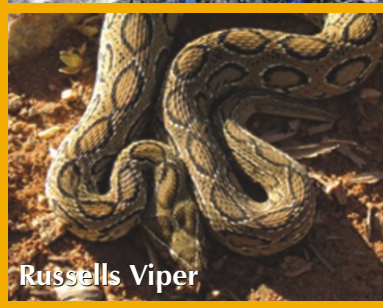
Guidelines

for the Clinical Management of Snake bites in the South-East Asia Region


Saw Scaled Viper


Spectacled Cobra


Common Krait


Russells Viper

World Health Organization
REGIONAL OFFICE FOR South-East Asia
New Delhi

Guidelines

for the Clinical Management of Snake Bite in the South-East Asia Region

Reprint of the 1999 edition written and edited for SEAMEOTROPMED – Regional Centre for Tropical Medicine, Faculty of Tropical Medicine, Mahidol University, Thailand.

Written and edited for SEAMEOTROPMED by David A Warrell with contributions by an international panel of experts, first published as a Supplement to the Southeast Asian Journal of Tropical Medicine & Public Health, Vol 30, Supplement 1, 1999

World Health Organization
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Preface

The geographical area specifically covered by this publication extends from Pakistan and the rest of the Indian subcontinent in the west through to the Philippines and Indonesia in the east, excluding Tibet, China, Taiwan, Korea, Japan, the eastern islands of Indonesia and New Guinea and Australia (Figure 1, inside of front cover).

Figure 1: Map of Asia showing the area specifically covered by the guidelines

In many parts of this region, snake bite is a familiar occupational hazard of farmers, plantation workers and others, resulting in tens of thousands of deaths each year and innumerable cases of chronic physical handicap. Much is now known about

the species of venomous snakes responsible for these bites, the nature of their venoms and the clinical effects of envenoming in human patients. This publication aims to pass on a digest of this knowledge to medical doctors, nurses, dispensers and community health workers who have the responsibility of treating victims of snake bite.

Any recommendations must be continually reconsidered in the light of new evidence and experience. Comments from readers are welcomed so that future editions can be updated and improved.

The guidelines are intended to provide enough practical information to allow any medicallytrained person to assess and treat a patient with snake bite at different levels of the health service. Recommendations are based on clinical experience and, where possible, on the results of clinical trials. The restrictions on the size of this document prevented the inclusion of detailed references to the original publications on which these recommendations were based. These can be found in the papers and reviews listed in "Further Reading".

I am grateful to the panel of experts who contributed to these Guidelines but I must take responsibility for the writing and editing of the document.

I acknowledge the exellent help provided by Miss Eunice Berry (Centre for Tropical Medicine, University of Oxford), who typed the several drafts of the manuscript, and by Ms Vimolsri Panichyanon (Assistant Programme Coordinator, SEAMEOTROPMED Network) and Drs Suvanee Supavej and Parnpen Viriyavejakul (Deputy Assistant Deans for International Relations, Faculty of Tropical Medicine, Mahidol University) who, under the overall direction of Professor Sornchai Looareesuwan, were responsible for organising the meeting of the international panel of experts in Bangkok on 29/30 November 1998.

David A Warrell
Oxford, December 1998

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*indicates countries of clinical experience with snake bite patients in the South-East Asian region.

1

Introduction

1.1 Venomous snakes of South-East Asia

The venom apparatus (Fig 2)

Venomous snakes of medical importance have a pair of enlarged teeth, the fangs, at the front of their upper jaw. These fangs contain a venom channel (like a hypodermic needle) or groove, along which venom can be introduced deep into the tissues of their natural prey. If a human is bitten, venom is usually injected subcutaneously or intramuscularly. Spitting cobras can squeeze the venom out of the tips of their fangs producing a fine spray directed towards the eyes of an aggressor.

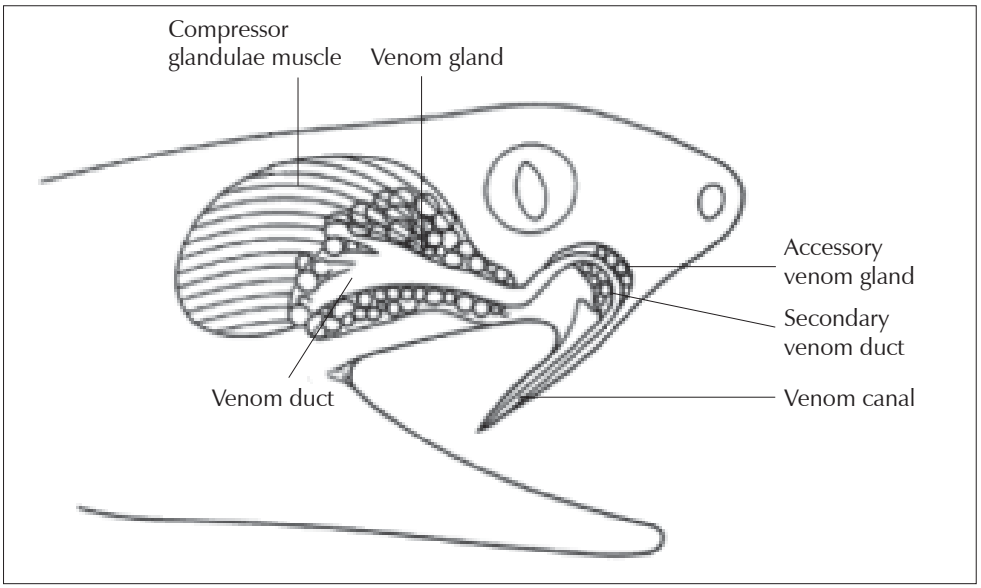

Figure 2: Venom apparatus of a saw-scaled viper (Copyright DA Warrell)

Classification

There are two important groups (families) of venomous snakes in South-East Asia – **Elapidae** have short permanently erect fangs (Fig 3). This family includes the cobras, king cobra, kraits, coral snakes and the sea snakes. The most important species, from a medical point of view, include the following:

cobras:	*N naja*	common spectacled Indian cobra (Fig 4)
(genus Naja)	*N oxiana*	North Indian or Oxus cobra (Fig 5)
	N kaouthia	monocellate cobra (Fig 6)
	N philippinensis	Philippine cobra
	N atra	Chinese cobra (Fig 7)
spitting cobras:	*N siamensis* (Fig 8)	
	N sumatrana (Fig 9)	
	N sputatrix etc	
king cobra:	*Ophiophagus hannah* (Fig 10)	
kraits:	B caeruleus	common krait (Fig 11)
(genus Bungarus)	B candidus	Malayan krait (Fig 12)
	B multicinctus	Chinese krait (Fig 13)
	B fasciatus	banded krait (Fig 14)

Sea snakes (important genera include Enhydrina, Lapemis and Hydrophis) (Fig 15)

Figure 3: **Short, permanently erect, fangs of a typical elapid (Thai monocellate cobra – *Naja kaouthia*) (Copyright DA Warrell)**

Figure 4: **Short, permanently erect, fangs of a typical elapid (Thai monocellate cobra – *Naja kaouthia* (Copyright DA Warrell)**

Figure 5: **North Indian or Oxus cobra (*Naja oxiana*) (Copyright DA Warrell) Viperdae have long fangs**

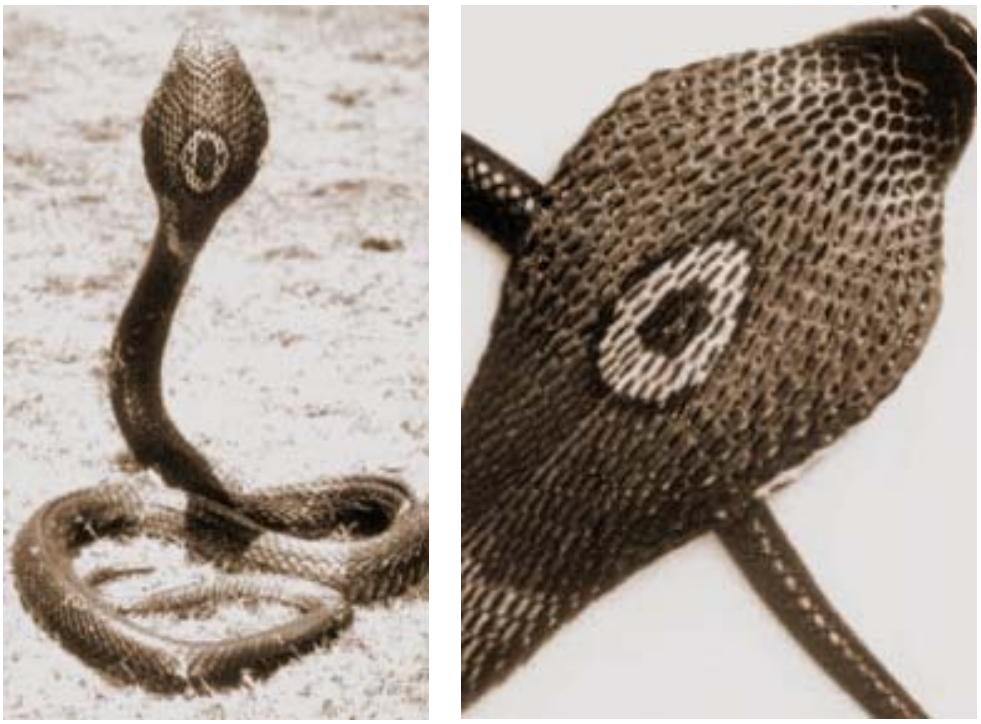

Figure 6: (Left) **Monocellate cobra** ***(Naja kaouthia), (Right)*** **Detail of hood (Copyright DA Warrell)**

Figure 7: **Chinese cobra** ***(Naja atra)*** **(Copyright DA Warrell)**

Figure 8(a): Indo-Chinese spitting cobra ***(Naja Saimensis)*** **(Copyright DA Warrell)**

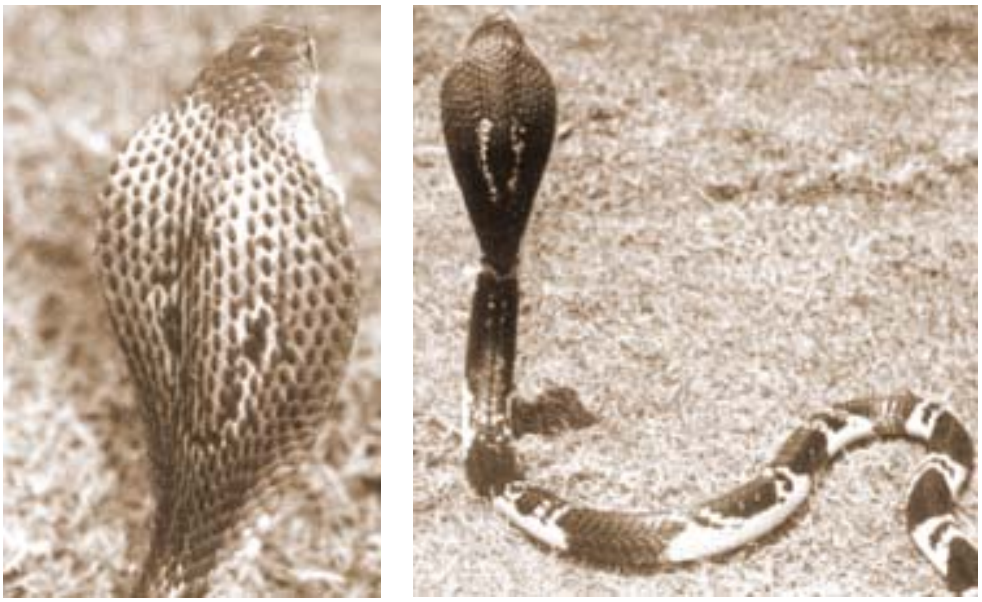

Figure 8(b): (Left) Brown coloured specimen with spectacle marking on hood. (Right) Black and white specimen with ill-defined spectacle marking on the hood. (Copyright DA Warrell)

Figure 9: Sumatran spitting cobra *(Naja sumatrana)* (a) black phase (b) golden phase (Copyright DA Warrell)

Figure 10: King cobra or hamadryad (Ophiophagus hannah). The famous king cobra dance in Yangon, Myanmar (Copyright DA Warrell)

Figure 11: Common krait *(Bungarus caeruleus)* (Copyright DA Warrell)

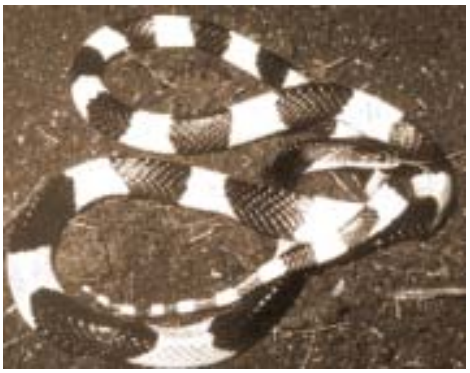

Figure 12: Malayan krait *(Bungarus candidus)* (Copyright DA Warrell)

Figure 13: Chinese krait *(Bungarus multicinctus)* (Copyright DA Warrell)

Figure 14: Banded krait *(Bungarus fasciatus)* (Copyright DA Warrell)

Figure 15: Blue spotted sea snake *(Hydrophis cyanocinctus)* (Copyright DA Warrell)

Viperidae have long fangs which are normally folded up against the upper jaw but, when the snake strikes, are erected (Fig 2). There are two subgroups, the typical vipers (Viperinae) and the pit vipers (Crotalinae). The Crotalinae have a special sense organ, the pit organ, to detect their warm-blooded prey. This is situated between the nostril and the eye (Fig 16).

