CHAPTER 19
POISONING

POISONS INFORMATION CENTRES

Poisons Information Helpline (national service)	24/7	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		
Tygerberg Poisons Information Centre Email: toxicology@sun.ac.za www.sun.ac.za/poisoncentre		
University of the Free State Poison Control and Medicine Information Centre	24/7	082 491 0160
Telephone numbers tested April 2022		

Access poisons information at: https://www.afritox.co.za/
The Afritox database is available free of charge to public hospitals in South Africa. If the above centres cannot be contacted, enquire at the nearest trauma and emergency unit.

ENVENOMATION

Envenomation is an instance of poisoning by venom resulting from a bite or sting from an animal such as a snake, spider, scorpion, insect, or marine life.

South African Vaccine Producers (SAVP): For procurement of Snake/spider/scorpion antivenom: Email: benita.mouton@nhls.ac.za	Office hours: (011) 386 6062/6063/6078 After hours 071 680 9897

19.1 INSECT BITES AND STINGS

T63.4 + (X23.99/X24.99/X25.99/X29.99)

DESCRIPTION

Toxicity due to insect bites and stings usually results in local effects only and systemic effects are rare. Occasionally, hypersensitivity reactions are encountered, varying from minor local inflammation to acute anaphylaxis. Multiple bee stings can result in systemic 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

Anaphylaxis: See section 20.7: Anaphylaxis/Anaphylactic Shock.

For pain:

- Paracetamol, oral, 500mg -1 g 4–6 hourly when required (to a maximum of 4 g in 24 hours) *LoE:IVb*
    - Maximum dose: 15 mg/kg/dose.

19.2 SNAKE BITES

T63.0 + (X20.99/W59.99)

DESCRIPTION

In the majority of snakebite incidents, the offending snake is not identified. The table below illustrates the three main envenomation syndromes seen in South Africa: cytotoxic, neurotoxic and haemotoxic.

	Envenomation syndromes			
	Cytotoxic	**Neurotoxic**	**Mixed cytotoxic & neurotoxic**	**Haemotoxic**
Snake species	Puff adder, Gaboon adder, spitting cobras (Mozambique, black-necked, zebra), stiletto snake, night adders, horned adders	Black and green mamba, non-spitting cobras (Cape, forest, snouted)	Rinkhals, Berg adder, Peringuey's adder, desert mountain adder, garter snakes, shield-nose snake, coral snake	Boomslang, vine snakes
Clinical features of envenomation	Pain, swelling, bruising, blisters, necrosis, regional lymphadenopathy, hypotension, coagulopathy, compartment syndrome	Pins and needles, metallic taste, visual disturbances, ptosis, drowsiness, sweating, drooling, dysphagia, progressive weakness,	Combined cytotoxic and neurotoxic features	Spontaneous bleeding (can present late >24 hours after bite), headaches, dizziness, fainting

		respiratory paralysis		
Antivenom (when indicated)	Polyvalent antivenom for Puff adder, Gaboon adder, and Mozambique spitting cobra only	Polyvalent antivenom for all species	Polyvalent antivenom for rinkhals only	Boomslang monovalent antivenom for boomslang bites only

Table 19.1: Presentation and management of envenomation syndromes
To find pictures for the identification of snakes:
http://www.cmej.org.za/index.php/cmej/article/view/2546/2581

GENERAL MEASURES

» Most snakebites will not result in death.
» Monitor all cases of snakebite for 24 hours.
» Supportive and symptomatic management with/without antivenom is required.
» Mechanical ventilation may be needed in some cases of neurotoxic envenomation.
» Cases of haemotoxic envenomation may require fluid resuscitation including blood products.
» True compartment syndrome is extremely rare in cytotoxic snakebites, as swelling is localised to the subcutaneous tissues. Fasciotomy is seldom indicated.

MEDICINE TREATMENT

Cleanse wound:

- Chlorhexidine 0.05% in water.

Antibiotics: T79.3 + (X20.99/W59.99)
Antibiotics are seldom indicated unless there is evidence of secondary infection.

- Amoxicillin/clavulanic acid, oral, 875/125 mg 12 hourly for 5 days.

LoE:IVb[i]

Immunisation, primary or booster: (Z23.5)

- Tetanus toxoid vaccine, IM, 0.5 mL immediately, if not previously immunised within the last 5 years.

In unimmunised or partially immunised patients:

- Tetanus immunoglobulin, human, IM, 250 units immediately.

Analgesia

For mild pain:

- Paracetamol, oral, 500mg -1 g 4–6 hourly when required (to a maximum of 4 g in 24 hours)
    - Maximum dose: 15 mg/kg/dose.

For severe pain:

ADD

- Morphine, IV, to a total maximum dose of 10 mg (See Appendix II, for individual dosing and monitoring for response and toxicity). *LoE:IVb*

CAUTION

Opioids increase the risk of respiratory depression particularly for neurotoxic envenomation, and if required, should only be used with caution in severe uncontrolled pain.

Note: NSAIDS are not recommended as they increase the risk of bleeding and renal failure, especially in patients with severe cytotoxic bites.

LoE:IVb[ii]

19.2.1 CYTOTOXIC AND NEUROTOXIC SNAKEBITE

T63.0 + (X20.99/W59.99)

MEDICINE TREATMENT

Polyvalent antivenom

Used in some cytotoxic and neurotoxic envenomations, only where indicated (see indications below).

Available from South African Vaccine Producers (refer to the table above for contact details). See package insert for full details.

LoE:IVb[iii]

Note:

» In most cases, patients do not need and should not be given antivenom.
» Adverse reactions to antivenom such as allergic reactions (10-30%) are common and may be severe. Pre-medication with adrenaline (epinephrine) may reduce the risk of severe adverse reactions to polyvalent snake antivenom.
» 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 that indicates ongoing venom activity.

LoE:IVb[iv]

Indications for polyvalent antivenom:

» Signs of neurotoxicity.
» Positively identified puff adder, Gaboon adder, Mozambique spitting cobra or rinkhals bites AND evidence of progressive severe cytotoxicity.

» Unidentified snakebite AND evidence of progressive severe cytotoxicity.
» Severe local cytotoxicity is defined as:
  – Swelling of the whole hand or foot within 1 hour
  – Swelling to the knee or elbow in less than 6 hours (or two joints above the bite site in 6 hours)
  – Swelling of the whole limb in less than 12 hours
  – Swelling progression > 5 cm/hour
  – Discolouration of the skin / necrosis at the bite site
  – A threatened airway due to swelling
  – Evidence of complications e.g. pseudo- or true compartment syndrome
  – Additional features of severe systemic cytotoxicity include:
    - Haematological abnormalities: Hb <8 g/dL, thrombocytopaenia, (<100 x 10^9/L), raised INR or abnormal thromboelastography (if available)
    - Arrhythmias (rare).
    - Shock

LoE:IIIb[v]

Note: Polyvalent antivenom is ineffective against the venom of: night adders, berg adders and other smaller adders, boomslang, and vine/twig snakes.

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

Administration and polyvalent antivenom dose:

- Pre-treat with adrenaline (epinephrine), SC, 0.25 mL of 1:1000 solution.

Note: This is contraindicated in patients with IHD, stroke, uncontrolled hypertension, and tachyarrhythmia.

LoE:IIa[vi]

- Polyvalent snake antivenom, slow IV infusion.
  - This guidance refers to the antivenom produced by South African Vaccine Producers. For any other product refer to the relevant package insert for guidance.

LoE:IVb[vii]

  - 1 ampoule contains 10 mL antivenom.
  - Cytotoxic snakebite (unidentified snake): give 50 mL,
  - For puff adder bites: the initial dose is 80 mL.
  - For Mozambique spitting cobras the initial dose is 100 mL
  - Neurotoxic snakebite: give 80–100 mL (and up to 200 mL in black mamba bites).
  - Dilute in sodium chloride, 0.9%, 200mL; for example, if 8 ampoules are required, remove 80 mL from 200 ml saline bag and replace with 80 mL antivenom.

LoE:IVb[viii]

  - Administer IV, over 30 minutes.
  - Reassess once the infusion is completed. A repeat dose may be given if there is ongoing neurotoxicity or cytotoxicity.

19.2.2 BOOMSLANG SNAKEBITE

T63.0 + (X20.99/W59.99)

DESCRIPTION

Boomslang venom is haemotoxic. A consumptive coagulopathy with hypofibrinogenaemia and bleeding usually sets in within 6–36 hours after the bite.

GENERAL MEASURES

» In suspected boomslang bite, a whole blood clotting time is a useful bedside test, especially in rural areas. Place 5 mL of blood in a dry glass test tube and leave at room temperature for 20 minutes. Normal clotting time varies from 5–20 minutes. It is important to repeat these over a few days.
» Other investigations include FBC, activated PTT, prothrombin time (INR), fibrinogen, D-dimer, and monomers.

Note: Polyvalent antivenom is not effective in boomslang bites.

