus stenodactylus		2		1																	
Rhynchoedura ornata			1		1				3	1											

Table 2.11 Trapline 1; Catch rate per day

Species	Day	1	2	3	4	5	6	7
	Date	17/12/81	18/12/81	19/12/81	20/12/81	21/12/81	22/12/81	23/12/81
Diplodactylus ciliaris				0/1*				
Simocelaps bertholdi			0/1					
Simocelaps fasciolatus			0/1					
Amphibolurus fordi		0/1						
Lucasium damaeum		0/1	0/1			0/1		
Nephrurus levis		0/1						
Diplodactylus conspicillatus							0/1	
Lerista labialis			0/2	1/1	1/2	0/3	1/0	0/2
Ctenotus brooksi (+ strauchi)		0/1	1/0	1/0				
Typhlina endotera								1/1
Ctenotus regius		0/2						
Diplodactylus stenodactylus		2/0	2/0	1/0			0/1	1/0
Rhynchoedura ornata		4/0	1/0	1/1	1/0		1/1	1/0

* 0/1 represents number collected swale/dune

Table 2.12 Trapline 2; Catch rate per day

Species	Day Date	1 18/12/81	2 19/12/81	3 20/12/81	4 21/12/81	5 22/12/81	6 23/12/81
Nephrurus levis		0/2*				1/0	
Erimiascincus richardsoni							0/1
Diplodactylus conspicillatus		3/0				2/0	2/0
Lerista labialis		0/1	0/1	1/0	2/0	1/2	0/2
Ctenotus brooksi (+ strauchi)		1/0					
Typhlina endotera							1/0
Ctenotus regius				1/0	3/0	2/0	
Diplodactylus stenodactylus		3/0		1/0	1/0		2/0
Rhynchoedura ornata		8/0	3/0		4/0	2/0	1/0

* 0/2 represents number collected swale/dune

Table 2.13 Trapline 3; Catch rate per day

Species	Day Date	1 19/12/81	2 20/12/81	3 21/12/81	4 22/12/81	5 23/12/81
Amphibolurus fordi			0/1*	0/1	1/2	
Erimiascincus richardsoni					0/1	
Lucasim damaeum		1/1				
Diplodactylus conspicillatus		2/0		2/0	1/0	
Lerista labialis		0/3	0/2	0/1	0/2	0/3
Amphibolurus vitticeps					0/1	
Ctenotus brooksi (+ strauchi)			2/1	1/0	2/0	
Typhlina endotera						0/2
Ctenotus regius			1/0			1/0
Diplodactylus stenodactylus		2/0		1/0		
Rhynchoedura ornata		1/0	1/0		2/0	2/0

* 0/1 represents number collected swale/dune

There was a clear division between swale and dune species as expected. Analysis of results using Morista's Index of Community Similarity (Bower and Zar 1977) was used to quantify this.

$$Im = \frac{2 \sum x_i y_i}{(\lambda_1 + \lambda_2) N_1 N_2}$$

The range of I_m the index of similarity, is from 0 (no similarity) to approximately 1 (identical).

Comparison of the three pitfall lines shows high similarity for each line (Table 2.14), thus confirming the impression of relative homogeneity of the reptile fauna throughout the region, within the dominant habitat.

Table 2.14 Morista's Index of Similarity (I_m) values for line comparison

	Line 1	Line 2	Line 3
Line 1	-	-	-
Line 2	0.943	-	-
Line 3	1.01	0.894	-

Comparison of dune to dune, dune to swale, and swale to swale (Table 2.15), shows the expected similarity of all dune samples, all swale samples, and the difference between dune and swale, this difference becoming less apparent with longer sampling. A high similarity between capture periods is also shown (Table 2.16).

Table 2.15 Morista's Index of Similarity (I_m) values for line dune and swale segment analysis

	Dune 1	Dune 2	Dune 3	Swale 1	Swale 2	Swale 3
Dune 1	-	-	-	-	-	-
Dune 2	1.05	-	-	-	-	-
Dune 3	0.993	1.045	-	-	-	-
Swale 1	0.576	-	-	-	-	-
Swale 2	-	0.203	-	0.961	-	-
Swale 3	-	-	0.0525	0.889	0.985	-

Table 2.16 Morista's Index of Similarity (I_m) values for different trap nights/days

	19/12	20/12	21/12	22/12
19/12	-	1.05	1.049	1.084

Sample numbers proved insufficient for quantitative population analysis. Plots of decrease in capture rate with time did not show a classic regression, but rather a tendency to straight line, possibly as a result of recruitment of sample from adjacent areas to fill the vacant niches. This suggests a locally mobile population.

Data is insufficient to make a quantitative assessment of species abundance or relative abundance of each species compared with the total reptile population. Catch rates confirm the impression that none of the species sampled is rare.

Certain species are clearly abundant, and readily sampled by pit trapping, and so may be useful for monitoring. These species are given in Table 2.17.

Table 2.17 Reptile species abundant on pit trap sampling, suitable for monitoring

Rhynchoedura ornata
Diplodactylus stenodactylus
Diplodactylus conspicillatus
Ctenotus sps.
Lerista labialis
Amphibolurus fordi
Simoselaps bertholdi

2.6 STOMACH CONTENT ANALYSIS

Little information on food preferences of reptiles is available. To provide baseline data on major food items of reptiles at Olympic Dam, a stomach content analysis was most appropriate.

2.6.1 Methods

Full details of methodology and results of the stomach content analysis are given in Appendix B.

Reptiles collected during the December 1981 trip were killed and preserved soon after capture, as discussed earlier. These specimens formed the resource for sampling.

All specimens were sorted into species and registered at the South Australian Museum. Then, working on specimens of each species in turn, stomachs were dissected out and emptied, stomach contents for each specimen being stored in individual containers in alcohol. Once all stomachs were emptied and contents stored, individual stomach contents were selected randomly, the examiner being unaware of the species of reptile involved. Contents were sorted and classified with the assistance of the Entomology Department, South Australian Museum. Once all contents were analysed, data on the reptile corresponding to each stomach was retrieved and collated.

Data was further analysed using the diversity index H' (Goldman 1953).

$$H' = - \quad p_i \log p_i$$

(p_i= frequency of prey of given category for given predator).

The number of reptiles examined totalled 139, covering 21 species of lizards and 5 species of snakes. Of these, 71 specimens had stomach contents. The remaining 68 specimens with empty stomachs were excluded from further analysis.

