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 March 2025		

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, 500 mg to 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. A *LoE:IVb*

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, 500 mg to 1 g 4 to 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). *LoE:IIIb[v]*
    - Shock

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 to 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 to 100 mL sodium chloride, 0.9% or dextrose 5%, administered over 5 to 10 minutes.
    - The dose of antivenom is the same for adults and children. *LoE:IVb*[ix]
    - Spontaneous systemic bleeding should stop within 15 to 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, 500 mg to 1 g 4 to 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 to 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 to 10 mL diluted in 50 to 100 mL sodium chloride, 0.9% or dextrose 5%, administered over 5 to 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, 500 mg to 1 g 4 to 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 to 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
- decreased bowel sounds
- hypothermia

- » bradycardia
- » altered (decreased) consciousness
- » hypotension

Salicylism: e.g. aspirin

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

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 to 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 to 24 hours, peaking in severity at about 72 to 96 hours. Patients may make a full recovery in 5 to 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]

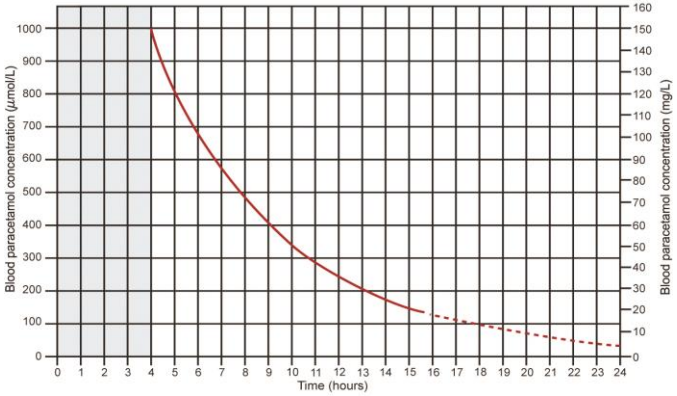
Repeated supratherapeutic ingestion (RSTI):

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 Smartphone 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. Ringer's 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. 100 mL).
  - 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%, 150 mL added to dextrose 5%, 1 L 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 to 200 mL/hour, targeting a urine output of 2 mL/kg/hour.
    - Titrate the sodium bicarbonate maintenance infusion to a urinary pH of 7.5 to 8.5 and blood pH of 7.45 to 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 to 0.1 mg 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 to 2 mL/kg administered in bolus doses. (Specialist consultation).
  - Aim to achieve a serum pH of 7.45 to 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 15 mg/kg/hour over about 6 hours.
  - Beware of hypotension.
  - **Note:** Prolonged use (>24 hours) of high doses is 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.

LoE:IIIa[xl]

- Deferoxamine can be used in pregnant women.

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*[xii]

Vomiting is common: (R11) *LoE:IVb*
- Metoclopramide, IV/oral, 10 mg 8 hourly as required.

Correct hypokalaemia cautiously: E87.6 + (T48.6+X44.99/X64.99/Y14.99)
- Potassium chloride, IV, 20 to 40 mmol/L in sodium chloride, 0.9%.
  - Maximum rate of infusion: 20 mmol/hour. *LoE:IIIa*[xiii]

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[xiii]

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*[xiv]

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[xiv]

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 to 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 to 10 minutes if necessary.

OR

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

OR

- Midazolam, IM/IV 10 mg, repeat once after 5 to 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[ii]

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 to 1 mg every 2 to 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 to 60 mL given over 15 to 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:IvbIx

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 500 mL of ethanol 40% with 500 mL water or juice to give a total volume of 1000 mL 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 to 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 to 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 to 1.3 g/L (100 to 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
- » bradycardia
- » respiratory depression
- » hypothermia
- » generalized seizures
- » constricted pupils or rarely, dilated pupils

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[lxv]

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 to 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]

STEP 1:

- Atropine bolus, IV
  - Administer 2mg atropine as an IV bolus.
  - 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.

ATROPINE : SECTION 21

A high strength formulation (100mg in 10mL) of atropine may be available at select facilities. Confirm the strength available before preparing and administering to the patient.

STEPS TO FOLLOW	**ATROPINE FORMULATIONS**		
Select formulation of atropine available	**100mg/10mL ampoule SECTION 21**	**1mg/mL ampoule**	**500mcg/mL ampoule**
Prepare atropine syringe for IV bolus	Dilute 1mL atropine with 9mL diluent for a 1mg/mL solution.	Use undiluted	
Atropine doses	**Volume of atropine**		
	Diluted 1mg/mL solution SECTION 21	**1mg/mL ampoule**	**500mcg/mL ampoule**
2mg	2mL	2mL	4mL
4mg	4mL	4mL	8mL
8mg	8mL	8mL	16mL
16mg	16mL	16mL	32mL
32mg	32mL	32mL	64mL
etc			
Total bolus dose = e.g. 62mg			

STEP 2:

- Atropine IV infusion
  - Calculate the total dose of atropine given as boluses. Give 10% of this dose per hour, titrating up to 20% per hour based on clinical response.

