5. Management of snake bites in Southeast Asia

The following steps or stages are often involved

Management of snake bite

5.1 First aid treatment
5.2 Transport to hospital
5.3 Rapid clinical assessment and resuscitation
5.4 Detailed clinical assessment and species diagnosis
5.5 Investigations/laboratory tests
5.6 Antivenom treatment
5.7 Observation of the response to antivenom:
   decision about the need for further dose(s) of antivenom
5.8 Supportive/ancillary treatment
5.9 Treatment of the bitten part
5.10 Rehabilitation
5.11 Treatment of chronic complications

5.1 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 (5.2)

● **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

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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!**

5.1.1 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.
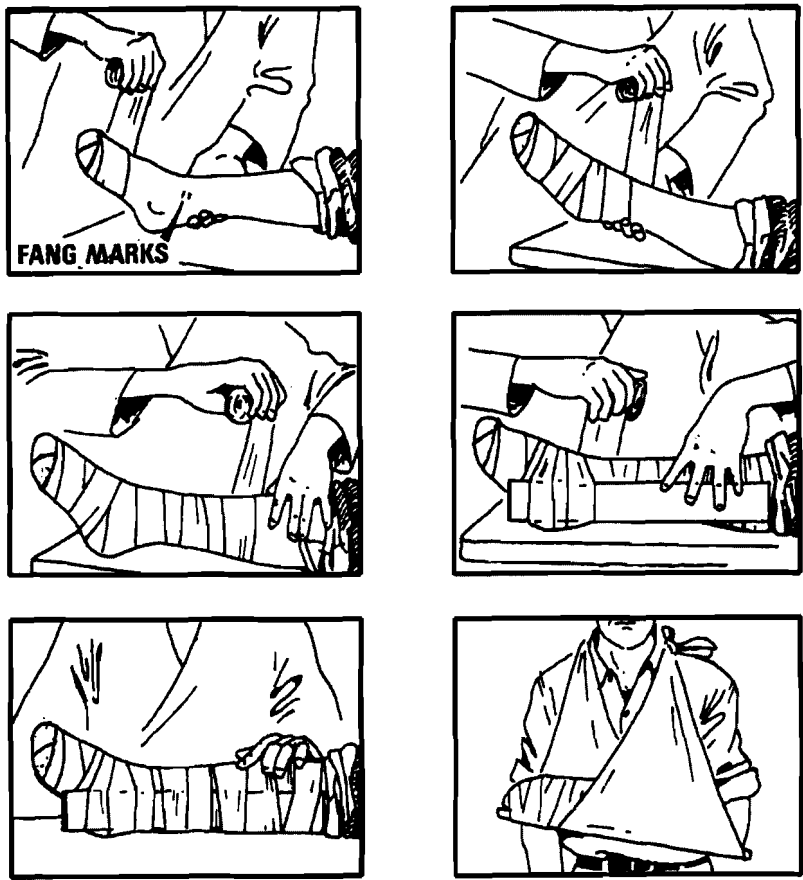

Figure 39 : Pressure immobilisation method. Recommended first-aid for bites by neurotoxic elapid snakes. *(by courtesy of the Australian Venom Research Unit, University of Melbourne)*

> **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)**.

5.1.2 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 !**

> ## ARTERIAL TOURNIQUETS ARE NOT RECOMMENDED

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

5.1.3 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.

5.2 Transport to hospital

Patients 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.

5.3 Treatment in the dispensary or hospital

5.3.1 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 :

    (a) Profound hypotension and shock resulting from direct cardiovascular effects of the venom or secondary effects such as hypovolaemia or haemorrhagic shock.

    (b) Terminal respiratory failure from progressive neurotoxic envenoming that has led to paralysis of the respiratory muscles.

    (c) Sudden deterioration or rapid development of severe systemic envenoming following the release of a tight tourniquet or compression bandage **(see Caution above).**

    (d) Cardiac arrest precipitated by hyperkalaemia resulting from skeletal muscle breakdown (rhabdomyolysis) after sea snake bite.

    (e) Late results of severe envenoming such as renal failure and septicaemia complicating local necrosis.

5.4 Detailed clinical assessment and species diagnosis

5.4.1 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 venepuncture sites and 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.

5.4.2 Physical examination

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

5.4.2.1 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).

5.4.2.2 General examination

Measure the blood pressure (sitting up and lying to detect a postural drop indicative of hypo-volaemia) 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 spontane-ous 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).

5.4.2.3 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 ophthalmo-plegia (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 mus-cles 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).

5.4.2.4 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" (abdo-

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)*

men 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 (FEV1 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.

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

5.4.2.5 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.

5.4.2.6 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.

5.4.3 Species diagnosis

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

5.5   Investigations/laboratory tests

5.5.1   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).