2.6.2 Results and discussion

Results of analysis are summarized in ~~Table 2.18~~ Appendix Table B.1. Of the 71 specimens' stomach contents, most were recognizable at least to order level. Ten arthropod orders were represented. Termites (Isoptera) and Ants (Hymenoptera) were the most numerous items, being 59% and 33% respectively of total prey items by number (but not necessarily volume).

Table 2.18 Reptile species particularly affected by road kills

Amphibolurus vitticeps
Varanus gouldii
Tiliqua occipitalis
Tiliqua rugosa
Pseudechis australis
Pseudonaja nuchalis

Unfortunately, in only a few species were there sufficient individuals sampled to be worthwhile analysing further. Rhynchoedura ornata had the highest number of sampled specimens (19) with an apparently specialized diet of termites. Diversity index for this species was the lowest (least diversity of diet) at H'=0.048. Two other species, Diplodactylus stenodactylus at H'=0.778, and Ctenotus regius at H'=0.709 had relatively large sample sizes, but showed high prey diversity in their diet.

The diversity of prey of diurnal compared with nocturnal reptiles showed almost twice the diversity of prey items for diurnal reptiles (H'=0.681 to H'=0.381).

The data from this study of food preference cannot be considered comprehensive, and has value mainly as initial baseline data. For a definitive study, much higher numbers of reptiles would need to be collected, and over a range of seasons at at least two different points in the sample habitat.

Nevertheless, this initial study illustrates the wide variety of prey items for small reptiles, and the importance of ants, the most numerous arthropod group in the Olympic Dam area, within that prey spectrum.

3 ENVIRONMENTAL IMPACT ASSESSMENT

3.1 RATIONALE AND LIMITATIONS

As discussed earlier, the reptile fauna is abundant and diverse, being the most populous ground vertebrate group. It is therefore of considerable significance in the ecology of the area. However, reptiles have not been studied as intensively in Australia as the two other major vertebrate groups, birds and mammals.

This imposes limitations on conclusions, as the data base from previous studies is very small. In addition, the time and survey energy input on reptiles at Olympic Dam was not sufficient to inspire absolute confidence in results. Nevertheless, the high number of species recorded compared with expected species which might be present, indicates a very successful survey of reptile species diversity for the area. In addition, the population studies and prey studies give an added data base for environmental impact assessment.

In making such an assessment most decisions must be empirical rather than based on mathematical analysis of data. Thus a considerable latitude of error may exist in any predictions of impact.

3.2 IMPACT OF PRIOR ACTIVITIES OF MAN ON THE OLYMPIC DAM AREA

The Olympic Dam area has been used extensively as pastoral lease in the past. In addition, feral animals exist in the area, particularly cats, rabbits and foxes.

Undoubtedly all of these have considerably modified the environment of the area and will continue to modify it until removed totally.

This modification almost certainly includes the structure of the reptile fauna of the area, to an extent which is not presently assessable. Certain reptile species, such as Moloch horridus would have been adversely affected by reduction in ground cover, and as a result may be extinct in the area now. Nearly all species of small reptiles would have suffered some reduction in total abundance as a result of modification of ground cover. Certain large reptiles such as Varanus gouldii and the dangerous venomous snakes, Pseudechis australis and Pseudonaja nuchalis may have been favoured by the reduction in shelter for prey reptiles, and the increase in mammal prey, particularly the introduced mouse, Mus musculus.

3.3 IMPACT OF EXPLORATION FOR MINERALS

Exploration activities include drilling, roads, clearing, and areas of total clearing such as that for buildings.

Destruction of habitat for reptiles will clearly cause a temporary, localized, major reduction in abundance. Animals on the periphery will then move in to recolonize, and certain species will be favoured. In particular, some small lizards such as Heteronotia binoei thrive around the detritus of man, especially in piles of rubbish and stored equipment. The high turnover of equipment and the policy of burying rubbish at Olympic Dam will have kept such localized population increases to a minimum. The influence of such local increases of small reptile abundance on dangerous venomous snake abundance is not known, but is probably locally signficant, as juveniles of these snakes are provided with increased prey availability and shelter from potential predators such as raptors. The increase in Mus musculus around mining camps would also allow larger population densities of adult dangerous venomous snakes to exist.

Vehicular traffic on roads would provide a further 'predator' for virtually all reptile species. However, most species with small ranges would be generally unaffected. Certain species, particularly those with extensive ranges, would be subject to considerable losses on the roads (Table 2.18). The extent of such deaths and the effects on total population for these species is unknown. However, as these pressures exist at much higher levels elsewhere in Australia, without having had any major documented impact on population, it can be presumed that the influence of road kills at Olympic Dam will not be catastrophic.

3.4 IMPACT OF OPERATIONS

The proposals for mine and associated developments at Olympic Dam will entail destruction of large tracts of habitat, with consequent decimation of reptile fauna. In particular, a large area will be totally destroyed for tailings storage and processing. Further, very large amounts of fill (up to 3.5×10^6 t/a) will need to be quarried, with resultant associated habitat and fauna destruction. In the process, considering the lifetime of the Project to be at least 30 years, an indeterminable number of reptiles will be destroyed. However, this reflects not only the scale of the Project, but the abundance of reptiles in the area, and areas adjacent.

There is no evidence to suggest that such destruction will significantly endanger any species of reptile now found in the area, as all are well represented extensively outside the area. As discussed earlier, certain species will recolonize many areas, but their numbers will be limited by food and shelter availability, and will not attain populations sufficient to constitute a direct interference with the project, with the possible exception of large venomous snakes. There is no data on which to base predictions for these snakes, but it is unlikely they will become any more than a minor nuisance in the context of overall Project functioning.

Ongoing effects of the mine Project will include habitat destruction for quarrying, road kill of larger reptiles (as discussed earlier), and possibly some effects on reptile populations outside the Project Area from particulate and gaseous emissions. The latter should be insignificant in view of the measures proposed to minimize such pollution.

Certain insects may flourish in and around the various dams and water reservoirs. The effect of such a prey increase on local reptile populations is unknown, but is unlikely to be of more than local significance. It may be that birds, including raptors, may be more favoured by such insect increases, and these could have a local deleterious impact on reptile populations. The total impact of quarrying on reptiles will depend on its nature and size.

