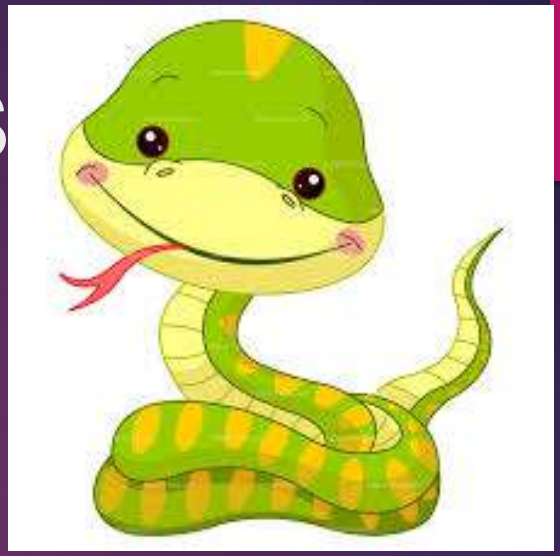
Snake bites

BY DR. SWATHI SWAROOPA. B

Contents

- Introduction
- Families of venomous snakes
- Clinical effects of venoms
- Early manifestations
- Complications and snake bite injuries
- General management as first aid

Introduction about SNAKES



- Limbless creatures
- Body is divided into head, trunk, and tail.
- Head bears two eyes, two nostrils, and a mouth
- External ears are absent since snakes do not posses auditory apparatus

Introduction about SNAKES

- it senses vibrations on the ground
- The mouth of a snake is extremely distensible
- Snakes are essentially carnivorous
- Some are cannibals and feed on other snakes, e.g. krait, king cobra, etc
- Survive for long periods of time without food, even up to several months or years

- They have four rows of teeth in the upper jaw, and two rows of teeth in the lower jaw.
- Venomous snakes have modified teeth called fangs in addition to ordinary teeth
- Fangs are usually two in number, invariably located one on each side of the upper jaw and connected to the venom glands.
- Tongue of a snake is forked and used to pick up scent particles from the exterior and transfer them to the Jacobson's organ in the roof of the mouth.

- The nostrils enable a snake to inhale air into its lungs.
- Hissing is accomplished by the forcible expulsion of air through the nostrils, and is an act of aggression or defence
- In some snakes, there is a pit between the eye and nostrils, which is a heat sensitive organ that helps in detecting warm blooded prey.
- Venomous snakes are found all over the world, except New Zealand, and most parts of the Arctic and Antarctic regions, as well as Ireland, Iceland, Chile, Hawaii, parts of Mediterranean and Caribbean regions, and some of the Pacific islands.

- Snakes are cold-blooded creatures, and their body temperature varies depending on the environmental conditions.
- Snakes regularly moult usually every 2 months
- Moulting enables a snake to become more alert and active.
- Most snakes lay eggs (oviparous) while a few species bring forth their young alive (viviparous).
- Snakes usually survive for many years, a few species living up to 20 years or more

Fangs

Classification of snakes

Snakes are classified on the basis of morphological characteristics

- Arrangement of scales
- Dentition
- Osteology,
- Myology,
- Sensory organs,
- Form of the hemipenes,
- Immunological analysis of venom and serum proteins, and
- Sequence analysis of DNA that encodes mitochondrial and other enzymes.

Venomous snakes belong to 5 families:

- **Colubridae**: Mountain racer, Western and Eastern hognose snakes, parrot snake, rat snake, wandering garter snake
- **Atractaspididae**: Eastern burrowing asps or stilleto snakes (also known as burrowing or mole vipers or adders, false vipers, side-stabbing snakes)
- **Elapidae**: a. Cobras (Naja) b. Kraits (Bungarus) c. Coral snakes (Calliophis, Maticora, Micrurus) d. Mambas (Dendroaspis)
- **Viperidae**: a)Viperinae or true vipers: Vipers and adders

  b) Crotalinae or pit vipers: Rattle snakes (Crotalus,Sistrurus), and Asian pit vipers (Trimeresurus,Hypnale).
- **Hydrophidae**: Sea snakes

- Approximately 330 species of snakes exist in India, of which about 70 species are venomous

The commonest Indian venomous snakes are referred to as the “Big Four”, and comprise

- Common krait (Bungarus caeruleus),
- Common cobra (Naja naja),
- Saw-scaled viper (Echis carinatus), and
- Russell’s viper (Vipera russelli).