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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 hypofibrinogenaemia ("incoagulable blood") as a result of venom-induced consumption coagulopathy

- In the Southeast 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)

5.5.2   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 cells", schistocytes) are seen when there is micro-angiopathic haemolysis.

Plasma/serum may be pinkish or brownish if there is gross haemoglobinaemia or myo-globinaemia.

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* renal 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. Micros copy 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.

5.6 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.**

5.6.1 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 the 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 of one 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.

5.6.2 Indications for antivenom treatment (see also Annex 1 and 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.

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Indications for antivenom

Antivenom treatment is recommended if and when a patient with proven or suspected snake bite 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 10^9/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 **5.5**)

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

5.6.3 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.**

5.6.4 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.

5.6.5 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.**

5.6.6 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.

5.6.6.1 Prevention of antivenom reactions

Recent studies have indicated that, while intramuscular promethazine is ineffective, subcutaneous epinephrine (adrenaline) (0.1% solution, adult dose 0.25 mg given immediately before the start of antivenom treatment) reduces the incidence of early antivenom reactions. In asthmatic patients, prophylactic use of an inhaled adrenergic β_2 agonist such as salbutamol may prevent bronchospasm.

5.6.7   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 antivenom.

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.

5.6.8   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

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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. This method has the advantage that intravenous access is available for the emergency treatment of reaction.

> **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).**

5.6.8.1 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.

5.6.8.2 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 :

1) 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;
2) on an expedition exploring a remote area very far from medical care;
3) 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.

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.

5.6.9 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.

5.6.10 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 hyper-sensitivity 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 adrenaline, antihistamines and corticosteroid are less likely to develop late reactions.

5.6.11 Treatment of early anaphylactic and pyrogenic antivenom reactions

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**

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.

5.6.11.1 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.

5.6.12 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

5.6.13 Observation of the response to antivenom

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

    (a) *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.

    (b) *Spontaneous systemic bleeding* (*eg* from the gums) usually stops within 15-30 minutes.

    (c) *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.

    (d) *In shocked patients*, blood pressure may increase within the first 30-60 minutes

and arrhythmias such as sinus bradycardia may resolve.

(e) *Neurotoxic envenoming* of the post-synaptic type (cobra bites) may begin to improve as early as 30 minutes after antivenom, but usually takes several hours. Envenoming with pre-synaptic toxins (kraits and sea snakes) is unlikely to respond in this way.

(f) *Active haemolysis and rhabdomyolysis* may cease within a few hours and the urine returns to its normal colour.

5.6.14 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 35-70 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.

5.6.15 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 mean 6 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.

5.6.16 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 **5.7.2.1**).

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 below **5.7.4**).

Dark brown urine (myoglobinuria or haemoglobinuria) : correct hypovolaemia and acidosis and consider a single infusion of mannitol (see below **5.7.4.2**).

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 life-threatening complications of local envenoming. Prophylactic broad spectrum antimicrobial treatment is justified (see below **5.8.1**).

5.7 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.

5.7.1 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.

5.7.2 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 effects 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.

5.7.2.1 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) or glycopyrronium bromide (adults 200 µg children 4 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, FEV_1 or maximum expiratory pressure) may improve.

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

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. Maintenance doses are atropine sulphate 50 µg/kg, neostigmine methylsulphate 50-100 µg/kg both by subcutaneous injection every four hours. Patients able to swallow tablets may be maintained on atropine 0.6 mg twice each day, neostigmine 15 mg four times each day or pyridostigmine 60 mg four times each day (inital adult doses).

5.7.3 Hypotension and shock

Snake bite: causes of hypotension and shock

1° Anaphylaxis
     Vasodilatation
     Cardiotoxicity
     Hypovolaemia

2° Antivenom reaction
     Respiratory failure
     Acute pituitary adrenal insufficiency
     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 pulmonary oedema when plasma extravasated in the bitten limb and elsewhere is reabsorbed into the circulation.

In patients with hypotension and 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.

5.7.4 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

5.7.4.1 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

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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 45°). 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" (500-1,000 ml/day). If possible, the patient should be referred to a renal unit. The diet should be bland, high in calories (1,700/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)**

(a)   give 10 ml of 10% calcium gluconate intravenously over 2 minutes (with ECG monitoring if possible) repeated up to three times

(b)   give 50 ml of 50% dextrose with 10 units of soluble insulin intravenously

(c)   sodium bicarbonate (40 ml of 8.4%) by slow intravenous infusion and a β_2 agonist aerosol by inhaler (*eg* salbutamol - "Ventolin" 5-10 mg) may also be used

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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

(a) Clinical uraemia
(b) Fluid overload
(c) 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

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5.7.4.2 Prevention of renal damage in patients with myoglobinuria or haemoglobinuria

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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)

5.7.4.3 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.