3.5 IMPACT OF THE TOWN

Town establishment will involve destruction of large areas of habitat with the same effects on reptile population as discussed earlier in relation to the Operations Area. Plans to leave significant areas of dune will allow dune reptiles to survive within the town, providing there is no increased predation from the two major town based reptile predators, namely feral cats and small boys. The former are controllable, and the latter far less controllable, except through behaviour modification by education. In any case, as for the mine site, no reptile species will become extinct or endangered even by total destruction of all its representatives within the town.

Assuming a high standard of development for the town, it is unlikely that conditions will favour venomous snakes, even in remaining dune areas, and a progressive decline in their numbers can be expected, but with continued encroachments from the periphery. With

correct populace education this should not prove a significant problem, although it may be expected that there will be some snakebites. With correct care this should not cause either fatalities (except in rare instances) or major loss of work time.

More important than direct effects within the town site and associated developments, is the potential effect of uncontrolled recreation on adjacent areas. Shooting of reptiles, habitat destruction by vehicles, particularly recreational vehicles, and predation on reptiles for pets could all have significant effects on reptile populations and abundance. Proposals to fence the area, and provide extensive recreational facilities, including an area for off-road vehicles may help to contain this potential impact.

3.6 IMPACT OF INCIDENTALS

In addition to the areas to be modified or destroyed for operations and the town, a number of other incidentals will entail habitat destruction. These are principally the access roads from Woomera, and to the borefields, and associated transmission lines and pipelines, and the borefields themselves.

These latter will entail very localized habitat destruction, probably of limited significance. Since these areas have not been examined for reptiles, prediction is difficult.

Similarly, the roads, pipelines and transmission lines will occasion only local habitat destruction, with variable effects on adjacent habitat dependent on control of the construction and maintenance process. Apart from disruption of territories of large reptiles as discussed earlier, which will not be of major significance, the effect of these incidentals will be of no real significance to the reptile population.

3.7 SUMMARY

Where the proposed development at Olympic Dam will cause the total or partial destruction of habitat, destruction of contained reptiles will result. However, as no reptiles exclusive to the area have been found, this will not have any major implication for the State's reptile fauna.

Many areas of total destruction are such that reptiles will not return. Some areas will retain some of their reptile fauna, such as untouched dunes within the town and Operations Area. The viability of such populations will depend on their size and predator control. Certain artificial areas such as dumps and outdoor equipment storage areas may favour a few species. Venomous snakes may be locally favoured around the mine and plant, but will probably not be favoured near the town. They are unlikely to be more than a minor problem, which will be further minimized by populace education.

Uncontrolled widespread recreational use of areas outside the Project may be cause for concern and should be strictly limited.

The effects of incidentals such as access roads, pipelines, transmission lines will be minimal. The effects of quarrying for fill are unknown and are of concern, as there is the potential for habitat destruction outside the current Project Area which could have adverse effects on the whole area's reptile population.

4 MITIGATION MEASURES

4.1 RATIONALE AND LIMITATIONS

As discussed earlier, severe limitations on data available make impact assessment in precise terms difficult, and consequently, precise mitigation measures are not possible. However, a set of guidelines can be given which covers problems foreseeable on the basis of current data.

The purpose of such mitigation measures is to minimize detrimental effects of the mining process and all its ramifications on the reptile fauna, and minimize the detrimental effects of reptiles on the operations, and in particular, its personnel.

4.2 GENERAL MITIGATION MEASURES

4.2.1 Reduction of undesired reptile abundance decrease

As discussed previously, total habitat destruction such as required for buildings, roads, tips and dumps, and towns, will cause total extermination of contained reptile fauna. However, as this is local only and the fauna is widely represented, such destruction is acceptable.

Nevertheless clearing should be minimized. Tight control of clearing operations must be maintained. All unnecessary clearing must be prevented. In particular, close supervision of earthmoving equipment activities should be ensured, so that unnecessary 'convenience' extensions of cleared areas are minimized.

Vehicular traffic should be confined to defined roads of sufficient width and construction to carry expected traffic, and without unnecessary side clearing. In the planning stages, roads should be kept to a minimum, and wherever possible should not be sited in sensitive areas, such as along the dune/swale interface. High lips or verges to roads both discourage unauthorized user exit from the road, and inhibit small reptile advance onto the road, but do limit movements of large reptiles. Prominent road verges are favoured as home sites for burrowing reptiles such as Amphibolurus inermis (Netted Dragon), and these may suffer significant population loss due to road kill. No data is available to confirm this however.

Fences may interfere with movement of larger reptiles, particularly goannas (Varanus gouldii), large skinks (Tiliqua rugosa, T. occipitalis), and large snakes. Where such fences are to exclude rabbits from an area to be rehabilitated to natural state, interference with reptile movement must be accepted. Clearing along fence lines, especially of low shrubs, should be kept to a minimum.

Where possible, introduced animals should be excluded. Cats in particular, are destructive to small animal populations, including reptiles, and every effort should be made to control the feral cat population. Pet cats in the town are liable to add to the problem, and should be banned. Pet dogs should be subject to stringent controls.

Specially fenced areas, with removal of all introduced animals, including rabbits would be of great interest. However, as rabbits have been a feature of this environment for many years, it is doubtful if any remaining reptile species are significantly endangered by rabbits. Nevertheless, control of rabbit populations should continue, as plague numbers of rabbits, induced by more ready availability of water and food, might endanger some local reptile populations.

Use of insecticides, defoliants, and so on should be very tightly controlled and monitored, as loss of small shrubs and insects would have a severe effect on reptile populations.

4.2.2 Prevention of reptile population explosions

It is unlikely that any reptile species will be so favoured by proposed development that generalized population explosion will occur, although small local population increases may occur in certain species in particular areas.

Rubbish and storage dumps will allow increases in local populations of some small reptiles, especially Heteronotia binoei. These increases are of no consequence in themselves, but may allow increased populations of predators on these lizards, including dangerous venomous snakes. Similarly, significant numbers of mice around buildings and dumps will attract adult venomous snakes. Therefore, dumps and outdoor stores of equipment providing habitat for mice and lizards should be minimized and sited well away from residential areas.

Increased insect populations around dams may similarly favour some reptile species, but the degree is not quantifiable, and is not likely to be of general significance.