Boomslang monovalent antivenom
Indicated for all boomslang bites with evidence of haemotoxicity.
Available from South African Vaccine Producers (refer to the table above for contact details). See full details in the package insert**.**

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

- Boomslang monovalent antivenom, slow IV infusion, 20 mL diluted in 50–100 mL sodium chloride, 0.9% or dextrose 5%, administered over 5–10 minutes.
    - The dose of antivenom is the same for adults and children. *LoE:IVb*[ix]
    - Spontaneous systemic bleeding should stop within 15–30 minutes and blood coagulability be restored within 6 hours of administering antivenom.
    - Re-evaluate regularly: Consider a repeat dose of 10 ml of antivenom if there is ongoing evidence of coagulopathy after 6 hours.

19.2.3 SNAKE VENOM IN THE EYE

S05.9 + (X20.99/W59.99)

DESCRIPTION

Snake venom in the eye, particularly from various species of spitting cobras and rinkhals, can cause local cytotoxic effects. Clinical presentation ranges

from periocular swelling and mild conjunctival and corneal inflammation, to frank corneal ulceration and perforation with eventual blindness.

MEDICINE TREATMENT

Instil local anaesthetic:

- Local anaesthetic ophthalmic drops, e.g.:
- Tetracaine 1%, drops (if available), instil 1 drop into the affected eye(s) before irrigation. *LoE:IIIb*[x]
    - Irrigate the eye thoroughly for 15–20 minutes with water or sodium chloride, 0.9% to dilute or remove the toxin.

Topical antibiotics:

- Chloramphenicol 1%, ophthalmic ointment 8 hourly for 7 days.
    - Apply chloramphenicol eye ointment and cover the affected eye with an eye patch. *LoE:IVb*

Note: Do not instil polyvalent antivenom in the eye or give systemically. *LoE:IVb*[xi]

REFERRAL

Refer all patients to an ophthalmologist.

19.3 SCORPION ENVENOMATION

T63.2 + (X22.99/W59.99)

DESCRIPTION

Medically important scorpions in Southern Africa are of the genus *Parabuthus* (*P. granulatus* and *P. transvaalicus*). These are large scorpions measuring 7–15 cm in length. Features useful in their identification are a relatively large tail and small pincers, so-called thick-tailed scorpions. Scorpions from the Scorpionidae family (e.g. Hadogenes, Opistophthalmus) are thin-tailed with large pincers.

To view pictures for the identification of scorpions:
http://www.cmej.org.za/index.php/cmej/article/view/2545/2580

A sting from thin-tailed scorpions is likely to result in local pain requiring analgesia only.
Clinical features of thick-tailed scorpion stings include:
Local effects:

» immediate and excruciating pain
» local paraesthesias and hyperaesthesia

Systemic effects:

» tremors, involuntary movements and fasciculations
» muscle pain, cramps, and weakness

» generalised paraesthesias and hyperaesthesia
» excessive sympathetic stimulation e.g. sweating, tachycardia
» excessive parasympathetic stimulation, e.g. hypersalivation, vomiting, diarrhoea, and priapism
» bulbar paralysis (dysphagia, dysarthria)
» respiratory difficulty/failure

GENERAL MEASURES

» Observe all cases of thick-tailed scorpion stings for at least 12 hours.
» Monitor respiratory function.
» Ventilatory support may be required.

MEDICINE TREATMENT

Scorpion antivenom therapy is recommended only in cases presenting with systemic neurotoxic effects.
Antivenom available from South African Vaccine Producers (refer to the table above for contact details). See full details in the package insert.

- Scorpion antivenom, IV infusion, 10 mL diluted in 100 mL sodium chloride 0.9% or dextrose 5%, administered over 10 minutes.
    - Response to antivenom may be slow and a repeat dose may be needed.

LoE:IVb

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

Immunisation, primary or booster: (Z23.5)
- Tetanus toxoid vaccine, IM, 0.5 mL, immediately, if not previously immunised within the last 5 years.

In unimmunised or partially immunised patients: (Z23.5)
- Tetanus immunoglobulin, human, IM, 250 units immediately.

Analgesia
For mild pain:
- Paracetamol, oral, 500mg -1 g 4–6 hourly when required (to a maximum of 4 g in 24 hours)
    - Maximum dose: 15 mg/kg/dose.

LoE:IVb[xii]

Severe local pain:
Application of ice, if tolerated.
- Lidocaine 1–2%, 2 mL: infiltrate affected area as a local anaesthetic.

LoE:IVb[xiii]

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

LoE:IVb[xiv]

Severe muscle pain and cramps:

LoE:IVb[xv]

- Calcium gluconate 10%, bolus IV infusion, 10 mL over 10 minutes.
    - Repeat if needed, only once i.e. maximum recommended dose of 2 grams.
    - **Note:** Effect may only last for 20–30 minutes and there is a limited amount that can be given.

LoE:IVb[xvi]

19.4 SPIDER ENVENOMATION

T63.3 + (X21.99)

DESCRIPTION

Local venomous spiders are divided into cytotoxic and neurotoxic groups.
To view pictures for the identification of spiders:
http://www.cmej.org.za/index.php/cmej/article/view/2547/2582

Cytotoxic spider group

The cytotoxic group includes sac, violin, and crab spiders.
Lesions may present with significant bite site necrosis, for which surgical debridement may be required. Bites can take weeks/months to heal.
Note: Antibiotics are reserved for secondary infection.

Neurotoxic spider group

The neurotoxic group is represented by the button spider (also known as widow spiders), genus *Latrodectus*. Black button spiders are more venomous than brown button spiders.

Features useful in the identification of the black button spider are:
» Black or dark brown colour.
» Variable red markings on the dorsal aspect of the abdomen, which diminish with age. It has no ventral markings.

Features of brown button spider:
» Light brown to creamy yellow to pitch black in colour.
» Typical red-orange hourglass-shaped marking on the ventral surface of the abdomen.

Envenomation from black button spiders may cause:
» Immediate local burning pain and tender regional lymph nodes within an hour.

» Severe general muscle pain, cramps, and rigidity especially of the large girdle muscles
  – Causes feeling of tightness of the chest and board-like rigidity of a non-tender abdomen.
  – Lasts for days to a week if antivenom is not given.
» Profuse sweating may be prominent.
» Diffuse paraesthesia, especially of the hands and feet.

GENERAL MEASURES

Observe all cases of potential neurotoxic spider bite for at least 24 hours.

MEDICINE TREATMENT

» Spider antivenom is only indicated for systemic symptoms of neurotoxicity in patients with button spider bites.
» Antivenom available from South African Vaccine Producers: (refer to the table above for contact details). See full details in the package insert**.**

- Spider antivenom, IV infusion, 5–10 mL diluted in 50–100 mL sodium chloride, 0.9% or dextrose 5%, administered over 5–10 minutes. *LoE:IVb*[xvii]

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

Immunisation, primary or booster: (Z23.5)
- Tetanus toxoid vaccine, IM, 0.5 mL immediately, if not previously immunised within the last 5 years.

In unimmunised or partially immunised patients: (Z23.5)
- Tetanus immunoglobulin, human, IM, 250 units immediately.

Analgesia

For mild pain:
- Paracetamol, oral, 500mg -1 g 4–6 hourly when required (to a maximum of 4 g in 24 hours)
  - Maximum dose: 15 mg/kg/dose.

Severe muscle pain and cramps:
- Calcium gluconate 10%, bolus IV infusion, 10 mL over 10 minutes.
  - Repeat if needed, only once, i.e. maximum recommended dose of 2 grams.
  - **Note:** Effect may only last for 20–30 minutes and there is a limited amount that can be given.

LoE:IVb[xviii]

For secondary infection:
See section 4.2: Cellulitis and Erysipelas.

POISONING

DESCRIPTION

Frequently encountered poisonings in adults are due to:

- analgesics
- anti-infectives
- anticonvulsants
- antihistamines
- cardiodepressants
- iron
- sedatives, antidepressants & antipsychotics
- ethanol/alcohol
- hydrocarbons e.g. paraffin
- irritants and corrosives
- pesticides
- toxic alcohols e.g. methanol, ethylene glycol

Maintain a high index of suspicion for intentional ingestion in adults presenting with poisoning.

DIAGNOSTIC CRITERIA

Clinical

Clinical presentations due to poisoning can be divided into 'toxidromes":

Anticholinergic: e.g. antihistamines, amanita pantherina/muscaria, atropine

- fever
- ileus
- flushing
- tachycardia
- urinary retention
- dry skin and mouth
- blurred vision
- mydriasis (dilated pupils)
- coma
- hallucinations and seizures

Cholinergic: e.g. organophosphates

- salivation
- lacrimation
- urination
- miosis (pinpoint pupils)
- diarrhoea
- vomiting
- bronchorrhoea
- bradycardia

Dystonic: e.g. haloperidol

- torticollis
- intermittent spasms and tongue thrusting
- opisthotonos

Opiates: e.g. morphine

- miosis (pinpoint pupils)
- respiratory depression
- bradycardia
- altered (decreased) consciousness
- decreased bowel sounds
- hypothermia
- hypotension

Salicylism: e.g. aspirin

- tachypnoea
- seizures
- metabolic acidosis and respiratory alkalosis
- agitation
- coma

Sedative-hypnotic: e.g. alcohol, benzodiazepines
- » obtundation or coma

Sympathomimetic: e.g. cocaine, amphetamines
- » hypertension
- » tachycardia
- » hyperthermia
- » agitation
- » sweating
- » dilated pupils

Sympathomimetic toxidrome partially 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 and anion gaps
- » visual disturbances (methanol)
- » inebriation and depressed level of consciousness.
- » nausea and vomiting
- » tachycardia and arrhythmias
- » renal failure (ethylene glycol)
- » hyperventilation

GENERAL MEASURES

It is very important to ascertain if a potentially TOXIC DOSE has been taken BEFORE instituting any potentially harmful decontamination procedures in an asymptomatic patient.