5.7.4.4 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.

5.7.4.5 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.

5.7.5 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, imminent urgent surgery, once specific antivenom has been given to neutralise venom procoagulants parturition or and other anti-haemostatic toxins, restoration of blood coagulability and platelet function can be accelerated

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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.

5.8 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.

5.8.1 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).

5.8.2 Necrosis (gangrene)

Once definite signs of necrosis have appeared (hyper-/hypo-pigmented, anaesthetic, demarcated area of skin with putrid odour or signs of sloughing), immediate thorough surgical débridement, split skin grafting and broad-spectrum antimicrobial cover are indicated. Bruised or haemorrhagic tissues can easily be mistaken for areas of necrosis by the inexperienced; débridement and amputation should be approached with caution in snake bite patients.

5.8.3 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

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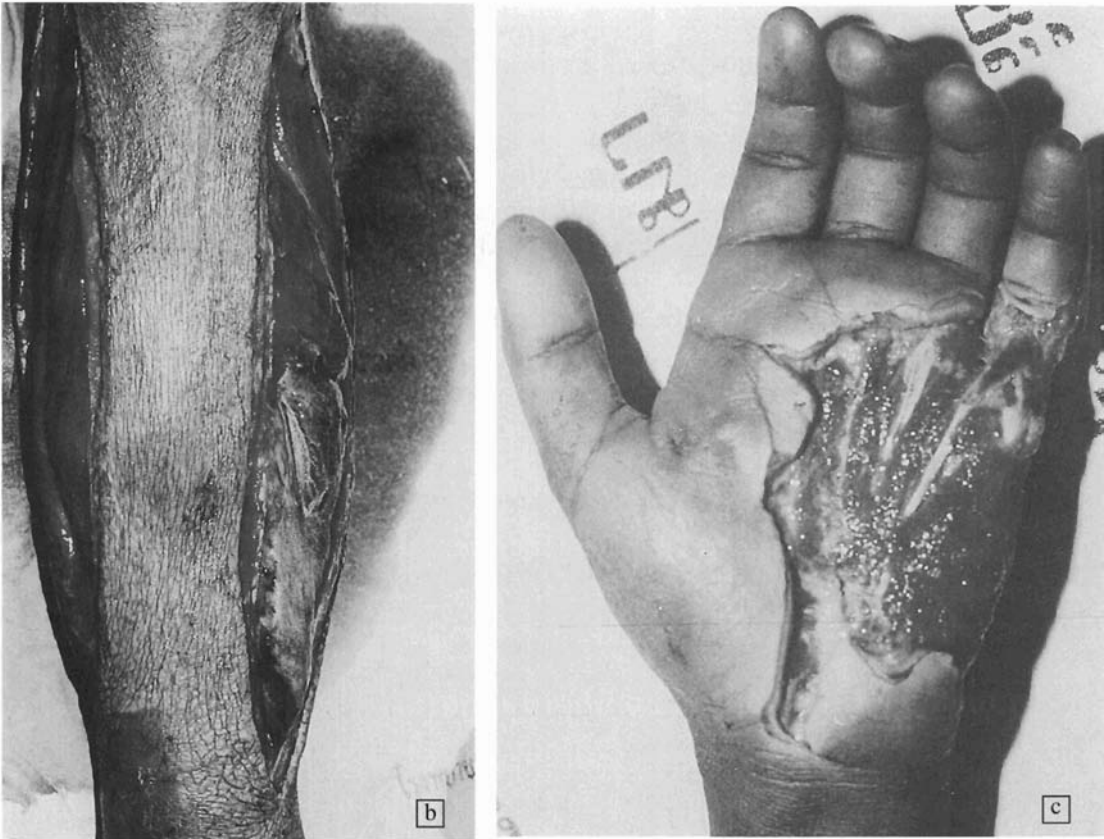
Figure 44b, c : Disastrous results of unnecessary fasciotomy in snake bite victims; (Left b) Persistent bleeding for 10 days, resulting in haemorrhagic shock despite transfusion of 20 units of blood, in a victim of Malayan pit viper bite in whom fasciotomy was performed before adequate antivenom treatment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

Detection of arterial pulses, by palpation or doppler ultrasound probes, does not exclude intracompartmental ischaemia. The most reliable test is to measure 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**
> **All three must be present**
>
> - haemostatic abnormalities have been corrected
>   (antivenom with or without clotting factors)
>
> - clinical evidence of an intracompartmental syndrome
>
> - intracompartmental pressure >40 mmHg (in adults)

5.9 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.

6. 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.

7. Conclusions and main recommendations

1. It is clear that in many parts of the Southeast 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 Southeast Asian region.**