Medically important species in South-East Asia are:

typical vipers	*Daboia russelii*	Russell's vipers (Fig 17)
	Echis carinatus and *E sochureki*	saw-scaled or carpet vipers (Figs 18, 19)
pit vipers	*Calloselasma rhodostoma*	Malayan pit viper (Fig 20)
	Hypnale hypnale	hump-nosed viper (Fig 21)

green pit vipers or bamboo vipers (genus Trimeresurus)

	T albolabris	white-lipped green pit viper (Fig 22)

T gramineus	Indian bamboo viper
T mucrosquamatus	Chinese habu (Fig 23)
T purpureomaculatus	mangrove pit viper (Fig 24)
T stejnegeri	Chinese bamboo viper

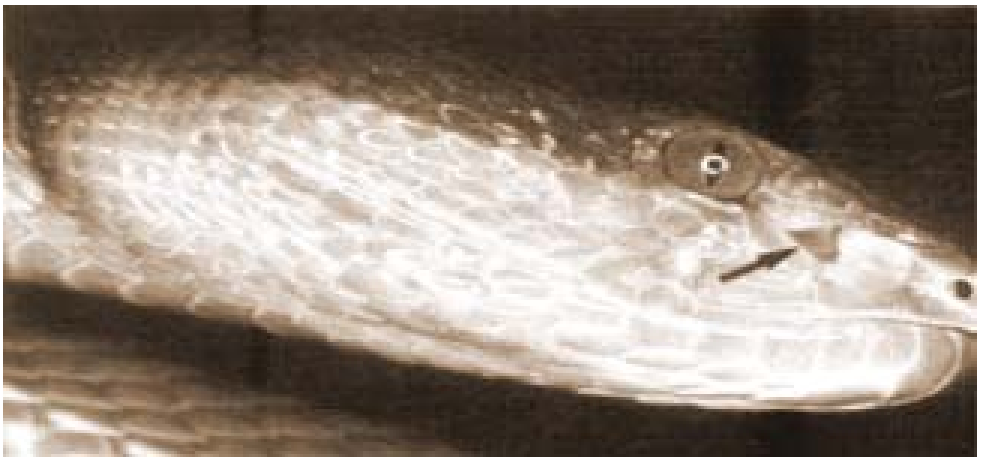

Figure 16: Head of a typical pit viper – white-lipped green pit viper *(Trimeresurus albolabris)* showing the pit organ situated between the nostril and the eye (arrow head) (Copyright DA Warrell)

Figure 17(a): Russell's vipers (Copyright DA Warrell) Eastern subspecies (Daboia russelii siamensis); specimen from Myanmar

Figure 17(b): Russell's vipers (Copyright DA Warrell) specimen from India

Figure 17(c): Russell's vipers (Copyright DA Warrell) Eastern subspecies (Daboia russelii siamensis); specimen from Thailand

Figure 17(d): Russell's vipers (Copyright DA Warrell) specimen from Burma

Figure 17(e): Russell's vipers (Copyright DA Warrell) details of fangs

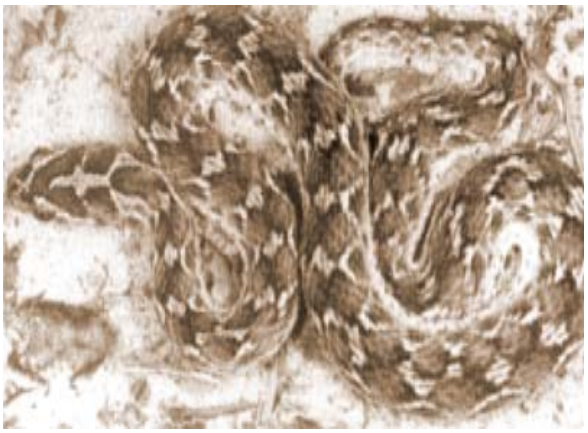

Figure 18: Saw-scaled viper *(Echis carinatus)* specimen from Sri Lanka (Copyright DA Warrell)

Figure 19: Northern saw-scaled viper *(Echis sochureki)* (Copyright DA Warrell)

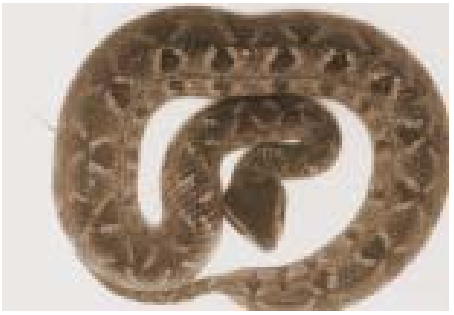

Figure 20: Northern saw-scaled viper *(Echis sochureki)* (Copyright DA Warrell)

Figure 21: Hump-nosed viper *(Hynpale hypnale)*. Specimen from Sri Lanka (Copyright DA Warrell)

Figure 22: White-lipped green pit viper *(Trimeresurus albolabris)* (Copyright DA Warrell)

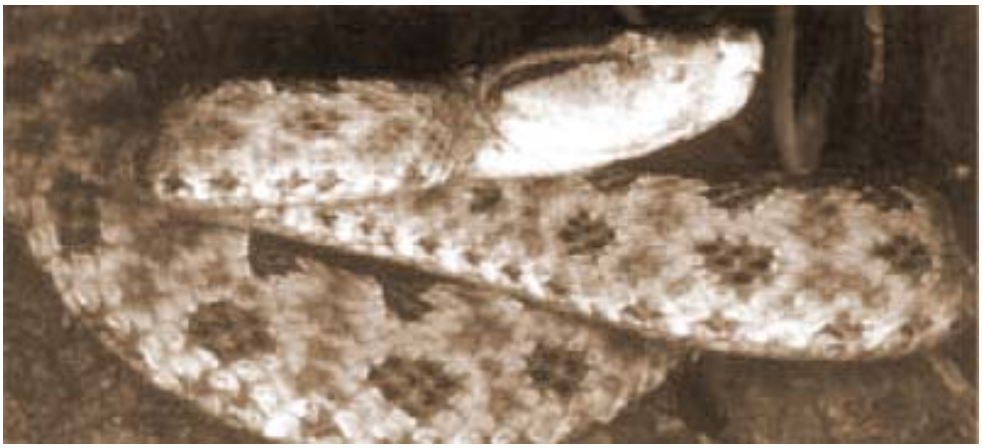

Figure 23: Chinese habu *(Trimeresurus microsquamatus)* (Copyright DA Warrell)

Figure 24: Mangrove pit viper *(Trimeresurus purpureomaculatus)* (Copyright DA Warrell)

How to identify venomous snakes

There is no simple rule for identifying a dangerous venomous snake. Some harmless snakes have evolved to look almost identical to venomous ones. However, some of the most notorious venomous snakes can be recognised by their size, shape, colour, pattern of markings, their behaviour and the sound they make when they feel threatened. For example, the defensive behaviour of the cobras is well known (Fig 8): they rear up, spread a hood, hiss and make repeated strikes towards the aggressor. Colouring can vary a lot. However, some patterns, like the large white, dark rimmed spots of the Russell's viper (Fig 17), or the alternating black and yellow bands of the banded krait (Fig 14), are distinctive. The blowing hiss of the Russell's viper and the grating rasp of the saw-scaled viper are warning and identifying sounds.

1.2 Snake venoms

Composition of venom

Snake venoms contain more than 20 different constituents, mainly proteins, including enzymes and polypeptide toxins. The following venom constituents cause important clinical effects:

Procoagulant enzymes (Viperidae) that stimulate blood clotting but result in incoagulable blood. Venoms such as Russell's viper venom contain several different procoagulants which activate different steps of the clotting cascade. The result is formation of fibrin in the blood stream. Most of this is immediately broken down by

the body's own fibrinolytic system. Eventually, and sometimes within 30 minutes of the bite, the levels of clotting factors have been so depleted ("consumption coagulopathy") that the blood will not clot.

Haemorrhagins (zinc metalloproteinases) that damage the endothelial lining of blood vessel walls causing spontaneous systemic haemorrhage.

Cytolytic or necrotic toxins - these digestive hydrolases (proteolytic enzymes and phospholipases A) polypeptide toxins and other factors increase permeability resulting in local swelling. They may also destroy cell membranes and tissues.

Haemolytic and myolytic phospholipases A_2 - these enzymes damage cell membranes, endothelium, skeletal muscle, nerve and red blood cells.

Pre-synaptic neurotoxins (Elapidae and some Viperidae) - these are phospholipases A_2 that damage nerve endings, initially releasing acetylcholine transmitter, then interfering with release.

Post-synaptic neurotoxins (Elapidae) - these polypeptides compete with acetylcholine for receptors in the neuromuscular junction and lead to curare-like paralysis.

Quantity of venom injected at a bite

This is very variable, depending on the species and size of the snake, the mechanical efficiency of the bite, whether one or two fangs penetrated the skin and whether there were repeated strikes. The snake may be able to control whether or not venom is injected. For whatever reason, a proportion of bites by venomous snakes do not result in the injection of sufficient venom to cause clinical effects. About 50% of bites by Malayan pit vipers and Russell's vipers, 30% of bites by cobras and 5-10% of bites by saw-scaled vipers do not result in any symptoms or signs of envenoming. Snakes do not exhaust their store of venom, even after several strikes, and they are no less venomous after eating their prey.

Although large snakes tend to inject more venom than smaller specimens of the same species, the venom of smaller, younger vipers may be richer in some dangerous components, such as those affecting haemostasis.

Bites by small snakes should not be ignored or dismissed. They should be taken just as seriously as bites by large snakes of the same species.

1.3 How common are snake bites?

It is difficult to answer this question because many snake bites and even deaths from snake bite are not recorded. One reason is that many snake bite victims are treated not in hospitals but by traditional healers.

To remedy the deficiency in reliable snake bite data, it is strongly recommended that snake bites should be made a specific notifiable disease in all countries in the South East Asian region.

Bangladesh – a survey of 10% of the country in 1988-9 revealed 764 bites with 168 deaths in one year. Cobra bites (34% of all bites) caused a case fatality of 40%.

Bhutan – (no data available)

Cambodia – (no data available)

India – estimates in the region of 200,000 bites and 15-20,000 snake bite deaths per year, originally made in the last century, are still quoted. No reliable national statistics are available. In 1981, a thousand deaths were reported in Maharashtra State. In the Burdwan district of West Bengal 29,489 people were bitten in one year with 1,301 deaths. It is estimated that between 35,000 and 50,000 people die of snake bite each year among India's population of 980 million.

Indonesia – no reliable data are available from this vast archipelago. Snake bites and deaths are reported from some islands, eg Komodo, but fewer than 20 deaths are registered each year.

Lao DPR – (no data available)

Malaysia – bites are common, especially in northwest peninsular Malaysia, but there are few deaths.

Myanmar (Burma) – snake bites and snake bite deaths have been reliably reported from colonial times. Russell's vipers are responsible for 90% of cases. In 1991, there were 14,000 bites with 1,000 deaths and in 1997, 8,000 bites with 500 deaths. Under-reporting is estimated at 12%. There are peaks of incidence in May and June in urban areas and during the rice harvest in October to December in rural areas.

Nepal – there are estimated to be at least 20,000 snake bites with about 200 deaths in hospitals each year, mainly in the Terai region. One survey suggested as many as 1,000 deaths per year. Among 16 fatalities recorded at one rural clinic during a monsoon season, 15 had died on their way to seek medical care.

Pakistan – there are an estimated 20,000 snake bite deaths each year

Philippines – there are no reliable estimates of mortality among the many islands of thearchipelago. Figures of 200-300 deaths each year have been suggested. Only cobras cause fatal envenoming, their usual victims being rice farmers.

Sri Lanka – epidemiological studies in Anuradhapura showed that only two-thirds of cases of fatal snake bite were being reported to the Government Agent

Statistical Branch. However, the Registrar General received reports of more than 800 deaths from bites and stings by venomous animals and insects in the late 1970s and the true annual incidence of snake bite fatalities may exceed 1,000.

Thailand – between 1985 and 1989, the number of reported snake bite cases increased from 3,377 to 6,038 per year, reflecting increased diligence in reporting rather than a true increase in snake bites; the number of deaths ranged from 81 to 183 (average 141) per year. In 1991 there were 1,469 reported bites with five deaths, in 1992, 6,733 bites with 19 deaths and, in 1994, 8,486 bites with eight deaths. Deaths reported in hospital returns were only 11% of the number recorded by the Public Health Authorities. In a national survey of dead snakes brought to hospital by the people they had bitten, 70% of the snakes were venomous species, the most commonly brought species being Malayan pit viper (*Calloselasma rhodostoma*) 38%, white-lipped green pit viper (*Trimeresurus albolabris*) 27%, Russell's viper (*Daboia russelii siamensis*) 14%, Indo-Chinese spitting cobra (*Naja siamensis*) 10% and monocellate cobra (*N kaouthia*) 7%. In an analysis of 46 fatal cases in which the snake had been reliably identified, Malayan kraits (*Bungarus candidus*) and Malayan pit vipers were each responsible for 13 cases, monocellate cobras for 12 and Russell's vipers for seven deaths.

Viet Nam – there are an estimated 30,000 bites per year. Among 430 rubber plantation workers bitten by Malayan pit vipers between 1993 and 1998, the case fatality was 22%, but only a minority had received antivenom treatment. Fishermen are still occasionally killed by sea snakes but rarely reach hospitals.

1.4 How do snake bites happen?

In South-East Asia, snake bite is an occupational hazard of rice farmers; rubber, coffee and other plantation workers; fishermen and those who handle snakes. Most snake bites happen when the snake is trodden on, either in the dark or in undergrowth, by someone who is bare-footed or wearing only sandals. The snake may be picked up, unintentionally in a handful of foliage or intentionally by someone who is trying to show off. Some bites occur when the snake (usually a krait) comes in to the home at night in search of its prey (other snakes, lizards, frogs, mice) and someone sleeping on the floor rolls over onto the snake in their sleep. Not all snake bites happen in rural areas. For example, in some large cities, such as Jammu in India, people who sleep in small huts (jhuggies) are frequently bitten by kraits.

1.5 How can snake bites be avoided?

Snake bite is an occupational hazard that is very difficult to avoid completely. However, attention to the following recommendations might reduce the number of accidents.

- Education! Know your local snakes, know the sort of places where they like to live and hide, know at what times of year, at what times of day/night or in what kinds of weather they are most likely to be active.

Snake bite: an occupational disease in South East Asia

Farmers (rice)
Plantation workers (rubber, coffee)
Herdsmen
Hunters
Snake handlers (snake charmers and in snake restaurants and traditional Chinese
pharmacies)
Fishermen and fish farmers
Sea snake catchers (for sea snake skins, leather)

- Be specially vigilant about snake bites after rains, during flooding, at harvest time and at night.
- Try to wear proper shoes or boots and long trousers, especially when walking in the dark or in undergrowth.
- Use a light (torch, flashlight or lamp) when walking at night.
- Avoid snakes as far as possible, including snakes performing for snake charmers. Never handle, threaten or attack a snake and never intentionally trap or corner a snake in an enclosed space.
- If at all possible, try to avoid sleeping on the ground.
- Keep young children away from areas known to be snake-infested.
- Avoid or take great care handling dead snakes, or snakes that appear to be dead.
- Avoid having rubble, rubbish, termite mounds or domestic animals close to human dwellings, as all of these attract snakes.
- Frequently check houses for snakes and, if possible, avoid types of house construction that will provide snakes with hiding places (e.g. thatched rooves with open eaves, mud and straw walls with large cracks and cavities, large unsealed spaces beneath floorboards).
- To prevent sea snake bites, fishermen should avoid touching sea snakes caught in nets and on lines. The head and tail are not easily distinguishable. There is a risk of bites to bathers and those washing clothes in muddy water of estuaries, river mouths and some coastlines.

2

Symptoms and Signs of Snake Bite

2.1 When venom has not been injected

Some people who are bitten by snakes or suspect or imagine that they have been bitten, may develop quite striking symptoms and signs, even when no venom has been injected. This results from an understandable fear of the consequences of a real venomous bite. Anxious people may overbreathe so that they develop pins and needles of the extremities, stiffness or tetany of their hands and feet and dizziness. Others may develop vasovagal shock after the bite or suspected bite – faintness and collapse with profound slowing of the heart. Others may become highly agitated and irrational and may develop a wide range of misleading symptoms. Another source of symptoms and signs not caused by snake venom is first aid and traditional treatments. Constricting bands or tourniquets may cause pain, swelling and congestion. Ingested herbal remedies may cause vomiting. Instillation of irritant plant juices into the eyes may cause conjunctivitis. Forcible insufflation of oils into the respiratory tract may lead to aspiration pneumonia, bronchospasm, ruptured ear drums and pneumothorax. Incisions, cauterisation, immersion in scalding liquid and heating over a fire can result in devastating injuries.

2.2 When venom has been injected

Early symptoms and signs

Following the immediate pain of mechanical penetration of the skin by the snake's fangs, there may be increasing local pain (burning, bursting, throbbing) at the site of the bite, local swelling that gradually extends proximally up the bitten limb and tender, painful enlargement of the regional lymph nodes draining the site of the bite (in the groin – femoral or inguinal, following bites in the lower limb; at the elbow (epitrochlear) or in the axilla following bites in the upper limb). However, bites by kraits, sea snakes and Philippine cobras may be virtually painless and may cause

negligible local swelling. Someone who is sleeping may not even wake up when bitten by a krait and there may be no detectable fang marks or signs of local envenoming.