- » Take a complete and accurate history, ascertain all relevant facts, and do a complete clinical examination.
- » Maintain a high index of suspicion.
- » Obtain a collateral history, especially for patients with impaired consciousness. A special effort should be made to obtain tablets, packets, containers, etc. to identify agents involved.
- » Stabilise the patient and monitor basic clinical parameters, i.e.:
  - – blood pressure and heart rate
  - – hydration
  - – airway and ventilation
  - – neurological status
  - – temperature
  - – glucose
- » Persistent or prolonged seizures may require medical management. Phenytoin should not be used in cases of poisoning due to substances known to be cardiotoxic e.g. tricyclic antidepressants, or where there is evidence of clinical cardiotoxicity.
- » Prevent physical injury in the restless – avoid excessive sedation.
- » Limit toxicological investigations to those that may influence/alter management. It is important to note the time after ingestion when blood was taken in order to correctly interpret results (e.g. paracetamol and iron levels).

LoE:IVb

Decontamination
Limit further exposure to poison for the patient and protect healthcare workers where necessary.

Topical exposure
In the case of skin exposure, remove clothes and wash the body. Showering may be useful.
Remove eye contaminants, especially alkalis, acids and other irritants, by continuous irrigation of the eye with sterile water or normal saline for 15–20 minutes. Analgesic eye drops may be required to perform this adequately.

Gut decontamination
Methods of gut decontamination include:
» Gastric lavage
» Activated charcoal administration
» Whole bowel irrigation

Gastric lavage
» If deemed beneficial, it should only be performed by experienced staff and
» within 60 minutes of ingestion. *LoE:IVb*[xix]
» Can be considered for cases with:
  – potentially life-threatening ingestions AND
  – a protected airway i.e. fully awake and cooperative or intubated with a depressed level of consciousness.
» Gastric lavage is contra-indicated 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 200mL warmed water or normal saline, and aspirate.
  – Continue until recovered solution is clear of particulate matter.

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.

LoE:IVb[xx]

Activated charcoal may be useful if these poisons are taken in toxic dose	Poisons where charcoal is ineffective and should not be given
» carbamazepine, barbiturates, phenytoin » dapsone, quinine » theophylline » salicylates » mushroom poisoning (Amanita phalloides) » slow-release preparations » digoxin » beta-blockers » NSAIDs	» ethanol, methanol, ethylene glycol » brake fluid » petroleum products (e.g. petrol or paraffin) » iron salts » lead, mercury, arsenic » lithium » strong acids or alkalis » other corrosive agents (e.g. household detergents)

Table 19.2: Appropriate use of activated charcoal

- Activated charcoal, oral, 50 g (equivalent to 36 level medicine measures) diluted in 100 mL water.
    - When mixing, add a small amount of water to charcoal in a container.
    - Cap and shake container to make a slurry and then dilute further.
    - Repeated doses of activated charcoal (i.e. 50 g every 4 hours) are effective in enhancing elimination of substances that undergo enterohepatic circulation, e.g. carbamazepine, dapsone, phenobarbitone, quinine or theophylline. *LoE:IIIb*[xxi]

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)
» modified-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, 1500-2000 mL/hour.
    - Continue until rectal effluent is clear.

LoE:IIIb[xxii]

Other treatment modalities

Sodium bicarbonate alkalinisation

Urine alkalinisation enhances renal elimination of certain toxins (salicylates) and serum alkalinisation improves acidosis enhancing myocardial functioning (TCAs) and reducing neurotoxicity (salicylates).

This is achieved by administering intravenous sodium bicarbonate ($NaHCO_3$) to maintain a urinary pH 7.5-8.5 or serum pH 7.45-7.55.

CAUTION
This is a high-risk procedure and should only be performed in consultation with a specialist.

Haemodialysis
Patients with symptomatically severe poisoning substances including salicylates, lithium, ethylene glycol, methanol, ethanol, and theophylline, may benefit from dialysis (http://www.extrip-workgroup.org/).
Refer patient to a hospital with dialysis facilities.

Antidotes
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.

Once medically stable:
Assess and manage intentional poisoning – self-harm or harm by others:
» Take a history of circumstances around the poisoning, substance use and mental illness, and examine the mental state.
» Assess further suicide risk – see Primary Health Care STGs and EML, section 16.7: Suicide risk assessment.
» Refer to social, psychological and/or psychiatric services.

Assess and manage a substance use disorder:
» Quantify the amount of substance used and related harms with these rating scales and discuss findings with the patient:
  – ASSIST:http://www.who.int/substance_abuse/activities/assist/en/
  – DUDIT:https://www.emcdda.europa.eu/system/files/attachments/12173/DUDIT-English-version.pdf
» Provide brief intervention with motivational interview.
» Refer for rehabilitation.

REFERRAL
» Severely ill patient for ventilatory/circulatory support.
» Relevant diagnostic testing not available, e.g. paracetamol levels, acid/base assessment.
» Relevant medication/antidote not available.
» Dialysis/haemoperfusion required.

19.5 ANALGESIC POISONING

19.5.1 PARACETAMOL POISONING

T39.1 + (X40.99/X60.99/Y10.99)

DESCRIPTION

Liver damage, due to the depletion of glutathione and accumulation of toxic metabolites, can occur in any individual with paracetamol overdose. Patients with predisposing risk factors for hepatotoxicity ("high risk" patients, see below) may experience toxicity at lower ingested doses.

Clinical features

Gastrointestinal symptoms (anorexia, nausea, vomiting, malaise) predominate in the first 24 hours. Patients with normal or only slightly raised serum paracetamol levels usually continue to full recovery. In patients with significantly raised paracetamol levels, hepatic toxicity (right upper quadrant abdominal pain and tenderness, elevated bilirubin, raised liver enzymes, coagulation defects, hypoglycaemia, encephalopathy, and metabolic acidosis) may manifest from 20-24 hours, peaking in severity at about 72-96 hours. Patients may make a full recovery in 5-7 days, or demise from hepatic failure, or less commonly, renal failure.

"High risk" patients include those with:

» Chronic alcoholism
» Chronic liver disease
» Use of enzyme-inducing medicines (e.g. carbamazepine, phenytoin, efavirenz, phenobarbitone, rifampicin etc.)
» Depletion of glutathione resources (e.g. malnutrition, starvation, AIDS, chronic illness, eating disorders etc.)
» Recent illness, dehydration

GENERAL MEASURES

The treatment of paracetamol overdose depends on the dose ingested and the time of presentation since ingestion. A serum paracetamol level is plotted on the nomogram to assess the risk for hepatotoxicity. Values which appear above the treatment line require the antidote N-acetylcysteine (NAC).

Acute single ingestion <8 hours post-ingestion:

» Toxic dose is defined as a paracetamol ingestion >200 mg/kg or 10 g (whichever is less).
» Give activated charcoal if the patient presents within 1-2 hours of ingestion.
» Perform a serum paracetamol level and ALT no earlier than 4 hours post-ingestion.
» If serum paracetamol level results will not be available before 8 hours post-ingestion, AND the patient has taken a toxic dose, 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.

Acute single ingestion >8 hours post-ingestion:

» Toxic dose defined as >200 mg/kg or 10 g (whichever is less).
» Start NAC infusion if a toxic dose has been ingested or the patient shows clinical signs of toxicity.
» Perform serum paracetamol level, ALT, and INR.
» Indications for continuing NAC infusion:
  – Serum paracetamol level above the treatment line on the nomogram.
  – Serum paracetamol level under the treatment line but abnormal ALT.
  – Measurable paracetamol level and/or abnormal ALT more than 24 hours post-ingestion.

Acute single ingestion with unknown time of ingestion:

Manage as for >8 hours post-ingestion, however, the nomogram is not applicable to this group.

LoE:IIIa[xxiii]

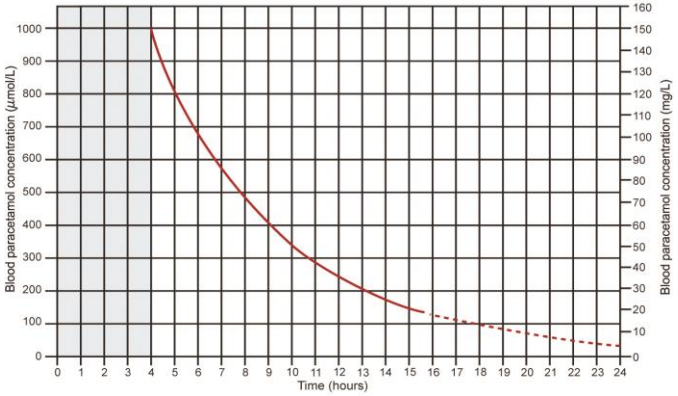
Repeated supratherapeutic ingestion (RSTI):

This may occur in patients using repeated high doses of the same product or concurrent use of multiple paracetamol-containing products such as during an acute febrile illness or in patients with chronic pain.