Worked example: Total bolus dose = 62mg. Administer 10-20% (6-12mg) per hour by IV infusion.			
STEPS TO FOLLOW	**ATROPINE FORMULATIONS**		
Select formulation of atropine available	**Atropine 100mg/10mL ampoule (SECTION 21)**	**Atropine 1mg/mL ampoule**	**Atropine 500mcg/mL ampoule**
Prepare solution for IV infusion	200mg atropine = 20mL (2 amps) Add to 180mL diluent to prepare a 1mg/mL atropine solution	40mg atropine = 40mL (40 amps) Add to 160mL diluent to prepare a 0.2mg/mL solution	40mg atropine = 80mL (80 amps) Add to 120mL diluent to prepare a 0.2mg/mL solution
Calculate 10% of total bolus dose to be given per hour	6mg	6mg	6mg
Start infusion at rate equivalent to 10% of bolus dose	6mL/hour	30mL/hour	30mL/hour

  - Titrate according to clinical response, by frequent reassessment and adjustments:
    - 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.

STEP 3 (May be required in some patients)

LoE:IVb

- Atropine bolus, IV
  - Some organophosphates are lipophilic in nature and cholinergic symptoms may recur even once the atropine infusion is in place. In such cases, repeat bolus doses, starting at 2mg, can be given in addition to the atropine infusion. Bolus doses may be increased incrementally as in Step 1 above.
  - Once the patient is restabilised, consider increasing the infusion rate, to 10% of the new total bolus doses i.e.

Infusion rate = 0.1 X (STEP 1 total bolus dose + STEP 3 total bolus dose).

- Titrate the infusion rate up to 20% based on clinical response.

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*[kix]

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 to 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://www.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 to 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 to 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 to 6 hours until the patient is stable, and thereafter, every 24 hours. INR (or PT) levels may take 3 to 4 days to normalise.

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

If INR 5 to 8:

- Temporarily discontinue any anticoagulant treatment.

If INR >8:

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

LoE:IVb[lxxii]

Note:

» 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 to 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.

References:

[i] Amoxicillin/clavulanic acid, oral: Blaylock RS. Antibiotic use and infection in snakebite victims. S Afr Med J. 1999 Aug;89(8):874-6. http://www.ncbi.nlm.nih.gov/pubmed/10488365

ii NSAID caution: World Health Organisation: Guidelines for the prevention and clinical management of snakebite in Africa. https://www.who.int/health-topics/snakebite#tab=overview

iii Polyvalent antivenom: Wood D, Webb C, DeMeyer J. Severe snakebites in northern KwaZulu-Natal: treatment modalities and outcomes. S Afr Med J. 2009 Nov;99(11):814-8. http://www.ncbi.nlm.nih.gov/pubmed/20218483

Polyvalent antivenom: Müller GJ, Modler H, Wium CA, Veale DJH, Marks CJ. Snake bite in southern Africa: diagnosis and management. CME Oct 2012; 30(10):362-82.http://www.cmej.org.za/index.php/cmej/article/view/2546/2581

SAMF 14th edition, page 653

[iv] Antivenom (period to administer): Müller GJ, Modler H, Wium CA, Veale DJH, Marks CJ. Snake bite in southern Africa: diagnosis and management. CME Oct 2012; 30(10):362-82. http://www.cmej.org.za/index.php/cmej/article/view/2546/2581

[v] Polyvalent snake antivenom (indications): Wood D, Sartorius B, Hift R. Snakebite in NE South Africa: clinical characteristics and risks for severity. S Afr Fam Prac 2016; 58(2):62-67. https://www.tandfonline.com/doi/full/10.1080/20786190.2015.1120934

Polyvalent snake antivenom (indications): Wood, Sartorius, Hift. Classifying snakebite in South Africa: validating a scoring system. S Afr Med J 2017;107(1):46-51. https://www.ncbi.nlm.nih.gov/pubmed/28112091

Hardcastle TC et al (progressive cytotoxic envenomation) . Approach to the diagnosis and management of snakebite envenomation in South Africa in humans: The hospital phase – emergency unit general principles. SAMJ June 2023 Vol 113, No6.