2.3 Clinical pattern of envenoming by snakes in South-East Asia

Symptoms and signs vary according to the species of snake responsible for the bite and the amount of venom injected. Sometimes the identity of the biting snake can be confirmed by examining the dead snake; it may be strongly suspected from the patient's description or the circumstances of the bite or from knowledge of the clinical effects of the venom of that species. This information will enable the doctor to choose an appropriate antivenom, anticipate the likely complications and therefore take appropriate action. If the biting species is unknown, recognition of the emerging pattern of symptoms, signs and results of laboratory tests ("the clinical syndrome"), may suggest which species was responsible.

Local symptoms and signs in the bitten part

- fang marks (Fig 25)
- local pain
- local bleeding (Fig 26)
- bruising
- lymphangitis
- lymph node enlargement
- nflammation (swelling, redness, heat)
- blistering (Fig 27)
- local infection, abscess formation
- necrosis (Fig 28)

Figure 25: Fang marks made by Russell's viper (Copyright DA Warrell)

Figure 26: Local bleeding from fang marks made by Malayan pit viper (Copyright DA Warrell)

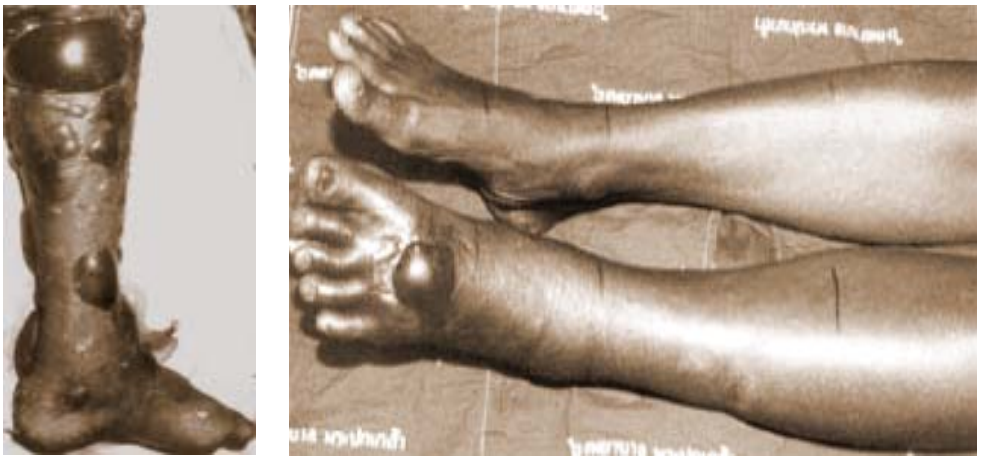

Figure 27 Local swelling and blistering (a) with bruising, following a bite by a Malayan pit viper (Copyright DA Warrell), (Bottom) Local swelling and blistering (b) with early necrosis following a bite by a monocellate cobra (Naja kaouthia) (Copyright DA Warrell)

Figure 28: Tissue necrosis following a bite by a Malayan pit viper (Copyright DA Warrell)

Figure 28(a): Tissue necrosis following a bite by an Indochinese spitting cobra (Naja siamensis) (Copyright *Sornchai Looareesuwan)*

Generalised (systemic) symptoms and signs

General

Nausea, vomiting, malaise, abdominal pain, weakness, drowsiness, prostration

Cardiovascular (Viperidae)

Visual disturbances, dizziness, faintness, collapse, shock, hypotension, cardiac arrhythmias, pulmonary oedema, conjunctival oedema (Fig 29)

Bleeding and clotting disorders (Viperidae)

- bleeding from recent wounds (including fang marks (Fig 26), venepunctures etc) and from old partly-healed wounds
- spontaneous systemic bleeding – from gums (Fig 30), epistaxis, bleeding into the tears, haemoptysis, haematemesis, rectal bleeding or melaena, haematuria, vaginal bleeding, bleeding into the skin (petechiae, purpura, ecchymoses) and mucosae (eg conjunctivae [Fig 31]), intracranial haemorrhage (meningism from subarachnoid haemorrhage, lateralising signs and/or coma from cerebral haemorrhage)

Neurological (Elapidae, Russell's viper)

Drowsiness, paraesthesiae, abnormalities of taste and smell, "heavy" eyelids, ptosis (Fig 32), external ophthalmoplegia (Fig 33), paralysis of facial muscles and other muscles innervated by the cranial nerves, aphonia, difficulty in swallowing secretions, respiratory and generalised flaccid paralysis

Skeletal muscle breakdown (sea snakes, Russell's viper)

Generalised pain, stiffness and tenderness of muscles, trismus, myoglobinuria (Fig 34), hyperkalaemia, cardiac arrest, acute renal failure

Renal (Viperidae, sea snakes)

Loin (lower back) pain, haematuria, haemoglobinuria, myoglobinuria, oliguria/anuria, symptoms and signs of uraemia (acidotic breathing, hiccups, nausea, pleuritic chest pain....)

Endocrine (acute pituitary/adrenal insufficiency) (Russell's viper)

Acute phase: shock, hypoglycaemia

Chronic phase (months to years after the bite): weakness, loss of secondary sexual hair, amenorrhoea, testicular atrophy, hypothyroidism etc (Fig 35)

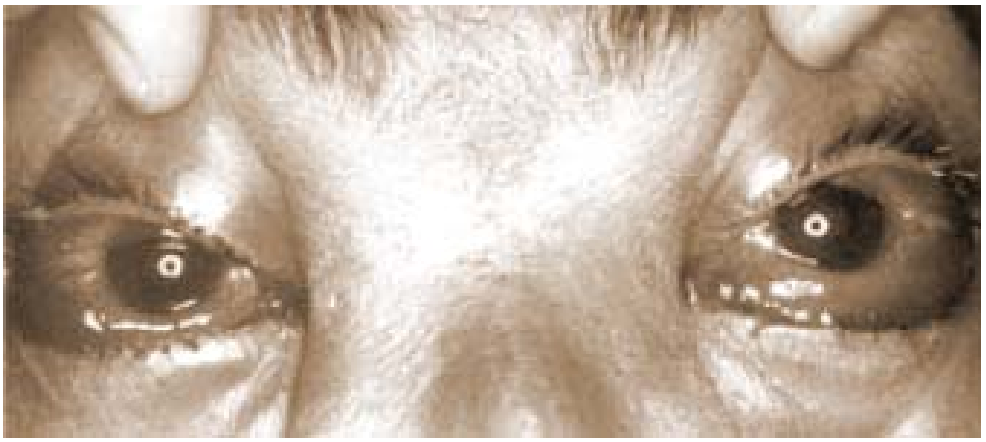

Figure 29: Bilateral conjunctival oedema (chemosis) after a bite by a Burmese Russell's viper (Copyright DA Warrell)

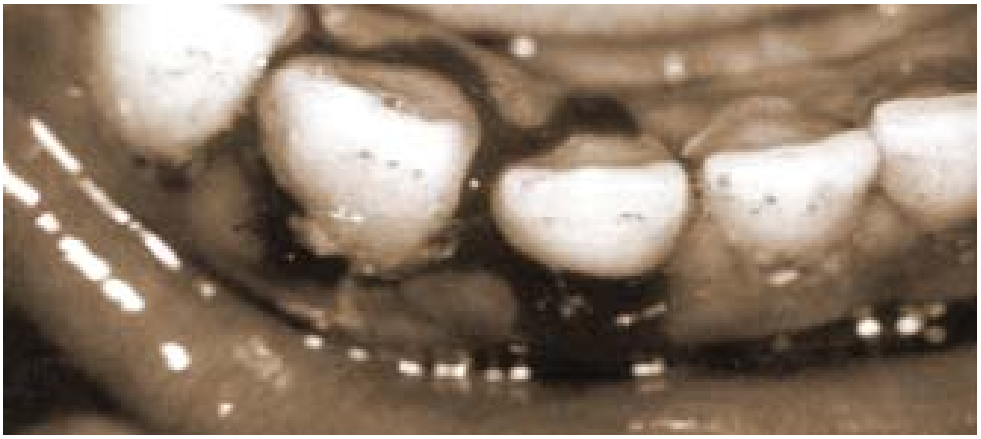

Figure 30: Bleeding from gingival sulci in a patient bitten by a saw-scaled viper (Copyright DA Warrell)

Figure 31: Subconjunctival haemorrhages in a patient bitten by a Burmese Russell's viper (Copyright DA Warrell)

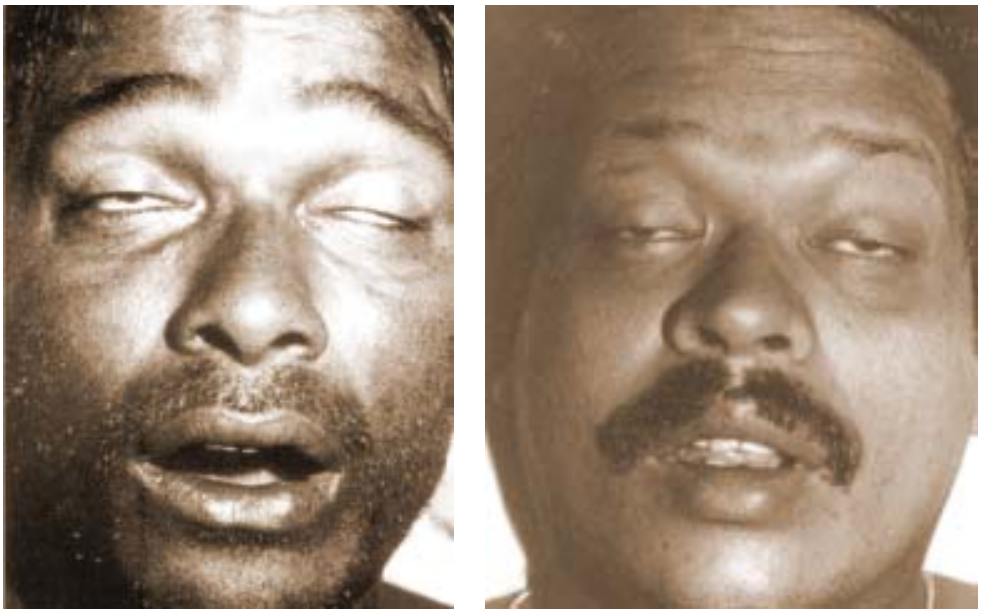

Figure 32: Bilateral ptosis (a) in a patient bitten by a common krait (Copyright DA Warrell), Bilateral ptosis (b) in a patient bitten by a Sri Lankan Russell's viper (Copyright DA Warrell)

Figure 33: External ophthalmoplegia in a patient bitten by a Russell's viper in Sri Lanka. The patient is attempting to look to his right. The eyes are held open because of the bilateral ptosis (Copyright DA *Warrell)*

Figure 34: Patient bitten by a Sri Lankan Russell's viper who began to pass dark brown urine containing myoglobin and haemoglobin 8 hours after the bite (Copyright DA Warrell)

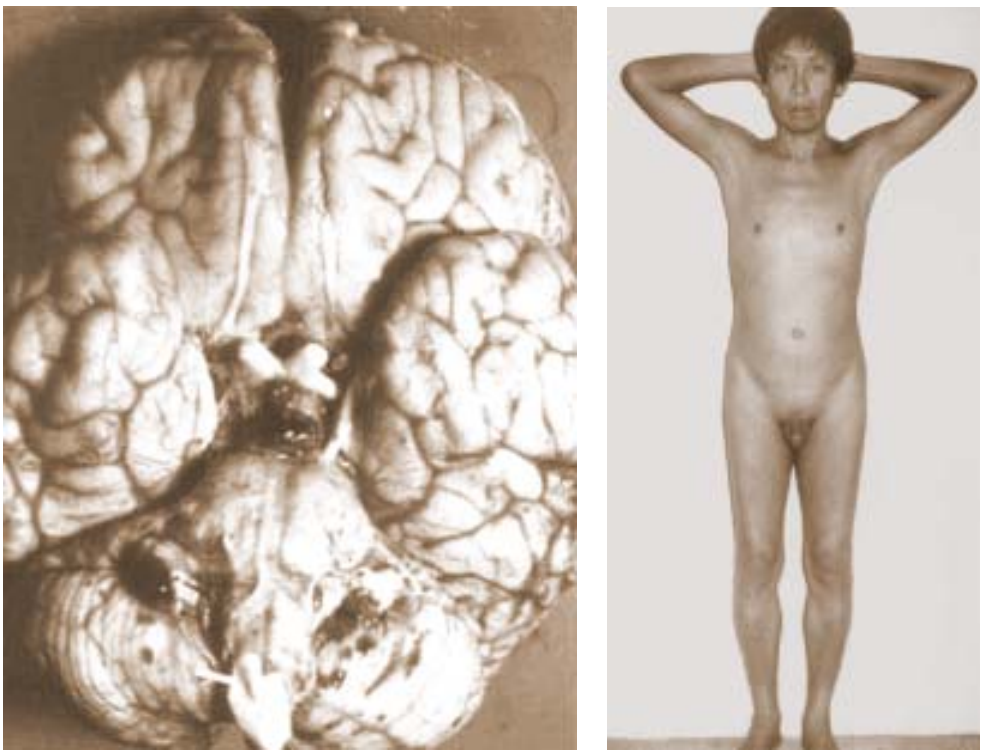

Figure 35: (a) Haemorrhagic infarction of the anterior pituitary (Sheehan's-like syndrome) after a bite by a Burmese Russell's viper, (b) Patient bitten by a Burmese Russell's viper three years previously, showing clinical signs of panhypopituitarism: loss of secondary sexual hair and testicular atrophy (Copyright DA Warrell)

2.4 Clinical syndromes of snake bite in South-East Asia

Limitations of syndromic approach

The more carefully the clinical effects of snake bites are studied, the more it is realised that the range of activities of a particular venom is very wide. For example, some elapid venoms, such as those of Asian cobras, can cause severe local envenoming

(Fig 28), formerly thought to be an effect only of viper venoms. In Sri Lanka and South India, Russell's viper venom causes paralytic signs (ptosis etc) (Fig 32), suggesting elapid neurotoxicity, and muscle pains and dark brown urine (Fig 34), suggesting sea snake rhabdomyolysis. Although thre may be considerable overlap of clinical features caused by venoms of different species of snake, a "syndromic approach" may still be useful, especially when the snake has not been identified and only monospecific antivenoms are available (see Annex 1 & 2).

Syndrome 1
Local envenoming (swelling etc) with bleeding/clotting disturbances = Viperidae (all species)

Syndrome 2
Local envenoming (swelling etc) with bleeding/clotting disturbances, shock or renal failure = Russell's viper (and possibly saw-scaled viper – Echis species – in some areas)

with conjunctival oedema (chemosis) and acute pituitary insufficiency = Russell's viper, Burma

with ptosis, external ophthalmoplegia, facial paralysis etc and dark brown urine = Russell's viper, Sri Lanka and South India

Syndrome 3
Local envenoming (swelling etc) with paralysis = cobra or king cobra

Syndrome 4
Paralysis with minimal or no local envenoming

Bite on land while sleeping, outside the Philippines = krait

in the Philippines = cobra (*Naja philippinensis*)

Bite in the sea = sea snake

Syndrome 5
Paralysis with dark brown urine and renal failure:

Bite on land (with bleeding/clotting disturbance) = Russell's viper, Sri Lanka/South India

Bite in the sea (no bleeding/clotting disturbances) = sea snake

2.5 Long term complications (sequelae) of snake bite

At the site of the bite, loss of tissue may result from sloughing or surgical débridement of necrotic areas or amputation: chronic ulceration, infection, osteomyelitis or arthritis may persist causing severe physical disability (Fig 36). Malignant transformation may occur in skin ulcers after a number of years (Fig 37).