4.2.3 Education programmes

A vigorous and ongoi progamme of education of all site personnel from management down through workers, spouses and children should be organized, to ensure a high level of awareness of ti ragility of the environment, and the animals within it. Personnel should be encouraged take an interest in native flora and fauna, and its preservation, and the extent of such preservation should be incorporated as a part of community spirit and pride.

Posters depicting environments, and animal types should be displayed prominently. Schools should incorporate exhibits, both static and dynamic, depicting facets of the local environment. Limited controlled keeping of suitable native fauna, particularly reptiles, should not be discouraged, but wholesale collection of fauna for pets should be discouraged.

A project environmental centre in the town could act as a focus for such education programmes, with displays on both the mining Project and the natural environment. The appointment of a full-time environmental officer with suitable tertiary training would be an essential part of this programme.

4.3 SPECIFIC MITIGATION PROBLEMS

Apart from the general measures outlined, there is one aspect of the reptile fauna which could have a significant effect on the Project; namely the dangerous venomous snakes, principally the Mulga Snake, Pseudechis australis, and the Western Brown Snake, Pseudonaja textilis. Both these species are potentially lethal to man, and both are reasonably common in the area. A snakebite from the Mulga Snake has already been incurred by personnel in the present temporary village for exploration staff. For reasons already discussed, these snakes may find a favourable environment in and around man's dwellings, especially dumps. Measures for reducing this have already been discussed. However, it will be impossible to exclude these snakes from either the Project Area or town site. Therefore, all personnel should be familiar with all the major snake species of the area, and the treatment of snakebite. The mining site and town should have readily available facilities for first-aid treatment of snakebite.

Given a projected town population of 7,500 to 9,000, a well equipped hospital (of perhaps 50 to 80 beds) will be needed for general medical purposes and should support a minimum of three general practitioners, plus visiting specialists.

This hospital should be the area centre for management of cases of snakebite, and not general practitioners' rooms. Adequate intensive monitoring facilities should be available, and a supply of antivenoms (Table 4.1). The hospital medical and nursing staff should be familiar with management of snakebite, and be prepared to institute treatment. All serious cases of snakebite should be managed in Adelaide at the Royal Adelaide Hospital (adults) or Adelaide Children's Hospital (children). This would necessitate transfer by air ambulance to Adelaide, after the immediate situation is stabilized. Further details of this are beyond the scope of this report, but should be discussed with the Consultant on Envenomation, Adelaide Children's Hospital (Dr J. White), when the appropriate stage in planning and developent has been reached.

Table 4.1 Antivenoms needed at hospital

Antivenom	Number of Vials
Black Snake AV	3
Brown snake AV	3
Death adder AV	1
Polyvalent AV	1

4.4 MONITORING

Possible reptile species for monitoring have already been discussed (Table 2.17). Baseline data in this report will assist in providing a data base for monitoring.

It is essential that co-ordinated monitoring be instituted, as present data is insufficient to allow accurate prediction of all effects of the Project on reptiles. Monitoring should provide ongoing data on reptile species abundance (relative and absolute), and diversity, and correlate this with food patterns and availability.

This will require a series of on-site and off-site control sample sites, using permanent pitfall lines, with pots capped between sample periods, and regular sampling encompassing all seasons. Monitoring personnel should develop non-destructive sampling techniques including marking of animals for capture/recapture, and stomach flushing for prey analysis rather than sacrificing animals for stomach analysis. These monitoring programmes should be correlated with monitoring of vegetation changes, and other relevant factors, including land use, and feral animal populations. Regular sampling of feral cat stomachs would provide valuable additional data.

Ongoing monitoring would best be performed by a combination of full time staff (e.g. the project environmental officer) and the consultants used for this assessment.

In addition to monitoring of the status of reptiles, some reptiles could be used for monitoring of pollutant levels. Small numerous lizards (e.g. Diplodlactylus stenodactylus, Ctenotus regius, and so on) and larger lizards (Varanus gouldii), could be sampled regularly to assay changes in levels of critical pollutants in the environment. This might also allow correlation back to population studies. Reptiles are probably the easiest local vertebrate group to use for such pollution monitoring, as they are abundant, readily sampled, and widespread, permitting sampling at different distances from causes of pollution.

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APPENDIX A

List of reptiles from Olympic Dam survey, registered in South Australian Museum, with registration and location data.

SAM Reg. No.	Species	Location	Data Sheet No.
R19830	Simoselaps bertholdi	30°27' x 136°53'	-
R19843	Suta suta	30°27' x 136°53'	-
R19980	Amphibolurus vitticeps	30°27' x 136°53'	02604
R19981	Morethia adelaidensis	30°27' x 136°53'	02869
R19982	Morethia adelaidensis	30°27' x 136°53'	02876
R19983	Morethia adelaidensis	30°27' x 136°53'	02876
R19984	Morethia adelaidensis	30°27' x 136°53'	02876
R19985	Lerista labialis	30°27' x 136°53'	02858
R19986	Lerista labialis	30°27' x 136°53'	02845
R19987	Lerista labialis	30°27' x 136°53'	02877
R19988	Lerista labialis	30°27' x 136°53'	02845
R19989	Menetia greyi	30°27' x 136°53'	02854
R19990	Ctenotus regius	30°27' x 136°53'	02901
R19991	Ctenotus schomburgkii	30°27' x 136°53'	02842
R19992	Ctenotus schomburgkii	30°27' x 136°53'	02861
R19993	Amphibolurus inermis	30°27' x 136°53'	02843
R19994	Amphibolurus inermis	30°27' x 136°53'	02850
R19995	Amphibolurus pictus	30°27' x 136°53'	02602
R19996	Amphibolurus fordi	30°27' x 136°53'	02856
R19997	Amphibolurus inermis	30°27' x 136°53'	02601
R19998	Tympanocryptis intima	30°27' x 136°53'	02862
R19999	Tympanocryptis intima	30°27' x 136°53'	02851
R20000	Tympanocryptis intima	30°27' x 136°53'	02853
R20001	Tympanocryptis tetraporophora	30°27' x 136°53'	02870
R20002	Tympanocryptis tetraporophora	30°27' x 136°53'	02870
R20003	Gehyra variegata	30°27' x 136°53'	02844
R20004	Gehyra variegata	30°27' x 136°53'	02846
R20005	Gehyra variegata	30°27' x 136°53'	02852
R20006	Gehyra variegata	30°27' x 136°53'	02846
R20007	Gehyra variegata	30°27' x 136°53'	02867
R20008	Gehyra variegata	30°27' x 136°53'	02855
R20009	Gehyra variegata	30°27' x 136°53'	02383
R20010	Lucaseum damaeum	30°27' x 136°53'	02606
R20011	Lucaseum damaeum	30°27' x 136°53'	02384
R20012	Lucaseum damaeum	30°27' x 136°53'	02863
R20013	Lucaseum damaeum	30°27' x 136°53'	02605
R20014	Lucaseum damaeum	30°27' x 136°53'	02386
R20015	Lucaseum damaeum	30°27' x 136°53'	02382
R20016	Lucaseum damaeum	30°27' x 136°53'	02386
R20017	Lucaseum damaeum	30°27' x 136°53'	02873
R20018	Rhynchoedura ornata	30°27' x 136°53'	02857
R20019	Rhynchoedura ornata	30°27' x 136°53'	02385
R20020	Nephrurus levis	30°27' x 136°53'	02387
R20021	Nephrurus levis	30°27' x 136°53'	02381
R20022	Heteronotia binoei	30°27' x 136°53'	02874
R20023	Heteronotia binoei	30°27' x 136°53'	02875
R20024	Heteronotia binoei	30°27' x 136°53'	02875
R20025	Ctenotus strauchii	30°27' x 136°53'	02868
R20026	Ctenotus atlas	30°27' x 136°53'	02872
R20027	Ctenotus atlas	30°27' x 136°53'	02866
R20028	Ctenotus regius	30°27' x 136°53'	02871
R20029	Ctenotus regius	30°27' x 136°53'	02841