Krait

Cobra

Saw-scaled viper

Russell's viper

FACTS

Snake bite

Majority (80%) is by non-venomous snakes

Venomous snakes

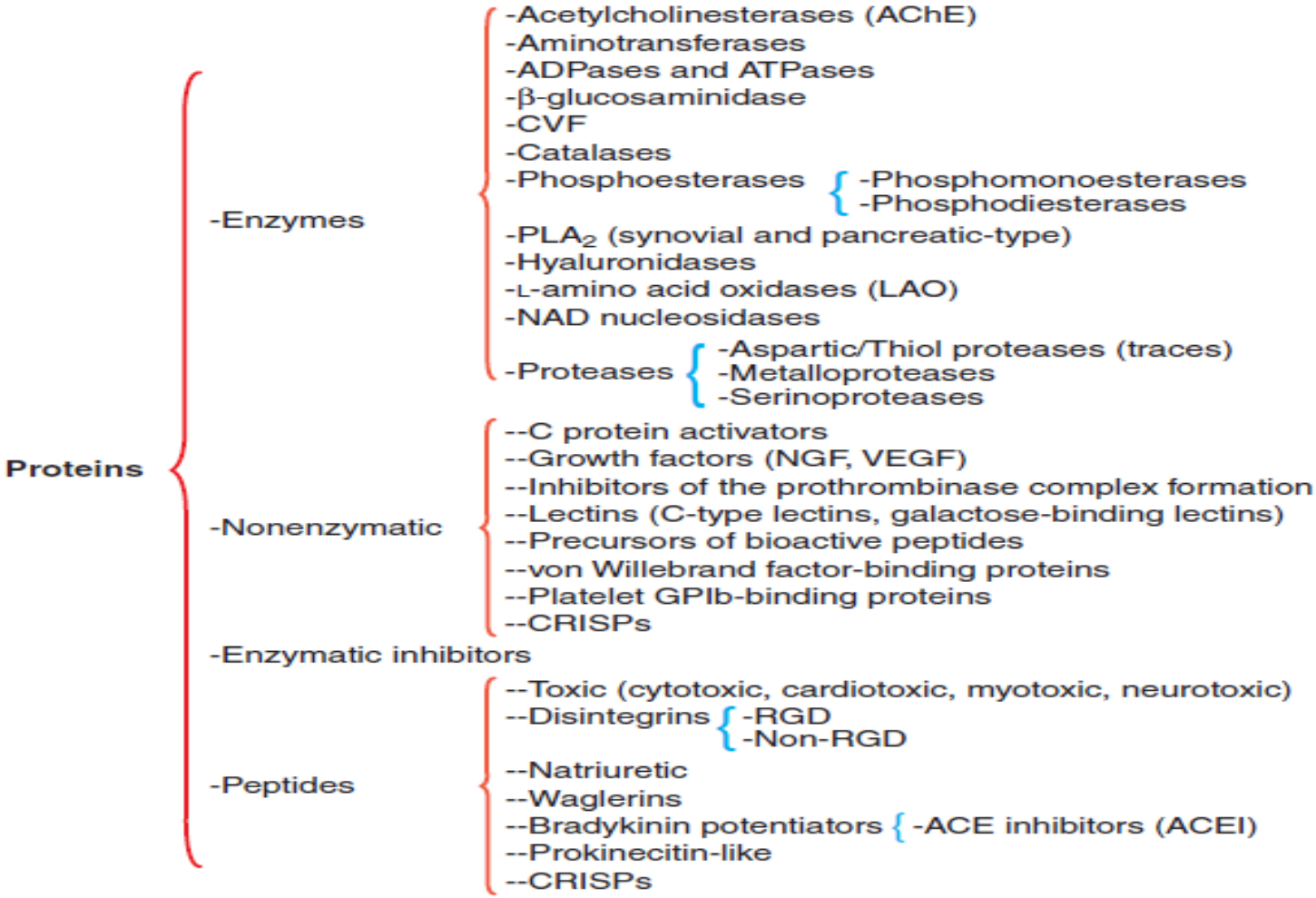
About 50% of bites are dry

Common Indian Venomous Snakes

The WHO classifies the following as Indian Snakes of Medical Importance:

- Class I - Commonly cause death or serious disability: Cobra/ Russells Viper/Saw-Scaled Viper
- Class II - Uncommonly cause bites but are recorded to cause serious effects (death or local necrosis): Krait/King Cobra
- Class III - Commonly cause bites but serious effects are very uncommon.

Venom composition of snakes



Venom composition of snakes

Organic compounds with low molecular mass	-Biogenic amines	-Serotonin, histamine
	-Amino acids	
	-Carbohydrates	
	-Citrate	
	-Nucleosides	
Inorganic compounds	-Calcium	
	-Cobalt	
	-Cooper	
	-Iron	
	-Phosphorus	
	-Potassium	
	-Magnesium	
	-Manganese	
	-Sodium	
	-Zinc	

Venom composition and mechanism

- More than 90% of snake venom (dry weight) is protein
- At least 26 different enzymes have been isolated from snake venoms
- No single snake venom contains all 26 enzymes

Venom composition and mechanism

Grouping the toxin components as

- Neurotoxins,
- Coagulants,
- Hemorrhagins,
- Hemolytics,
- Myotoxins,
- Cytotoxins, and
- Nephrotoxins

Venom composition and mechanism

Neurotoxins:

- Produce neuromuscular paralysis ranging from dizziness to ptosis; to ophthalmoplegia, flaccid facial muscle paralysis, and inability to swallow; to paralysis of larger muscle groups; and finally to paralysis of respiratory muscles and death by asphyxiation.

Coagulants

- May have initial procoagulant action that uses up clotting factors leading to bleeding.
- Anticoagulants may directly inhibit normal clotting at several places in the clotting cascade or via inhibition of platelet aggregation.
- In addition, some venom components may damage the endothelial lining of blood vessels leading to hemorrhage.

Venom composition and mechanism

Myotoxins

- They directly impact muscle contraction leading to paralysis or cause rhabdomyolysis or the breakdown of skeletal muscle.
- Myoglobinuria, or a dark brown urine, and hyperkalemia may be noted.

Cytotoxic agents

- They have proteolytic or necrotic properties leading to the breakdown of tissue.
- Typical signs include massive swelling, pain, discoloration, blistering, bruising, and wound weeping.

Venom composition and mechanism

- Sarafotoxins, which are found only in of Afro-Arabia, cause coronary artery constriction that can lead to reduced coronary blood flow, angina, and myocardial infarction.

Nephrotoxins

- They cause direct damage to kidney structures leading to bleeding, damage to several parts of the nephron, tissue oxygen deprivation, and renal failure.

Venom composition and mechanism

Enzymes

Proteolytic enzymes; Catalyze the breakdown of tissue proteins and peptides associated with marked tissue destruction.

Peptide hydrolases,

Proteases,

Endopeptidases,

Peptidases, and

Proteinases

Venom composition and mechanism

Collagenase is a specific kind of proteinase that digests collagen.

Hyaluronidase cleaves internal glycoside bonds in certain acid mucopolysaccharides resulting in a decrease in the viscosity of connective tissues.

The breakdown in the hyaluronic barrier allows other fractions of venom to penetrate the tissues, causing hyaluronidase to be called “spreading factor

Venom composition and mechanism

fi brin(ogen)olytic enzymes are two types -metalloproteinases and the serine proteinases

Some of the proteinases act on blood as

pro-coagulant or anti-coagulant since they may exert activating or inhibiting effects of plasma factors.

They are also endowed with fibrino(geno) lytic activities as well as thrombin and plasmin.

Venom proteases are divided into two broad classes of enzymes:

Snake Venom Serine Proteases (SVSPs) and

Snake Venom Metalloproteinases (SVMPs)

Venom composition and mechanism

Snake Venom Serine Proteases (SVSPs)

- They are called "Thrombinic enzymes from snake venom" or Snake Venom Thrombin-Like Enzymes (SVTLEs).
- SVSPs target mainly the coagulation cascade and act as potent platelet aggregating molecules.
- These enzymes stimulate blood clotting with formation of fibrin in the blood stream.

- Paradoxically, this process results in incoagulable blood because most of the fibrin clot is broken down immediately by the body's own plasmin fibrinolytic system and, sometimes within 30 minutes of the bite, the levels of clotting factors are so depleted ("consumption coagulopathy") that the blood will not clot.

Venom composition and mechanism

Snake Venom Metalloproteinases (SVMPs) (Zn2+- metalloproteinases)

- Have Procoagulant and anti-coagulant activities,
- Zinc metalloproteinase : Damage vascular endothelium, causing bleeding.
- SVMPs are also involved in the pathogenesis of edema, inflammation, myonecrosis, skin damage and the development of cardiovascular shock
- These are called hemorragins because of systemic bleeding after bites;

Venom composition and mechanism

Procoagulant enzymes:

- Contain serine proteases and other procoagulant enzymes that are thrombin-like or activate factor X, prothrombin and other clotting factors.
- These enzymes stimulate blood clotting with formation of fibrin in the blood stream.
- Paradoxically, this process results in incoagulable blood because most of the fibrin clot is broken down immediately by the body's own plasmin fibrinolytic system and, sometimes within 30 minutes of the bite, the levels of clotting factors are so depleted ("consumption coagulopathy") that the blood will not clot.