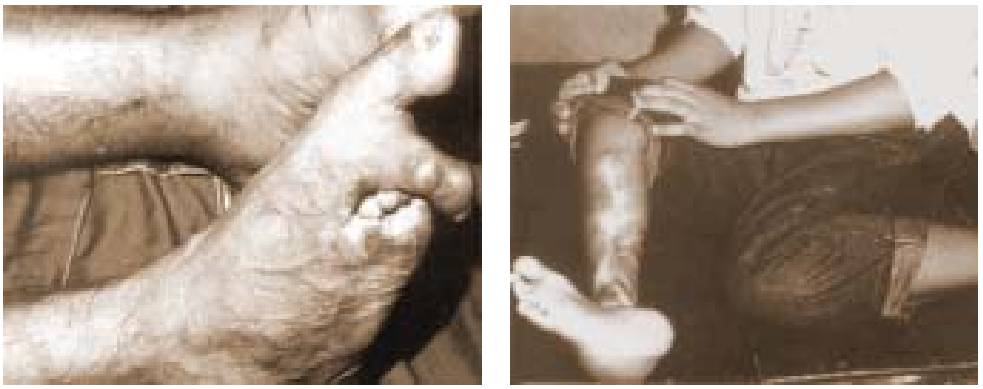

Figure 36: (a) and (b) Chronic physical handicap resulting from necrotic envenoming by Malayan pit vipers (Copyright DA Warrell)

Figure 37: Squamous cell carcinoma developing at the site of a chronic skin ulcer with osteomyelitis 8 years after a bite by a Malayan pit viper (Copyright DA Warrell)

Chronic renal failure occurs after bilateral cortical necrosis (Russell's viper bites) and chronic panhypopituitarism or diabetes insipidus after Russell's viper bites in Myanmar and South India (Fig 35b). Chronic neurological deficit is seen in the few patients who survive intracranial haemorrhages (Viperidae).

3

Symptoms and Signs of Cobra-spit Ophthalmia

(Eye injuries from spitting cobras) (Fig 38)

If the "spat" venom enters the eyes, there is immediate and persistent intense burning, stinging pain, followed by profuse watering of the eyes with production of whitish discharge, congested conjunctivae, spasm and swelling of the eyelids, photophobia and clouding of vision. Corneal ulceration, permanent corneal scarring and secondary endophthalmitis are recognised complications of African spitting cobra venom but have not been described in Asia.

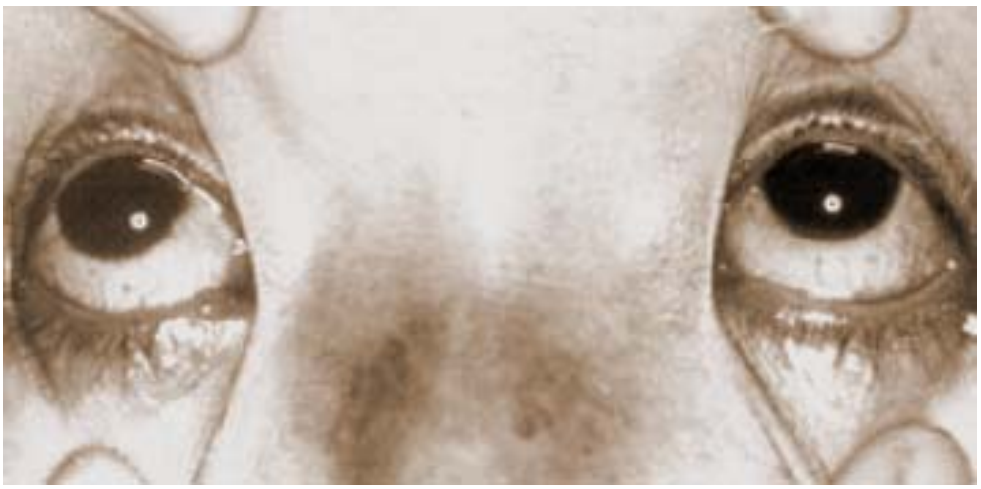

Figure 38: Bilateral conjunctivitis in a patient who had venom spat into both eyes by an Indo-Chinese spitting cobra (Naja siamensis) (Copyright DA Warrell)

4

Management of Snake Bites in South-East Asia

The following steps or stages are often involved

Management of snake bite

- First aid treatment
- Transport to hospital
- Rapid clinical assessment and resuscitation
- Detailed clinical assessment and species diagnosis
- Investigations/laboratory tests
- Antivenom treatment
- Observation of the response to antivenom: decision about the need for further dose(s) of antivenom
- Supportive/ancillary treatment
- Treatment of the bitten part
- Rehabilitation
- Treatment of chronic complications

4.1 First aid treatment

First aid treatment is carried out immediately or very soon after the bite, before the patient reaches a dispensary or hospital. It can be performed by the snake bite victim himself/herself or by anyone else who is present.

Aims of first aid

- attempt to retard systemic absorption of venom
- preserve life and prevent complications before the patient can receive medical care (at a dispensary or hospital)
- control distressing or dangerous early symptoms of envenoming
- arrange the transport of the patient to a place where they can receive medical care (4.2)
- **Above all, do no harm!**

Unfortunately, most of the traditional, popular, available and affordable first aid methods have proved to be useless or even frankly dangerous. These methods include: making local incisions or pricks/punctures ("tattooing") at the site of the bite or in the bitten limb, attempts to suck the venom out of the wound, use of (black) snake stones, tying tight bands (tourniquets) around the limb, electric shock, topical instillation or application of chemicals, herbs or ice packs.

Local people may have great confidence in traditional (herbal) treatments, but they must not be allowed to delay medical treatment or to do harm.

Most traditional first aid methods should be discouraged:
They do more harm than good !

Recommended first aid methods

- Reassure the victim who may be very anxious
- Immobilise the bitten limb with a splint or sling (any movement or muscular contraction increases absorption of venom into the bloodstream and lymphatics)
- Consider pressure-immobilisation (Fig 39) for some elapid bites
- Avoid any interference with the bite wound as this may introduce infection, increase absorption of the venom and increase local bleeding

As far as the snake is concerned – do not attempt to kill it as this may be dangerous. However, if the snake has already been killed, it should be taken to the dispensary or hospital with the patient in case it can be identified. However, do not handle the snake with your bare hands as even a severed head can bite!

The special danger of rapidly developing paralytic envenoming after bites by some elapid snakes: use of pressure-immobilisation

Bites by cobras, king cobras, kraits or sea snakes may lead, on rare occasions, to the rapid development of life-threatening respiratory paralysis. This paralysis might be

delayed by slowing down the absorption of venom from the site of the bite. The following technique is currently recommended:

Pressure immobilisation method (Fig 39). Ideally, an elasticated, stretchy, crepe bandage, approximately 10 cm wide and at least 4.5 metres long should be used. If that it not available, any long strips of material can be used. The bandage is bound firmly around the entire bitten limb, starting distally around the fingers or toes and moving proximally, to include a rigid splint. The bandage is bound as tightly as for a sprained ankle, but not so tightly that the peripheral pulse (radial, posterior tibial, dorsalis pedis) is occluded or that a finger cannot easily be slipped between its layers.

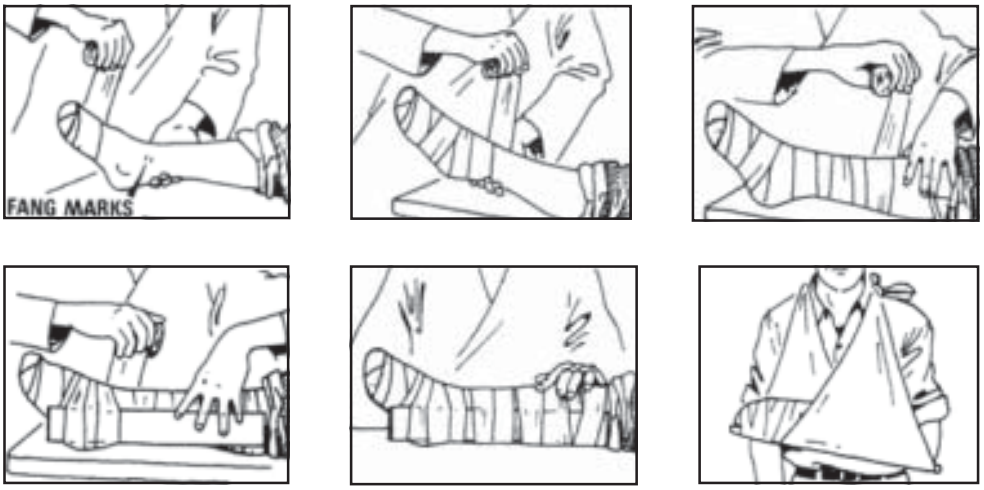

Figure 39: **Pressure immobilisation method. Recommended first-aid for bites by neurotoxic elapid snakes (by courtesy of the Australian Venom Research Unit, University of Melbourne)**

Pressure immobilisation is recommended for bites by neurotoxic elapid snakes, including sea snakes but should not be used for viper bites because of the danger of increasing the local effects of the necrotic venom.

Ideally, compression bandages should not be released until the patient is under medical care in hospital, resuscitation facilities are available and antivenom treatment has been started (**see Caution below**).

Caution: Release of a tight tourniquet or compression bandage may result in the dramatic development of severe systemic envenoming.

Tight (arterial) tourniquets are not recommended!

Traditional tight (arterial) tourniquets. To be effective, these had to be applied around the upper part of the limb, so tightly that the peripheral pulse was occluded. This method was extremely painful and very dangerous if the tourniquet was left on for too long (more than about 40 minutes), as the limb might be damaged by ischaemia. **Many gangrenous limbs resulted!**

Viper and cobra bites

The pressure-immobilisation method as described above will increase intracompartmental pressure and, by localising the venom, might be expected to increase the locally-necrotic effects of viper venoms and some cobra venoms.

Pressure bandaging is not recommended for bites by vipers and cobras whose venoms cause local necrosis.

The use of a local compression pad applied over the wound, without pressure bandaging of the entire bitten limb, has produced promising results in Myanmar and deserves further study.

Arterial tourniquets are not recommended

4.2 Transport to hospital

The patient must be transported to a place where they can receive medical care (dispensary or hospital) as quickly, but as safely and comfortably as possible. Any movement, but especially movement of the bitten limb, must be reduced to an absolute minimum to avoid increasing the systemic absorption of venom. Any muscular contraction will increase this spread of venom from the site of the bite. A stretcher, bicycle, cart, horse, motor vehicle, train or boat should be used, or the patient should be carried.

4.3 Treatment in the dispensary or hospital

Rapid clinical assessment and resuscitation

Cardiopulmonary resuscitation may be needed, including administration of oxygen and establishment of intravenous access. **A**irway, respiratory movements (**B**reathing) and arterial pulse (**C**irculation) must be checked immediately. The level of consciousness must be assessed.

The following are examples of clinical situations in which snake bite victims might require urgent resuscitation:

- Profound hypotension and shock resulting from direct cardiovascular effects of the venom or secondary effects such as hypovolaemia or haemorrhagic shock.
- Terminal respiratory failure from progressive neurotoxic envenoming that has led to paralysis of the respiratory muscles.
- Sudden deterioration or rapid development of severe systemic envenoming following the release of a tight tourniquet or compression bandage (see Caution above).

- Cardiac arrest precipitated by hyperkalaemia resulting from skeletal muscle breakdown (rhabdomyolysis) after sea snake bite.
- Late results of severe envenoming such as renal failure and septicaemia complicating local necrosis.

4.4 Detailed clinical assessment and species diagnosis

History

A precise history of the circumstances of the bite and the progression of local and systemic symptoms and signs is very important. Three useful initial questions are:

"In what part of your body have you been bitten?"

The doctor can see immediately evidence that the patient has been bitten by a snake (eg fang marks) and the nature and extent of signs of local envenoming.

"When were you bitten?"

Assessment of the severity of envenoming depends on how long ago the patient was bitten. If the patient has arrived at the hospital soon after the bite, there may be few symptoms and signs even though a large amount of venom may have been injected.

"Where is the snake that bit you?"

If the snake has been killed and brought, its correct identification can be very helpful. If it is obviously a harmless species (or not a snake at all!), the patient can be quickly reassured and discharged from hospital.

Early clues that a patient has severe envenoming:

- Snake identified as a very dangerous one
- Rapid early extension of local swelling from the site of the bite
- Early tender enlargement of local lymph nodes, indicating spread of venom in the lymphatic system
- Early systemic symptoms: collapse (hypotension, shock), nausea, vomiting, diarrhoea, severe headache, "heaviness" of the eyelids, inappropriate (pathological) drowsiness or early ptosis/ophthalmoplegia
- Early spontaneous systemic bleeding
- Passage of dark brown urine

Patients who become defibrinogenated or thrombocytopenic may begin to bleed from old, partially-healed wounds as well as bleeding persistently from the fang marks.

The patient should be asked how much urine has been passed since the bite and whether it was a normal colour.

An important early symptom of sea snake envenoming that may develop as soon as 30 minutes after the bite is generalised pain, tenderness and stiffness of muscles and trismus.

Physical examination

This should start with careful assessment of the site of the bite and signs of local envenoming.

Examination of the bitten part

The extent of swelling, which is usually also the extent of tenderness to palpation, should be recorded. Lymph nodes draining the limb should be palpated and overlying ecchymoses and lymphangitic lines noted.

A bitten limb may be tensely oedematous, cold, immobile and with impalpable arterial pulses. These appearances may suggest intravascular thrombosis, which is exceptionally rare after snake bite, or a compartmental syndrome, which is uncommon. If possible, intracompartmental pressure should be measured (see Annex 5) and the blood flow and patency of arteries and veins assessed (eg by doppler ultrasound).

Early signs of necrosis may include blistering, demarcated darkening (easily confused with bruising) or paleness of the skin, loss of sensation and a smell of putrefaction (rotting flesh).

General examination

Measure the blood pressure (sitting up and lying to detect a postural drop indicative of hypovolaemia) and heart rate. Examine the skin and mucous membranes for evidence of petechiae, purpura, ecchymoses and, in the conjunctivae, chemosis. Thoroughly examine the gingival sulci, using a torch and tongue depressor, as these may show the earliest evidence of spontaneous systemic bleeding. Examine the nose for epistaxis. Abdominal tenderness may suggest gastrointestinal or retroperitoneal bleeding. Loin (low back) pain and tenderness suggests acute renal ischaemia (Russell's viper bites). Intracranial haemorrhage is suggested by lateralising neurological signs, asymmetrical pupils, convulsions or impaired consciousness (in the absence of respiratory or circulatory failure).

Neurotoxic envenoming

To exclude early neurotoxic envenoming, ask the patient to look up and observe whether the upper lids retract fully (Fig 40). Test eye movements for evidence of early external ophthalmoplegia (Fig 33). Check the size and reaction of the pupils. Ask the patient to open their mouth wide and protrude their tongue; early restriction in mouth opening may indicate trismus (sea snake envenoming) or more often paralysis of pterygoid muscles (Fig 41). Check other muscles innervated by the cranial nerves (facial muscles, tongue, gag reflex etc). The muscles flexing the neck may be paralysed, giving the "broken neck sign" (Fig 42).