Miscellaneous

SAM Reg. No.	Species	Location	Data Sheet No.
R19277	Ctenotus strauchii	30°27' x 137°00	Feb. 1981
R19440	Tympanocryptis intima	30°27' x 136°53'	Mar. 1981
R21108	Pseudonaja sp.	30°34' x 136°55'	6/11/81
R21109	Pseudechis australis	30°23' x 136°51'	12/11/81
R21110	Pseudechis australis	30°23' x 136°51'	12/11/81
R21111	Pseudechis australis		Nov. 1981 town site

September, 1981

SAM Reg No	Date	Species	Location	Data Sheet No.
R20696	19	Amphibolurus fordi	30°27' x 136°53'	SW Pitfall
R20697	20	Ctenotus atlas	30°27' x 136°53'	NE Pitfall
R20698	20	Menetia greyii	30°27' x 136°53'	NE Pitfall
R20699	19	Lucasium damaeum	30°27' x 136°53'	NE Pitfall
R20700	19	Lucasium damaeum	30°27' x 136°53'	SW Pitfall
R20701	20	Diplodactylus stenodactylus	30°27' x 136°53'	SW Pitfall
R20702	21	Rhynchoedura ornata	30°27' x 136°53'	NE Pitfall
R20703	20	Ctenotus regius	30°27' x 136°53'	SW Pitfall
R20704	20	Ctenotus regius	30°27' x 136°53'	SW Pitfall
R20705	19	Rhynchoedura ornata	30°27' x 136°53'	NE Pitfall
R20706	19	Rhynchoedura ornata	30°27' x 136°53'	NE Pitfall
R20707	19	Rhynchoedura ornata	30°27' x 136°53'	NE Pitfall
R20708	19	Rhynchoedura ornata	30°27' x 136°53'	NE Pitfall
R20709	19	Rhynchoedura ornata	30°27' x 136°53'	NE Pitfall
R20710	19	Lucasium damaeum	30°27' x 136°53'	NE Pitfall
R20711	21	Pygopus nigriceps	30°27' x 136°53'	NE Pitfall
R20712	21	Diplodactylus conspicillatus	30°27' x 136°53'	NE Pitfall
R20713	19	Ctenotus brooksi	30°27' x 136°53'	SW Pitfall
R20714	21	Lerista labialis	30°27' x 136°53'	NE Pitfall
R20715	21	Lerista labialis	30°27' x 136°53'	NE Pitfall
R20716	18	Ctenotus regius	30°27' x 136°53'	NE Pitfall
R20717	20	Lucasium damaeum	30°27' x 136°53'	NE Pitfall
R20718	20	Diplodactylus stenodactylus	30°27' x 136°53'	NE Pitfall
R20719	20	Lerista labialis	30°27' x 136°53'	NE Pitfall
R20720	21	Diplodactylus stenodactylus	30°27' x 136°53'	SE Pitfall
R20721	21	Heteronotia binoei	30°27' x 136°53'	Salvage Yard
R20722	18	Lerista labialis	30°27' x 136°53'	NE Pitfall
R20723	18	Lerista labialis	30°27' x 136°53'	NE Pitfall
R20724	20	Lerista labialis	30°27' x 136°53'	NE Pitfall
R20725	20	Lerista labialis	30°27' x 136°53'	NE Pitfall
R20726	20	Ctenotus schomburgkii	30°27' x 136°53'	NE Pitfall
R20727	20	Ctenotus schomburgkii	30°27' x 136°53'	NE Pitfall
R20745	19	Amphibolurus vitticeps	30°27' x 136°53'	NE Pitfall
R20826		Amphibolurus inermis	10km from Olympic Dam on road to Andamooka	
R20827		Amphibolurus inermis	30°27' x 136°53'	SE Pitfall
R20828		Amphibolurus inermis	30°27' x 136°53'	Airstrip
R20739		Simoselaps bertholdi	30°27' x 136°53'	
R20741		Simoselaps bertholdi	30°27' x 136°53'	SW Pitfall
R20742		Lerista labialis	30°27' x 136°53'	SW Pitfall
R20743		Amphibolurus vitticeps	30°27' x 136°53'	Salvage Yard