Venom composition and mechanism

- Some venoms contain multiple anti-haemostatic factors. For example, Russell's viper venom contains toxins that activate factors V, X, IX and XIII, fibrinolysis, protein C, platelet aggregation, anticoagulation and haemorrhage.

Snake Venom Proteins Active on the Hemostatic System

GENERAL FUNCTIONAL ACTIVITY	SPECIFIC BIOLOGICAL ACTIVITY
Procoagulant	Activates factors II, V, IX, X, and protein C Fibrinogen clotting
Anticoagulant	Factor IX/factor X–binding protein Thrombin inhibitor Phospholipase A
Fibrinolytic	Fibrin(ogen) degradation Plasminogen activation
Vessel wall interactive	Hemorrhagic

SOURCES: Data from Markland (1998) and Russell (2001).

Venom composition and mechanism

- SV-PLA2s (Phospholipase A 2)) induced various biological effects such as neurotoxic, myotoxic, cytolytic, edematic, cardiotoxic and anticoagulant effects

- Acetylcholinesterase: Although found in most elapid venoms, it does not contribute to their neurotoxicity.

Venom composition and mechanism

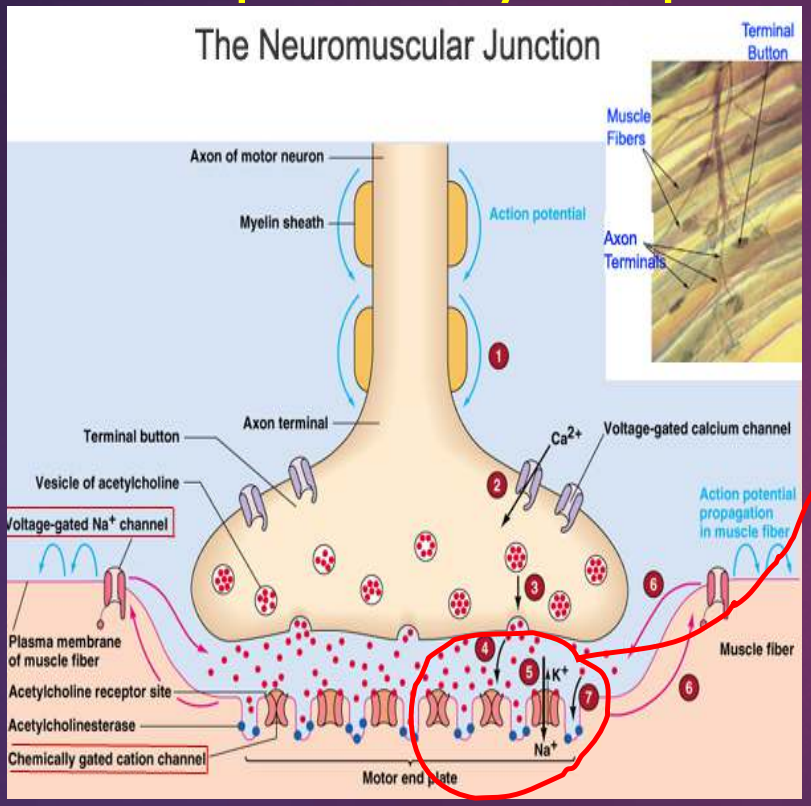
Venom polypeptide toxins (“neurotoxins”)

- Postsynaptic (α) neurotoxins (Elapidae) such as α-bungarotoxin and cobrotoxin, They bind to acetylcholine receptors at the motor endplate and lead to curare-like paralysis.

- Presynaptic (β) neurotoxins (Elapidaeand some Viperidae) such as β-bungarotoxin, crotoxin, and taipoxin, phospholipase A subunit damage nerve endings, initially releasing acetylcholine transmitter, then interfering with release of acetylcholine at the nerve endings at neuromuscular junctions

.

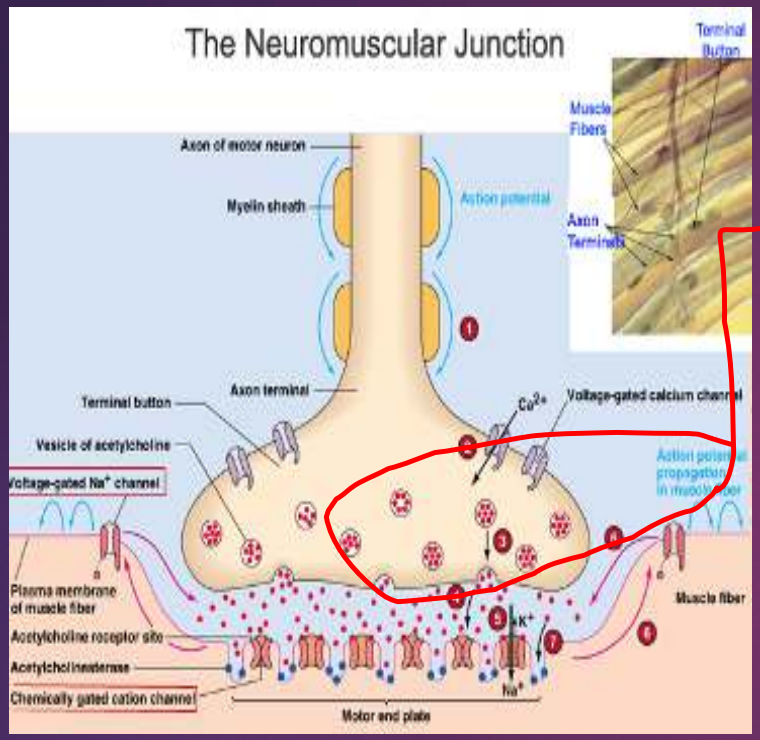
Cobra – post-synaptic



- alpha-neurotoxins "Curare - mimetic toxins"
- Bind specifically to Ach receptors, preventing the interaction between Ach and receptors on postsynaptic membrane.
- Prevents the opening of the sodium channel associated with the Ach receptor and results in neuromuscular blockade.
- ASV -rapid reversal of paralysis.
- Dissociation of the toxin-receptor complex, which leads to a reversal of Paralysis

Anticholinesterases reverse the neuromuscular blockade

Krait- Pre-synaptic action



Beta-bungarotoxin- Phospholipases A2

1) Inhibiting the release of Ach from the presynaptic membrane

2) Presynaptic nerve terminals exhibited signs of irreversible physical damage and are devoid of synaptic vesicles

3) ASV & anticholinesterases have no effect

Paralysis lasts several weeks and frequently requires prolonged MV. Recovery is dependent upon regeneration of the terminal axon.