Bulbar and respiratory paralysis

Can the patient swallow or are secretions accumulating in the pharynx, an early sign of bulbar paralysis? Ask the patient to take deep breaths in and out. "Paradoxical respiration" (abdomen expands rather than the chest on attempted inspiration) indicates that the diaphragm is still contracting but that the intercostal muscles and accessory muscles of inspiration are paralysed. Objective measurement of ventilatory capacity is very useful. Use a peak flow metre, spirometer (FEV_1 and FVC) or ask the patient to blow into the tube of a sphygmomanometer to record the maximum expiratory pressure (mmHg). Remember that, provided their lungs are adequately ventilated, patients with profound generalised flaccid paralysis from neurotoxic envenoming are fully conscious. Because their eyes are closed and they do not move or speak, they are commonly assumed to be unconscious. They may still be able to flex a finger or toe and so simple communication is possible.

Figure 40: Examination for ptosis, usually the earliest sign of neurotoxic envenoming (Copyright DA Warrell)

Figure 41: Inability to open the mouth and protrude the tongue in a patient with neurotoxic envenoming from the Malayan krait (Copyright DA Warrell)

Figure 42: Broken neck sign in a child envenomed by a cobra in Malaysia (Copyright the late HA Reid)

Do not assume that patients have irreversible brain damage because they are areflexic, unresponsive to painful stimuli, or have fixed dilated pupils.

Generalised rhabdomyolysis

In victims of envenoming by sea snakes and Russell's vipers in Sri Lanka and South India, muscles, especially of the neck, trunk and proximal part of the limbs, may become tender and painful on active or passive movement and later may become paralysed. In sea snake bite there is pseudotrismus that can be overcome by sustained pressure on the lower jaw. Myoglobinuria may be evident 3 hours after the bite.

Examination of pregnant women

There will be concern about fetal distress (revealed by fetal bradycardia), vaginal bleeding and threatened abortion. Monitoring of uterine contractions and fetal heart rate is useful. Lactating women who have been bitten by snakes should be encouraged to continue breast feeding.

Species diagnosis

If the dead snake has been brought, it can be identified. Otherwise, the species responsible can be inferred indirectly form the patient's description of the snake and the clinical syndrome of symptoms and signs (see above and Annex 1 & 2). This is specially important in Thailand where only monospecific antivenoms are available.

4.5 Investigations/laboratory tests

20 minute whole blood clotting test (20WBCT)

This very useful and informative bedside test requires very little skill and only one piece of apparatus - a new, clean, dry, glass vessel (tube or bottle).

20 minute whole blood clotting test (20WBCT)

- Place a few mls of freshly sampled venous blood in a small glass vessel
- Leave undisturbed for 20 minutes at ambient temperature
- Tip the vessel once
- If the blood is still liquid (unclotted) and runs out, the patient has hypo-fibrinogenaemia ("incoagulable blood") as a result of venom-induced consumption coagulopathy
- In the South East Asian region, incoagulable blood is diagnostic of a viper bite and rules out an elapid bite
- **Warning! If the vessel used for the test is not made of ordinary glass, or if it has been used before and cleaned with detergent, its wall may not stimulate clotting of the blood sample in the usual way and test will be invalid**
- If there is any doubt, repeat the test in duplicate, including a "control" (blood from a healthy person)

Other tests

Haemoglobin concentration/haematocrit: a transient increase indicates haemoconcentration resulting from a generalised increase in capillary permeability (eg in Russell's viper bite). More often, there is a decrease reflecting blood loss or, in the case of Indian and Sri Lankan Russell's viper bite, intravascular haemolysis.

Platelet count: this may be decreased in victims of viper bites.

White blood cell count: an early neutrophil leucocytosis is evidence of systemic envenoming from any species.

Blood film: fragmented red cells ("helmet cell", schistocytes) are seen when there is microangiopathic haemolysis.

Plasma/serum may be pinkish or brownish if there is gross haemoglobinaemia or myoglobinaemia.

Biochemical abnormalities: aminotransferases and muscle enzymes (creatine kinase, aldolase etc) will be elevated if there is severe local damage or, particularly, if there is generalised muscle damage (Sri Lankan and South Indian Russell's viper bites, sea snake bites). Mild hepatic dysfunction is reflected in slight increases in other serum enzymes. Bilirubin is elevated following massive extravasation of blood. Creatinine, urea or blood urea nitrogen levels are raised in the renal failure of Russell's viper and saw-scaled viper bites and sea snake bites. Early hyperkalaemia may be seen following extensive rhabdomyolysis in sea snake bites. Bicarbonate will be low in metabolic acidosis (eg enal failure).

Arterial blood gases and pH may show evidence of respiratory failure (neurotoxic envenoming) and acidaemia (respiratory or metabolic acidosis).

Warning: arterial puncture is contraindicated in patients with haemostatic abnormalities (Viperidae)

Desaturation: arterial oxygen desaturation can be assessed non-invasively in patients with respiratory failure or shock using a finger oximeter.

Urine examination: the urine should be tested by dipsticks for blood/haemoglobin/myoglobin. Standard dipsticks do not distinguish blood, haemoglobin and myoglobin. Haemoglobin and myoglobin can be separated by immunoassays but there is no easy or reliable test. Microscopy will confirm whether there are erythrocytes in the urine. Red cell casts indicate glomerular bleeding. Massive proteinuria is an early sign of the generalised increase in capillary permeability in Russell's viper envenoming.

4.6 Antivenom treatment

Antivenom is the only specific antidote to snake venom. A most important decision in the management of a snake bite victim is whether or not to give antivenom.

What is antivenom?

Antivenom is immunoglobulin (usually the enzyme refined $F(ab)_2$ fragment of IgG) purified from the serum or plasma of a horse or sheep that has been immunised with the venoms of one or more species of snake. "Specific" antivenom, implies that the antivenom has been raised against the venom of the snake that has bitten the patient and that it can therefore be expected to contain specific antibody that will neutralise that particular venom. Monovalent or monospecific antivenom neutralises the venom of only one species of snake. Polyvalent or polyspecific antivenom neutralises the venoms of several different species of snakes, usually the most important species, from a medical point of view, in a particular geographical area. For example, Haffkine, Kasauli, Serum Institute of India and Bengal "polyvalent anti-snake venom serum" is raised in horses using the venoms of t he four most important venomous snakes in India (Indian cobra, *Naja naja*; Indian krait, *Bungarus caeruleus*; Russell's viper, *Daboia russelii*; saw-scaled viper, *Echis carinatus*). Antibodies raised against the venom ofone species may have cross-neutralising activity against other venoms, usually from closely related species. This is known as paraspecific activity. For example, the manufacturers of Haffkine polyvalent anti-snake venom serum claim that this antivenom also neutralises venoms of two Trimeresurus species.

Indications for antivenom treatment (see also Annex 1 & 2)

Antivenom treatment carries a risk of severe adverse reactions and in most countries it is costly and may be in limited supply. It should therefore be used only in patients in whom the benefits of antivenom treatment are considered to exceed the risks.

Indications for antivenom vary in different countries.

Inappropriate use of antivenom

In some parts of the world, antivenom is given to any patient claiming to have been bitten by a snake, irrespective of symptoms or signs of envenoming. Sometimes the local community are so frightened of snake bite that they compel the doctor to give antivenom against medical advice. These practices should be strongly discouraged as they expose patients who may not need treatment to the risks of antivenom reactions; they also waste valuable and scarce stocks of antivenom.

Indications for antivenom

Antivenom treatment is recommended if and when a patient with proven or suspected snake develops one or more of the following signs

Systemic envenoming

- *Haemostatic abnormalities:* spontaneous systemic bleeding (**clinical**), coagulopathy (**20WBCT or other laboratory**) or thrombocytopenia (<100 x 109/litre) (**laboratory**)
- Neurotoxic signs: ptosis, external ophthalmoplegia, paralysis etc (**clinical**)
- *Cardiovascular abnormalities*: hypotension, shock, cardiac arrhythmia (**clinical**), abnormal ECG
- *Acute renal failure:* oliguria/anuria (clinical), rising blood creatinine/ urea (**laboratory**)
- (*Haemoglobin-/myoglobin-uria:*) dark brown urine (**clinical**), urine dipsticks, other evidence of intravascular haemolysis or generalised rhabdomyolysis (muscle aches and pains, hyperkalaemia) (**clinical, laboratory**)
- Supporting laboratory evidence of systemic envenoming (see 4.5, page 30)

Local envenoming

- Local swelling involving more than half of the bitten limb (in the absence of a tourniquet) Swelling after bites on the digits (toes and especially fingers)
- Rapid extension of swelling (for example beyond the wrist or ankle within a few hours of bites on the hands or feet)
- Development of an enlarged tender lymph node draining the bitten limb

How long after the bite can antivenom be expected to be effective?

Antivenom treatment should be given as soon as it is indicated. It may reverse systemic envenoming even when this has persisted for several days or, in the case of haemostatic abnormalities, for two or more weeks. However, when there are signs of local envenoming, **without** systemic envenoming, antivenom will be effective only if it can be given within the first few hours after the bite.

Prediction of antivenom reactions

Skin and conjunctival "hypersensitivity" tests may reveal IgE mediated Type I hypersensitivity to horse or sheep proteins but do not predict the large majority of early (anaphylactic) or late (serum sickness type) antivenom reactions. Since they may delay treatment and can in themselves be sensitizing, these tests should not be used.

Contraindications to antivenom

There is no absolute contraindication to antivenom treatment, but patients who have reacted to horse (equine) or sheep (ovine) serum in the past (for example after treatment with equine anti-tetanus serum, equine anti-rabies serum or equine or ovine antivenom) and those with a strong history of atopic diseases (especially severe asthma) should be given antivenom only if they have signs of systemic envenoming.

Prophylaxis in high risk patients

In the absence of any prophylactic regimen that has proved effective in clinical trials, these high risk patients may be pre-treated **empirically** with subcutaneous epinephrine (adrenaline), intravenous antihistamines (both anti-H_1, such as promethazine or chloramphenicol; and anti- H_2, such as cimetidine or ranitidine) and corticosteroid. In asthmatic patients, prophylactic use of an inhaled adrenergic $\exists_2$ agonist such as salbutamol may prevent bronchospasm.

Selection of antivenom

Antivenom should be given only if its stated range of specificity includes the species known or thought to have been responsible for the bite. Liquid antivenoms that have become opaque should not be used as precipitation of protein indicates loss of activity and an increased risk of reactions.

Expiry dates quoted by manufacturers are often very conservative. Provided that antivenom has been properly stored, it can be expected to retain useful activity for many months after the stated “expiry date”.

If the biting species is known, the ideal treatment is with a monospecific/ monovalent antivenom, as this involves administration of a lower dose of antivenom protein than with a polyspecific/ polyvalent antivenoms. Polyspecific/polyvalent antivenoms are preferred in many countries because of the difficulty in identifying species responsible for bites. Polyspecific antivenoms can be just as effective as monospecific ones, but since they contain specific antibodies against several different venoms, a larger dose of antivenom protein must be administered to neutralise a particular venom.

Administration of antivenom

- Epinephrine (adrenaline) should always be drawn up in readiness before antivenom is administered.
- Antivenom should be given by the intravenous route whenever possible.

Freeze-dried (lyophilised) antivenoms are reconstituted, usually with 10 ml of sterile water for injection per ampoule. The freeze-dried protein may be difficult to dissolve. Two methods of administration are recommended:

(1) *Intravenous "push" injection*: reconstituted freeze-dried antivenom or neat liquid antivenom is given by slow intravenous injection (not more than 2 ml/minute). This method has the advantage that the doctor/nurse/dispenser giving the antivenom must remain with the patient during the time when some early reactions may develop. It is also economical, saving the use of intravenous fluids, giving sets, cannulae etc.

(2) *Intravenous infusion*: reconstituted freeze-dried or neat liquid antivenom is diluted in approximately 5-10 ml of isotonic fluid per kg body weight (ie 250-500 ml of isotonic saline or 5% dextrose in the case of an adult patient) and is infused at a constant rate over a period of about one hour.

Local administration of antivenom at the site of the bite is not recommended!

Although this route may seem rational, it should not be used as it is extremely painful, may increase intracompartmental pressure and has not been shown to be effective.

Intramuscular injection of antivenom

Antivenoms are large molecules ($F(ab)_2$ fragments or sometimes whole IgG) which, after intramuscular injection, are absorbed slowly via lymphatics. Bioavailability is poor, especially after intragluteal injection and blood levels of antivenom never reach those achieved rapidly by intravenous administration. Other disadvantages are the pain of injection of large volumes of antivenom and the risk of haematoma formation in patients with haemostatic abnormalities.

Antivenom must never be given by the intramuscular route if it could be given intravenously.

Situations in which intramuscular administration might be considered :

- at a peripheral first aid station, before a patient with obvious envenoming is put in an ambulance for a journey to hospital that may last several hours;
- on an expedition exploring a remote area very far from medical care;
- when intravenous access has proved impossible.

Although the risk of antivenom reactions is less with intramuscular than intravenous administration, epinephrine (adrenaline) must be readily available. Patients must be closely observed *for at least one hour* after starting intravenous antivenom administration, so that early anaphylactic antivenom reactions can be detected and treated early with epinephrine (adrenaline).

Under these unusual circumstances, the dose of antivenom should be divided between a number of sites in the upper anterolateral region of both thighs. A maximum of 5-10 ml should be given at each site by deep intramuscular injection followed by massage to aid absorption. Local bleeding and haematoma formation is a problem in patients with incoagulable blood.

Finding enough muscle mass to contain such large volumes of antivenom is particularly difficult in children.

Antivenom should never be injected into the gluteal region (upper outer quadrant of the buttock) as absorption is exceptionally slow and unreliable and there is always the danger of sciatic nerve damage when the injection is given by an inexperienced operator.

Dose of antivenom

Snakes inject the same dose of venom into children and adults. Children must therefore be given exactly the same dose of antivenom as adults.

Manufacturers' recommendations are usually based on inappropriate animal tests in which venom and antivenom are incubated before being injected into the test animal. The recommended dose is often the amount of antivenom required to neutralise the average venom yield when captive snakes are milked of their venom. In practice, the choice of an initial dose of antivenom is usually empirical.

Antivenom manufacturers, health institutions and medical research organisations should encourage and promote the proper clinical testing of antivenoms as with other therapeutic agents. This is the only reliable guide to the initial dose (and safety) of an antivenom.

Since the neutralising power of antivenoms varies from batch to batch, the results of a particular clinical trial may soon become obsolete if the manufacturers change the strength of the antivenom.

Antivenom reactions

A proportion of patients, usually more than 20%, develop a reaction either early (within a few hours) or late (5 days or more) after being given antivenom.

Early anaphylactic reactions: usually within 10-180 minutes of starting antivenom, the patient begins to itch (often over the scalp) and develops urticaria, dry cough, fever, nausea, vomiting, abdominal colic, diarrhoea and tachycardia. A minority of these patients may develop severe life-threatening anaphylaxis: hypotension, bronchospasm and angio-oedema. Fatal reactions have probably been under-reported as death after snake bite is usually attributed to the venom.

In most cases, these reactions are not truly "allergic". They are not IgE-mediated type I hypersensitivity reactions to horse or sheep proteins as there is no evidence of specific IgE, either by skin testing or radioallergosorbent tests (RAST). Complement activation by IgG aggregates or residual Fc fragments or direct stimulation of mast cells or basophils by antivenom protein are more likely mechanisms for these reactions.