RSTI toxic doses are defined as:

» >200 mg/kg or 10 g (whichever is less) over a single 24-hour period.
» >300 mg/kg or 12 g (whichever is less) over a single 48-hour period.
» >60 mg/kg/day for more than 48 hours ***and*** patients have symptoms suggestive of liver injury.

LoE:IIIa[xxiv]



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.

Figure 19.1: Paracetamol treatment nomogram. (Access the paracetamol nomogram tool on the EML Clinical Guide cellphone application).

MEDICINE TREATMENT

N-acetylcysteine is the antidote of choice and should be given intravenously. Although it is more effective when given within 8 hours of ingestion of paracetamol, there may be benefit even if liver failure has developed. Histamine may be released, which mimics an allergic reaction. If this occurs and the patient is stable, infusion may continue at a slower rate under antihistamine cover. Stop the infusion if bronchospasm occurs.

- N-acetylcysteine, IV:
  - Initial infusion: 200 mg/kg in 500 mL dextrose, 5% over 4 hours.
  - Second infusion: 100 mg/kg in 1000 mL dextrose, 5% over 16 hours.
  - Any further N-acetylcysteine is given according to the second infusion regimen. *LoE:IIIa*[xxv]

If N-acetylcysteine IV formulation is unavailable:

- N-acetylcysteine, oral, 140 mg/kg immediately.
  - Followed by 70 mg/kg 4 hourly, for up to seventeen doses. *LoE:IIIa*[xxvi]

Note:

» As anaphylactoid reactions to N-acetylcysteine do occur, the loading dose should preferably be administered in a monitored area. *LoE:IVb*[xxvii]

» Avoid giving oral N-acetylcysteine together with activated charcoal as systemic absorption and effect of N-acetylcysteine is reduced. *LoE:IIIa*[xxviii]

Further investigations and referral

Blood tests such as renal function, clotting profile, serum glucose and acid/base status should only be done where clinically indicated.

Patients who develop liver failure must be referred for further management and/or possible transplant.

19.5.2 SALICYLATE POISONING

T39.0 + (X40.99/X60.99/Y10.99)

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% methyl salicylate.

Diagnosis:

Mild to moderate toxicity:

» Nausea, vomiting, tinnitus, fever, tachypnoea, and respiratory alkalosis

Severe toxicity:

» Metabolic acidosis, altered mental status, seizures, coma, non-cardiogenic pulmonary oedema.

» Monitor salicylate levels if possible (do not always correlate with clinical severity):

Severity of toxicity	Peak plasma salicylate concentrations	
	mmol/L	mg/dL
Asymptomatic	<2.2 mmol/L	<30 mg/dL
Mild toxicity	2.2-4.3 mmol/L	30-60 mg/dL
Moderate toxicity	4.3-5.8 mmol/L	60-80 mg/dL
Severe toxicity	>5.8 mmol/L	>80 mg/dL

Table 19.3: Severity of toxicity by peak plasma salicylate concentrations.

» Serial monitoring until declining levels are documented.
» Monitor and treat hypoglycaemia; patients with normoglycaemia may still be neuroglycopaenic.

GENERAL MEASURES

» Assess severity with history, clinical examination, and salicylate levels if possible.
» Correct hydration using dextrose-containing fluids.
» Ensure hypokalaemia treated early
» Consider ICU admission for pulmonary and/or cerebral oedema.

MEDICINE TREATMENT

- Salicylates delay gastric emptying, therefore activated charcoal may be effective for a longer period than usual.
- Whole bowel irrigation maybe useful for enteric-coated or modified-release preparations.

LoE:IIIb[xxix]

For mild toxicity:

» Rehydrate and correct hypovolaemia with dextrose-containing fluids.
  - Add dextrose 50%, 100mL to every litre of balanced crystalloid solution (e.g. ringers lactate) or sodium chloride 0.9%, and administer by IV infusion. *LoE:IIIb*[xxx]
  - During preparation of the infusion fluid, ensure the equivalent volume of rehydration fluid (e.g. 100mL) is removed from the bag before adding the total dextrose 50% volume (e.g. 100mL).
  - The rate and duration of IV fluids should be guided by clinical assessment of fluid balance. *LoE:IVb*

In patients with moderate to severe toxicity and/or acidosis:

- Sodium bicarbonate 8.4%, IV, 1–2 mL/kg over 30 minutes to manage acidosis. *LoE:IIIb*[xxxi]
- Simultaneously fluid resuscitate with sodium bicarbonate 8.4%, 150mL added to dextrose 5%, 1L and administer by IV infusion to correct hypovolaemia.
  - During preparation of the infusion fluid, ensure the equivalent volume of dextrose 5% (i.e. 150 mL) is removed from the bag before adding the total sodium bicarbonate 8.4% volume of 150 mL.

- Continue a maintenance infusion at 150 – 200 mL/hour, targeting a urine output of 2mL/kg/hour.
- Titrate the sodium bicarbonate maintenance infusion to a urinary pH of 7.5 – 8.5 and blood pH of 7.45 – 7.5.
- Monitor for and correct hypokalaemia.

LoE:IVb

REFERRAL

» Discuss with specialist and consider ICU admission
» Where acidosis does not respond to sodium bicarbonate, refer for haemodialysis.

LoE:IIIa[xxxii]

19.5.3. OPIOID POISONING

T40.0/T40.1/T40.2/T40.3 + (X42.99/X62.99/Y12.99)

DESCRIPTION

Patients present with the triad of CNS depression, respiratory depression, and constricted pupils. Non-cardiogenic pulmonary oedema can occur.

GENERAL MEASURES

Supportive management aimed at maintaining cardiorespiratory function.

Body packers/stuffers:
» Patients may ingest packages of illicit opioids and are at increased risk of life-threatening toxicity in the event of rupture.
» Abdominal X-rays or CT scan may show packages.
» Conservative management is recommended, as any attempt at removal risks package rupture.
» Activated charcoal and whole bowel irrigation may aid in expelling packets.
» Surgery is reserved for those who develop obstruction or perforation.

MEDICINE TREATMENT

- Naloxone, IV, 0.4 mg immediately, in patients with significant respiratory depression.
    - Effectiveness is limited by a half-life (± 1 hour) that is shorter than most opioids.
    - Repeated incremental doses (e.g.: 0.4 mg, 0.8 mg, 2 mg, 4 mg etc.) may be required at 2 to 3 minute intervals, up to a maximum of 10 mg. If a response is noted, a maintenance infusion of 0.4 mg/hour should be initiated.
    - If there is no response after a maximum total dose of 10 mg of naloxone is administered, the diagnosis of opioid-induced or partial opioid-induced toxicity should be re-assessed.
    - Consider intramuscular or subcutaneous administration if the intravenous route is not available.

Note:
- Clinical response is measured by reversal of respiratory depression rather than complete reversal of sedation.
- Continuous monitoring is required for all patients who receive naloxone.
- Naloxone in an opioid-dependent person may precipitate a withdrawal syndrome with agitation, hypertension, tachycardia, emesis, and potential aspiration. These patients usually require lower doses when initiating naloxone (0.04-0.1mg IV).

LoE:IVb[xxxiii]

19.6 ANTIDEPRESSANT POISONING

19.6.1. TRICYCLIC ANTIDEPRESSANT POISONING

T43.0 + (X41.99/X61.99/Y11.99)

DESCRIPTION

TCAs may be life threatening at relatively low doses. Cardiovascular and neurological impairment are the most serious consequences of TCA toxicity, and patients can deteriorate rapidly depending on the severity.

LoE:IVb[xxxiv]

Mild to moderate poisoning:

» Sedation
» Anticholinergic effects:
- delirium,
- urinary retention
- tachycardia
- dilated pupils
- dry mouth

Severe Poisoning:

» Widened QRS duration, ventricular dysrhythmias
» Seizures
» Coma
» Pulmonary oedema
» Hypotension

GENERAL MEASURES

» Do a baseline ECG in all patients.
» ICU admission for ventilatory/circulatory support, when indicated. Be prepared to intubate symptomatic patients early.
» Discharge patients only when:
  – asymptomatic, or
  – mild symptoms/signs of toxicity and ECG has normalised for at least 24 hours.

MEDICINE TREATMENT

Tricyclic antidepressants delay gastric emptying, therefore activated charcoal may be effective for a longer period than usual.
Indications for serum alkalinisation:
» ventricular dysrhythmias,

» prolonged QRS >100 msec,
» hypotension unresponsive to fluids, or
» seizures.

- Sodium bicarbonate 8.4% solution, IV 1–2 mL/kg administered in bolus doses (Specialist consultation).
  - Aim to achieve a serum pH of 7.45–7.55.
  - Monitor acid-base status, serum potassium and sodium.
  - If sodium bicarbonate is unavailable or fluid restrictions limit intake, consider hyperventilation of intubated patients. *LoE:IIIa*[xxxv]

In severe cases, inotropic support and anti-arrhythmics may be required (See section 3.3: Cardiac dysrhythmias) in addition to serum alkalinisation. Hypotension is due to myocardial dysfunction and alpha-adrenergic vasodilation; be careful not to fluid overload the patient. *LoE:IVb*

For seizures or if sedation is required for restlessness:
Treat with benzodiazepines - see section: 14.4.1 Status epilepticus.
Note: Phenytoin should be avoided (due to potential cardiotoxicity).
LoE:IVb[xxxvi]

Note: Flumazenil is not recommended in any patient with mixed overdoses possibly including tricyclic antidepressants as it increases the risk of convulsions and dysrhythmias.
LoE:Ia[xxxvii]

19.7 IRON POISONING

T45.4 + (X44.99/X64.99/Y14.99)

DESCRIPTION

Iron is a commonly prescribed drug, especially in pregnancy, and in overdose causes initial gastrointestinal toxicity. Patients may have a stage of “apparent recovery” 6–36 hours post-ingestion. This should not be confused with true recovery as patients may subsequently deteriorate.

