CHAPTER 18

POISONING

For advice contact:

POISON INFORMATION CENTRES		
Poisons Information Helpline (National service)	24 hours/day, every day for poisons queries	0861 555 777
Red Cross War Memorial Children's Hospital Poisons Information Centre Email: poisonsinformation@uct.ac.za http://www.paediatrics.uct.ac.za/poisons-information-centre	Office Hours	(021) 658 5308
Tygerberg Poison Information Centre Email: toxicology@sun.ac.za www.sun.ac.za/poisoncentre	Office Hours	(021) 938 9596
University of the Free State Poison Control and Medicine Information Centre	24 hours/day	082 491 0160

The Afritox poisons information database is available free of charge to public hospitals in South Africa: see www.afritox.co.za for information on how to access it.

18.1 POISONING

DESCRIPTION

Frequently encountered poisonings in children include:

- analgesics,
- hydrocarbons,
- pesticides,
- plant material,
- household cleaning products.
- vitamins and minerals,
- anticonvulsants,
- antipsychotics,
- sedatives and antidepressants,

Suspect intentional ingestion in older children and adolescents.

DIAGNOSTIC CRITERIA

Clinical

Can be divided into 'toxidromes':

Cholinergic, e.g. organophosphates:

- salivation,
- lacrimation,
- urination,
- pinpoint pupils,
- diarrhoea,
- vomiting,
- bronchorrhoea,
- bradycardia.

Salicylism, e.g. aspirin:

- tachypnoea,
- metabolic acidosis,
- seizures.
- agitation,
- coma,

Anticholinergic, e.g. antihistamines, *Amanita pantherina*, atropine:

- fever,
- ileus,
- flushing,
- tachycardia,
- urinary retention,
- dry/warm skin,
- blurred vision,
- dilated pupils,
- coma,
- hallucinations and seizures.

Sedative-hypnotic, e.g. alcohol, benzodiazepines:

- Obtundation or coma.

Opiates, e.g. morphine:

- pinpoint,
- respiratory depression,
- bradycardia,
- hypotension.
- decreased bowel sounds,
- hypothermia,
- altered (decreased) mental status,

Dystonic reaction, e.g. haloperidol, antihistamines, anti-emetics:

- torticollis,
- opisthotonus,
- intermittent spasms and tongue thrusting.

Sympathomimetic, e.g. cocaine, amphetamines:

- hypertension,
- tachycardia,
- hyperthermia,
- agitation,
- sweating,
- dilated pupils.

Sympathomimetic toxidrome resembles anticholinergic toxidrome, i.e. fight, flight and fright response, however, the sympathomimetic toxic patient is sweaty as opposed to hot, dry skin seen with anticholinergic toxicity.

Toxic alcohols, e.g. ethylene glycol, methanol:

- » metabolic acidosis,
- » increased osmolar gap,
- » increased anion gap,
- » visual disturbances (methanol),
- » hypoglycaemia,
- » convulsions,
- » renal failure (ethylene glycol),
- » depressed level of consciousness.

TREATMENT

- If the ingestion has definitely occurred: establish whether toxicity is expected and act accordingly.
- If the possibility of ingestion was remote: only observation is necessary.

Principles of treatment

- » Stabilise the patient if necessary.
- » Decontaminate the patient if indicated (see below) and contra-indications are not present.
- » Give antidote if available. There are a limited number of antidotes for poisoning by certain substances, e.g. N-acetylcysteine for paracetamol, naloxone for opioids. Each antidote has specific criteria and indications for use.
- » Enhance elimination if possible.
- » Monitor hydration status carefully.

Decontamination:

1. Gastric lavage

Gastric lavage is seldom indicated and may cause more harm than benefit. If indicated, it should only be performed by experienced staff and within 60 minutes of ingestion.

Indicated only if patient:

- has ingested a potentially life-threatening poison,
- has a protected airway, i.e. fully awake and cooperative or intubated with a depressed level of consciousness.

Gastric lavage is contraindicated after ingestion of corrosive substances and volatile hydrocarbons such as paraffin.

Technique:

- Place patient in left lateral head down position.
- Insert orogastric tube if possible, with largest bore and rounded tip.
- Insert 200 mL warmed water or normal saline, and aspirate.

Continue until recovered solution is clear of particulate matter.

2. Activated charcoal

May reduce systemic absorption of a variety of poisonous substances. The greatest benefit is achieved if activated charcoal is given within one hour after ingestion; however, where gastric emptying is delayed by certain substances, there may be a longer period of time in which it is effective.

Activated charcoal must only be given in cases where the airway is protected, i.e. fully awake and cooperative patient or intubated with a depressed level of consciousness.

Repeated doses of activated charcoal every 4 hours are effective in enhancing elimination of substances that undergo enterohepatic circulation, e.g. carbamazepine, dapsone, phenobarbital, quinine or theophylline overdose.

- Activated charcoal, oral, given as a slurry:
  - If < 6 years of age: 1 g/kg in 50–100 mL water.
  - If > 6 years of age: 20–50 g in 100–300 mL water.

LoE III[1]

Note: In the intubated patient with a protected airway, the activated charcoal can be administered via a nasogastric tube (the slurry is thick and requires administration to be pushed through a syringe).

Contra-indications:
» If patient is unconscious and the airway is not protected.

Poisons where charcoal is **ineffective** and should not be given	Charcoal may be useful if these poisons are taken in toxic doses
» ethanol » methanol » brake fluid » petrol or paraffin » iron salts » lithium » bleach and caustic alkalis » boric acid	» carbamazepine, barbiturates, phenytoin » dapsone, quinine » theophylline » salicylates » mushroom poisoning (*Amanita phalloides*) » slow-release preparations » digoxin » beta-blockers » NSAIDs

3. Whole bowel irrigation

Whole bowel irrigation can be done for potentially toxic ingestions of substances that are:
» not absorbed by activated charcoal (e.g. iron and lithium),
» sustained-release and enteric-coated products, or
» for removal of illicit drugs in body packers.
Patients must have a protected airway, i.e. fully awake and cooperative or intubated with a depressed level of consciousness.

- Polyethylene glycol (PEG) balanced electrolyte solution, NGT:
  - Child (9 months to 6 years): 500 ml per hour
  - Child (6 to 12 years): 1000 ml per hour
  - Continue until rectal effluent is clear.

LoE III[2]

REFERRAL

» Severely ill patient for ventilatory/circulatory support.
» If relevant diagnostic testing is not available, e.g. paracetamol levels.
» If relevant medication/antidotes are not available.
» If dialysis/haemoperfusion is required.
» For psychiatric evaluation where deliberate self-harm is suspected.

SECONDARY PREVENTION

All cases of accidental poisoning require an assessment of home circumstances. The opportunity must be taken to educate childcare providers on safe storage practices, particularly of medications and household products.

18.1.1 ANTICHOLINERGIC POISONING

T44.3

DESCRIPTION

Various plant species and pharmaceutical preparations can cause anti-cholinergic toxicity.

Plants: *Datura stramonium*, e.g. 'stinkblaar' and 'malpitte'.

Medicines: atropine, diphenoxylate with atropine and diphenhydramine. Other classes of medicines include antiparkinsonism agents, antispasmodics, antipsychotics, antihistamines and tricyclic antidepressants.

DIAGNOSTIC CRITERIA

Clinical

» Alteration of mental status, including delirium, hallucinations, agitation and seizures.
» Peripheral anticholinergic effects include:
  > dilated pupils,
  > tachycardia and arrhythmias,
  > flushing,
  > urinary retention,
  > decreased GIT motility,
  > dry skin and mucous membranes.

Investigations

» ECG and continuous cardiac monitoring.
» Pulse oximetry.