Pyrogenic (endotoxin) reactions usually develop 1-2 hours after treatment. Symptoms include shaking chills (rigors), fever, vasodilatation and a fall in blood pressure. Febrile convulsions may be precipitated in children. These reactions are caused by pyrogen contamination during the manufacturing process. They are commonly reported.

Late (serum sickness type) reactions develop 1-12 (mean 7) days after treatment. Clinical features include fever, nausea, vomiting, diarrhoea, itching, recurrent urticaria, arthralgia, myalgia, lymphadenopathy, periarticular swellings, mononeuritis multiplex, proteinuria with immune complex nephritis and rarely encephalopathy. Patients who suffer early reactions and are treated with antihistamines and corticosteroid are less likely to develop late reactions.

Treatment of early anaphylactic and pyrogenic antivenom reactions

Epinephrine (adrenaline) is given intramuscularly (into the deltoid muscle or the upper lateral thigh) in an initial dose of 0.5 mg for adults, 0.01 mg/kg body weight for children. Severe, life-threatening anaphylaxis can evolve very rapidly and so epinephrine (adrenaline) should be given at the very first sign of a reaction, even when only a few spots of urticaria have appeared or at the start of itching, tachycardia or restlessness. The dose can be repeated every 5-10 minutes if the patient's condition is deteriorating.

At the earliest sign of a reaction:

- Antivenom administration must be temporarily suspended
- Epinephrine (adrenaline) (0.1% solution, 1 in 1,000, 1 mg/ml) is the effective treatment for early anaphylactic and pyrogenic antivenom reactions

Additional treatment

After epinephrine (adrenaline), an anti H_1 antihistamine such as chlorpheniramine maleate (adults 10 mg, children 0.2 mg/kg by intravenous injection over a few minutes) should be given followed by intravenous hydrocortisone (adults 100 mg, children 2 mg/kg body weight). The corticosteroid is unlikely to act for several hours, but may prevent recurrent anaphylaxis.

There is increasing evidence that anti H_2 antihistamines such as cimetidine or ranitidine have a role in the treatment of severe anaphylaxis. Both drugs are given, diluted in 20 ml isotonic saline, by slow intravenous injection (over 2 minutes).

Doses: cimetidine – adults 200 mg, children 4 mg/kg;
ranitidine – adults 50 mg, children 1 mg/kg.

In pyrogenic reactions the patient must also be cooled physically and with antipyretics (for example paracetamol by mouth or suppository). Intravenous fluids should be given to correct hypovolaemia.

Treatment of late (serum sickness) reactions

Late (serum sickness) reactions usually respond to a 5-day course of oral antihistamine. Patients who fail to respond in 24-48 hours should be given a 5-day course of prednisolone.

Doses: Chlorpheniramine: adults 2 mg six hourly, children 0.25 mg/kg /day in divided doses

Prednisolone: adults 5 mg six hourly, children 0.7 mg/kg/day in divided doses for 5-7 days

Observation of the response to antivenom

If an adequate dose of appropriate antivenom has been administered, the following responses may be seen.

- *General*: the patient feels better. Nausea, headache and generalised aches and pains may disappear very quickly. This may be partly attributable to a placebo effect.
- *Spontaneous systemic bleeding* (eg from the gums) usually stops within 15-30 minutes.
- *Blood coagulability* (as measured by 20WBCT) is usually restored in 3-9 hours. Bleeding from new and partly healed wounds usually stops much sooner than this.
- *In shocked patients*, blood pressure may increase within the first 30-60 minutes and arrhythmias such as sinus bradycardia may resolve.
- *Neurotoxic envenoming* of the post-synaptic type (cobra bites) may begin to improve as early as 30 minutes after antivenom, but usually take several hours. Envenoming with presynaptic toxins (kraits and sea snakes) is unlikely to respond in this way.
- *Active haemolysis and rhabdomyolysis* may cease within a few hours and the urine returns to its normal colour.

Recurrence of systemic envenoming

In patients envenomed by vipers, after an initial response to antivenom (cessation of bleeding, restoration of blood coagulability), signs of systemic envenoming may recur within 24-48 hours.

This is attributable to:

(1) continuing absorption of venom from the "depot" at the site of the bite, perhaps assisted by improved blood supply following correction of shock, hypovolaemia etc, after elimination of antivenom (range of elimination half-lives: IgG 45 hours; $F(ab)_2$ 80-100 hours; Fab 12-18 hours);

(2) a redistribution of venom from the tissues into the vascular space, as the result of antivenom treatment.

Recurrent neurotoxic envenoming after treatment of cobra bite has also been described.

Criteria for repeating the initial dose of antivenom

Criteria for giving more antivenom
• Persistence or recurrence of blood incoagulability after 6 hr of bleeding after 1-2 hr • Deteriorating neurotoxic or cardiovascular signs after 1-2 hr

If the blood remains incoagulable (as measured by 20WBCT) six hours after the initial dose of antivenom, the same dose should be repeated. This is based on the observation that, if a large dose of antivenom (more than enough to neutralise the venom procoagulant enzymes) is given initially, the time taken for the liver to restore coagulable levels of fibrinogen and other clotting factors is 3-9 hours.

In patients who continue to bleed briskly, the dose of antivenom should be repeated within 1-2 hours.

In case of deteriorating neurotoxicity or cardiovascular signs, the initial dose of antivenom should be repeated after 1-2 hours, and full supportive treatment must be considered.

Conservative treatment when no antivenom is available

This will be the situation in many parts of the region, where supplies of antivenom run out or where the bite is known to have been inflicted by a species against whose venom there is no available specific antivenom (for example for bites by the Malayan krait (*Bungarus candidus*), coral snakes - genera Calliophis and Maticora), sea snakes, the mangrove/shore pit viper *T purpureomaculatus* and the mountain pit viper *Ovophis monticola*.

The following conservative measures are suggested:

Neurotoxic envenoming with respiratory paralysis: assisted ventilation. This has proved effective, and has been followed by complete recovery, even after being maintained for periods of more than one month. Manual ventilation (anaesthetic bag) by relays of doctors, medical students, relatives and nurses has been effective where no mechanical ventilator was available. Anticholinesterases should always be tried (see below Trial of anticholinesterase, p 41).

Haemostatic abnormalities – strict bed rest to avoid even minor trauma; transfusion of clotting factors and platelets; ideally, fresh frozen plasma and cryoprecipitate with platelet concentrates or, if these are not available, fresh whole blood. Intramuscular injections should be avoided.

Shock, myocardial damage: hypovolaemia should be corrected with colloid/crystalloids, controlled by observation of the central venous pressure. Ancillary pressor

drugs (dopamine or epinephrine-adrenaline) may also be needed. Patients with hypotension associated with bradycardia should be treated with atropine.

Renal failure: conservative treatment or dialysis (see Oliguria and renal failure, page 42).

Dark brown urine (myoglobinuria or haemoglobinuria): correct hypovolaemia and acidosis and consider a single infusion of mannitol (see Prevention of renal damage in patients with myoglobinuria or haemoglobinuria, page 45).

Severe local envenoming: local necrosis, intracompartmental syndromes and even thrombosis of major vessels is more likely in patients who cannot be treated with antivenom. Surgical intervention may be needed but the risks of surgery in a patient with consumption coagulopathy, thrombocytopenia and enhanced fibrinolysis must be balanced against the lifethreatening complications of local envenoming. Prophylactic broad spectrum antimicrobial treatment is justified (see Bacterial infections, page 46).

4.7 Supportive/ancillary treatment

Antivenom treatment can be expected to neutralise free circulating venom, prevent progression of envenoming and allow recovery. However, these processes take time and the severely envenomed patient may require life support systems such as assisted ventilation and renal dialysis until the severely damaged organs and tissues have had time to recover.

Dangers of venepuncture in patients with haemostatic abnormalities

In patients with incoagulable blood, any injection (subcutaneous, intramuscular) and, particularly venepuncture, carries a risk of persistent bleeding and haematoma formation. Arterial puncture is contraindicated in such patients.

Repeated venepuncture can be avoided by using an indwelling cannula and three-way tap system. When blood coagulability has been restored, the dead space should be filled with heparinised saline, but beware! If this is not flushed out before blood sampling, misleading results will be obtained in clotting tests, including the 20WBCT.

In patients with coagulopathy, sites of venous access and placement of intravenous cannulae or catheters should be chosen where haemostasis by external pressure is most likely to be effective, eg the antecubital fossa. If possible, avoid jugular, subclavian and femoral vein puncture. A pressure pad must be applied at the site of any venepuncture.

Neurotoxic envenoming

Antivenom treatment alone cannot be relied upon to save the life of a patient with bulbar and respiratory paralysis.

Death may result from aspiration, airway obstruction or respiratory failure. A clear airway must be maintained. Once there is loss of gag reflex and pooling of secretions in the pharynx, failure of the cough reflex or respiratory distress, a cuffed endotracheal tube should be inserted. If this is impossible for any reason, a tracheostomy should be performed and a snugly-fitting or cuffed tracheostomy tube inserted.

Although artificial ventilation was first suggested for neurotoxic envenoming 125 years ago, patients continue to die of asphyxiation because some doctors believe that antivenom is sufficient treatment.

Anticholinesterase drugs have a variable, but potentially very useful effect in patients with neurotoxic envenoming, especially those bitten by cobras.

A trial of anticholinesterase (eg "Tensilon test") should be performed in every patient with neurotoxic envenoming, as it would be in any patient with suspected myasthenia gravis.

Trial of anticholinesterase

Anticholinesterase (eg "Tensilon"/edrophonium) test

- Baseline observations
- Give atropine intravenously
- Give anticholinesterase drug
- Observe effect
- If positive, institute regular atropine and (long acting) anticholinesterase

Ideally, a short acting anticholinesterase, such as edrophonium ("Tensilon"), should be used. Baseline observations or measurements are made against which to assess the effectiveness of the anticholinesterase. Atropine sulphate (adults 0.6 mg, children 50 μg/kg body weight) is given by intravenous injection (to prevent the undesirable muscarinic effects of acetylcholine such as increased secretions, sweating, bradycardia and colic) followed immediately by edrophonium chloride (adults 10 mg, children 0.25 mg/kg body weight) given intravenously over 3 or 4 minutes. The patient is observed over the next 10-20 minutes for signs of improved neuromuscular transmission. Ptosis may disappear (Fig 43) and ventilatory capacity (peak flow, FEV1 or maximum expiratory pressure) may improve.

If edrophonium chloride is not available, any other anticholinesterases (neostigmine – "Prostigmine", distigmine, pyridostigmine, ambenomium) can be used for this assessment but a longer period of observation will be needed (up to 1 hour).

Patients who respond convincingly can be maintained on a longer-acting anticholinesterase such as neostigmine methylsulphate combined with atropine.

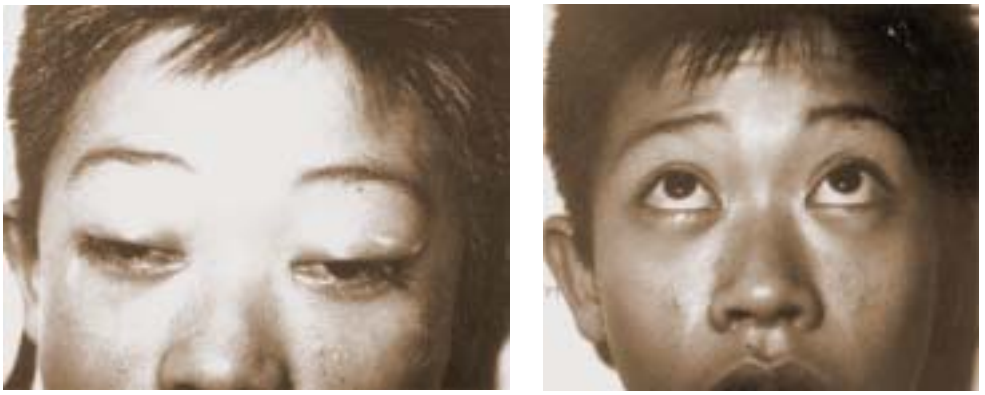

Figure 43: (a) before and (b) after intravenous atropine followed by intravenous edrophonium chloride in a patient envenomed by a Malayan krait (Bungarus candidus) (Copyright DA Warrell)

Hypotension and shock

Snake bite: causes of hypotension and shock	
(1) Anaphylaxis	(2) Antivenom reaction
Vasodilatation	Respiratory failure
Cardiotoxicity	Acute pituitary adrenal insufficiency
Hypovolaemia	Septicaemia

This is usually the result of hypovolaemia (from loss of circulating volume into the swollen limb, or internal/external haemorrhage), venom-induced vasodilatation or direct myocardial effects with or without arrhythmias. Ideally, treatment with plasma expanders (colloids or crystalloid) should be controlled by observation of the central venous pressure (jugular venous pressure or direct measurement of pressure in the superior vena cava via a catheter connected to a saline manometer, see Annex 4). Excessive volume replacement may cause pulmonaryoedema when plasma extravasated in the bitten limb and elsewhere is reabsorbed into the circulation.

In patients with evidence of a generalised increase in capillary permeability, a selective vasoconstrictor such as dopamine may be given by intravenous infusion, preferably into a central vein (starting dose 2.5-5 μg/kg/minute).

In victims of Russell's viper bites in Myanmar and South India, acute pituitary adrenal insufficiency resulting from haemorrhagic infarction of the anterior pituitary may contribute to shock. Hydrocortisone is effective in these cases.

Oliguria and renal failure

Detection of renal failure

- Dwindling or no urine output
- Rising blood urea/creatinine concentrations
- Clinical "uraemia syndrome"

  nausea, vomiting, hiccups, fetor, drowsiness, confusion, coma, flapping tremor, muscle twitching, convulsions, pericardial friction rub, signs of fluid overload

In patients with any of these features, the following should be monitored

- pulse rate
- blood pressure, lying and sitting, to detect postural hypotension
- respiratory rate
- temperature
- height of jugular venous pulse
- auscultation of lung bases for crepitations

Oliguric phase of renal failure

Most, but not all, patients with acute renal failure are oliguric, defined as a urine output of less than 400 ml/day or less than 20 ml/hour. Conservative management may tide the patient over, avoiding the need for dialysis. If the patient is hypovolaemic, indicated by supine or postural hypotension, empty neck veins, sunken eyeballs, loss of skin turgor and dryness of mucosae, proceed as follows:

(1) Establish intravenous access

(2) Insert a urethral catheter (full sterile precautions!)

(3) Determine the central venous pressure. This can be achieved either by observing the vertical height of the jugular venous pulsation above the sternal angle with the patient propped up on pillows at 45°; or by direct measurement of central venous (superior vena caval) pressure through a long catheter preferably inserted at the antecubital fossa (see Annex 4). The catheter is connected to a saline manometer, the 0 point of which must be placed at the same level as the right atrium (that is, at the sternal angle when the patient is propped up at 45°). In someone who is obviously volume-depleted, resuscitation should start immediately, and not be delayed until a central venous line has been inserted.

(4) Fluid challenge: depending on the initial state of hydration/dehydration, an adult patient can be given two litres of isotonic saline over one hour or, until the jugular venous pressure/central venous pressure has risen to 8-10 cm above the sternal angle (with the patient propped up at 45o). The patient must be closely observed while this is being done. The fluid challenge must be stopped immediately if pulmonary oedema develops. If the urine output does not improve, try furosamide challenge.