Significant exposure may be associated with:

» severe vomiting and diarrhoea
» metabolic acidosis,
» CNS depression,
» hepatitis.
» gastrointestinal haemorrhage
» hypotension, shock
» renal failure, and

Ferrous salt	Amount	Elemental iron
Ferrous sulphate	170 mg	± 65 mg
Ferrous gluconate	300 mg	35 mg
Ferrous fumarate	200 mg	± 65 mg

Table 19.4: Elemental iron content available in different iron salts

GENERAL MEASURES

» Gastrointestinal decontamination by whole bowel irrigation is recommended:
  – if >60 mg/kg elemental iron has been ingested
  – if modified-release preparations ingested
  – undissoved tablets still visible on abdominal X-ray
» Activated charcoal does not bind iron and is not indicated in isolated iron overdose.
» Serum iron concentration should be measured 4–6 hours after ingestion and repeated every 6 hours until peak. The use of deferoxamine (desferrioxamine) interferes with the interpretation of further serum iron levels.
» Give intravenous fluids for hypotension.

MEDICINE TREATMENT

Chelation therapy

» Patients with serum iron levels <54 µmol/L and absence of symptoms >6 hours after overdose do not require chelation therapy.
» Deferoxamine (desferrioxamine) may be used for the following indications (if in doubt, consult the Poisons Information Helpline):
  – Severe symptoms (altered mental status, haemodynamic instability, metabolic acidosis).
  – Serum iron concentration >90 µmol/L.
  – Peak serum iron concentration >60 µmol/L, AND persistent gastrointestinal symptoms.

LoE:IIIa[xxxviii]

- Deferoxamine (desferrioxamine), IV infusion, 80 mg/kg. *LoE:IVb*[xxxix]
  - Administer at 15mg/kg/hour over about 6 hours.
  - Beware of hypotension.
  - **Note:** Prolonged use (>24 hours) of high doses are associated with acute lung injury and should be avoided. However, additional doses may be required in severe poisonings – A benefit-risk assessment is required in these patients.
  - Where IV access is not obtainable, deferoxamine can be given by IM injection as follows: deferoxamine, IM injection 1 g immediately, followed by 500 mg every 4 to 12 hours, as needed based on clinical response.
  - For cardiogenic shock, the IV route is preferred and should be used as soon as IV access is possible.
  - Deferoxamine can be used in pregnant women. *LoE:IIIa*[xl]

REFERRAL

Haemodialysis may be needed to remove deferoxamine-iron complexes in patients with renal insufficiency.

19.8 THEOPHYLLINE POISONING

T48.6 + (X44.99/X64.99/Y14.99)

DESCRIPTION

Patients present with:

- » tachycardia and tachyarrhythmias,
- » nausea and vomiting
- » agitation
- » seizures
- » hyperventilation
- » tremor
- » profound hypokalaemia
- »

GENERAL MEASURES

- » Monitor ECG and treat dysrhythmias.
- » Monitor and correct fluid status and electrolyte abnormalities.
- » Monitor theophylline concentrations, if available. Levels may continue to rise up to 24 hours after ingestion of modified release preparations.

MEDICINE TREATMENT

- Activated charcoal, oral, 50 g diluted in 100 mL water.
  - Multiple doses of activated charcoal enhance elimination.

LoE:IIIa[xli]

Vomiting is common: (R11)

- Metoclopramide, IV/oral, 10 mg 8 hourly as required.

LoE:IVb

Correct hypokalaemia cautiously: E87.6 + (T48.6+X44.99/X64.99/Y14.99)

- Potassium chloride, IV, 20-40 mmol/L in sodium chloride, 0.9%
  - Maximum rate of infusion: 20 mmol/hour.

LoE:IIIa[xlii]

For seizures: R56.8 + (T48.6+X44.99/X64.99/Y14.99)

Treat with benzodiazepines - see section: 14.4.1 Status epilepticus.

Note: Phenytoin should be avoided (due to potential cardiotoxicity).

LoE:IVb[xliii]

REFERRAL

In patients with symptoms of severe overdose (severe hypokalaemia, seizures, refractory hypotension, dysrhythmias, theophylline level >555 µmol/L (100 mg/L), refer for haemodialysis.

LoE:IIIa[xliv]

19.9 SEDATIVE HYPNOTIC POISONING

19.9.1 BENZODIAZEPINE POISONING

T42.4 + (X41.99/X61.99/Y11.99)

DESCRIPTION

Patients present with depressed levels of consciousness, confusion, ataxia, and dysarthria. Benzodiazepines are unlikely to cause significant respiratory

depression unless co-ingested with alcohol or other CNS depressants. However, there is a risk of respiratory depression due to overdose in the elderly.

GENERAL MEASURES

Management is supportive, and ventilation may be required.

Note: The use of flumazenil is not recommended in any patient with possible benzodiazepine poisoning as it increases the risk of convulsions and dysrhythmias.

LoE:Ia[xlv]

19.9.2 LITHIUM POISONING

T43.8 + (X41.99/X61.99/Y11.99)

DESCRIPTION

Lithium toxicity mostly occurs with chronic therapy and may be precipitated by decreased excretion due to renal dysfunction, diuresis, dehydration, hyponatraemia, or drug-drug interactions (e.g. NSAIDs, diuretics, ACE-inhibitors, and ARBs).

Signs and symptoms include:

- » nausea, vomiting, and diarrhoea
- » nystagmus
- » CNS symptoms: tremor, hyperreflexia, choreoathetoid movements, fasciculations, ataxia, agitation, confusion and lethargy

In severe toxicity:

- » Coma
- » Seizures
- » Dysrhythmias
- » Hypotension

GENERAL MEASURES

Monitor:

- » Vitals signs, mental status, and urine output.
- » If available, do serial lithium levels 6 hourly until peaked and declining.
- » Electrolytes and renal function.
- » Cardiac function and treat dysrhythmias (see chapter 3.3: Cardiovascular dysrhythmias).
- » Thyroid function, in chronic toxicity.

MEDICINE TREATMENT

If ingested dose is potentially toxic or modified-release products were ingested, consider WBI.

LoE:IIIb[xlvi]

- » Hydration: administer sodium chloride, 0.9 % to maintain urine flow of 1–2 mL/kg/hour while preventing hypernatremia.

» Correct electrolyte abnormalities: see section: 7.2 Major electrolyte abnormalities.
» For seizures: Treat with benzodiazepines – see section: 14.4.1 Status epilepticus.
Note: Phenytoin should be avoided (due to potential cardiotoxicity).

LoE:Ivb[xlvii]

REFERRAL

Early referral for haemodialysis is indicated in severe lithium poisoning and in patients with renal impairment. Discuss with a specialist.

LoE:IIIa[xlviii]

19.10 ISONIAZID POISONING

T37.1 + (X44.99/X64.99/Y14.99)

DESCRIPTION

Acute toxicity can present with the classic triad of seizures, metabolic acidosis, and coma. Seizures are of a generalised tonic-clonic type, and often refractory to standard anticonvulsant therapy.

GENERAL MEASURES

Supportive management aimed at preventing and managing complications. Treat hyperthermia.

MEDICINE TREATMENT

For seizures:

- Pyridoxine, crushed tablets orally or via NGT in unconscious patient(s).
    - Known amount: Pyridoxine dose is 1 g for every gram of isoniazid ingested (maximum of 5 g)
    - Unknown amount: Pyridoxine dose is 5 g for unknown amount ingested.

LoE:IIIa[xlix]

Benzodiazepines may be used as an interim measure to control seizures:

- Lorazepam, IV/IM, 4 mg, repeat once after 5–10 minutes, if necessary. *LoE:Ivb*[l]

OR

- Diazepam, IV, 10 mg, not faster than 2 mg/minute, repeat once after 5-10 minutes if necessary.

OR

- Clonazepam, IV, 2 mg, repeat once after 5–10 minutes if necessary.

OR

- Midazolam, IM/IV 10 mg, repeat once after 5–10 minutes if necessary.

OR

- Midazolam buccal, 10 mg using the parenteral formulation.

CAUTION
Phenytoin should not be used to control seizures in INH poisoning, as it does not have GABA agonist properties.

LoE:IVb[ii]

REFERRAL
» Uncontrolled seizures

19.11 CALCIUM CHANNEL BLOCKER AND BETA BLOCKER POISONING
T44.7/T46.1 + (X43.99/X63.99/Y14.99)

DESCRIPTION
Cardiovascular toxicity results in profound hypotension, bradycardia, decreased systemic vascular resistance and cardiogenic shock. Depressed level of consciousness and metabolic acidosis are due to poor tissue perfusion. Hyperglycaemia and hypokalaemia may occur. Patients who have co-ingested other cardiac medicines and those with pre-existing cardiac disease are at increased risk of morbidity.