GENERAL AND SUPPORTIVE MEASURES

» Stabilise patient, i.e. airway, breathing and circulation.
» Cooling for hyperthermia.
» Perform decontamination depending on route of exposure.

MEDICINE TREATMENT

- Activated charcoal, see section 18.1: Poisoning.

For agitation:

- Diazepam, IV/oral, 0.1–0.2 mg/kg.
  - Maximum dose: 10 mg.

For seizures:
See Chapter 13: The Nervous System, section 13.3: Status epilepticus (convulsive).

REFERRAL

» Cardiac dysrhythmia.
» No response to treatment.

18.1.2 ANTICOAGULANT POISONING

T45.5
*Notifiable condition (if poisoning due to agricultural or stock remedy, e.g. rodenticide).

DESCRIPTION

Poisoning due to warfarin and 'super-warfarins' (long-acting warfarin) (products such as pellets, granules, wedges, blocks and powder marketed as rodent pesticides). These may be accidentally ingested by toddlers or young children.

Note: Where the history is of an unspecified rat poison or pesticide ingestion, consider other active ingredients such as amitraz and organophosphates.

DIAGNOSTIC CRITERIA

Clinical

» Signs and symptoms depend on the potency, and onset of coagulopathy may be delayed by 48–72 hours. May be asymptomatic if a small quantity has been ingested.
» Bruising or bleeding.

Investigations

» Measure prothrombin time.
  > Obtain baseline INR if symptoms/signs present.
  > A baseline or repeat INR should be done in all cases of significant ingestion at 36–72 hours post-ingestion.

GENERAL AND SUPPORTIVE MEASURES

» Observe an asymptomatic child: may be as outpatient depending on

history (amount ingested) and ability to return if symptoms develop.

MEDICINE TREATMENT

ONLY if INR deranged (> 2.5 IU):
Vitamin K1, IV/oral, 1–5 mg/dose administered slowly.

Note: Intravenous solution can be used orally.

Oral vitamin K1 is usually preferred to intravenous vitamin K1 unless more rapid reversal is required (e.g. the patient is bleeding). Intravenous vitamin K1 may cause hypersensitivity reactions.

If significant bleeding present:
ADD

- Lyophilised plasma, IV, 20 mL/kg.

OR

- Fresh frozen plasma, IV, 20 mL/kg.

Repeat vitamin K_1 dosing and length of therapy is dependent on INR response to treatment and clinical response—contact poison center for patient specific advice.

Ingestion of 'super-warfarins' may be refractory to large doses of vitamin K_1 and therapy may be required for several weeks after ingestion.

18.1.3 TRICYCLIC ANTIDEPRESSANT POISONING

T43.0

DESCRIPTION

Poisoning with tricyclic antidepressants (TCAs) represent a large portion of poisoning fatalities. There is a high risk of tricyclic antidepressant toxicity in children because of its narrow therapeutic index. Serious toxicity may occur with low doses in children. Patients can deteriorate rapidly.

DIAGNOSTIC CRITERIA

» Can cause anticholinergic syndromes.
» Mainly affects the cardiovascular and nervous systems leading to:
> QRS widening,
> ventricular dysrhythmias,
> hypotension,
> altered mental status,
> seizures.

GENERAL AND SUPPORTIVE MEASURES

» Gastric lavage for large ingestions or patients presenting within a few hours post ingestion, unless the patient is unconscious and the airway is not protected.
» Circulatory and respiratory support as needed.
» Cardiac and ECG monitoring for 48 hours.

MEDICINE TREATMENT

- Activated charcoal: see section 18.1: Poisoning.
  - TCAs delay gastric emptying, therefore, activated charcoal may be effective for a longer period than usual.

For hypotension:

- Sodium chloride 0.9% or Ringer's Lactate, IV bolus, 20 mL/kg.

Serum alkalinisation for all patients with:

» ventricular dysrhythmias,
» prolonged QRS > 100 ms,
» hypotension unresponsive to fluids, or
» seizures.

- Sodium bicarbonate, bolus doses (1–2 mEq/kg as an 8.4% solution), to achieve a pH of 7.45–7.55. (Specialist consultation with Poisons Information Centre if possible.)
- Monitor acid-base status, serum potassium and sodium.

LoE III[3]

In severe cases, inotropic support and anti-arrhythmic agents may be required in addition to serum alkalinisation. Hypotension is due to myocardial dysfunction and alpha-adrenergic vasodilation, therefore be careful to avoid fluid overload.

For seizures:

» See Chapter 13: The Nervous System, section 13.1: Seizures.
Note: Phenytoin should be avoided (due to potential cardiotoxicity).

LoE III[4,5]

For circulatory and respiratory support:
See Chapter 1: Emergencies and Trauma, section 1.1.4: Cardiorespiratory arrest.

REFERRAL

» Any cardiac arrhythmia.

18.1.4 INGESTION OF CAUSTIC OR CORROSIVE AGENTS

T54

DESCRIPTION

Alkalis, e.g. sodium hydroxide, potassium permanganate.
Acids, e.g. hydrochloric acid.
The severity of the injury is dependent on the concentration and duration of exposure to the acid or alkali.

DIAGNOSTIC CRITERIA

Clinical

» Chief symptom is pain.
» Young children may present with:
  > crying, > refusal to swallow,
  > drooling, > vomiting.
» Stridor or hoarseness indicates laryngeal injury.
» The presence of oral or pharyngeal burns does not predict the presence of oesophageal or gastric injury.
» Oesophageal or gastric injury can cause perforation or subsequent fistula formation.
» Asymptomatic patients are unlikely to have significant oesophageal or gastric injury.

GENERAL AND SUPPORTIVE MEASURES

Asymptomatic

» Monitor for development of symptoms:
  > A 12-hour symptom-free period usually indicates that no intervention is necessary.

Symptomatic

» Gastric lavage/emesis/activated charcoal are contraindicated in all cases.
» Keep patient nil per mouth.
» Insertion of a NGT is contraindicated.
» Airway injury may necessitate endotracheal intubation.
» Endoscopic evaluation.

MEDICINE TREATMENT

» Prophylactic antibiotics are not indicated.
» Empiric steroid therapy is not indicated, however, based on endoscopy findings, may be appropriate (sub-specialist initiated).

For pain control:
See Chapter 20: Pain Control, section 20.1.1: Management of pain.

REFERRAL

» All symptomatic cases for endoscopic evaluation as soon as possible.

18.1.5 VOLATILE SOLVENTS

T53

DESCRIPTION

Inhalants include: spray-paint, glue and paint thinners that may contain hydrocarbons such as toluene and/or n-Hexane. If these are ingested, hydrocarbon poisoning with possible chemical pneumonitis must also be considered.

DIAGNOSTIC CRITERIA

» distinctive odour,
» discolouration around mouth/nose,
» palpitations,
» dizziness,
» cardiac arrhythmias,
» euphoria,
» headaches,
» progressive CNS depression,
» syncope,
» hypokalaemia,
» mucous membrane irritation, i.e. sneezing, coughing and tearing,
» GIT complaints, i.e. nausea, vomiting and abdominal pain,
» distal renal tubular acidosis, i.e. hyperchloraemic metabolic acidosis with a normal anion gap.
» Complications include peripheral neuropathy and hepatotoxicity.

GENERAL AND SUPPORTIVE MEASURES

» Stabilise airway, breathing and circulation.
» Perform a chest X-ray if respiratory symptoms present.
» Monitor patient for respiratory symptoms: if absent after 6–8 hours, child can be discharged.
» Correct fluid and electrolyte abnormalities.