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SAM Reg. No.	Species	Location	Data Sheet No.
R20871	Tympanocryptis tetraporophora	30°48' x 136°52'	02776 VP2
R20872	Tympanocryptis tetraporophora	30°48' x 136°52'	02776 VP2
R20873	Tympanocryptis tetraporophora	30°48' x 136°52'	02776 VP2
R20874	Tympanocryptis tetraporophora	30°48' x 136°52'	02776 VP2
R20875	Tympanocryptis tetraporophora	30°48' x 136°52'	02776 VP2
R20876	Eremioscincus richardsoni	30°23' x 136°51'	02772 12
R20877	Nephrurus laevis	30°22' x 136°56'	02625 14
R20878	Nephrurus laevis	30°22' x 136°56'	02625 14
R20879	Nephrurus laevis	30°22' x 136°56'	02625 14
R20880	Nephrurus laevis	30°32' x 136°48'	02946 W
R20881	Diplodactylus conspicillatus	30°32' x 136°48'	02946
R20882	Diplodactylus conspicillatus	30°23' x 136°53'	02780
R20883	Diplodactylus conspicillatus	30°23' x 136°53'	02780
R20884	Diplodactylus conspicillatus	30°23' x 136°53'	02780
R20885	Diplodactylus conspicillatus	30°23' x 136°53'	02780
R20898	Diplodactylus tessellatus	30°23' x 136°53'	02780
R20899	Diplodactylus stenodactylus	30°23' x 136°53'	02780
R20900	Diplodactylus stenodactylus	30°23' x 136°53'	02780
R20901	Tympanocryptis intima	30°20' x 136°50'	02763
R20902	Heteronotia binoei	30°20' x 136°50'	02763
R20903	Menetia greyii	30°20' x 136°50'	02763
R20904	Lerista frosti	30°55' x 136°53'	02778 VP3
R20905	Tympanocryptis tetraporophora	30°55' x 136°53'	02778 VP3
R20906	Morethia adelaidensis	30°55' x 136°53'	02778 VP3
R20907	Morethia adelaidensis	30°48' x 136°52'	02765 Sth
R20908	Morethia adelaidensis	30°22' x 136°56'	02729 13
R20909	Morethia adelaidensis	30°22' x 136°56'	02729 13
R20910	Morethia adelaidensis	30°22' x 136°56'	02729 13
R20911	Morethia adelaidensis	30°49' x 136°52'	02724 Sth
R20912	Morethia adelaidensis	30°49' x 136°52'	02724 Sth
R20913	Menetia greyii	30°19' x 136°57'	02887 13
R20914	Morethia adelaidensis	30°19' x 136°57'	02887 13
R20915	Morethia adelaidensis	30°19' x 136°57'	02773 13
R20916	Morethia adelaidensis	30°19' x 136°57'	02773 13
R20917	Ctenotus strauchii	30°19' x 136°57'	02773 13
R20918	Menetia greyii	30°19' x 136°57'	02771 12
R20919	Rhynchoedura ornata	30°23' x 136°51'	02771 12
R20920	Ctenotus atlas	30°22' x 136°56'	02761 14
R20921	Ctenotus atlas	30°22' x 136°56'	02761 14
R20922	Ctenotus atlas	30°22' x 136°56'	02761 14
R20923	Ctenotus regius	30°22' x 136°56'	02761 14
R20924	Ctenotus regius	30°22' x 136°56'	02761 14
R20925	Ctenotus regius	30°22' x 136°56'	02886 14
R20926	Ctenotus atlas	30°22' x 136°56'	02886 14
R20927	Lialis burtonis	30°22' x 136°56'	02886 14
R20928	Morethia adelaidensis	30°22' x 136°56'	02886 14
R20929	Ctenotus brooksi	30°22' x 136°56'	02886 14
R20930	Lerista labialis	30°22' x 136°56'	02886 14
R20931	Lerista labialis	30°22' x 136°56'	02886 14
R20932	Lerista labialis	30°22' x 136°56'	02886 14
R20933	Lerista labialis	30°22' x 136°56'	02886 14
R20934	Lerista labialis	30°22' x 136°56'	02886 14
R20935	Lerista labialis	30°22' x 136°56'	02886 14

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SAM Reg. No.	Species	Location	Data Sheet No.
R20936	Ctenotus sp.	30°19' x 136°57'	02890 13
R20937	Lerista labialis	30°27' x 136°53'	02881 4
R20938	Lerista labialis	30°27' x 136°53'	02881 4
R20939	Lerista labialis	30°22' x 136°56'	02627 14
R20940	Lerista labialis	30°22' x 136°56'	02627 14
R20941	Lerista xanthura	30°22' x 136°56'	02627 14
R20942	Morethia adelaidensis	30°55' x 136°53'	02888 Sth
R20943	Ctenotus strauchii	30°55' x 136°53'	02888 Sth
R20944	Ctenotus uber	30°48' x 136°52'	02777 VP2
R20945	Ctenotus uber	30°48' x 136°52'	02777 VP2
R20946	Tympanocryptis tetraporophora	30°48' x 136°52'	02777 VP2
R20947	Morethia adelaidensis	30°48' x 136°52'	02777 VP2
R20948	Eremioscincus richardsoni	30°23' x 136°51'	02762 12
R20949	Ctenotus uber	30°48' x 136°52'	02722 Sth
R20950	Diplodactylus tesselatus	30°27' x 136°51'	02767 11
R20951	Menetia greyii	30°27' x 136°51'	02767 11
R20952	Ctenotus schomburgkii	30°27' x 136°51'	02767 11
R20953	Ctenotus schomburgkii	30°27' x 136°51'	02767 11
R20954	Diplodactylus conspicillatus	30°27' x 136°51'	02767 11
R20955	Ctenotus atlas	30°22' x 136°56'	02626 14
R20956	Ctenotus atlas	30°22' x 136°56'	02626 14
R20957	Ctenotus schomburgkii	30°22' x 136°56'	02626 14
R20958	Diplodactylus conspicillatus	30°27' x 136°51'	02882 11
R20959	Ctenotus schomburgkii	30°27' x 136°51'	02882 11
R20960	Lerista labialis	30°27' x 136°53'	02721 SW
R20961	Lerista labialis	30°27' x 136°53'	02721 SW
R20962	Lerista xanthura	30°22' x 136°56'	02728 14
R20963	Lerista xanthura	30°22' x 136°56'	02728 14
R20964	Lerista labialis	30°22' x 136°56'	02728 14
R20965	Lerista labialis	30°22' x 136°56'	02728 14
R20966	Morethia adelaidensis	30°22' x 136°56'	02728 14
R20967	Morethia adelaidensis	30°22' x 136°56'	02728 14
R20968	Ctenotus schomburgkii	30°27' x 136°51'	02947 11
R20969	Ctenotus schomburgkii	30°27' x 136°51'	02947 11
R20970	Menetia greyii	30°27' x 136°51'	02947 11
R20971	Tympanocryptis lineata	30°23' x 136°51'	02889 12
R20972	Diplodactylus conspicillatus	30°23' x 136°51'	02889 12
R20973	Diplodactylus ciliaris	30°23' x 136°51'	02769 12
R20974	Ctenotus brooksi	30°22' x 136°56'	02775 14
R20975	Menetia greyii	30°22' x 136°56'	02775 14
R20976	Diplodactylus tessellatus	30°22' x 136°56'	02775 14
R20977	Ctenotus atlas	30°22' x 136°56'	02775 14
R20978	Tympanocryptis tetraporophora	30°55' x 136°53'	02726 Sth
R20979	Lerista frosti	30°55' x 136°53'	02945 Sth
R20980	Ctenotus strauchii	30°27' x 136°54'	02628
R20981	Pseudonaja nuchalis	30°22' x 136°56'	02621 14
R20982	Pseudonaja nuchalis	30°22' x 136°56'	02623 14
R20984	Lucasium damaeum	30°23' x 136°51'	02883 12
R20985	Ctenotus schomburgkii	30°23' x 136°51'	02883 12
R20986	Ctenotus brooksi	30°23' x 136°51'	02883 12
R20987	Lucasium damaeum	30°27' x 136°53'	02780
R20989	Lerista frosti	30°57' x 136°53'	P. Bird 5