(5) Furosamide (frusemide) challenge: 100 mg of furosamide is injected slowly (4-5 mg/minute). If this does not induce a urine output of 40 ml/hour, give a second dose of furosamide, 200 mg. If urine output does not improve, try mannitol challenge.

(6) Mannitol challenge: 200 ml of 20% mannitol may be infused intravenously over 20 minutes but this must not be repeated as there is a danger of inducing dangerous fluid and electrolyte imbalance. An improvement in urine output to more than 40 ml/hr or more than 1 litre/day is considered satisfactory.

(7) Conservative management: If the urine output does not improve, despite these challenges, no further diuretics should be given and fluid intake should

be restricted to a total of the previous day's output plus "insensible losses" (500-1000 ml/day). If possible, the patient should be referred to a renal unit. The diet should be bland, high in calories (1700/day), low in protein (less than 40g/day), low in potassium (avoid fruit, fruit juices and potassium-containing drugs) and low in salt. Infections will cause tissue breakdown and increase urea levels. They should be prevented or treated promptly with non-nephrotoxic antibiotics (ie avoid aminoglycosides such as gentamicin).

(8) Biochemical monitoring: Serum potassium, urea, creatinine and, if possible, pH, bicarbonate, calcium and phosphate should be monitored frequently. If this is not possible the electrocardiogram (ECG) should be examined for evidence of hyperkalaemia, especially following bites by sea snakes, or Sri Lankan or South Indian Russell's vipers or if the patient is passing dark brown urine, indicating rhabdomyolysis or intravascular haemolysis.

ECG evidence of hyperkalaemia: tall peaked T waves, prolonged P-R interval, absent P waves, wide QRS complexes.

Emergency treatment of hyperkalaemia (serum potassium >6.5 mmol/l or ECG changes)

- give 10 ml of 10% calcium gluconate intravenously over 2 minutes (with ECG monitoring if possible) repeated up to three times
- give 50 ml of 50% dextrose with 10 units of soluble insulin intravenously
- sodium bicarbonate (40 ml of 8.4%) by slow intravenous infusion and a â2 agonist aerosol by inhaler (eg salbutamol – "Ventolin" 5-10 mg) may also be used

These emergency treatments will control hyperkalaemia for 3-6 hours only. If the patient is hypotensive and profoundly acidotic (deep sighing "Kussmaul" respirations, very low plasma bicarbonate concentration or very low pH - <7.10), 40 ml of 8.4% sodium bicarbonate (1 mmol/ml) may be infused intravenously over 30 minutes. If this leads to circulatory improvement, the dose can be repeated.

Caution: Intravenous bicarbonate may precipitate profound hypocalcaemia and fitting, especially in patients with rhabdomyolysis.

(9) Dialysis

Indications for dialysis

- Clinical uraemia
- Fluid overload
- Blood biochemistry – one or more of the following
  creatinine >6 mg/dl (500 μmol/l)
  urea >200 mg/dl (400 mmol/l)
  potassium >7 mmol/l (or hyperkalaemic ECG changes)
  symptomatic acidosis

Prevention of renal damage in patients with myoglobinuria or haemoglobinuria

To minimise the risk of renal damage from excreted myoglobin and/or haemoglobin:

- correct hypovolaemia (see above) and maintain saline diuresis (if possible)
- correct severe acidosis with bicarbonate (see above)
- give a single infusion of mannitol (200 ml of 20% solution over 20 minutes)

Diuretic phase of renal failure

Urine output increases following the period of anuria. The patient may become polyuric and volume depleted so that salt and water must be carefully replaced. Hypokalaemia may develop, in which case a diet rich in potassium (fruit and fruit juices) is recommended.

Renal recovery phase

The diuretic phase may last for months after Russell's viper bite. In Myanmar and South India, hypopituitarism may complicate recovery of Russell's viper bite victims. Corticosteroid, fluid and electrolyte replacement may be needed in these patients.

Persisting renal dysfunction

In Myanmar, persistent tubular degenerative changes were observed in Russell's viper bite victims who showed continuing albuminuria, hypertension and nocturia for up to 11 months after the bite, despite apparent recovery in renal function. In India, 20-25% of patients referred to renal units with acute renal failure following Russell's viper bite suffered oliguria for more than 4 weeks suggesting the possibility of bilateral renal cortical necrosis. This can be confirmed by renal biopsy or contrast enhanced CT scans of the kidneys. Patients with patchy cortical necrosis show delayed and partial recovery of renal function but those with diffuse cortical necrosis require regular maintenance dialysis and eventual renal transplantation.

Haemostatic disturbances

Bleeding and clotting disturbances usually respond satisfactorily to treatment with specific antivenom, but the dose may need to be repeated several times, at six hourly intervals, before blood coagulability (assessed by the 20WBCT) is finally and permanently restored.

In exceptional circumstances, such as severe bleeding or imminent urgent surgery, once specific antivenom has been given to neutralise venom procoagulants and other antihaemostatic toxins, restoration of coagulability and platelet function can be accelerated by giving fresh frozen plasma, cryoprecipitate (fibrinogen, factor VIII), fresh whole blood or platelet concentrates.

Heparin is ineffective against venom-induced thrombin and may cause bleeding on its own account. It should never be used in cases of snake bite.

Antifibrinolytic agents are not effective and should not be used in victims of snake bite.

4.8 Treatment of the bitten part

The bitten limb, which may be painful and swollen, should be nursed in the most comfortable position, preferably slightly elevated, to encourage reabsorption of oedema fluid. Bullae may be large and tense but they should be aspirated only if they seem likely to rupture.

Bacterial infections

Infection at the time of the bite with organisms from the snake's venom and buccal cavity is a problem with some species such as the Malayan pit viper. In this case, a prophylactic course of penicillin (or erythromycin for penicillin-hypersensitive patients) and a single dose of gentamicin or a course of chloramphenicol, together with a booster dose of tetanus toxoid is recommended. Interference with the wound (incisions made with an unsterilised razor blade/knife etc) creates a risk of secondary bacterial infection and justifies the use of broad spectrum antibiotics (eg amoxycillin or a cephalosporin plus a single dose of gentamicin plus metronidazole).

Compartmental syndromes and fasciotomy

The appearance of an immobile, tensely-swollen, cold and apparently pulseless snake-bitten limb may suggest to surgeons the possibility of increased intracompartmental pressure, especially if the digital pulp spaces or the anterior tibial compartment are involved. Swelling of envenomed muscle within such tight fascial compartments could result in an increase in tissue pressure above the venous pressure, resulting in ischaemia. However, the classical signs of an intracompartmental pressure syndrome may be difficult to assess in snake bite victims.

Clinical features of a compartmental syndrome

- Disproportionately severe pain
- Weakness of intracompartmental muscles
- Pain on passive stretching of intracompartmental muscles
- Hypoaesthesia of areas of skin supplied by nerves running through the compartment
- Obvious tenseness of the compartment on palpation

Detection of arterial pulses by palpation or doppler ultrasound probes, does not exclude intracompartmental ischaemia. The most reliable test is to measure

Figure 44: Disastrous results of unnecessary fasciotomy in snake bite victims (a) profuse bleeding in a patient with mild local envenoming but severe coagulopathy following a bite by a green pit viper (Trimeresurus albolabris) (Copyright Sornchai Looareesuwan). Disastrous results of unnecessary fasciotomy in snake bite victims (b) Persistent bleeding for 10 days, resulting in haemorrhagic shock despite transfusion of 20 unites of blood, in a victim of Malayan pit viper bite in whom fasciotomy was performed before adequate antivenom treatment had been given to correct the coagulopathy (Copyright DA Warrell). (Right) Disastrous results of unnecessary fasciotomy in snake bite victims (c) Residual skin loss and exposure of tendons following fasciotomy for mild local envenoming in a patient bitten by a green pit viper (Trimeresurus albolabris) (Copyright Sornchai Looareesuwan)

intracompartmental pressure directly through a cannula introduced into the compartment and connected to a pressure transducer or manometer (Annex 5). In orthopaedic practice, intracompartmental pressures exceeding 40 mmHg (less in children) may carry a risk of ischaemic necrosis (eg Volkmann's ischaemia or anterior tibial compartment syndrome). However, fasciotomy should not be contemplated until haemostatic abnormalities have been corrected, otherwise the patient may bleed to death (Fig 44). Animal studies have suggested that muscle sufficiently envenomed and swollen to cause intracompartmental syndromes, may already be irreversibly damaged by the direct effects of the venom. Early treatment with antivenom remains the best way of preventing irreversible muscle damage.

Criteria for fasciotomy in snake-bitten limbs

Haemostatic abnormalities have been corrected (antivenom with or without clotting factors)

- clinical evidence of an intracompartmental syndrome
- intracompartmental pressure >40 mmHg (in adults)

4.9 Rehabilitation

Restoration of normal function in the bitten part after the patient has been discharged from hospital is not usually supervised. Conventional physiotherapy may well accelerate this process. In patients with severe local envenoming, the limb should be maintained in a functional position. For example, in the leg, equinus deformity of the ankle should be prevented by application of a back slab.

5

Management of Cobra Spit Ophthalmia

First aid consists of irrigating the affected eyes and other mucous membranes with liberal quantities of water or any other available bland liquid. Instillation of 0.5% adrenaline drops relieves pain and inflammation. In view of the risk of corneal abrasion, fluorescein staining or slit lamp examination is essential. Otherwise, topical antimicrobials (tetracycline or chloramphenicol) should be applied to prevent endophthalmitis or blinding corneal opacities. Some ophthalmologists recommend the use of a dressing pad to close the eye.

The instillation of diluted antivenom may cause local irritation and is of uncertain benefit. It is not recommended.

6

Conclusions and Main Recommendations

1. It is clear that in many parts of the South East Asian region, snake bite is an important medical emergency and cause of hospital admission. It results in the death or chronic disability of many active younger people, especially those involved in farming and plantation work. However, the true scale of mortality and acute and chronic morbidity from snake bite remains uncertain because of inadequate reporting in almost every part of the region.

To remedy this deficiency, it is strongly recommended that snake bite should be made a specific notifiable disease in all countries in the South East Asian region.

2. Snake bite is an occupational disease of farmers, plantation workers, herdsmen, fishermen and other food producers. It is therefore a medical problem that has important implications for the nutrition and economy of the countries where it occurs commonly.

It is recommended that snake bite should be formally recognised as an important occupational disease in the South East Asian region.

3. Despite its importance, there have been fewer proper clinical studies of snake bite than of almost any other tropical disease. Snake bites probably cause more deaths in the region than do *Entamoeba histolytica* infections but only a small fraction of the research investment in amoebiasis has been devoted to the study of snake bite.

It is recommended that governments, academic institutions, pharmaceutical, agricultural and other industries and other funding bodies, should actively encourage and sponsor properly designed clinical studies of all aspects of snake bite.

4. Some ministries of health in the region have begun to organise training of doctors and other medical workers in the clinical management of snake bite patients. However, medical personnel throughout the region would benefit from more formal instruction on all aspects of the subject. This should include the identification of medically-important species of snake, clinical diagnosis and the appropriate use of antivenoms and ancillary treatments.

It is recommended that education and training on snake bite should be included in the curriculum of medical schools and should be addressed specifically through the organisation of special training courses and other educational events.

5. Community education on snake bite is outside the terms of eference of this publication. However, it is clear that this is an essential component of any community programme for prevention of snake bite.

Community education about venomous snakes and snake bite is strongly recommended as the method most likely to succeed in preventing bites.

6. Most of the familiar methods for first-aid treatment of snake bite, both western and "traditional/herbal", have been found to result in more harm (risk) than good (benefit). Their use should be discouraged and they should never be allowed to delay the movement of the patient to medical care at the hospital or dispensary.

Recommended first-aid methods emphasise reassurance, immobilisation of the bitten limb and movement of the patient to a place where they can receive medical care as soon as possible.

7. Diagnosis of the species of snake responsible for the bite is important for optimal clinical management. This may be achieved by identifying the dead snake or by inference from the "clinical syndrome" of envenoming.

A syndromic approach should be developed for diagnosing the species responsible for snake bites in different parts of the region.

8. Antivenom is the only effective antidote for snake venom. However, it is usually expensive and in short supply and its use carries the risk of potentially dangerous reactions.

- It is recommended that antivenom should be used only in patients in whom the benefits of treatment are considered to exceed the risks. Indications for antivenom include signs of systemic and/or severe local envenoming.
- Skin/conjunctival hypersensitivity testing does not reliably predict early or late antivenom reactions and is not recommended.

- It is recommended that whenever possible antivenom should be given by slow intravenous injection or infusion.
- Epinephrine (adrenaline) should always be drawn up in readiness in case of an early anaphylactic antivenom reaction.
- Subcutaneous epinephrine (adernaline) may reduce the incidence of early antivenom reactions if given immediately before the start of antivenom treatment.

9.

When no antivenom is available, judicious conservative treatment can in many cases save the life of the patient.

10.

In the case of neurotoxic envenoming with bulbar and respiratory paralysis, antivenom alone cannot be relied upon to prevent early death from asphyxiation. Artificial ventilation is essential in such cases.

11.

Conservative management and, in some cases, dialysis, is an effective supportive treatment for acute renal failure in victims of Russell's viper, saw-scaled viper and sea snake bites.

12.

Fasciotomy should not be carried out in snake bite patients unless or until haemostatic abnormalities have been corrected, clinical features of an intracompartmental syndrome are present and a high intracompartmental pressure has been confirmed by direct measurement.