The treatment of suspected cardiogenic shock in calcium channel blocker and beta blocker poisoning follows similar therapeutic principles. The mainstay of treatment is high-dose insulin euglycaemic therapy (HIET) and inotrope and vasopressor infusions.

LoE:IVb[iii]

GENERAL MEASURES
» Monitor vital signs, ECG, and blood glucose.
» Treat symptomatic patients in consultation with a specialist.

MEDICINE TREATMENT
» Caution is advised for all decontamination procedures as they increase vagal tone and may exacerbate bradycardia.
» Activated charcoal may be considered before the onset of symptoms.
» Whole bowel irrigation can be considered for ingestion of modified-release preparations.

LoE:IIIa[iii]

Bradycardia: R00.1 + (T46.1/X44.99/X64.99/Y14.99)

- Atropine, IV 0.5–1 mg every 2–3 minutes to a maximum of 3 mg. *LoE:IVb[iv]*

Hypotension: I95.9 + (T46.1/X44.99/X64.99/Y14.99)

- Start with sodium chloride 0.9%, IV. *LoE:IVb[iv]*

If not effectively controlled

ADD

- Calcium gluconate 10%, IV, 30–60 mL given over 15–30 minutes, with ECG monitoring.

- This may be repeated a maximum of 4 times. *LoE:IVb[iv]*

- Simultaneously use vasopressors and inotropes as needed, e.g. adrenaline (epinephrine) infusion for persistent hypotension (section 20.1: Cardiac arrest in adults) or dobutamine for bradycardia (section 20.11.3: Cardiogenic shock) and refer patient immediately.

REFERRAL
All patients requring HIET should be treated in a High Care or ICU setting

19.12 COTRIMOXAZOLE POISONING
T37.0 + (X44.99/X64.99/Y14.99)

DESCRIPTION
Acute overdose is associated with a low probability of clinically relevant toxicity. Symptoms include nausea and vomiting, dizziness, headache, and neurological symptoms (such as drowsiness, confusion, and mental depression). Other signs include bone marrow depression, haematuria, and renal insufficiency. Hypersensitivity reactions may occur.

GENERAL MEASURES
» Treatment is symptomatic and supportive.
» Monitor FBC, electrolytes, glucose, hepatic, and renal function in symptomatic patients.

19.13 ANTIRETROVIRAL AGENTS POISONING
T37.5 + (X44.99/X64.99/Y14.99)

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

19.14 ILLICIT DRUGS

19.14.1 COCAINE POISONING

T40.5 + (X42.99/X62.99/Y12.99)

DESCRIPTION

Cocaine may be absorbed through any mucous membrane, smoked, ingested, or injected intravenously.

Clinical features:

Mild toxicity: euphoria, anxiety, altered mental status, tachycardia, mild hypertension.

Moderate toxicity: agitation, paranoia, hallucinations, cardiac dysrhythmias.

Severe toxicity: severe headache, seizure, hyperthermia, rhabdomyolysis, severe acidosis, vascular incidents (stroke, MI, intestinal ischaemia etc.), pulmonary oedema.

GENERAL MEASURES

» Supportive management aimed at preventing and managing complications.
» Cool patients with hyperthermia.
» Raised serum creatinine kinase may indicate rhabdomyolysis or myocardial infarction.

» Body packers/stuffers:
  – Patients may ingest packages of cocaine and are at increased risk of life-threatening toxicity in the event of rupture.
  – Abdominal X-rays or CT scan may be helpful in identifying packages.
  – Conservative management is recommended, as any attempt at removal risks package rupture.
  – Activated charcoal and whole bowel irrigation may aid in expelling packets.
  – Surgery is reserved for those who develop obstruction or perforation.

MEDICINE TREATMENT

Benzodiazepines play a key role in the management of sympathetic and psychomotor features of cocaine poisoning.

For sedation and seizures:

Treat with benzodiazepines - see section: 14.4.1 Status epilepticus.

Note: Phenytoin should be avoided (due to potential cardiotoxicity).

LoE:IVb[lvii]

Delirium with severe agitation:

See section 20.8: Delirium with perceptual disturbances.

Arrhythmias:
See section 3.3: Cardiac dysrhythmias.

Hypertension unresponsive to benzodiazepines:
See section 3.6.1: Hypertension, severe.

CAUTION
ß–blockers (other than labetalol) may worsen vasoconstriction and should not be used.

19.14.2 AMPHETAMINE DERIVATIVES POISONING

T43.6 + (X41.99/X61.99/Y11.99)

DESCRIPTION

These include:
» "Ecstasy": 3,4-methylenedioxymethamphetamine (MDMA).
» "Ice" and "Eve": 3,4-methylenedioxy-N-ethylamphetamine (MDEA).
» "Tik": Methamphetamine.

Drug effects are due to the increased release of noradrenaline, dopamine, and serotonin. Patients present with:
» hyperthermia, especially with MDMA
» tachycardia
» hypertension
» angina pectoris and myocardial infarction
» stroke
» hyperactivity
» sweating
» dilated pupils
» teeth grinding
» delirium
» tremors
» seizures and coma

Additional complications include:
» rhabdomyolysis
» hyperkalaemia
» acute tubular necrosis
» hyponatraemia
» dehydration

GENERAL MEASURES

Supportive management aims to maintain stable cardiorespiratory function. Manage hyperthermia, hypoglycaemia, dehydration, and electrolyte abnormalities.

MEDICINE TREATMENT

LoE:IVb[lviii]

For seizures: R56.8 + (T43.6 + X41.99/X61.99/Y11.99)
Treat with benzodiazepines - see section 14.4.1: Status epilepticus.
Note: Phenytoin should be avoided (due to potential cardiotoxicity).

Severe hypertension:
See section 3.6.1: Hypertension, severe.

Haemodialysis may be required for acute renal failure.

19.15 HYDROCARBON POISONING

T52.0 + (X46.99/X66.99/Y16.99)

Note: This section does not include information on aromatic hydrocarbons (e.g. benzene, toluene, xylene) often used by glue sniffers to get high.

DESCRIPTION

Poisoning due to petroleum products, including paraffin, turpentine, petrol, and mineral spirits.

Clinical signs include:

- » chemical pneumonitis
- » arrhythmias
- » nausea and vomiting
- depression, seizures, coma

GENERAL MEASURES

- » If contaminated, remove clothing and wash skin.
- » Do not induce emesis or attempt gastric emptying/lavage.

MEDICINE TREATMENT

- » Activated charcoal is of no value.
- » Observe and examine for chemical pneumonitis. Prophylactic antibiotics are not indicated.

19.16 INGESTION OF CAUSTIC SUBSTANCES

T54.1A/T54.2/T54.3/T54.9 + (X46.99/X66.99/Y16.99)

DESCRIPTION

- » Alkaline: Toilet bowl cleaners, drain cleaners, oven cleaners.
- » Acids: Various e.g. domestic descalers.
- » Caustic substances can cause necrosis of the gut mucosa and underlying tissue, resulting in acute perforation (particularly strong alkalis), and possible strictures later (which can occur with acids and alkalis). Concentrated caustic substances are more corrosive and present a higher risk for necrosis.

GENERAL MEASURES

- » No activated charcoal, forced emesis, or gastric lavage.
- » Rinse mouth with copious amounts of cold water.
- » Make patient nil by mouth and set up IV access.
- » If persistent vomiting, drooling or any difficulty in swallowing, patient may require endoscopic evaluation within 24-48 hours and possible surgical intervention. (Discuss with a specialist).

LoE:IvB[lix]

19.17 ALCOHOLS

19.17.1 ETHANOL POISONING

T51.0 + (X45.99/X65.99/Y15.99)

DESCRIPTION

Acute poisoning usually presents with:

- » Nausea and vomiting
- » Hypoglycaemia
- » Hypokalaemia
- » Depression, seizures, coma
- » Hypothermia
- » Acidosis

Consider other causes for the patient's condition, including hypoglycaemia and head trauma.

GENERAL MEASURES

- » Supportive management is aimed at maintaining stable cardiorespiratory function.
- » Protect the airway (ventilation may be needed).
- » Manage hypothermia, hypoglycaemia, and electrolyte abnormalities.

MEDICINE TREATMENT

- Thiamine, IV, 100 mg in 1 L dextrose, 5%.

19.17.2 ETHYLENE GLYCOL POISONING

T52.3 + (X46.99/X66.99/Y16.99)

DESCRIPTION

Ethylene glycol is the main component of motor vehicle radiator coolant/antifreeze and is occasionally found in brake fluid. It is also found in homemade toilet and drain cleaners.

Mild to moderate intoxication: resembles alcohol intoxication, with nausea and vomiting, nystagmus, ataxia, and somnolence.

Severe intoxication: associated with more severe CNS depression (coma, hypotonia, hyporeflexia) and high anion gap metabolic acidosis. Cardiovascular signs include tachycardia and hypertension. Calcium oxalate crystals cause renal failure and hypocalcaemia, which may manifest with prolongation of the QT interval on ECG or tetany.

$$Anion\ gap = Na - (CL + HCO_3)\ [Normal = 8 - 16]$$

GENERAL MEASURES

- » Consult the Poisons Information Helpline for assistance with management.
- » Treat early to reduce the risk of forming toxic metabolites.