MEDICINE TREATMENT

For agitation:

- Diazepam, IV/oral, 0.1–0.2 mg/kg.
  - Maximum dose: 10 mg.

For cardiac dysrhythmias, e.g. ventricular fibrillation, see Chapter 4: Cardiovascular System, section 4.1: Cardiac dysrhythmias.

REFERRAL

» Cardiac dysrhythmia.

18.1.6 ETHANOL POISONING

T51.0

DESCRIPTION

Ethanol is a selective CNS depressant at low concentrations, and a generalised depressant at high concentrations.

DIAGNOSTIC CRITERIA

Clinical

- » lack of co-ordination,
- » ataxia,
- » slurred speech,
- » gait disturbances,
- » drowsiness.
- » stupor,
- » coma,
- » hypoglycaemia,
- » convulsions,

Investigations

» Monitor blood glucose levels.

MEDICINE TREATMENT

Obtunded patients with hypoglycaemia:

- Dextrose 10%, IV, 2 mL/kg followed by 10% dextrose maintenance infusion. Titrate until blood glucose is controlled.

If patient responds to glucose administration, perform serial glucose levels to detect recurrent hypoglycaemia.

REFERRAL

» Persistent hypoglycaemia despite treatment.
» Depressed level of consciousness despite treatment.

18.1.7 IRON POISONING

T45.4

DESCRIPTION

Iron is widely available as an over-the-counter product and is commonly ingested accidentally by toddlers.

DIAGNOSTIC CRITERIA

» Toxicity is related to the ingested dose of elemental iron.
» A single dose of elemental iron > 20 mg/kg requires hospital assessment and management.

Clinical

- » gastrointestinal features,
- » shock and metabolic acidosis,
- » coma,
- » hepatic necrosis.

Elemental iron per preparation

Iron product	Strength	Elemental content	Elemental content per mL or tablet
Ferrous gluconate syrup	350 mg/5 mL	40 mg elemental iron per 5 mL	8 mg elemental iron per mL
Ferrous lactate drops	125 mg/mL	25 mg elemental iron per mL	25 mg elemental iron per mL
Ferrous sulphate compound tablets	170 mg	55 mg elemental iron per tablet	± 55 mg elemental iron per tablet

Categories of iron toxicity

Low risk	Medium risk	High risk
» No history of: • abdominal pain, • nausea, • vomiting, or diarrhoea. » Asymptomatic for 6 hours. • 20 mg/kg of elemental iron ingested.	» Clinical features of toxicity and serum iron > 300 mcg/dL (60 µmol/L).	Any of these features present: » Lethargy/decreased level of consciousness. » Metabolic acidosis. » Shock/hypotension. » Evidence of haematemesis or melaena. » Serum iron > 500 mcg/dL (90 µmol/L) irrespective of clinical features.

» Low risk patients are unlikely to have ingested enough iron to lead to serious poisoning and can be discharged.
» Admit high and medium risk patients.

Investigations

Medium and high risk:

» Abdominal X-ray; if history is uncertain or to assess the efficacy of gut decontamination.
» Arterial blood gas.
» Serum electrolytes.
» Liver function tests.
» Serum iron levels within 2–6 hours after ingestion.

GENERAL AND SUPPORTIVE MEASURES

General supportive treatment, including airway management if required.

MEDICINE TREATMENT

Medium and high risk

Fluid resuscitation:

- Sodium chloride 0.9%, IV, 20 mL/kg as an initial bolus followed by maintenance therapy.

If no signs of gastrointestinal dysfunction, e.g. perforation/haemorrhage:

» Whole bowel irrigation is recommended if:
  > > 60 mg/kg elemental iron has been ingested.
  > Modified-release preparations are ingested.
  > Undissolved tablets are still visible on abdominal X-ray.

LoE III[2]

Chelation therapy

All medium and high-risk cases (see table above).
For iron ingestion > 60 mg/kg of elemental iron:

- Desferrioxamine, IV, 15 mg/kg/hour as a continuous infusion until acidosis resolves and urine is no longer pink.
  - Beware of hypotension.

REFERRAL

» All medium and high-risk cases should be managed in a high care unit or ICU with access to serial serum iron measurement. Chelation therapy should preferably be initiated prior to urgent referral/transfer.

18.1.8 NEUROLEPTIC POISONING

T43.5

DESCRIPTION

Neuroleptic overdose may cause a depressed level of consciousness, hypotension, tachycardia and cardiac dysrhythmias and seizures.
Commonly used neuroleptics include chlorpromazine, haloperidol and phenothiazine anti-emetics (e.g. promethazine).

Acute dystonic reactions/extrapyramidal symptoms are distressing adverse reactions (sustained muscle spasms) occurring after an overdose or during chronic therapy with neuroleptics. A typical dystonic reaction includes hyperextension or hyperflexion of the limbs with abnormal posturing of the trunk. Other extrapyramidal symptoms may occur.
The neuroleptic malignant syndrome is uncommon following an overdose and is an idiosyncratic life threatening reaction, presenting with:

» temperature dysregulation,
» altered mental state,
» musculoskeletal effects (pipe-like rigidity).
» autonomic instability,
» diaphoresis,

DIAGNOSTIC CRITERIA

» Dystonic reactions.
» Other extrapyramidal symptoms.

GENERAL AND SUPPORTIVE MEASURES

» Observe asymptomatic patients for a minimum of 6 hours.
» Admit all symptomatic patients for continuous cardiac monitoring.

» Patients with hyperthermia, muscular rigidity or seizures are more at risk for rhabdomyolysis and subsequent renal failure; test urine for myoglobin (urine test strip for haemoglobin) and serum for creatine kinase and creatinine.

MEDICINE TREATMENT

- Activated charcoal, see section 18.1: Poisoning.

For acute dystonic reactions:

- Biperidin, IV, slow injection.
  - If < 1 year of age: 1 mg.
  - If 1–6 years of age: 2 mg.
  - If 6–10 years of age: 3 mg.

If concomitant significant anticholinergic findings are present, such as fever and dry skin and mucous membranes, a benzodiazepine is preferred.

REFERRAL

» Patients with neuroleptic malignant syndrome.
» Patients with conduction abnormalities (prolonged QT).
» Patients with acute kidney injury.

18.1.9 ORGANOPHOSPHATE POISONING

T60.0
*Notifiable condition

DESCRIPTION

Organophosphates are potent inhibitors of acetylcholinesterase.

Note: Where the history is of an unspecified rat poison or pesticide ingestion, consider other active ingredients such as amitraz and 'super-warfarin' anticoagulants.

DIAGNOSTIC CRITERIA

Clinical

» Acute cholinergic toxidrome has central and peripheral effects.

Peripheral effects:

> Muscarinic: diarrhea, vomiting, urinary incontinence, lacrimation, pinpoint pupils, bronchorrhoea, bronchoconstriction, hyper-salivation, sweating, bradycardia, hypotension.

> Nicotinic: tachycardia, hypertension, dilated pupils, muscle weakness and fasciculations.

Cardiac features of bradycardia and tachycardia depend on whether muscarinic or nicotinic effects predominate.

Central effects:
> Nicotinic: confusion, coma, convulsions.

» Intermediate syndrome can occur within 1–4 days after recovery from cholinergic crisis. Patients develop weakness or paralysis predominantly affecting the muscles of respiration (including the neck flexors and bulbar muscles) and proximal limb muscles, sparing the distal muscles. Cranial nerves may also be affected. Supportive care is the mainstay of therapy.

LoE III[6]

» Signs depend on dose and route of exposure (vapour or liquid) as well as the time exposed (vapour).

Investigations

» Decreased levels of pseudocholinesterase.
  > Use for confirmation only.
  > Do not wait for levels before treating.