Venom composition and mechanism

- Proteolytic enzymes (hydrolases) may be responsible for local changes in vascular permeability leading to oedema, blistering, and bruising, and to necrosis.

- Biological amines such as histamine and 5-hydroxytryptamine may contribute to local pain and permeability changes at the site of a snakebite.

Venom composition and mechanism

- These include digestive hydrolases, hyaluronidase, and activators or inactivators of physiological processes, such as kininogenase.

- Most venoms contain l -amino acid oxidase, phosphomono- and diesterases, 5’-nucleotidase, DNAase, NAD-nucleosidase, phospholipase A 2 and peptidases.

Bites

Venomous Snakebite

Without Envenomation:

- Dry bite
- Protective gear
- Leakage of venom
- Superficial bite

Bites

With Envenomation:

Colubrid bite

Elapid bite

Viperid bite

Hydrophid bite

Clinical Features

A significant proportion of snakebites is said to be due to non-venomous snakes.

Due to fear and apprehension associated with snakes, every bite (venomous or otherwise) is attended by some degree of shock characterised by

Giddiness,

Syncope,

Sweating,

Palpitation,

Tachycardia, and

Hypotension.

Colubrid bite

- Clinical effects of colubrid snakebite are generally localized, and comprise pain, oedema, erythema, ecchymosis and numbness, which resolve over one to two weeks.

- Excessive salivation with metallic taste, and headache have also been reported.

- **Colubridae: Mountain racer, Western and Eastern hognose snakes, parrot snake, rat snake, wandering garter snake**

Elapid bite (cobra, kraits)

Local effects

- Minimal local manifestations and Pain, swelling are relatively less intense.
- Serosanguinous ooze from the bite site with mild pain, tenderness, and blistering
- Cobras-can occasionally cause significant local swelling, blistering, and regional lymphadenopathy
- The lesion may emit a putrid smell, and break down with loss of skin and subcutaneous tissue
- Early onset of gangrene (wet type)

Elapid bite

Systemic Effects:

- Neurotoxicity
  - Occur earlier (within 15 minutes to ½ hour) in cobra bite
  - Delayed (up to several hours) in krait bite.

Preparalytic Stage

- Vomiting
- Ptosis
- Blurred vision, external ophthalmoplegia

Elapid bite

Preparalytic Stage

- Paraesthesiae around the mouth
- Hyperacusis
- Headache, myalgia
- Vertigo
- Hypersalivation (due to autonomic stimulation).

Elapid bite

Paralytic Stage

- The facial muscles, palate, jaws, tongue, vocal cords, neck muscles, and muscles of deglutition all become progressively flaccidly paralysed
- Difficult to open their mouths and speak.
- Respiratory arrest
- Unconscious, most are able to follow simple commands.

Elapid bite

Paralytic Stage

- Neurotoxicity, if it develops, generally begins 1 to 5 hours after envenomation
- Platelet aggregation and coagulation-fibrinolysis system may occur after envenomation by cobras.
- Renal complications are rare in elapid bites

Summary of elapid bites

- Less local manifestations- Serosanguinous ooze observed
- Neurotoxicity
- Pre-paralytic and paralytic stage
- Coagulation defects with cobra bite
- rare renal manifestations

Viperid bite (Vipers, Rattle snakes)

Local Effects:

- Pitless as well as pit vipers cause marked local manifestations which develop rapidly, usually within ½ hour, but may occasionally be delayed for several hours
- Swelling and blisters first appears around the bite site, and then spreads quickly to involve the whole limb
- Pain, tenderness, and regional lymphadenopathy, Bruising
- Blisters may contain either clear or bloodstained fluid or extensive necrosis of skin

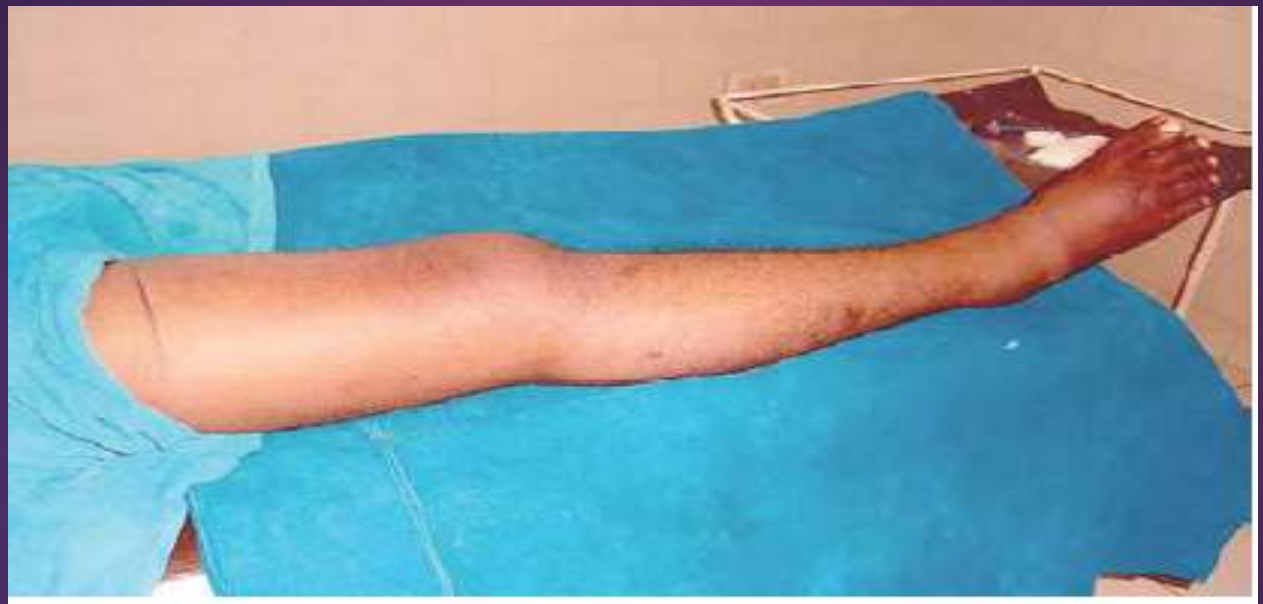


Fig 12.20: Viper bite—Extensive swelling of lower limb

Viperid bite

Local Effects:

- Raised intracompartmental pressure leads to severe pain, tense swelling, subcutaneous anaesthesia, and increased pain on stretching intracompartmental muscles.

Systemic Effects:

- Haemostatic abnormalities are very characteristic of viperid bites

- Epistaxis, haemoptysis (relatively rare), ecchymoses, intracranial and sub-conjuctival haemorrhages, and bleeding into the floor of the mouth, tympanic membrane, gastrointestinal tract, and genito-urinary tract.