» Monitor blood gases and administer sodium bicarbonate.
» Early haemodialysis is the treatment of choice for severe poisoning with profound acidosis.

MEDICINE TREATMENT

Ethanol

LoE:Ivb[lx]

Indications:

History of ingestion, plus any two of the following criteria:
» Arterial pH <7.3
» Serum bicarbonate <20 mmol/L
» Presence of urinary oxalate crystals (ethylene glycol only) or visual disturbances (methanol only)

Preparation and administration of ethanol:

Step 1: Prepare an ethanol 20% solution:

If using Ethanol 96% BP, oral,

- Add 1 part ethanol 96% to 4 parts juice or water e.g. 250 mL of ethanol 96% with 1000mL water or juice to give a total volume of 1250 mL ethanol 20%.

If using Ethanol 40% v/v (gin, whiskey, vodka), oral

- Add 1 part ethanol 40% to 1 part juice or water e.g. dilute 500mL of ethanol 40% with 500mL water or juice to give a total volume of 1000mL ethanol 20%.
- **Note:** Spirit liquor products in South Africa are frequently bottled at 43% v/v. These can be used interchangeably.

Step 2: Administer a loading dose:

- Ethanol 20% (the solution prepared in Step 1), oral, 4 mL/kg over 15-30 minutes.

Step 3: Continue with maintenance doses:

- Ethanol 20% (the solution prepared in Step 1), oral:
    - Non-drinker: 0.5 mL/kg/hour
    - Chronic drinker: 1 mL/kg/hour

WORKED EXAMPLES

For a 60kg patient who is a non-drinker:
Loading dose: 240 ml of the ethanol 20% solution orally over 15-30 minutes.
Maintenance dose: 30 mL per hour orally of the ethanol 20% solution.

For a 60kg patient who is a chronic drinker:
Loading dose: 240 ml of the ethanol 20% solution orally over 15-30 minutes.
Maintenance dose: 60 mL per hour orally of the ethanol 20% solution.

Note:
» If patients are not co-operative, administer ethanol via a nasogastric tube.

CAUTION
Locally available commercial ethanol products are not approved for IV administration and should not be administered via this route.

» Maintain ethanol levels of 1–1.3 g/L (100–130 mg/dL).
» Where ethylene glycol, methanol (see Section 19.17.3: Methanol poisoning), and ethanol levels are not available for monitoring purposes, titrate the ethanol rate of administration according to improvement in metabolic acidosis and signs of systemic toxicity.
» Increase the dose of ethanol if the patient is receiving concomitant haemodialysis.
» Several days of ethanol therapy may be required until clinical condition improves.
» Alcoholic beverages are sometimes labelled as "percentage proof". Alcohol proof values are double the alcohol percentage (volume/volume) values. i.e. an 80 proof alcohol would be 40% (v/v).

LoE:IVb[lxi]

Cofactor therapy:

- Thiamine, oral, 100 mg daily.
- Pyridoxine, oral, 100 mg daily.

LoE:IVb[lxii]

Metabolic acidosis: E87.2 + (T52.8/X46.99/X66.99/Y16.99)

- Sodium bicarbonate 8.4%, IV, 50–100 mmol/L administered over 30–45 minutes.

Note:

» Rapid correction of acidosis may precipitate seizures in a hypocalcaemic patient. Correct severe or clinically evident hypocalcaemia.
» Monitor glucose levels and correct hypoglycaemia, if necessary. *LoE:IVb*[lxiii]

REFERRAL

Severe poisoning with profound acidosis for early haemodialysis.

19.17.3 METHANOL POISONING

T51.1 + (X45.99/X65.99/Y15.99)

DESCRIPTION

Methanol, once present in methylated spirits, was replaced with less toxic agents 10-20 years ago. However, it may still be found in stove or model fuels, as well as in antifreeze and windscreen washes.

Presentation:

» Ingestion of methanol results in initial mild inebriation (headache, confusion, nausea, and vomiting) similar to ethanol intoxication followed by an asymptomatic/latent period.

» After a latent period of about 12-24 hours, toxic metabolite (formic acid) formation results in severe high anion gap metabolic acidosis, and retinal toxicity (from visual impairment to total blindness).

$$Anion\ gap = Na - (CL + HCO_3)\ [Normal = 8 - 16]$$

MEDICINE TREATMENT

If acidotic or patient has visual disturbances;
Start with immediate ethanol antidote therapy (See section 19.17.2: Ethylene glycol poisoning), and evaluate for urgent dialysis, if available.

LoE:IIIa[lxiv]

19.18 PESTICIDES AND RODENTICIDES

19.18.1 AMITRAZ POISONING

T44.4 + (X43.99/X63.99/Y13.99)
* Notifiable condition.
Poisoning from all pesticides (i.e. agricultural stock remedies) is a notifiable medical condition. Please visit https://nicd.ac.za/nmc-overview/notification-process for further information.

DESCRIPTION

Amitraz is a pesticide/insecticide with α_2-adrenergic agonist properties. It is usually formulated as a tick dip for dogs, cattle, and sheep. Commercial formulations contain up to 20% of amitraz in organic solvents. Poisoning may occur when amitraz is ingested or absorbed via the skin or by inhalation.

A history of an unspecified rat poison or pesticide exposure warrants consideration of other active ingredients such as super-warfarin anticoagulants and organophosphates.

Patients with acute poisoning present with:

- » impaired consciousness
- » drowsiness
- » vomiting
- » hypotension
- » constricted pupils or rarely, dilated pupils
- » bradycardia
- » respiratory depression
- » hypothermia
- » generalized seizures

Other complications include:

- » hyperglycaemia
- » glycosuria
- » mild increase in transaminases

Patients usually regain consciousness within 24 hours.

Note: Amitraz poisoning can be confused with organophosphate poisoning; whilst amitraz causes central nervous system depression, bradycardia, miosis and respiratory depression, 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.

GENERAL MEASURES

» Decontamination of skin and clothes where applicable.
» Supportive and symptomatic treatment to maintain patent airway, adequate respiration and circulation.
» Mechanical ventilation may be needed in some cases.
» Keep patient warm.

MEDICINE TREATMENT

- Activated charcoal, once patient is stabilised.

For severe bradycardia: R00.1 + (T44.4 + X43.99/X63.99/Y13.99)
Manage with atropine - see section 3.3.3: Heart block (second or third degree).

For seizures: R56.8 + (T44.4 + X43.99/X63.99/Y13.99)
Treat with benzodiazepines - see section: 14.4.1 Status epilepticus.
Note: Phenytoin should be avoided (due to potential cardiotoxicity).

LoE:IVb[xv]

19.18.2 ORGANOPHOSPHATE POISONING

T60.0 + (X48.99/X68.99/Y18.99)
* Notifiable condition.
Poisoning from all pesticides (i.e. agricultural stock remedies) is a notifiable medical condition. Please visit https://nicd.ac.za/nmc-overview/notification-process for further information.

DESCRIPTION

Absorption may occur through the skin, gastrointestinal tract if taken orally, or by inhalation.

Patients present with muscarinic and nicotinic manifestations of intoxication.

» Peripheral effects:
  – *Muscarinic overstimulation:* bradycardia, hypotension, salivation, lacrimation, vomiting, diarrhoea, increased bronchial secretions, bronchospasm, and miosis (pinpoint pupils).
  – *Nicotinic overstimulation:* muscle weakness and fasciculations, tachycardia, hypertension, mydriasis (dilated pupils).
» Central effects: coma, confusion, convulsions.

Diagnosis is supported by low serum pseudocholinesterase levels.

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:IIIa[lxvi]

CAUTION

A history of an unspecified rat poison or pesticide exposure warrants consideration of other active ingredients such as super-warfarin anticoagulants and amitraz.

GENERAL MEASURES

» Ensure use of personal protective equipment for staff – gloves, gowns, and eye protection. If staff come into contact with body fluids, wash off immediately. *LoE:IIIa*[lxvii]

» Decontamination procedures for the patient should only be done once the patient is fully resuscitated.

» Remove patient's clothes and wash the body with soap and water. Place clothes in closed bags.

» Maintain adequate ventilation and circulation. Ventilatory support in ICU may be required. Suction secretions frequently.

» Note: If using suxamethonium for intubation, consider that metabolism may be delayed and prolonged respiratory support may be required. Use alternative agents if possible (See section 12.3: Muscle relaxants).

MEDICINE TREATMENT

- Activated charcoal, once patient is stabilised.

For bronchorrhoea, bronchospasm, or bradycardia: *LoE:IIIa*[lxviii]

- Atropine bolus, IV, 2 mg.
  - 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. 2 mg, 4 mg, 8 mg, 16 mg etc.
    - If no clinical response, give double the dose.
    - If some response, give the same or reduced dose.
- Follow atropinisation with atropine, IV infusion.
  - Titrate according to clinical response, starting at 10% of the total bolus dose. Calculate the total dose of atropine given as boluses (as described above). Give 10–20% of this dose per hour, titrating according to clinical response.

- e.g.: 40 mg of Atropine in 200 mL sodium chloride 0.9% (0.2 mg/mL) 10-20 mg/hour = 50-100 mL/hr.