GENERAL AND SUPPORTIVE MEASURES

» Ensure use of personal protective equipment.
» Remove all patient's clothing and wash clothes thoroughly.
» Wash affected skin with soap and water.
» Suction secretions frequently.
» Monitor respiratory function closely and ventilate if necessary. If using suxamethonium or mivacurium for intubation, consider that metabolism may be delayed and prolonged respiratory support may be required. Use alternative agents if possible.

LoE III[7]

» Also monitor heart rate, pupillary size and level of consciousness.

MEDICINE TREATMENT

For bradycardia, bronchorrhoea or bronchospasm:

- Atropine bolus, IV, 0.05 mg/kg.
  - Reassess after 3–5 minutes for evidence of atropinisation as indicated by reduced bronchial secretions, dry skin, increasing heart rate and blood pressure, and dilating pupils (note: pupil dilatation may be delayed).
- Give repeated atropine boluses, incrementally doubling the dose until adequate clinical response achieved, e.g.:
  - 10 kg child: 0.5 mg, 1 mg, 2 mg, 4 mg, 8 mg, (no maximum dose).
  - If no clinical response, give double the dose.
  - If some response, give the same or reduced dose.

» Follow with infusion. Calculate the total dose of atropine given as boluses (as described above). Give 10–20% of this dose per hour.

» Reassess frequently and adjust atropine infusion as follows:
  > Bronchial secretions, bronchospasm or bradycardia recur—increase dose.
  > Good control of bronchial secretions and signs of atropine overdose (tachycardia, dilated pupils, agitation, pyrexia, reduced bowel sounds and urinary retention): decrease dose.
  > No recurrence of bronchial secretions and no signs of atropine overdose: reduce dose slowly.

Note: Do not stop atropine infusion abruptly, but wean over at least 24 hours. Tachycardia and dilated pupils are not contraindications for giving atropine in the acute resuscitation setting.

Glycopyrrolate is not a substitute for atropine. However, it does not penetrate the CNS and therefore, may be useful in patients who are suffering from central cholinergic toxicity as a result of atropine but still require control of peripheral muscarinic symptoms.

- Glycopyrrolate, 0.025 mg/kg, IV.

LoE III [6]

Patients with organophosphate poisoning may be extremely agitated or develop seizures due to central toxicity. Treat both with a benzodiazepine. See Chapter 13: The Nervous System, section 13.3: Status epilepticus (convulsive).

REFERRAL

» All severe cases for ICU care.

18.1.10 OPIOID POISONING

T40.2

DESCRIPTION

Codeine is a common drug of abuse.

The duration of action of morphine is 3–6 hours. Other oral agents, e.g. codeine and long acting morphine, demonstrate a delayed effect of up to 4–12 hours.

DIAGNOSTIC CRITERIA

» Altered level of consciousness.
» Classic triad of CNS depression, respiratory depression and pinpoint pupils.
» Hypotension, hypothermia, bradycardia and hyporeflexia.
» Vomiting is common with the risk of aspiration, especially in patients with depressed level of consciousness.

Note: Symptoms may take time to develop. May be awake and alert in the early phase 1–2 hours after ingestion. Neonates of mothers using heroin may present with withdrawal, manifested as jitteriness.

GENERAL AND SUPPORTIVE MEASURES

» Airway protection is a priority.
» Supportive care, ventilate with bag-mask device.
» Monitor oxygen saturation constantly.
» Observe for urinary retention.

MEDICINE TREATMENT

- Activated charcoal.

If respiratory depression or depressed level of consciousness:

» Provide airway support.
» Ventilate until $PaCO_2$ normal.

- Naloxone, IV, 0.1 mg/kg:
  - If no response after 5 minutes, repeat dose and titrate according to response.
  - Duration of action of naloxone is 20–30 minutes.
  - If repeated doses of naloxone are necessary, a continuous IV infusion of naloxone can be instituted (0.01 mg/kg/hour).

CAUTION

All patients treated with naloxone should be observed for at least 12 hours for relapse, especially if a long acting opioid has been ingested.

REFERRAL

» Patients requiring multiple doses of naloxone.

18.1.11 PARACETAMOL POISONING

T39.1

DESCRIPTION

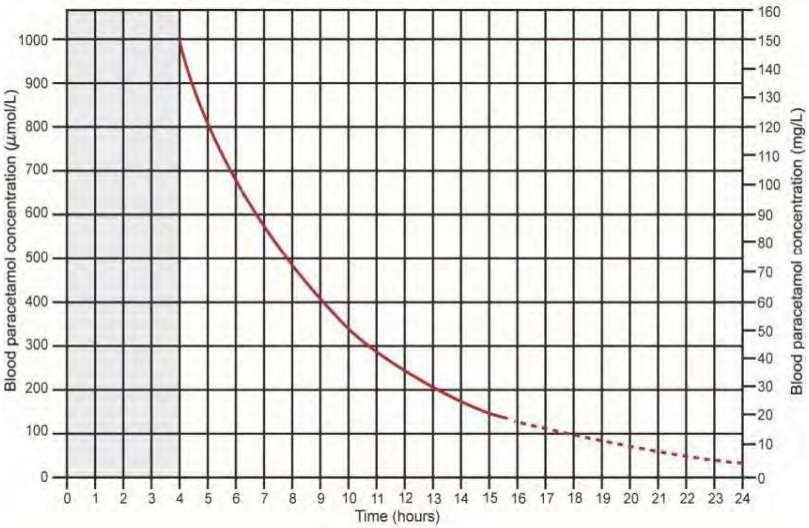
Poisoning due to paracetamol by adolescents is generally due to intentional ingestion. The accidental ingestion of paracetamol elixir preparations by toddlers very rarely causes toxicity. Toxicity can be due to acute ingestions or repeated supratherapeutic ingestion (RSTI). Toxicity due to IV paracetamol may also occur.

Patients with predisposing risk factors for hepatotoxicity, so-called ‘high-risk’ patients (glutathione deficiency, liver disease, use of enzyme- inducing drugs, patients with recent illness or dehydration) may experience toxicity at lower doses.

DIAGNOSTIC CRITERIA

» An acute ingestion in excess of 200 mg/kg per 24-hour period in healthy children is potentially toxic.
» Serum paracetamol concentration must be measured at least four hours following ingestion.
» Use nomogram to assess risk of toxicity.

Paracetamol treatment nomogram



Source: Daly FF, Fountain JS, Murray L, Graudins A, Buckley NA; Panel of Australian and New Zealand clinical toxicologists. Guidelines for the management of paracetamol poisoning in Australia and New Zealand—explanation and elaboration. A consensus statement from clinical toxicologists consulting to the Australasian poisons information centres. Med J Aust. 2008 Mar 3;188(5):296-301.

» Cautions for use of this chart:
  > The time co-ordinates refer to time since ingestion.
  > Serum levels drawn before 4 hours may not represent peak levels.
  > Use the graph only in relation to a single acute ingestion.
  > Do not use when there is a history of RSTI, or delayed presentation (> 24 hours post-ingestion).

Repeated supratherapeutic ingestions (RSTI)

Can occur with repeated high doses of the same product or the concurrent use of multiple paracetamol-containing products.

RSTI is defined as:

  > > 200 mg/kg or 10 g (whichever is less) over a single 24-hour period.
  > > 150 mg/kg or 6 g (whichever is less) per 24-hour period for the preceding 48 hours.
  > > 100 mg/kg or 4 g/day (whichever is less) per 24-hour period for

more than 48 hours AND patients with symptoms suggestive of liver injury.

This nomogram is not designed for use in RSTI. Management of RSTI is complex; contact the Poisons Information Helpline for advice.