Systemic Effects with Viperid bite

Fig 12.21: Viper bite – Haematuria

g 12.22: Viper bite – Gingival bleeding

Fig 12.23: Viper bite – Haematemesis

Viperid bite

Systemic Effects:

- Bleeding into anterior pituitary (causing a Sheehan-like syndrome) has been reported.
- Subarachnoid haemorrhage manifests as severe headache and meningism.
- Intracerebral haemorrhage may cause hemiplegia, loss of consciousness, and convulsions.
- Retroperitoneal and intraperitoneal haemorrhages cause abdominal distension, tenderness, and peritonism, with signs of haemorrhagic

Viperid bite

Systemic Effects:

- Intravascular haemolysis causing haemoglobinuria and renal failure is a frequent occurrence, especially in bites by Russell's viper
- Acute renal failure is often associated with the presence of DIC which results in severe renal tubular and cortical necrosis with widespread microvascular fibrin deposition (microthrombi).
- Direct toxic effect produced by the venom of Russell's viper may produce renal damage.

Viperid bite

Systemic Effects:

- Hypotension is an important manifestation in all viper bites and is usually accompanied by tachycardia.
- Haemorrhagic manifestations could more commonly be due to primary pathological fibrinolysis (PPF) than disseminated intravascular coagulation (DIC).
- Cardiotoxicity (which may be seen in elapid bite also) produces a wide variety of ECG changes.

Common ECG Changes

- Sinus bradycardia
- Sinus tachycardia
- Sinus arrhythmia
- Tall T waves
- ³ ST depression %1mm with flat or inverted T in all chest leads
- ³ ST depression %1mm with T inversion in inferior leads, or in anterior leads.
- ST elevation in leads V_1 to V_6, I, aVL; Q wave V_1 to V_4 and ST depression in II, III, aVF
- First degree or second degree heart block

Viperid bite

Systemic Effects:

- Ptosis and neurological symptoms may occur in the case of Russell's viper
- Generalised flaccid paralysis can develop after envenomation
- Neurotoxic effects are caused by the presence of phospholipases A2 with presynaptic neurotoxic activity.

Fig 12.24: Ptosis in Russell's viper bite

Summary of viperid bite

- Local manifestations- blisters, bruising, swelling, necrosis and increased intracompartmental pressure
- Haemostatic manifestations are characterstic feature-DIC, PPF, Hemorrages (Intacerebral, subarachonoid, intravascular, pituitary, intraperitonial and Retroperitonial.)
- Renal failure and Neurotoxicity mainly due to Russels viper
- Hypotension, tachycardia and cardiac toxicity
- Pre-synaptic neurotoxicity

Hydrophid bite (sea snakes)

Local Effects:

- Sea snakebites are well-known to produce minimal local effects
- However teeth are often left behind in the wound
- It is important to note that in some cases, there may be no clear fang marks, but a vague scratch mark, and yet serious poisoning may occur.
- Fang marks may appear as one, two or more small circular dots, as though made by a pin or hypodermic needle

Hydrophid bite

Systemic effects

- Hydrophid venom is predominantly myotoxic leads to rhabdomyolysis
- Myalgia with stiffness and tenderness of muscles will occur
- Myoglobinaemia and myoglobinuria occur resulting in acute tubular necrosis and renal failure
- Trismus is an early feature

Hydrophid bite

Systemic effects:

- Passive stretching of muscles is painful
- Flaccid paralysis develops, beginning with ptosis
- Hyperkalaemia -This may besevere enough to cause cardiac arrest. Tall, peaked T waves and QRS prolongation suggest severe hyperkalaemia.
- Other effects may include dizziness, nausea, vomiting, headache, and diaphoresis.

Hydrophid bite

Systemic effects:

- Neurotoxicity may include ptosis, ophthalmoplegia,dysarthria, blurred or double vision, mydriasis, inability to sit unassisted, depressed muscle stretch reflexes, and flaccid paralysis
- Paralysis of respiratory muscles causes death due to respiratory failure.
- Failing vision is considered to be a terminal sign.
- The fatality rate is estimated to be about 3%.

Summary of Hydrophid bite

- Minimal local effects
- Mainly myotoxic
- Neurotoxicity observed
- Failure of respiratory muscles
- Failing vision
- Hyperkalemia

Diagnosis of Snakebite

Fang Marks

- Two puncture wounds separated from each other by a distance varying from 8 mm to 4 cm
- Sideswipe may produce only a single puncture
- Many venomous species possess more than one set of fangs and thus multiple fang marks may be present
- Wolf Snake have large front teeth which inflict bites that look similar to fang marks
- Occasionally, fang marks may not be clearly evident

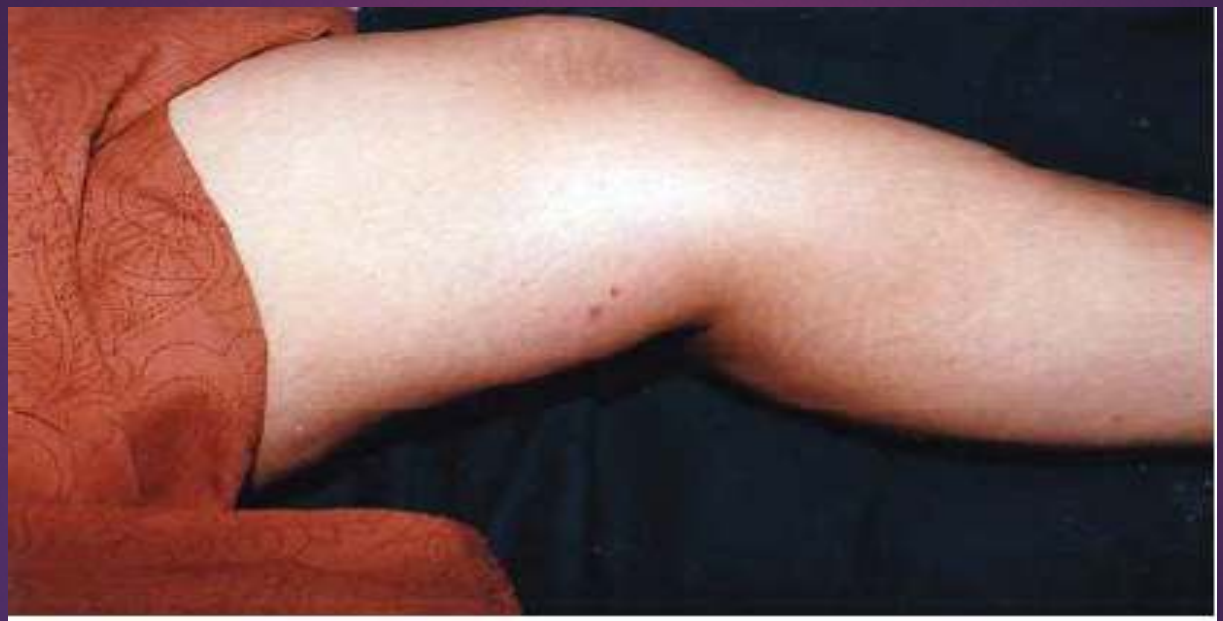


Fig 12.25: Classical fang marks

Diagnosis of Snakebite

Identification of Snake:

- Difficult to find though victim or his attendants bring with them the culprit (dead) snake

Laboratory Investigations:

- Haematological
- Anaemia
- Leucocytosis
- Thrombocytopenia
- High haematocrit initially; later it falls
- Evidence of haemolysis: fragmented RBC (schistocytes or helmet cells)

Diagnosis of Snakebite

Laboratory Investigations:

Haematological

- Prolonged clotting time and prothrombin time
- Prolonged partial thromboplastin time
- Depressed fibrinogen levels
- Elevated FDP (Fibrin degradation products).

Diagnosis of Snakebite

Laboratory Investigations:

ECG

- Common ECG changes include bradycardia with ST segment elevation or depression, T wave inversion, QT prolongation, and changes due to hyperkalaemia.

Metabolic

- Hyperkalaemia
- Hypoxaemia with respiratory acidosis
- Metabolic acidosis or lactic acidosis (increased anion gap).

Diagnosis of Snakebite

Laboratory Investigations:

Urine

- Haematuria
- Proteinuria
- Haemoglobinuria
- Myoglobinuria.

Renal

- In acute renal failure, all features of azotaemia will be present.

.

Diagnosis of Snakebite

Laboratory Investigations:

Chest X-ray

- Pulmonary oedema
- Intrapulmonary haemorrhages
- Pleural effusion.

X-ray of bitten part

- For bitten areas that remain tender, plain radiographs may reveal the presence of embedded snake tooth/fang fragments.

Diagnosis of Snakebite

Laboratory Investigations:

Immunodiagnosis

- Immunological detection of venom antigens in body fluids can be accomplished by ELISA

- This is highly sensitive, but specificity may be inadequate to distinguish between different species of snakes

General management as first aid

- Verbal reassurance

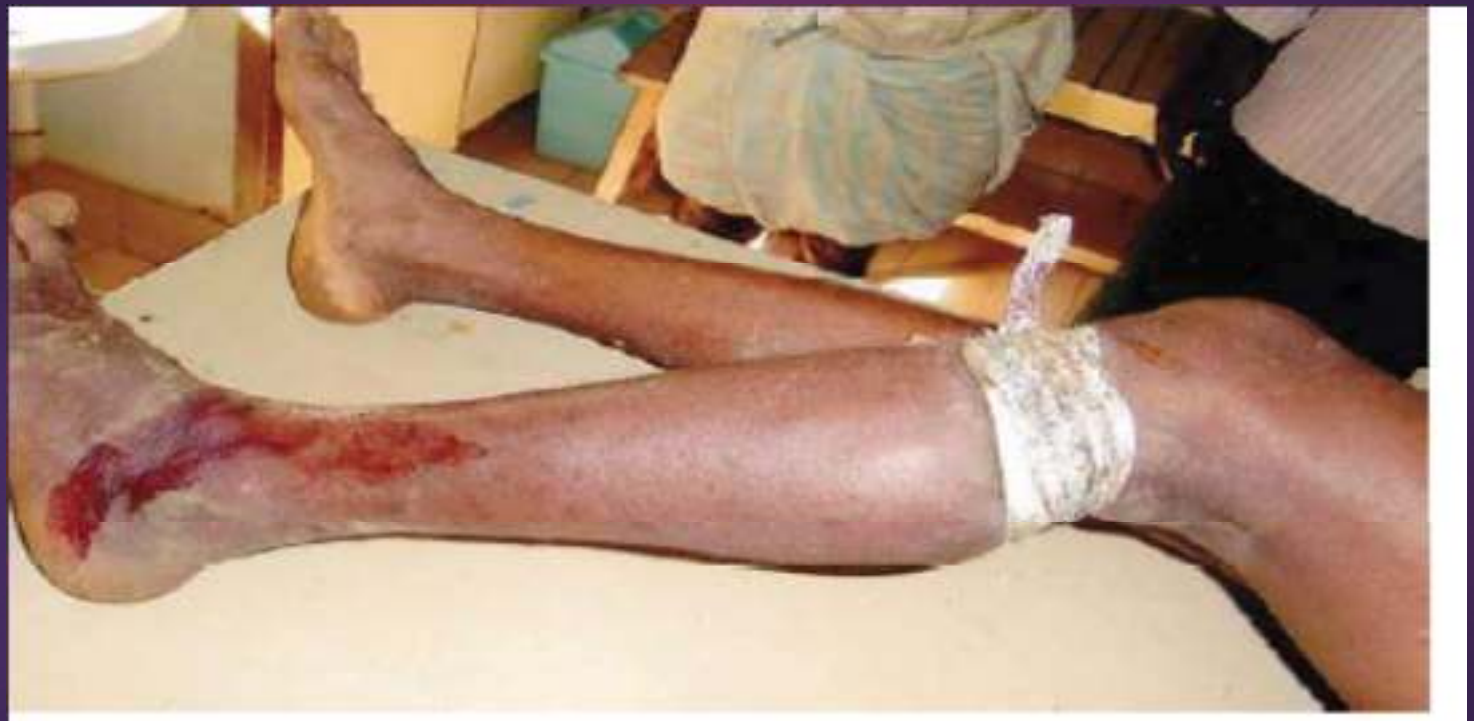
- Immobilisation: Mobilization enhance systemic absorption of venom, there is universal consensus that the patient should be put at rest

  - Firm binding of the splint with a crepe bandage is an effective form of immobilization (Sutherland wrap; Pressure Immobilisation Method).

  - Local Compression Pads (Monash method): useful in victims of bites by Russell's Viper (A firm rubber pad is applied with cotton bandaging over the site of the bite and the limb is then immobilised with a splint.