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SAM Reg. No.	Species	Location	Data Sheet No.
R20990	Lerista frosti	30°57' x 136°53'	P. Bird 5
R20999	Typhlina endotera	30°35' x 136°53'	P. Aitkin 16
R21009	Varanus gouldii flavirufus	30°27' x 136°53'	02779 OD
R21016	Morethia adelaidensis	30°57' x 136°53'	P. Bird
R21018	Lerista labialis	30°20' x 136°43'	P. Bird
R21019	Lerista labialis	30°20' x 136°43'	P. Bird
R21020	Lerista labialis	30°20' x 136°43'	P. Bird
R21025	Pseudonaja nuchalis	30°43' x 136°53'	P. Aitkin 19
R21026	Pseudonaja modesta	30°47' x 136°54'	P. Aitkin 18
R21027	Lialis burtonis	30°20' x 136°55'	P. Aitkin 14
R21028	Pseudonaja modesta	30°33' x 136°53'	P. Aitkin 16
R21029	Pygopus nigriceps	30°47' x 136°54'	P. Aitkin 18
R21030	Typhlina endotera	30°30' x 136°48'	P. Aitkin 15
R21031	Typhlina endotera	30°20' x 136°55'	P. Aitkin 14
R21032	Typhlina endotera	30°33' x 136°53'	P. Aitkin 16
R21033	Pygopus nigriceps	30°27' x 136°51'	P. Aitkin 11
R21034	Phyllurus milii	30°45' x 136°52'	P. Aitkin 20
R21035	Pygopus nigriceps	30°20' x 136°55'	P. Aitkin 14
R21036	Ctenotus regius	30°33' x 136°53'	P. Aitkin 17
R21037	Lerista desertorum	30°33' x 136°53'	P. Aitkin 17
R21038	Lerista labialis	30°33' x 136°53'	P. Aitkin 16
R21039	Lerista labialis	30°33' x 136°53'	P. Aitkin 17
R21040	Menetia greyii	30°33' x 136°53'	P. Aitkin 17
R21041	Ctenotus regius	30°33' x 136°53'	P. Aitkin 17
R21042	Eremioscincus richardsoni	30°43' x 136°53'	P. Aitkin 19
R21043	Typhlina endotera	30°45' x 136°55'	P. Aitkin
R21044	Ctenotus atlas	30°47' x 136°54'	P. Aitkin 18
R21045	Ctenotus brooksi	30°47' x 136°54'	P. Aitkin 18
R21046	Menetia greyii	30°47' x 136°54'	P. Aitkin 18
R21047	Simoselaps bertholdi	30°33' x 136°53'	P. Aitkin 16
R21048	Eremioscincus richardsoni	30°20' x 136°55'	P. Aitkin 14
R21049	Eremioscincus richardsoni	30°43' x 136°53'	P. Aitkin 19
R21080	Lerista labialis	30°23' x 136°51'	02883 12
R21081	Lerista labialis	30°23' x 136°51'	02883 12
R21082	Lerista labialis	30°23' x 136°51'	02883 12
R21083	Lerista labialis	30°23' x 136°51'	02883 12
R21166	Varanus gilleni	30°45' x 136°52'	P. Aitkin 20
R20869	Simoselaps fasciolatus	30°23' x 136°51'	02884 12
R20858	Pseudonaja modesta	30°23' x 136°51'	02770 12
R20859	Pseudonaja modesta	30°27' x 136°53'	02768 OD
R20860	Simoselaps bertholdi	30°23' x 136°51'	02950 12
R20861	Simoselaps fasciolatus	30°23' x 136°51'	02950 12
R20862	Simoselaps bertholdi	30°27' x 136°53'	02885 4
R20863	Simoselaps bertholdi	30°27' x 136°53'	02624 4
R20864	Typhlina endotera	30°27' x 136°53'	02766 OD

APPENDIX B

Report on stomach content analysis, January 1982.

Juliet Davies
Terry Schwanner
Julian White

AN ANALYSIS OF THE STOMACH CONTENTS OF THE REPTILES FROM ROXBY DONWS STATION

Introduction

The Olympic Dam site, centred on 36°26'S x 136°53'E (1:250,000 Andamooka map) on Roxby Downs Station, was determined to contain a number of minerals including tin, copper, gold and uranium. An investigation is being carried out by the Roxby Downs Management Group with view to a major mine on the site, which contains arid vegetation typical of northern South Australia. As a result of these investigations an Environmental Impact Assessment has been prepared, and, as part of that assessment, studies are being undertaken on the reptile fauna in the area. As part of these studies, analysis of stomach contents was made to act as a baseline for future studies on the effect of mining on this environment.

Materials and methods

Specimens were collected by Kingsley Turner and Mark Galliford, using lines of pitfall traps (connected by mesh fencing) and general collecting (on foot, and by vehicle along roads) both day and night. The localities of the traplines are listed below. Collecting was done during one week in December, 1981.