LoE III[8]

Investigations

If toxic dose ingested or patient symptomatic, do:

» Serum paracetamol level.
» Baseline electrolytes.
» ALT.
» INR, if abnormal ALT or showing signs of hepatotoxicity.

MEDICINE TREATMENT

Acute ingestion:

» Gastric lavage is unlikely to be required.
» Activated charcoal can be considered for large intentional overdoses.
» For acute ingestion, initiate treatment with N-acetyl cysteine (NAC) if the blood paracetamol concentration for the time since ingestion falls to the right of the curved line on the nomogram.
» If a patient has taken a potentially toxic dose [≥ 10 g (20 tablets) or ≥ 200 mg/kg, whichever is smaller] AND the serum paracetamol level results will not be available before 8 hours post-ingestion OR the patient presents > 8 hours post-ingestion, do not delay initiation of NAC. It can always be stopped if the serum level plotted on the nomogram does not indicate its continued use.
» If the time of ingestion is unknown, start treatment for any detectable level of paracetamol or any elevation of AST or ALT.

RSTI:

Management of RSTI is complex; contact the Poisons Information Helpline for advice.

- N-Acetylcysteine, IV:
  20-hour regimen:
  - 200 mg/kg in 7 mL/kg of 5% dextrose over 4 hours.
  - Followed by 100 mg/kg in 14 mL/kg 5% dextrose over 16 hours.

  Repeat infusions according to second dose.

REFERRAL

Patients with severe hepatotoxicity as indicated by any of the following:

» INR > 2 IU at 24 hours or > 3 IU at any time after overdose,
» pH < 7.3, bicarbonate < 18 mmol/L or lactate > 3 mmol/L,
» hypotension despite adequate fluid resuscitation,
» encephalopathy,
» creatinine > 200 µmol/L.

18.1.12 PETROCHEMICAL POISONING

T53.6

DESCRIPTION

Accidental ingestion of paraffin, particularly by toddlers, is common in South Africa.

DIAGNOSTIC CRITERIA

Clinical

» Paraffin is volatile and inhalation of the fumes or aspiration of liquid can cause respiratory distress due to chemical pneumonitis.
» CNS symptoms: depressed level of consciousness.

Investigations

» Chest X-ray if respiratory distress present.

GENERAL AND SUPPORTIVE MEASURES

CAUTION
Do not attempt gastric lavage.

» Observe patient for up to 6–8 hours if asymptomatic.
» Administer oxygen, if necessary.
» Remove contaminated clothes and wash skin to prevent chemical burns.

MEDICINE TREATMENT

If infection develops 48 hours after ingestion:

» See Chapter 15: Respiratory, section 15.1.1: Pneumonia.

REFERRAL

» For ventilatory support.

18.1.13 SALICYLATE POISONING

T39.0

DESCRIPTION

Salicylate poisoning may result from oral and/or topical exposure. Salicylate products vary widely in concentration, e.g. oil of wintergreen is almost 100% methylsalicylate. As little as 4 mL of oil of wintergreen may be fatal in a child.

DIAGNOSTIC CRITERIA

Clinical

» Ingestion of less than 150 mg/kg of aspirin will not cause toxicity except in a child with hepatic or renal disease.
» Ingestion of 150–300 mg/kg of aspirin may result in mild to moderate toxicity.

» Ingestion of > 300 mg/kg of aspirin may result in severe toxicity.
» Ingestion of > 500 mg/kg of aspirin should be considered a potentially lethal dose.
» Features include:
  » fever,
  » nausea,
  » epigastric pain,
  » vomiting,
  » tinnitus,
  » hyperventilation,
  » renal failure,
  » hypoglycaemia,
  » CNS depression,
  » respiratory alkalosis (initially) followed by metabolic acidosis.

» Monitor blood gases and electrolytes, urine output and urine pH.
» Monitor salicylate levels if possible (do not always correlate with clinical severity):
  > Asymptomatic: peak plasma salicylate level of < 20 mg/dL (< 30 mg/dL in adolescents).
  > Mild toxicity: Peak plasma salicylate level 20 to <45 mg/dL in child (30 to <60 mg/dL in adolescents).
  > Moderate toxicity: Peak plasma salicylate 45 to 70 mg/dL in child (60 to 80 mg/dL in adolescents).
  > Severe toxicity: Peak plasma salicylate level > 70 mg/dL in child (> 80 mg/dL in adolescents).
» Serial monitoring until declining levels are documented.
» Monitor and treat hypoglycaemia; patients with normoglycaemia may still be neuroglycopaenic.

GENERAL AND SUPPORTIVE MEASURES

» Consider gastric lavage, see section 18.1: Poisoning.
» Correct hydration.

MEDICINE TREATMENT

After gastric lavage:

- Activated charcoal.
  - May be used for up to 12 hours due to delayed gastric emptying or if sustained-release/enteric-coated preparations were ingested.

Urinary alkalinisation

If metabolic acidosis (pH < 7.3) is present and/or salicylate levels are high, give:

- Sodium bicarbonate 8.4%, IV, 1 mL/kg bolus dose to increase pH to 7.4, administered over 1 hour (with maintenance fluid).
  - Repeat bolus doses, if necessary, to maintain urine pH above 7.5.
  - Monitor urine pH hourly and potassium levels 3 hourly.

For hydration:

- 5% dextrose saline, IV.

For bleeding:

- Vitamin K_1, IV/oral, 1–5 mg/dose administered slowly 6 hourly.

Note: Intravenous solution can be used orally.

REFERRAL

» Severe cases for ICU care: if arterial pH remains < 7.2, refer for urinary alkalinisation and possible haemodialysis.

18.1.14 BENZODIAZEPINE POISONING

T42.4

DESCRIPTION

Young children or toddlers are typically involved in accidental exposure and ingest small amounts of sedatives.

Adolescents may ingest large amounts during suicide, suicidal gesture or for recreational use.

DIAGNOSTIC CRITERIA

Clinical

» Cardiorespiratory depression.
» Decreased level of consciousness.

Investigations

» Serum drug levels are of no value in the acute treatment phase.
» Urine test: may have medico-legal implications.

GENERAL AND SUPPORTIVE MEASURES

» If there is respiratory depression, intubate, ventilate and transfer.
» Only supportive treatment is necessary in most patients.

REFERRAL

» Respiratory depression.

18.1.15 SULFONYLUREA POISONING

T38.3

DESCRIPTION

Sulfonylureas may cause severe and protracted hypoglycaemia. The half-life of the sulfonylureas varies:

» Glibenclamide	T½ = 10 hours
» Gliclazide	T½ = 10–12 hours
» Glimepiride	T½ = 5–8 hours

DIAGNOSTIC CRITERIA

Clinical

» Coma and seizures.
» Profound hypoglycaemia, usually within 4 hours of ingestion.

Investigations

» Glucose monitoring is the mainstay of diagnostic testing.

GENERAL AND SUPPORTIVE MEASURES

» Observe for at least 24 hours, even if a single tablet is ingested.
» Glucose-containing fluid orally.

MEDICINE TREATMENT

- Activated charcoal; see section 18.1: Poisoning.

If symptoms of hypoglycaemia are present or blood glucose is below 2.6 mmol/L:

- Dextrose 10% (2 mL/kg), IV bolus followed by 10% dextrose maintenance infusion. Titrate until blood glucose is controlled.

If desired response not achieved,

ADD

- Octreotide 1–1.5 mcg/kg IV or SC.

Note: Corticosteroids are not indicated.

REFERRAL

» Patients not responding to intravenous glucose.