General management as first aid

- Beverages:
  - Use of “stimulating” beverages such as coffee is inadvisable and ineffective
  - Alcohol must never be administered, since it increases the absorption of venom.
- Tourniquet:
  - Application of a tourniquet proximal to the bite site of a bitten limb in order to prevent the spread has often been advocated



g 12.27: Tight tourniquet compromising blood circulation

General management as first aid

- Serious risk associated with Tourniquet
  - Ischemia and gangrene, damage to peripheral nerves (especially lateral popliteal nerve),
  - Increased fibrinolytic activity,
  - Congestion, swelling, increased bleeding, and increased local effect of venom
  - Release of a tourniquet, leads to a flooding of accumulated venom from the bite site into the systemic circulation with life-threatening consequences.
- Today the general consensus about Tourniquet is against application of a tourniquet

General management as first aid

- Incision and suction:
  - This is under controversy
  - Serious risks associated with it, including uncontrolled bleeding in patients with incoagulable blood, damage to nerves, blood vessels and tendons, and introduction of infection.
  - Some practitioners still advocate the method in selected cases, especially if it is done within the first 5 to 10 minutes following the bite.
  - If it is decided to be done, cruciate incisions must be avoided.
  - Parallel incisions may be made through the fang marks, about 1 cm long and no deeper than 3 mm, in the long axis of the limb

General management as first aid

- Cyrotherapy:
  - Local cooling (application of ice) in the region of the bitesite was previously recommended for minimising the absorption of venom.
  - Today not recommended because of serious risk of necrosis leading to gangrene.

General management as first aid

- Electric shock:
  - It has been suggested that if snake antivenom is not available to treat a venomous bite, local electric treatment may be done which is claimed to be life saving.
  - The electric shock (25 kv, 1 ma) is to be applied direct to the bite by means of an insulated probe for a couple of seconds, and repeated 4 to 5 times at 5to 10 second intervals, taking care to ground the area as closely to the site of the bite as possible.
  - Today, the universal view is that it is a useless and dangerous method.

General management as first aid

Drugs

- Mild to moderate pain, paracetamol can be given.
- If pain is severe, several authorities recommend judicious use of narcotic analgesics such as pentazocine or pethidine, even though in some cases this can be hazardous, e.g. elapid bites, where there may be CNS depression.
- Aspirin and non-steroidal anti-inflammatory drugs must not be used as it cause gastric erosions which lead to persistent gastric bleeding
- Use of corticosteroids and antihistamines are advocated in allergic reactions to antivenom.

General management as first aid

Drugs

- Persistent vomiting can be treated with intravenous chlorpromazine
- Patients should be made to lie on their side with the head down to avoid aspiration.
- Intramuscular and subcutaneous injections should be avoided, especially in patients with incoagulable blood, since they can lead to haematoma formation.
- Pressure dressings should be applied to venepuncture sites to prevent oozing.

General management as first aid

Drugs

- Though Clostridium tetani has not been isolated, its ubiquitous nature prompts most authorities to emphasise the importance of tetanus prophylaxis in the form of tetanus toxoid.

What should not be done after snake bite

The following measures are potentially harmful, and must not be undertaken:

- Application of a tight tourniquet which occludes arterial supply
- Cauterisation of bitesite
- Multiple, deep incisions through bitesite
- Suction by mouth, vacuum pump, or syringe
- Application of injurious substances such as potassium permanganate, phenol, etc.
- Application of electric shock
- Application of ice (cryotherapy)
- Use of herbal, folk, or Ayurvedic medicines or remedies

Antivenom (Antivenin) therapy

Use of antivenom therapy as routine is irrational because

- Many cases of snakebite involve nonvenomous snakes
- Envenomation is not the rule even in venomous bites.
- Antivenoms available are usually effective against specific species of snakes, and are of no benefit against other species.
- Antivenom is usually in short supply, and has limited shelf-life

Antivenom (Antivenin) therapy

Reid's criteria (modified by Persson) for antivenom therapy are as follows:

- Prolonged or recurring hypotension
- Persistent or recurring shock in spite of treatment
- Pronounced leukocytosis
- Protracted gastrointestinal symptoms

Antivenom (Antivenin) therapy

Reid's criteria (modified by Persson) for antivenom therapy are as follows:

- ECG changes
- Raised serum creatine phosphokinase
- Early extensive swelling in adults
- Haemolysis
- Pregnant women, small children.
- Acidosis

Antivenom (Antivenin) therapy

Indications for Antivenom Therapy

Systemic envenomation	*Severe local envenomation*
1. Haemostatic disturbances: Spontaneous systemic bleeding or caogulopathy	1. Local envenomation associated with neutrophil leukocytosis, elevated creatine phosphokinase and aminotransferases, haemoconcentration, uraemia, hypercreatinaemia, oliguria, hypoxaemia, acidosis, vomiting
2. CVS abnormalities: Shock, hypotension, abnormal ECG, arrhythmia, cardiac failure, pulmonary oedema	2. Local swelling involving more than half the bitten limb
3. Neurotoxicity	3. Extensive blistering or bruising
4. Generalised rhabdomyolysis	4. High-risk of necrosis
5. Impaired consciousness	

Antivenom (Antivenin) therapy

Timing of antivenom therapy

- Antisnake venom (ASV) must be administered as early as possible when signs of systemic or severe local envenomation develop.

- Some investigators have reported beneficial effects even after a lapse of 1 week or more.

Antivenom (Antivenin) therapy

Availability of antivenom therapy

- In India, polyvalent antivenom is commonly available which is effective against the Big Four.

- The best form of antivenom is a lyophilized (freeze-dried) powder, which is produced by immunisation of horses with the venom of the snakes mentioned.

Antivenom (Antivenin) therapy

Administration of antivenom therapy

- The powder must be reconstituted in distilled water or saline just before use. If the resulting solution is opaque (turbid) to any extent, it has lost its efficacy and should be discarded
- The antivenom must always be administered intravenously.
- The only indication for intramuscular injection of antivenom is in the case of a remote field site involving a great many hours of transportation to a medical facility.

Fig 12.31: Turbid antivenom solution (left)

Antivenom (Antivenin) therapy

Administration of antivenom therapy

- If ASV is administered intramuscularly, a number of sites in the thigh should be used, and the area should be massaged to aid absorption.

- Before beginning antivenom therapy, a skin test is conventionally advised for detecting hypersensitivity

Antivenom (Antivenin) therapy

Administration of antivenom therapy

- Current consensus is NOT to perform any test for hypersensitivity for the following reasons:
  - Most of the reactions to antivenom, i.e. anaphylactic and late serum reactions, are not IgE-mediated hypersensitivity reactions to horse or sheep protein.
  - In fact, the very act of administering an ASV test dose may pre-sensitise the victim and therefore make an allergic reaction more likely.
  - There is also the logical argument which states that even if the patient shows some evidence of early sensitivity, antivenom is going to be required in any case, as it is the only known cure for envenomation.