(1) Andamooka (1:250,000)
30°26'S x 136°54'E
Olympic Dam area
(Trapline one)

(2) Andamooka (1:250,000)
30°26'S x 136°53'E
Olympic Dam area
(Trapline two)

(3) Andamooka (1:250,000)
30°26'S x 136°54'E
Olympic Dam area
(Trapline three)

Most specimens were killed on collection to obtain fresh stomach contents, and subsequently preserved in formaldehyde solution. The specimens were then labelled with serial numbers (for the collection of the Herpetology Department of the South Australian Museum). They were then placed in 70% alcohol solution.

The stomachs were exposed by making a lateral incision along the body wall. The stomachs were then prised out with forceps and split with a scalpel. Those stomachs containing material were carefully scraped clean with a spatula and emptied into a small glass bottle along with a label (corresponding to the Museum serial number for the animal) and a 70% alcohol solution. When these glass bottles containing the stomach contents were later analysed the information was written on cards containing the serial number alone to avoid prejudicing any findings by knowing from what species of reptile the stomach contents were taken. After the stomachs were replaced in the specimens, the specimens were placed in the collection of the South Australian Museum.

Stomach contents were analysed under a binocular microscope and information about the classification of insects and crustaceans found in the contents were written on cards. When all stomach contents had been analysed the species names of the animals corresponding to the serial numbers were written on these cards. The information from

these cards was compiled into a table (below). The diversity index (H') is a measure of the equability of diet (Goldman 1953). It is calculated as $H' = \sum p_i \log p_i$, where p_i is the frequency of prey of a given category for a given reptile species. H' values usually range from 0 (low diversity in the diet) to 1 (high diversity).

Of 139 specimens of twenty-six species of reptiles (twenty-one lizards and five snakes) examined, only 71 specimens had contents in their stomachs.

Results

Stomachs of 71 individuals contained food items which represented ten arthropod orders and miscellaneous items of animal and plant origin, some of which were unidentifiable (Table B-1).

Termites and ants formed the greater percentages of the total prey of all reptile species considered collectively (termites: 59%; ants: 33%). When reptiles are grouped into nocturnal and diurnal species, 60% of the prey of diurnal species are ants. Nocturnal species consumed 30% of the total prey as ants and 64% as termites. Grouped as genera, the following total proportions of termites and ants in stomachs were obtained:

Amphibolurus	(79%)
Ctenotus	(52%)
Diplodactylus	(38%)
Lerista	(83%)
Rhynchoedura	(99%)

Reptile species whose stomachs contained relatively large numbers of one or both of these two prey categories (termites and ants) were:

Amphibolurus fordi	(92%)
Amphibolurus nuchalis	(57%)
Ctenotus brooksi	(66%)
Diplodactylus conspicillatus	(60%)
Lerista labialis	(61%)
Rhynchoedura ornata	(99%)

Species for which sufficient numbers of stomachs (five or more) containing food were examined exhibited markedly differing diets. In terms of the H' statistics, the most specialized (least diverse) diet was that of Rhynchoedura ornata (H'= 0.048) which consisted almost entirely of ants. Alternatively, Ctenotus regius (H'= 0.709) and Diplodactylus stenodactylus (H'= 0.778) exhibited broad (diverse) prey preferences. Other species had intermediate H' values.

Interestingly, diurnal species appeared to have almost twice the diversity of diets as nocturnal species (H'= 0.681 and H'= 0.381 respectively). This might be expected if more diurnal thermoactive arthropod prey are active. This assumption was not tested during these studies.

Discussion

The only species for which a large series of specimens was obtained were Diplodactylus stenodactylus (eighteen individuals), Lerista labialis (thirty-two individuals) and Rhynchoedura ornata (thirty-nine individuals). Because of the small numbers of specimens (of which roughly one half had full stomachs) it is difficult, if not impossible, to come to any conclusions about the diets of different species. If such conclusions are to be drawn it

Table B.1 **Prey items in stomach contents of reptiles from Olympic Dam/Roxby Downs**

Prey Categories	Diurnal						Nocturnal										DIURNAL	NOCTURNAL	ALL SPECIES
	Amphibolurus fordi	*Amphibolurus nuchalis*	*Ctenotus atlas*	*Ctenotus brooksi*	*Ctenotus regius*	*Ctenotus strauchii*	*Diplodactylus conspicillatus*	*Diplodactylus intermedius*	*Diplodactylus stenodactylus*	*Erlmiascincus richardsonii*	*Heteronotia binoei*	*Lerista labialis*	*Lucasium dameum*	*Nephrus laevis*	*Rhynchoedura ornata*	*Simoselaps bertholdi*			
Amphipoda				1													1		1
Araneida				1	4				8				1				5	9	14
Coleoptera					1			2		1			2				1	5	6
Hemiptera		2	1		2		1		2								5	3	8
Homoptera				1					1								1	1	2
Hymenoptera	11	4		16	3		1		10			5		188	5		34	209	243
Isoptera				1			3		1			14			424		1	442	443
Odonata				2											1		2	1	3
Orthoptera	1				1				1								2	1	3
Thysanura				2					9								2	9	11
Misc. (animal)						1										1	1	1	2
Misc. (plant)		1			1				1								2	1	3
Misc. (unident.)											1	4	1		2			8	8
Total no. items	12	7	1	24	12	1	5	2	33	1	1	23	4	188	432	1	57	690	747
No. stomachs/ with items	5	4	1	6	4	1	2	1	13	1	1	9	2	1	19	1	21	50	71
Total stomachs	8	4	2	7	6	1	9	1	18	2	1	32	6	1	39	2	28	111	139
H' (individuals)	.121	.415	-	.518	.709	-	.413		.778	-	-	.407	.452	-	.048	-			
H' (genera)	.319	.777	.809	-	-	.407	.452	-	.048	-									
H' (nocturnal vs diurnal)			.681								.393								

will be necessary to collect larger numbers of specimens from the same locality at different times of the year (to account for any seasonal fluctuations in arthropod populations). Thus, any hypotheses stated in the results can only be based on a small sample.

Large numbers of lizards (especially the geckoes) were found with small ants clinging to their bodies and limbs. It is possible that the large number of ants noted in the stomach contents is due to the lizards picking ants off their bodies. It is also possible that the ants congregate in pitfall traps because they are attracted by the lizards, which, once they have fallen into the traps, are unable to climb out again. It would be necessary to collect insects in the area (by netting, trapping in pots of a sticky substance and trapping in pots of water) to determine whether the large numbers of ants found in lizard's stomachs reflect a large ant population in the area, or a fault in the pitfall trapping system when it is used to trap individuals for analysis of stomach contents.