7

Further Reading

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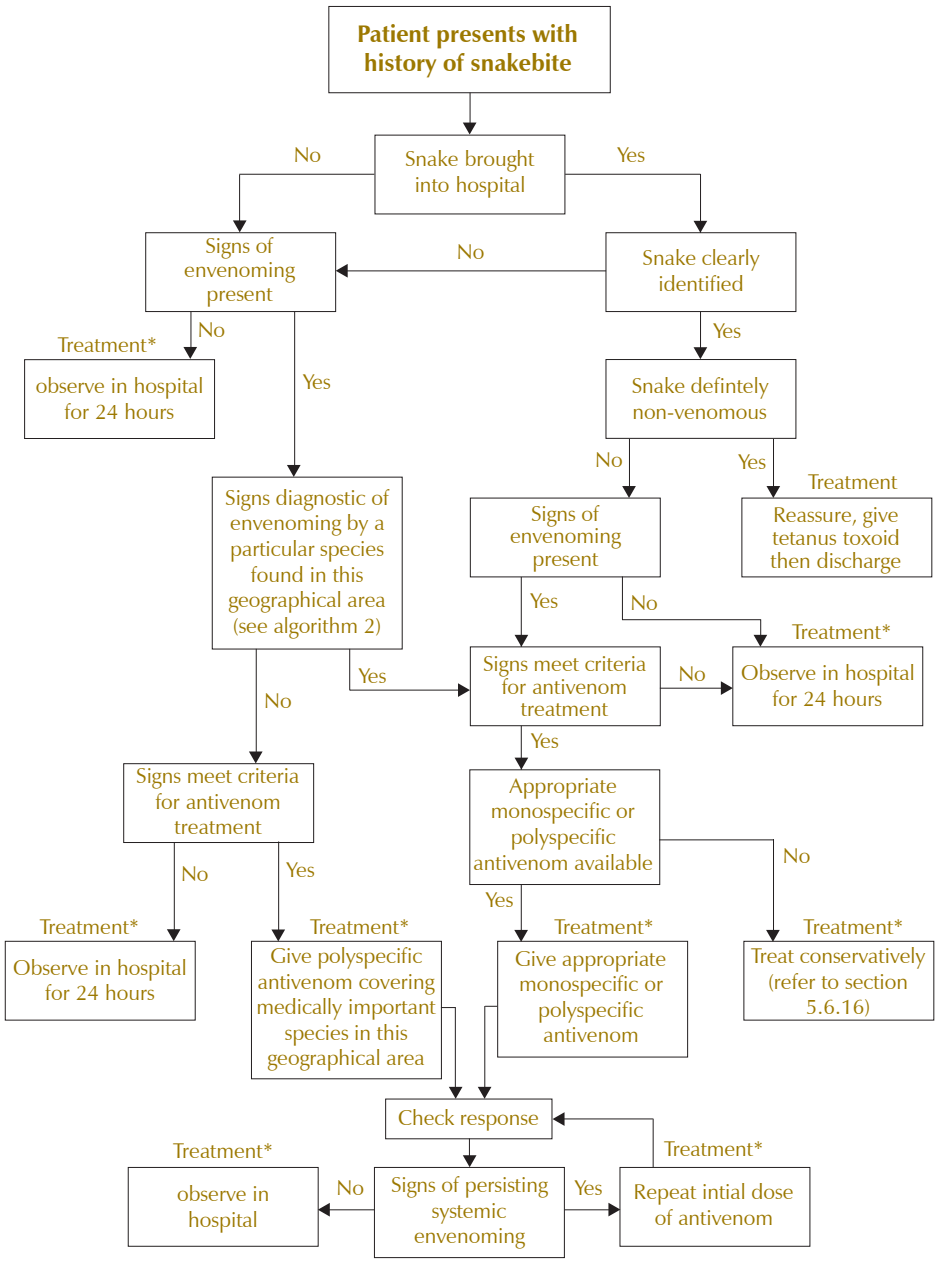
Annex 1

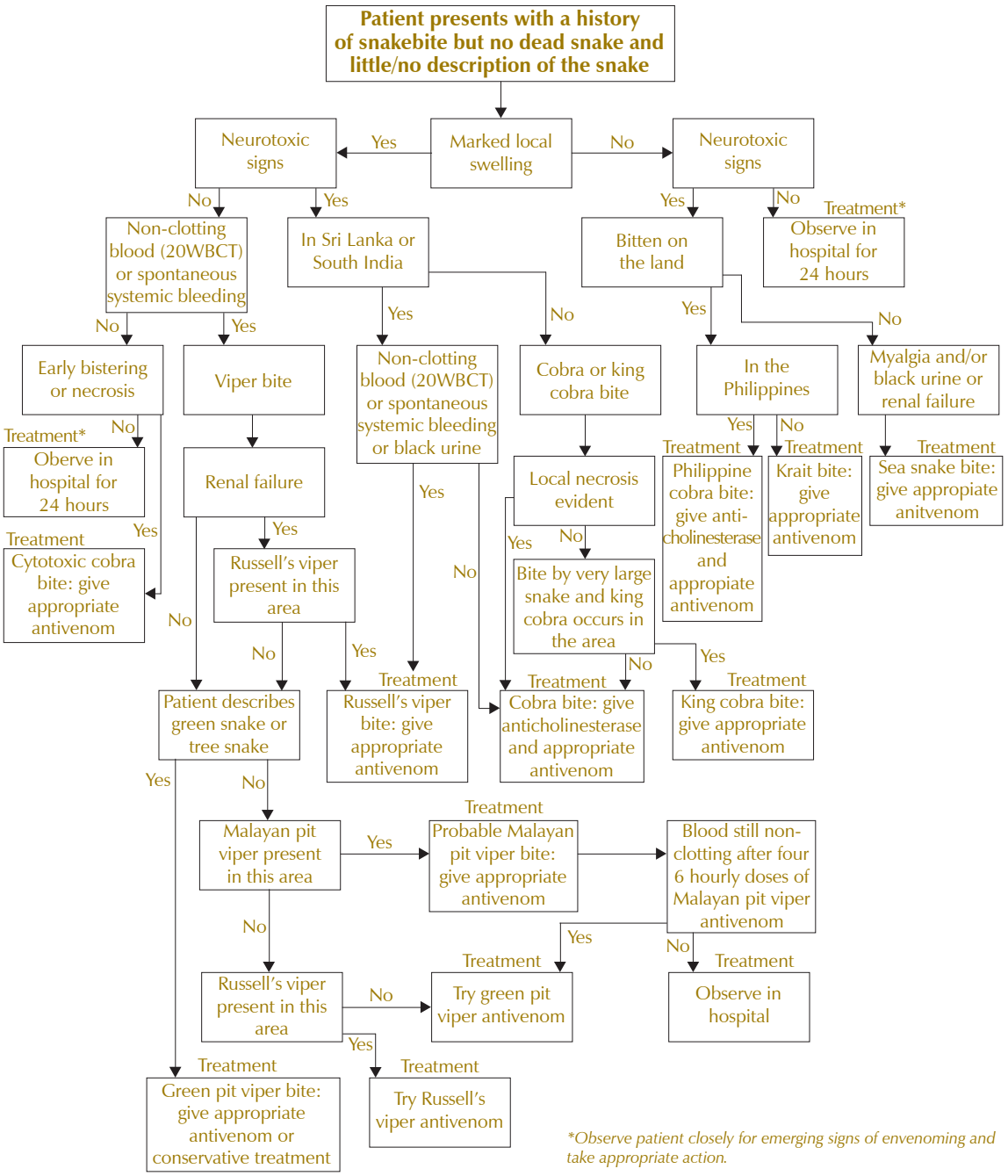
Algorithm: Antivenom Treatment of Snakebite Cases



**Patients must be assessed carefully for signs of emerging or recurrent envenoming and appropriate action taken.*

Annex 2

Algorithm: Differentiating Major Asian Snake Species by Clincal Syndrome



**Observe patient closely for emerging signs of envenoming and take appropriate action.*

Annex 3

Antivenoms for Treatment of Bites by South-East Asian Snakes

(List by Country of Manufacture)

1. China

Shanghai Institute of Biological Products, Ministry of Health, 1262 Yan An Road (W), Shanghai 200052, China (Tel ++ 8621-62803189; Fax ++ 8621-62801807). Contact: Ms Minzhi Lu, Manager, International Affairs & Trade Department (Tel ++ 8621-62805234)

(liquid antivenoms, 10-15 ml/ampoule)

- *Agkistrodon acutus* antivenin (purified) (= *Deinagkistrodon acutus*, found in North Viet Nam). Recommended dose 8,000 IU (= 4 ampoules)
- "*Agkistrodon halys*" antivenin (purified) (said to be active against venoms of *Trimeresurus mucrosquamatus* and *T stejnegeri*). Recommended dose 6,000 IU (= 1 ampoule)
- *Bungarus multicinctus* antivenin (purified) (said to be effective against the venom of *Ophiophagus hannah*). Recommended dose for bites by both species 10,000 IU (= 1.25 ampoules)
- "*Naja naja*" antivenom (purified) (= *Naja atra*). Recommended dose 2,000 IU (= 2 ampoules)

2. Germany

Knoll AG, Postfach 21 08 05, 67008 Ludwigshafen, Germany (Tel ++ 49621-5892688; Fax ++ 49621-5893707). Contact: Mr Lok, Managing Director

(liquid antivenom, 10 ml/ampoule)

- Cobra monospecific antivenom (*Naja naja sputatrix* = Malaysian *N sumatrana*)

3. India

(a) Bengal Chemicals & Pharmaceuticals, 6 Ganesh Chunder Avenue, Calcutta (Fax ++91 33 2257697)
(liquid antivenom)

- Polyvalent (*Bungarus caeruleus*, *Naja naja, Vipera russelli, Echis carinatus*)

(b) Central Research Institute (Simla Hills), 173 204 (HP) Kasauli (Tel ++ 91-17932060; Fax ++ 91-179272049)

(liquid and lyophilised antivenoms, 10 ml/ampoule)

- Polyvalent (*B caeruleus, N naja, V russelli, E carinatus*)

(c) Haffkine Biopharmaceutical Company Ltd, Acharya Donde Marg, Parel, Bombay 400012 (Tel ++ 91-224129320 and 234129224; Fax ++ 91 41 68578; Telex 11.71427 HBPC IN)

(lyopholised antivenoms, 10 ml/ampoule)

- Polyvalent anti-snake venom serum (*B caeruleus, E carinatus, N naja, V russelli*)

(d) King's Institute of Preventive Medicine, Guindi, Madras NA5

- Polyvalent

(e) Serum Institute of India Ltd, 212/2 Hadapsar, Pune-411 028 (Tel ++ 91-212672016; Fax ++ 91-212672040; Telex 145-7317 SERA IN, 145-7216 SEAL IN) Contact: Dr SS Jadhav, Executive Director (QA)

(lyopholized antivenoms)

- Polyvalent (*B caeruleus, N naja, V russelli, E carinatus*)
- Bivalent (*E carinatus, V russelli*)

4. Indonesia

Perum Bio Farma (Pasteur Institute), Jl Pasteur 28, Post Box 1136, Bandung 40161 (Tel ++ 6222-83755; Fax ++ 6222-210299; Telex 28432 BIOFAR IA)

(liquid antivenom, 5 ml/ampoule)

- Polyvalent antivenom serum (*Calloselasma rhodostoma, B fasciatus, N sputatrix*)

5. Iran

State Serum & Vaccine Institute, Razi Hessarek, bP 656, Teheran (Tel ++ 98 2221 2005)

(liquid antivenoms, 10 ml/ampoule)

- Polyvalent snake antivenom (equine) (said to neutralise the venoms of two South-East Asian species – *Naja oxiana* and *Echis carinatus* (probably *E sochureki*), *Vipera lebetina* (= *Macrovipera lebetina*) and *Pseudocerastes persicus*

6. Myanmar (Burma)

Myanmar Pharmaceutical Factory, Yangon

(lyophilized and liquid antivenoms, 10 ml/ampoule)

- Viper antivenom (*V russelli*)
- Cobra antivenom (*N kaouthia*)

7. Pakistan

National Institute of Health, Biological Production Division, Islamabad (Tel ++ 9251-240946; Fax ++ 9251-20797; Telex 5811-NAIB-PK) Contact: Shahid Akhtar
(liquid and lyophilized antivenoms, 10 ml/ampoule)

- Polyvalent anti-snake venom serum (*B caeruleus, E carinatus, N naja, V lebetina, V russelli*)

8. Philippines

Biologicals Production Service, Dept of Health, Los Baños, Laguna
(liquid antivenom)

- Philippine cobra antivenin (*Naja philippinensis*)

9. Taiwan

National Institute of Preventive Medicine, 161 Kun-Yang Street, Nan-Kang, Taipei, ROC 11513 (Tel ++ 8862-7859215; Fax ++ 8862-7853944). Contact: Dr Gong-Ren Wang, Director
(lyophilised antivenoms, 10 ml/ampoule)

- *Bungarus multicinctus* and *N atra* bivalent antivenom
- *Trimeresurus muquosquamatus* and *Trimeresurus grammineus* (= *T stejnegeri*) bivalent antivenom
- *Agkistrodon acutus* (= *Deinagkistrodon acutus*) antivenom

10. Thailand

The Thai Red Cross Society, Queen Saovabha Memorial Institute, 1871 Rama VI Road, Bangkok 10330 (Tel ++ 662-2520161-4; Fax ++ 662-2540212; Telex 82535 THRESCO TH)
(freeze dried monovalent antivenoms, 10 ml/ampoule)

- Cobra antivenom
- King cobra antivenin
- Banded krait antivenin
- Russell's viper antivenin
- Malayan pit viper antivenin
- Green pit viper antivenin

Annex 4

Measurement of Central Venous Pressure

In seriously ill patients with shock or renal failure in whom clinical assessment of the jugular venous pressure is difficult or considered inaccurate, a central venous catheter should be inserted percutaneously. In those with no haemostatic problems, a catheter may be inserted into the jugular or subclavian vein provided adequate facilities for a sterile procedure and subsequent nursing are available. However, patients who have been bitten by vipers may have obvious haemostatic problems or may develop coagulopathy. In these cases, the antecubital approach is by far the safest as haemostasis can be achieved by local pressure. A long catheter (at least 50-70 cm for an adult) is required (Fig 45). The catheter is connected via a three-way tap and pressure tubing to a manometer. The whole system is filled with sterile isotonic saline. Before readings can be taken, the zero on the manometer must be aligned as accurately as possible with the horizontal plane of the left atrium. A simple spiritlevel (eg a 20 ml glass ampoule with bubble, taped to a ruler) can be used to locate the manometer zero at the same height as an appropriate chest-wall landmark, such as the midaxillary line, in the supine patient (Fig 46) or the sternal angle in a patient sitting up at 45°. There should be strict attention to asepsis. Infection and thrombosis are potential complications; especially if the catheter remains in place for a long time.

Figure 45: Central venous pressure monitoring in a patient with shock after Russell's viper bite, in a township hospital in rural Myanmar. A 70 cm long catheter was inserted into an antecubital vein (Seldinger percutaneous guidewire technique) and advanced until its tip was in the superior vena cava. An extension tube connects with a simple saline manometer whose zero point is at the level of the mid-axillary line (Copyright DA Warrell)

Figure 46: Adjusting the zero point of the central venous pressure manometer to the mid-axillary line, using a home-made ruler-plus-glass-ampoule "spirit level" (Copyright DA Warrell)

Annex 5

Measurement of Intracompartmental Pressure in Tensely Swollen Snake-bitten Limbs

To confirm a clinical suspicion of intracompartmental syndrome (see Compartmental syndromes and fasciotomy, page 46) the pressure inside the particular compartment should be measured directly. The threshold pressure required to initiate the flow of liquid into the fascial compartment is a measure of the tissue pressure inside that compartment. With full sterile precautions and after infiltrating local anaesthetic, a 21 or 22 gauge cannula, approximately 3-4 cm long, is inserted into the compartment through or around an introducing 20 or 21 gauge needle. The cannula is connected through narrow pressure tubing to a syringe or low speed infusion pump. Through a three-way tap, the system is connected, through a side arm to a blood pressure transducer or saline or mercury manometer (Fig 47). The system is filled with sterile isotonic saline. If a syringe-type infusion pump and arterial blood pressure transducer with monitor is used, the pressure can be measured continuously at a very slow rate of infusion (eg 0.7 ml/day). If a saline or mercury manometer is used, a much higher rate of infusion is required to initiate flow into the compartment. These systems are not suitable for continuous intracompartmental pressure monitoring.

Alternatively, the simple but expensive Stryker pressure monitor can be used (Fig 48). Whatever system is employed, the zero point in the pressure measuring device must be aligned to the level at which the cannula enters the fascial compartment.

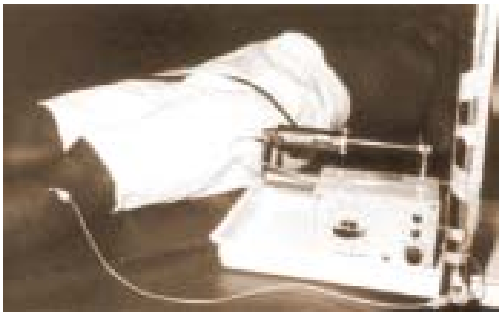


Figure 47: Infusion pump, saline manometer system in use for measuring the tissue pressure inside the anterior tibial compartment (Copyright DA Warrell)

Figure 48: Stryker pressure monitor in use for measurement of intracompartmental pressure (Copyright DA Warrell)

World Health Organization

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