18.1.16 SYMPATHOMIMETIC AGENT POISONING

T43.6/F14

DESCRIPTION

Pseudoephedrine in decongestants, methylphenidate and illicit drugs such as cocaine and amphetamines ('Tik') are sympathomimetic agents. These agents are frequently abused as recreational drugs.

DIAGNOSTIC CRITERIA

Clinical

» hypertension,
» tachycardia,
» tachypnoea,
» agitation,
» psychosis,
» dilated pupils,
» diaphoresis,
» hyperthermia: effects of sympathomimetics that predispose to hyperthermia include:

- peripheral vasoconstriction and impaired cutaneous heat loss,
- agitation,
- seizures,
- increased muscle activity,
- impaired behavioural response.

» With cocaine toxicity, cardiovascular manifestations predominate, including:
- supraventricular and ventricular dysrhythmias,
- myocardial ischaemia.

» Neonates of mothers using cocaine may present with withdrawal signs, manifested by jitteriness.

Investigations

» ECG monitoring to evaluate dysrhythmias.

GENERAL AND SUPPORTIVE MEASURES

» Admit all seriously ill children to ICU.
» Maintain hydration.
» Cooling for hyperthermia.
» Mildly toxic patients require no specific treatment.

MEDICINE TREATMENT

- Activated charcoal, see section 18.1: Poisoning.

For agitation and tachycardia:

- Diazepam, IV/oral, 0.1–0.2 mg/kg.
  - Maximum dose of 10 mg.

For severe hypertension:

See Chapter 4: Cardiovascular System, section 4.11.1: Hypertension, acute severe.

For seizures:

See Chapter 13: The Nervous System, section 13.3: Status epilepticus (convulsive).

REFERRAL

» Status epilepticus requiring ICU.
» Hypertensive crisis.

18.1.17 ISONIAZID POISONING

T37.1

DESCRIPTION

INH interferes with pyridoxine and niacin metabolism, leading to impaired synthesis of gamma aminobutyric acid (GABA). Acute poisoning, which may follow intentional or accidental ingestions, may be severe.

DIAGNOSTIC CRITERIA

Clinical

Triad of refractory seizures, metabolic acidosis and coma within 2–3 hours of ingestion. Hyperthermia and rhabdomyolysis can develop after prolonged seizure activity.

Investigations

» Metabolic acidosis - high anion gap due to lactate accumulation.

GENERAL AND SUPPORTIVE MEASURES

» Respiratory and circulatory support.

MEDICINE TREATMENT

- Activated charcoal, see section 18.1: Poisoning.

For seizures:

- Pyridoxine is the primary treatment of seizures and coma, which once controlled, should help resolve metabolic acidosis.
- Asymptomatic patients presenting within 2 hours, give an initial prophylactic dose of 70 mg/kg of oral pyridoxine up to a maximum dose of 5 g.
- Symptomatic patients with significant symptoms or seizures: replace INH with pyridoxine gram-for-gram, up to a maximum of 5 g.
- Oral pyridoxine 25 mg tablets can be crushed and given with fluids via nasogastric tube.
- If seizures recur, repeated doses of pyridoxine may be given up to a maximum daily dose of 15-30 g.

Note: Benzodiazepines and phenobarbitone may be used to control seizures (whilst pyridoxine is being prepared/given). Phenytoin should be avoided (due to potential cardiotoxicity).

LoE III[4]

Metabolic acidosis should improve with seizure control, but additional sodium bicarbonate may be required.

REFERRAL
» Refractory seizures.

18.1.18 THEOPHYLLINE POISONING
T48.6

DESCRIPTION
Agents such as aminophylline and caffeine have similar features in overdose. Sustained release preparations can cause prolonged toxicity. Toxicity can occur with therapeutic dosing.

DIAGNOSTIC CRITERIA
Clinical
» Mainly affects the gastrointestinal, cardiovascular and central nervous systems:
  > Central nervous system: agitation, tremor, seizures, coma, hyperventilation.
  > Gastrointestinal tract: nausea and vomiting.
  > Cardiovascular: tachycardia, arrhythmias, hypotension.

Investigations
» Serum levels; a theophylline level 111 µmol/L (> 20 mg/L) is considered toxic.
» Hyperglycaemia.
» Hypokalaemia.
» Respiratory alkalosis and/or metabolic acidosis.

GENERAL AND SUPPORTIVE MEASURES
» Observe all patients who have ingested 10 mg/kg or more of theophylline for at least 4 hours for a normal release preparation and at least 12 hours for a sustained release preparation.
» Manage hypotension.
» Cardiac monitoring.
» Potassium levels should be monitored and replaced if required.

MEDICINE TREATMENT
- Ondansetron for vomiting. See Chapter 21: Palliative care.
- Activated charcoal. Repeated doses may be required to enhance elimination of theophylline. See section 18.1: Poisoning.
- **Note**: Phenytoin should be avoided (due to potential cardiotoxicity).

LoE III[4]

REFERRAL
Refer for consideration of haemodialysis, severe poisonings as evidenced by:
» serum theophylline > 555 µmol/L (> 100 mg/L),
» seizures,

» refractory shock,
» life-threatening dysrhythmias,
» rising theophylline level and/or clinical deterioration despite optimal care.

18.1.19 AMITRAZ POISONING

T60.9
*Notifiable condition.

DESCRIPTION

Amitraz is a pesticide used in tick-dips for animals and as an insecticide in crop sprays. Liquid formulations often contain solvents that may cause additional clinical effects. Significant skin contact may lead to systemic effects.

Note: Where the history is of an unspecified rat poison or pesticide ingestion, consider other active ingredients such as 'super-warfarin' anticoagulants and organophosphates.

DIAGNOSTIC CRITERIA

Clinical

Symptoms occur between 30 minutes to 4 hours.

» Gastrointestinal: vomiting.
» Central nervous system: ataxia, drowsiness (leading to coma), seizures. No excessive secretions. Pinpoint pupils or dilated pupils may be present.
» Cardiovascular: bradycardia, hypotension (or hypertension).
» Respiratory depression, or tachypnoea, aspiration and chemical pneumonitis.
» Hypothermia and hyperglycaemia are common.

Amitraz poisoning can be confused with organophosphate poisoning, but it does not cause excessive sweating and salivation, urinary and faecal incontinence or muscle fasciculation which are seen with organophosphate poisoning. Furthermore, organophosphate toxicity results in reduced serum pseudocholinesterase levels.

Investigations

» Acidosis (respiratory or metabolic).
» Liver enzymes.
» Chest X-ray, if respiratory symptoms.

GENERAL AND SUPPORTIVE MEASURES

» Decontaminate skin and clothes if applicable.
» Monitoring (blood pressure, pulse, respiration, level of consciousness, temperature, blood gas, blood sugar).

- > **Asymptomatic:** observe for 4 hours.
- > **Symptomatic:** supportive treatment as required.

MEDICINE TREATMENT

- Activated charcoal, see section 18.1: Poisoning.

Specific treatment should only be used if there is inadequate response to standard resuscitation measures.

- Atropine may be used for severe bradycardia.

REFERRAL

» Severe cases requiring intensive care.

18.1.20 ANTIRETROVIRAL AGENTS POISONING

T37.5

DESCRIPTION

» Limited data is available regarding overdose of these medicines.
» Toxicological effects are generally extensions of adverse effects.

GENERAL MEASURES

» Monitor FBC, serum electrolytes, renal and liver function.
» Monitor serum lipase in patients with abdominal pain.
» Lactic acid and serum pH should be monitored in acidotic patients.

TREATMENT

» There are no specific antidotes.
» Treatment is symptomatic and supportive.

18.1.21 CARBON MONOXIDE POISONING

Y17

DESCRIPTION

Poisoning caused by accidental or intentional exposure to fires in poorly ventilated areas, combustion engines, faulty stoves and faulty heating systems.