- Reassess frequently and adjust atropine infusion as follows:
  - Bronchial secretions, bronchospasm or bradycardia recurs: increase dose.
  - Good control of bronchial secretions and signs of atropine overdose (tachycardia, mydriasis, 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; instead, wean over at least 24 hours.

» Tachycardia and mydriasis are not contraindications for giving atropine in the acute resuscitation setting. *LoE:IIIa*[lxix]

For severe agitation:

- Diazepam, IV, 5–10 mg, immediately.
  - Repeat after 30–60 minutes if needed. *LoE:IIIa*[lxx]

REFERRAL

Refer if ventilatory support is unavailable.

19.18.3 PARAQUAT POISONING

T60.3 + (X48.99/X68.99/Y18.99)

* Notifiable condition.

Poisoning from all pesticides (i.e. agricultural stock remedies) is a notifiable medical condition. Please visit https://nicd.ac.za/nmc-overview/notification-process for further information.

DESCRIPTION

Paraquat is the most toxic herbicide known, and toxicity causes multi-organ failure which is often fatal. Following oral ingestion, patients present with oral, oesophageal, and gastric erosions with severe gastroenteritis. Multi-organ failure develops within 1–3 days, particularly renal and respiratory failure. Patients surviving the initial phase usually develop pulmonary fibrosis.

GENERAL MEASURES

» Supportive and symptomatic management to maintain patent airway, adequate respiration, and circulation.

» Mechanical ventilation may be needed in some cases.

» Palliative care is the mainstay of treatment.

CAUTION
High inspiratory fraction of inspired oxygen (FiO2) may worsen pulmonary toxicity. Supplemental oxygen should only be provided if the patient is confirmed hypoxic.

MEDICINE TREATMENT
- Activated charcoal

19.19 ANTICOAGULANT (WARFARIN AND RODENTICIDE SUPERWARFARIN) POISONING

T45.5 + (X44.99/X64.99/Y14.99)
* Notifiable condition – rodenticide superwarfarin poisoning
Poisoning from all pesticides (i.e. agricultural stock remedies) is a notifiable medical condition. Please visit https://nicd.ac.za/nmc-overview/notification-process for further information.

DESCRIPTION
Poisoning due to ingestion of warfarin and superwarfarins, e.g. rat poison and other vermin poisons. Warfarin toxicity can occur with either acute overdose or unintentionally, during therapeutic use, whereby drug interactions increase warfarin bioavailability (e.g. concomitant enzyme inhibitor), or concomitant anticoagulant drugs are administered (e.g. NSAIDS). Bleeding is the main clinical presentation e.g. gastrointestinal or intracranial bleeding; however bleeding may be occult. Superwarfarins are more potent than warfarin and may have a long duration of effect; small doses of concentrated formulations may cause significant anticoagulation.

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

GENERAL MEASURES
» Resuscitation.
» Stop warfarin in patients on therapy.
» Measure INR at baseline and 48 hours post ingestion, as the anticoagulant effect may be delayed by 1–2 days.

MEDICINE TREATMENT
Do NOT give vitamin K_1 prophylactically. It is only indicated when there is active bleeding or a specifically raised INR (INR > 4).

Active bleeding:
R58 + (T45.5 + X44.99/X64.99/Y14.99)
- Lyophilised plasma, IV, 15 mL/kg.

OR *LoE:IIIa*[lxxi]
- Fresh Frozen Plasma, IV, 15 mL/kg.

AND

- Vitamin K_1, IV, 10 mg
    - Administer as a slow IV injection.
    - Do not dilute or mix with other injectables.

For patients on long term vitamin K antagonist anticoagulants, e.g. warfarin:

- Temporarily discontinue anticoagulant therapy.
- Decrease Vitamin K dose by half, i.e. Vitamin K1, IV, 5 mg. Administer as a slow IV injection.

LoE:IV

No bleeding but INR is raised (INR > 4):

Note: If Vitamin K_1 is only available as a parenteral preparation, administer the same preparation orally as this is safest in anticoagulant poisoning.

Patients NOT on long-term therapeutic anticoagulants and INR > 4.0:

- Vitamin K_1, oral, 10-20 mg.
    - Check INR at least 12 hours after vitamin K_1 has been administered. Repeated doses should be guided by further INR (or PT) measurements every 4-6 hours until the patient is stable, and thereafter, every 24 hours. INR (or PT) levels may take 3-4 days to normalise.

Patients on long-term vitamin K antagonist anticoagulant drugs (e.g. warfarin therapy):

If INR 5-8:

- Temporarily discontinue any anticoagulant treatment.

If INR > 8:

- Vitamin K_1, oral, 0.5 – 1.0 mg (one tenth of the normal dose).
    - A repeat dose may be given 12-24 hrs later if the INR remains ≥ 8.

Note:

LoE:IVb[lxxii]

» These patients are complex and require management in consultation with a haematologist.
» Patients with prosthetic heart valves receiving high-dose vitamin K have a higher risk for increased resistance to warfarin and development of thromboembolism. Treat as above but monitor INR frequently to prevent overcorrection. Treat in consultation with a specialist.
» For patients on other anticoagulant therapies, additional antagonists may be required.
» In all patients on therapeutic warfarin, a major overdose or bleeding episode should prompt careful review of the need for anticoagulation.
» Warfarin should be re-started once the INR is in the therapeutic range if it is still indicated.
» In patients with superwarfarin toxicity, treatment with vitamin K_1 may need to be prolonged for several months as superwarfarins are very long acting. Discuss with the Poisons Information Centre or haematologist for advice on dosing and duration of treatment.

19.20 CARBON MONOXIDE POISONING

T58 + (X47.99/X67.99/Y17.99)

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
- » metabolic acidosis (severe)
- » high arterial carboxyhaemoglobin levels
- » impaired level of consciousness
- » tachycardia
- » chest pain
- » retinal haemorrhages
- » respiratory alkalosis (mild)

Note: There may be a normal arterial PaO2, 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

- Oxygen, 100%, via positive pressure facemask.

For seizures: R56.8 + (T58 + X47.99/X67.99/Y17.99)
Treat with benzodiazepines - see section: 14.4.1 Status epilepticus.
Note: Phenytoin should be avoided (due to potential cardiotoxicity).

LoE:IVb[lxxiii]

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.

19.21 HEAVY METAL POISONING

T56.0/T56.1/T56.4/T56.8/T57.0

DESCRIPTION

This includes mercury, arsenic, gold, copper, lead poisoning, thallium etc. Frequent/occupational inhalation of metal fumes and particles can cause metal fume fever, a flu-like syndrome with fever, malaise, bronchospasm, and bi-weekly variations in severity that may be mildest on the weekend and most severe on Monday or Tuesday after returning to work. This may be confused with an acute viral illness with fever, cough, sweating, myalgia, headache etc. The course of the illness is usually benign.

The management of heavy metal toxicity depends on the specific metal, route of exposure and length of time between exposure and clinical presentation of symptoms. Discuss all potential patients with the Poisons Information Helpline for further investigation, treatment options and possible referral.

LoE:IVb[lxxiv]

Metal	Signs and symptoms
Copper salts	GIT irritation, hepatotoxicity, and haemolysis.
Arsenic	Impairs cellular respiration, resulting in multi-organ dysfunction.
Mercury	Clinical effects depend on the route of exposure and type of mercury (inorganic versus organic).
Lead	Chronic toxicity more common. Affects nervous, gastrointestinal, renal, and haematopoietic systems.
Gold	Deposition of immune complexes in kidneys and skin; mucus membrane inflammation
Thallium	Alopecia and painful ascending peripheral neuropathy.

Table 19.6: Clinical features of heavy metal poisoning

LoE:IVb[lxxv]

19.22 POISONING WITH SUBSTANCES THAT CAUSE METHAEMOGLOBINAEMIA

D74.9 + (T41.3/T41.4/T46.3 + X44.99/X64.99/Y14.99)

DESCRIPTION

- Substances causing methaemoglobinaemia include nitrites, nitroglycerine, dapsone, mothballs (naphthalene), local anaesthetics, phenazopyridine, chlorates, and anilines.
- Nitrites are used to cure meat in the formal and informal butchery sector.
- Patients present with:
  - Deep cyanosis with only mildly reduced oxygen saturation
  - CNS depression, and
  - arrhythmias.

Note: Methaemoglobinaemia causes patients to appear cyanosed with falsely high conventional pulse oximetry readings and normal PaO2. Blood gas analysis using co-oximetry is required to specifically measure methaemoglobin levels.

MEDICINE TREATMENT

- Oxygen via facemask.

In symptomatic cases or patients with high methaemoglobin values > 30%:

- Methylene blue (methylthionine chloride) 1% dilute solution, slow IV infusion, 1–2 mg/kg administered over 5 minutes.
    - Repeat in 1 hour and, if necessary, 4 hourly up to a total dose of 7 mg/kg.
    - Side effects include precordial pain, restlessness, and dyspnoea.
    - After administration of methylene blue, oxygen saturation may drop initially.

In life-threatening cases not responding to methylene blue, or if methylene blue is not available, exchange transfusion may be considered. Refer to the Poisons Information Helpline for advice on treatment and possible alternatives to methylene blue.

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