Antivenom (Antivenin) therapy

Dose of antivenom

1. Two methods of antivenom administration are recommended:

   IV injection: 2 ml/ minute

   IV infusion: 5–10ml of isotonic fluid/kg body weight

2. Haemotoxic/Neurotoxic Envenomation (< 3 hrs since bite):

   10 Vials ASV

3. Haemotoxic/Neurotoxic Envenomation (> 3 hrs since bite):

   7 Vials ASV

- No further antivenom is given over the next five hours

Antivenom (Antivenin) therapy

Administration of antivenom therapy

- Six hours after the first administration of antivenom a further clotting and blood analysis test is carried out in the case of haemotoxic envenomation to determine whether coagulability has been restored.
- If the blood test shows that coagulability has indeed been restored, no further antivenom is administered.
- In the event that coagulation has not been restored, a further dose of antivenom is administered over the next 30 to 60 minutes, and blood tests carried out again after six hours.
- This six-hour period is crucial as it represents the time it takes the liver to restore clotting factors to normal levels.

Antivenom (Antivenin) therapy

Administration of antivenom therapy

- In the case of neurotoxic envenomation there is considerable confusion as to the regime to be adopted
- At the first sign of systemic neurotoxic symptoms such as ptosis or ophthalmoplegia, ASV should be administered to the patient, again over 30 to 60 minutes
- If the patient presents with respiratory failure at the hospital, ASV should be administered and assisted and mechanical ventilation are the primary means of treatment.

Antivenom (Antivenin) therapy

Administration of antivenom therapy

- In the case of post-synaptic envenomations, antivenom can reverse neurotoxic symptoms in the early stages.
- If after 1–2 hours the symptoms have worsened, or have not reduced, a second dose of ASV should be administered
- If the patient undergoes respiratory failure or the symptoms have worsened to this extent, then ASV should be stopped after the second dose.
- At this stage assisted and mechanical ventilation are the primary means of treatment

Antivenom (Antivenin) therapy

Administration of antivenom therapy

- With regard to pre-synaptic neurotoxic envenomation, nerve terminals have been destroyed, and mechanical ventilation is required until these terminals can be rebuilt

- NOTE: Once coagulation has been restored, the victim should be kept under observation, blood monitoring should continue, and in the rare event that recurrence occurs, further antivenom must be administered.

Antivenom (Antivenin) therapy

Reactions/Adverse Effects of antivenom therapy

- Early (anaphylactic) reaction: Develops in 10 minutes to 1 hour of beginning the antivenom therapy.
- It begins with cough, urticaria, tachycardia, palpitations, nausea, vomiting, headache, and fever.
- The full-blown anaphylactic reaction is characterised by hypotension, bronchospasm, and angioedema

Antivenom (Antivenin) therapy

Reactions/Adverse Effects of antivenom therapy

- Treatment involves administration of adrenaline subcutaneously, 0.5 to 1 ml of 0.1% solution (1 in 1000) for adults; 0.01 mg/kg for children. This is followed by an antihistamine (e.g. chlorpheniramine maleate, 10 mg in adults; 0.3 mg/kg in children).

- Pyrogenic reaction: Develops in 1 to 2 hours of beginning the therapy.

- It is characterized by chills, goose fleshing, shivering, rise in temperature, sweating, vomiting, and diarrhoea.

Antivenom (Antivenin) therapy

Reactions/Adverse Effects of antivenom therapy

- Treatment involves fanning, tepid sponging, hypothermia blankets, or antipyretic drugs such as paracetamol.

- Late (serum sickness) reaction: Develops about 7 days after treatment. It usually responds to antihistamines and corticosteroids.

Antivenom (Antivenin) therapy

If antivenom is unavailable, the following conservative measures can be undertaken:

- Haemostatic abnormalities: Give clotting factors and platelets (i.e. fresh frozen plasma and cryoprecipitate with platelet concentrates). Blood transfusion may be indicated.

- Shock/Hypotension:
  - Give colloids/crystalloids as needed.
  - Monitor central venous pressure and cardiac output.
  - Dopamine and other pressor agents may be required.
  - Blood transfusions may be indicated in the presence of systemic bleeding.

Antivenom (Antivenin) therapy

If antivenom is unavailable, the following conservative measures can be undertaken:

- Myoglobinuria: Correct hypovolaemia and acidosis.
- Acute renal failure: Supportive care or haemodialysis.

Antivenom (Antivenin) therapy

Other considerations

Secondary Infection with the bite can be prevented with

- Erythromycin or penicillin.
- Gentamicin
- Amoxycillin , Cephalosporine & Metronidazole

- Leave blisters alone. They will break spontaneously and heal.

- If there is local necrosis, excise the slough and apply saline dressings

Antivenom (Antivenin) therapy

Other considerations

Intracompartmental syndrome

- This results from swelling of muscles within tight fascial compartments.
- It manifests as severe pain, weakness of compartmental muscles,resistance to passive stretching, hyperaesthesia of areas of skin supplied by local nerves, and tenseness of the compartment.
- Treatment : Fasciotomy should be done to relieve the pressure (but performed only when blood coagulability has been restored.)

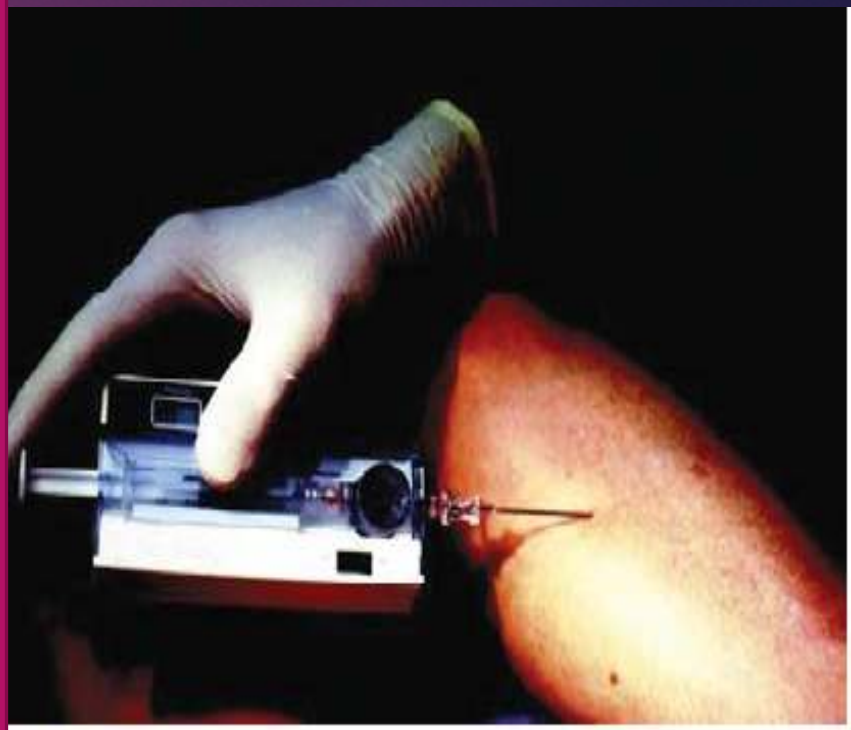


Fig 12.32: Stryker intracompartmental pressure monitor