Patients present with:

» dizziness,
» headache,
» seizures and other CNS symptoms,
» nausea and vomiting,
» chest pain,
» tachycardia,
» high arterial carboxyhaemoglobin levels,
» impaired level of consciousness,
» retinal haemorrhages,
» respiratory alkalosis (mild),
» metabolic acidosis (severe).

Note: There may be a normal arterial PaO_2, but low oxygen saturation on pulse oximetry. Neither are useful in assessing severity of carbon monoxide poisoning. Ideally, a blood gas sample should be sent for co-oximetry to specifically detect carboxyhaemoglobin levels.

GENERAL MEASURES

» Remove patient from toxic environment.
» Ventilation may be needed in deeply comatosed patients.
» Monitor ECG and neurological status.

MEDICINE TREATMENT

» Give 100% oxygen via positive pressure facemask.
» Evidence for the benefit of hyperbaric oxygen therapy is unclear, therefore, it cannot be routinely advised.

For seizures:

- Benzodiazepines. See Chapter 13: The Nervous System, section 13.3: Status epilepticus (convulsive).
- **Note**: Phenytoin should be avoided (due to potential cardiotoxicity).

LoE:III[4]

Metabolic acidosis:
Metabolic acidosis shifts the oxygen-dissociation curve to the right and, therefore, aids in maintaining tissue oxygenation despite reduced haemoglobin carrying capacity. Metabolic acidosis should only be treated if profound and persistent, following standard treatment protocols.

Patients should be followed up after discharge for the persistence of neurocognitive symptoms.

In patients not responding to 100% oxygen, consider exposure to cyanide during the fire and refer patient urgently.

18.2 ENVENOMATION

» The management of severe envenomation, particularly by snakes and scorpions, is complex.
» Please contact the Poisons Information Helpline for advice.
  - **0861 555 777.**

18.2.1 INSECT BITES AND STINGS

T63.4 + (X29.99/X23.99) + External Cause Code (V,W,X,Y)

DESCRIPTION

Toxicity due to insect bites and stings usually results in local effects only; systemic effects are rare. Occasionally, hypersensitivity reactions are

encountered, which may vary from minor local inflammation to acute anaphylaxis.

Multiple bee stings can result in toxicity and may require ICU care.

GENERAL MEASURES

» Allergic reactions may be acutely life-threatening.
» Patients with multiple stings may develop delayed systemic toxicity. Beware of premature discharge from the healthcare facility.

MEDICINE TREATMENT

For anaphylaxis:
See Chapter 1: Emergencies and Trauma, section 1.1.3: Anaphylaxis/Anaphylactic reactions.

For pain:

- Paracetamol, oral, 15 mg/kg/dose 6 hourly as required.

18.2.2 SCORPION STINGS

T63.2

DESCRIPTION

Some scorpion species can cause serious systemic toxicity. Thick-tailed scorpions with small pincers are extremely toxic, resulting in both local and systemic features. Thin-tailed scorpions with large pincers are much less toxic and usually cause local symptoms only.

DIAGNOSTIC CRITERIA

» Pain and paraesthesia occur immediately after envenomation.
» Autonomic and motor findings may differentiate scorpion stings from other causes of pain.
» In severe cases, cranial nerve dysfunction, blurred vision, pharyngeal muscle incoordination, drooling and respiratory compromise can occur.
» Excessive motor activity may present as restlessness, or uncontrollable jerking of extremities.
» Nausea, vomiting, tachycardia and severe agitation can also occur.
» Other serious effects include cardiac dysfunction, pulmonary oedema, and pancreatitis.

GENERAL AND SUPPORTIVE MEASURES

» If unidentified scorpion or confirmed thick-tailed scorpion, observe for a minimum of 12 hours in hospital.
» Monitor airway, breathing and circulation.
» Ventilatory support may be required.

MEDICINE TREATMENT

For pain:

- Paracetamol, oral, 15 mg/kg/dose, 6 hourly as required.

Very painful scorpion stings

- Lidocaine (lignocaine) 2%, 2 mL injected around the sting as a local anaesthetic.

Caution
Opiates increase the risk of respiratory depression and if required, should only be used with caution in severe uncontrolled pain.

For muscle cramps:

- Calcium gluconate 10%, IV, 0.5 mL/kg by slow intravenous injection.
  - Give 0.5–1 mL/minute.
  - Monitor ECG.
  - Monitor response and repeat as needed.

If not immunised in the past 5 years:

- Tetanus toxoid, IM, 0.5 mL.

Complete course in previously unvaccinated patients.

Antivenom therapy

Antivenom therapy is recommended only in cases with systemic signs.

Obtainable from South African Vaccine Producers (Tel.: +2711 386 6063/2/78 or after hours 071 680 9897 or 082 884 2971). See full details in the package insert.

- Scorpion antivenom, slow IV, 10 mL administered over 3–5 minutes. If consistency is too thick, can be diluted in sodium chloride or 5% dextrose.

CAUTION
Never administer antivenom without being prepared to manage acute anaphylaxis.

REFERRAL

» Severe cases requiring intensive care.

18.2.3 SNAKEBITE

T63.0

DESCRIPTION

The effects of snakebites may be cytotoxic, neurotoxic and/or haemotoxic.

The overall effect is determined by the predominant toxin in the snake venom.

In the majority of cases, the species of snake is unknown. The patients can be divided into:

» no evidence of bite, no envenomation,
» evidence of bite, minor envenomation, i.e. fang marks, minimal pain, minimal swelling and no systemic signs,
» evidence of serious envenomation.

DIAGNOSTIC CRITERIA

Cytotoxic venom

» Puff adder, spitting cobra, gaboon adder.
» Rinkhals has both cytotoxic and neurotoxic features.
» Venom causes severe local damage to tissues and vascular endothelium.
» Severe swelling and local necrosis occurs.

Neurotoxic venom

» Mamba, non-spitting cobra, e.g. Cape cobra, berg adder.
» Rinkhals has both cytotoxic and neurotoxic features.
» Venom causes a paresis and paralysis of skeletal muscles.
» Paralysis of respiratory muscles with respiratory failure may occur.
» Preceded by severe pain and paraesthesias.
» Ophthalmoplegia occurs when ocular muscles become paralysed.
» Speech and swallowing may be affected.
» Signs and symptoms start within 15–30 minutes.

The bite site can be rather unremarkable, except for the berg adder, which also has some swelling.

Haemotoxic venom

» Boomslang, vine snake.
» Venom may cause: spontaneous bleeding, headache, dizziness, fainting.

GENERAL AND SUPPORTIVE MEASURES

» Patients with no evidence of bite and patients with evidence of bite but only minor envenomation should be admitted for observation. No anti-venom is indicated.
» Do not suck or cut the wound.
» Do not apply tourniquet.
» Where serious envenomation is suspected, immediate treatment includes:
  > minimising movement of affected limb,
  > emergency treatment by bandaging affected limb with crepe bandage without compromising blood supply,
  > rapid transportation to a facility with antivenom available is the most important principle of pre-hospital care,

- optimal therapy consisting of placing the patient at rest with the affected body part raised to the level of the heart,
- stabilising circulation and blood pressure.

» For cytotoxic envenomation, surgical intervention, i.e. decompression surgery for established compartment syndrome and debridement of necrotic tissue should only be done when absolutely necessary and as conservatively as possible.

» For neurotoxic envenomation, ventilatory and cardiovascular support may be needed in an ICU.

MEDICINE TREATMENT

Analgesia:

- Paracetamol, oral, 15 mg/kg/dose, 6 hourly as required.

Avoid NSAIDS and aspirin due to concerns of coagulopathy.
Opioids can be used for severe pain, but should be used cautiously in neurotoxic snakebite.

All patients not immunized within the past 5 years:

- Tetanus toxoid, IM, 0.5 mL.

In children with a penetrating wound and who are not completely immunised:

- Tetanus immunoglobulin, IM.
  - If < 5 years of age: 75 IU.
  - If 5–10 years of age: 125 IU.
  - If > 10 years of age: 250 IU.

Clean wound:

- Chlorhexidine 0.05% solution in water.

Antibiotics are seldom needed, except for secondary infection.

Antivenom therapy

Two types of snake antivenom are available:

- Polyvalent antivenom: active against puff adder, gaboon adder, rinkhals, green mamba, black mamba, Jameson's mamba, Cape cobra, forest cobra, snouted cobra, Mozambique spitting cobra.
- Monovalent antivenom: for boomslang bites only.

» Obtainable from South African Vaccine Producers (Tel.: +2711 386 6063/2/78 or afterhours 071 680 9897 or 082 884 2971). See full details in the package insert.

Indications:

» Consider antivenom in children who are persistently and severely affected even after the first day.

» Polyvalent antivenom:
  > Positively identified snake included in polyvalent antivenom AND evidence of severe cytotoxic envenomation.
  > Unidentified snake and evidence of progressive severe cytotoxic envenomation:
    - Painful swelling of whole hand/foot within 1 hour.
    - Swelling to the elbow/knee in less than 6 hours.
    - Swelling of the whole limb in less than 12 hours.
    - Swelling progression > 2.5 cm per hour.
    - A threatened airway due to swelling.
    - Evidence of complication, e.g. compartment syndrome.
    - Systemic evidence of severe cytotoxicity.
      - Shock.
      - Haematological abnormalities: INR > 1.5 IU, Hb < 8 g/dL, thrombocytopaenia (< 100 x 10^9/L) or leukocytosis (> 10 x 10^9/L).
      - Arrhythmias (rare).
  > Any signs of neurotoxicity, i.e. weakness or paralysis.

» Monovalent antivenom:
  > Positively identified boomslang AND clinical or laboratory features of coagulopathy.
  > Unidentified snakebite with evidence of coagulopathy AND no swelling at the bite site.

Administration and antivenom dose:

CAUTION
Never administer antivenom without being prepared to manage acute anaphylaxis.

» In most cases patients do not need and should not be given antivenom.
» Adverse reactions to antivenom are common and may be severe.
» Pre-medication with adrenaline (epinephrine) may reduce the risk of severe adverse reactions to polyvalent snake antivenom.

- Adrenaline (epinephrine) 1:1000, SC, 0.01 mL/kg, to a maximum of 0.25 mL.

» The dose of antivenom is the same for adults and children.
» Monitor for any deterioration in respiratory function as patients may need ventilation whether or not polyvalent antivenom has been given.
» Antivenom should be given as soon as possible, however, administration may be considered even as late as 48–72 hours after the bite, if there is continued clinical deterioration indicating ongoing venom activity.

- Polyvalent snake antivenom, IV.
  - 1 ampoule contains 10 mL antivenom.
    - Cytotoxic snakebite: give 50 mL.
    - Neurotoxic snakebite: give 80–100 mL (and up to 200 mL in black mamba bites).
  - Dilute in sodium chloride 0.9%, 50–100mL.
  - Administer IV, over 30 minutes.

LoE III[9]

- Boomslang monovalent antivenom:
  - Slow IV, 10 mL administered over 3–5 minutes.

  OR
  - IV infusion, 10–20 mL diluted in sodium chloride 0.9% or dextrose 5%, 50–100 mL administered over 5–10 minutes.

Spontaneous systemic bleeding should stop within 15–30 minutes and blood coagulability be restored within 6 hours.

» After administration of antivenom, observe patient for 24 hours.
» Contact the Poisons Information Helpline for further advice.
» Correct anaemia and bleeding tendency.

REFERRAL

» Snakebite with neurotoxic or haemotoxic manifestations may need intensive care.

18.2.4 SNAKE VENOM IN THE EYE

S05.9 + (X20.99)

DESCRIPTION

Direct or indirect snake venom exposure to the eye, particularly from various species of spitting cobras and rinkhals, can cause chemical injury with varying clinical presentations ranging from periocular swelling and mild conjunctival and corneal inflammation to frank corneal ulceration and perforation with eventual blindness.

GENERAL MEASURES

» Instill local anaesthetic and promptly perform copious irrigation for 15–20 minutes to dilute or remove the toxin with sodium chloride 0.9%.
» Apply chloramphenicol ointment and cover the affected eye with an eye patch.
» **Note**: Do not instill polyvalent antivenom in the eye or give systemically.

LoE:III[9]

REFERRAL

» Refer all patients to an ophthalmologist.

18.2.5 SPIDER BITES

T63.3

The vast majority of spiders are not harmful to humans.

18.2.5.1 SPIDER BITES, NEUROTOXIC (BUTTON/WIDOW SPIDERS)

DESCRIPTION

The term latrodectism is used to describe the systemic symptoms and signs following envenomation by the bite of the *Latrodectus* species (button or widow spiders). Most cases are caused by the bite of a black button spider; brown button spider bites are usually milder and characterized by local symptoms and signs.

DIAGNOSTIC CRITERIA

» Bites are felt immediately as a pinprick sensation, followed by increasing local pain that may spread to include the entire extremity.
» Typical target lesions, i.e. erythematous ring surrounding a pale center.
» Spasms in large muscle groups, abdominal pain or rigidity, progressing to generalised pain involving the trunk and abdomen have been described.
» Paraesthesia of hands and feet.
» Sweating and anxiety may occur.
» Priapism may occur, especially in children.

GENERAL AND SUPPORTIVE MEASURES

» Supportive care of airway, breathing and circulation.

MEDICINE TREATMENT

Analgesia:

- Paracetamol, oral, 15 mg/kg/dose, 6 hourly as required.

For pain and muscle cramps:

- Calcium gluconate 10%, IV, 0.5 mL/kg by slow intravenous injection.
  - Give 0.5–1 mL/minute.
  - Monitor ECG and respiration.

For severe envenomation (if systemic symptoms are present):

- *Latrodectus* spider antivenom, IV infusion, 5–10 mL diluted in sodium chloride 0.9% or dextrose 5%, 50–100 mL administered over 5–10 minutes.
  - Obtainable from South African Vaccine Producers (Tel.: +2711 386 6063/2/78 or afterhours 071 680 9897 or 082 884 2971). See full details in the package insert.

CAUTION
Never administer antivenom without being prepared to manage acute anaphylaxis.

18.2.5.2 SPIDER BITES, NECROTIC ARACHNIDISM

T63.3

DESCRIPTION

Violin/recluse (*Loxosceles)* spiders and sac (*Cheiracanthium*) spiders can produce local necrotic skin lesions that are mediated by enzymes.

DIAGNOSTIC CRITERIA

» Bites are initially painless.
» Skin lesions can vary from mildly erythematous lesions to severe local reactions, i.e. blistering, bluish discolouration progressing to frank necrosis.
» Systemic effects occasionally include nausea, vomiting, fever, chills, arthralgia, haemolysis, thrombocytopaenia, haemoglobinuria and renal failure.

GENERAL AND SUPPORTIVE MEASURES

» Supportive care.
» Surgical debridement may be required once clear margins around the necrotic lesions are established.

MEDICINE TREATMENT

For pain:

- Paracetamol, oral, 15 mg/kg/dose, 6 hourly as required.

Antibiotic therapy for septic lesions.
Surgical debridement may be considered for large necrotic lesions.

References

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