[vi] Adrenaline (epinephrine), SC: Nuchpraryoon I, Garner P. Interventions for preventing reactions to snake antivenom. Cochrane Database Syst Rev. 2000;(2):CD002153. Review. http://www.ncbi.nlm.nih.gov/pubmed/21572992

Adrenaline (epinephrine), SC: de Silva HA, Pathmeswaran A, Ranasinha CD, Jayamanne S, Samarakoon SB, Hittharage A, Kalupahana R, Ratnatilaka GA, Uluwatthage W, Aronson JK, Armitage JM, Lalloo DG, de Silva HJ. Low-dose adrenaline, promethazine, and hydrocortisone in the prevention of acute adverse reactions to antivenom following snakebite: a randomised, double-blind, placebo-controlled trial. PLoS Med. 2011 May;8(5):e1000435. http://www.ncbi.nlm.nih.gov/pubmed/21572992

[vii] Polyvalent antivenom (neurotoxic, cytotoxic or unidentified snakebites): Müller GJ, Modler H, Wium CA, Veale DJH, Marks CJ. Snake bite in southern Africa: diagnosis and management. CME Oct 2012; 30(10):362-82.http://www.cmej.org.za/index.php/cmej/article/view/2546/2581

Polyvalent antivenom: South African Medicines Formulary. 14th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.

viii Polyvalent antivenom (cytotoxic Mozabique spitting cobra bite): World Health Organisation: Guidelines for the prevention and clinical management of snakebite in Africa. https://www.who.int/health-topics/snakebite#tab=overview

[ix] Boomslang monovalent antivenom (neurotoxic, cytotoxic or unidentified snakebites): Müller GJ, Modler H, Wium CA, Veale DJH, Marks CJ. Snake bite in southern Africa: diagnosis and management. CME Oct 2012; 30(10):362-82. http://www.cmej.org.za/index.php/cmej/article/view/2546/2581

Boomslang monovalent antivenom: Guidelines for the prevention and clinical management of snakebite in Africa. https://www.who.int/health-topics/snakebite#tab=overview

[x] Local anaesthetic ophthamlmic drops (therapeutic group): Lawrenson JG, Edgar DF, Tanna GK, Gudgeon AC. Comparison of the tolerability and efficacy of unit-dose, preservative-free topical ocular anaesthetics. Ophthalmic Physiol Opt. 1998 Sep;18(5):393-400. https://pubmed.ncbi.nlm.nih.gov/10023471/

[xi] Polyvalent antivenom (snake venom in the eye): Müller GJ, Modler H, Wium CA, Veale DJH, Marks CJ. Snake bite in southern Africa: diagnosis and management. CME Oct 2012; 30(10):362-82. http://www.cmej.org.za/index.php/cmej/article/view/2546/2581

[xii]Paracetamol, oral: Müller GJ, Modler H, Wium CA, Veale DJH, Marks CJ. Scorpion sting in southern Africa: diagnosis and management. CME Oct 2012; 30(10):356-61.http://www.cmej.org.za/index.php/cmej/article/view/2545/2580

[xiii] Lidocaine. 1-2%, infiltration:Aksel G, Güler S, Doğan N, Corbacioğlu S. A randomized trial comparing intravenous paracetamol, topical lidocaine, and ice application for treatment of pain associated with scorpion stings. Hum ExpToxicol. 2014 Oct 10. pii: 0960327114551394. [Epub ahead of print] http://www.ncbi.nlm.nih.gov/pubmed/25304965

Lidocaine. 1-2%, infiltration: Chippaux JP. Emerging options for the management of scorpion stings. Drug Des Devel Ther. 2012;6:165-73. http://www.ncbi.nlm.nih.gov/pubmed/22826633

[xiv] Opiates (caution): Müller GJ, Modler H, Wium CA, Veale DJH, Marks CJ. Scorpion sting in southern Africa: diagnosis and management. CME Oct 2012; 30(10):356-61. http://www.cmej.org.za/index.php/cmej/article/view/2545/2580

[xv] Scorpion sting: Muller, G J et al. Scorpion sting in southern Africa: diagnosis and management. Continuing Medical Education, [S.l.], v. 30, n. 10, p. 356-361, sep. 2012. ISSN 2078-5143. Available at: <http://www.cmej.org.za/index.php/cmej/article/view/2545/2580>. Date accessed: 18 Apr. 2023.

[xvi] Calcium gluconate 10%, bolus IV: South African Medicines Formulary. 14th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.

Package Insert: Calcium Gluconate Injection Fresenius solution for injection. Fresenius Kabi Manufacturing SA (Pty) Ltd. Last revised 7 February 2023.

Shann F. Drug doses: 17th Edition. 2017.

[xvii] Spider antivenom: Muller GJ, Wium CA, Marks CJ, du Plessis CE, Veale DJH. Spider bite in southern Africa: diagnosis and management. CME October 2012;30(10): 382-91. http://www.cmej.org.za/index.php/cmej/article/view/2547/2582
[xviii] Calcium gluconate 10%, bolus IV: South African Medicines Formulary. 14th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.
Package Insert: Calcium Gluconate Injection Fresenius solution for injection. Fresenius Kabi Manufacturing SA (Pty) Ltd. Last revised 7 February 2023.
Shann F. Drug doses: 17th Edition. 2017.
[xix] Gastric lavage: Benson BE, Hoppu K, Troutman WG, Bedry R, Erdman A, Höjer J, Mégarbane B, Thanacoody R, Caravati EM; American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Position paper update: gastric lavage for gastrointestinal decontamination. Clin Toxicol (Phila). 2013 Mar;51(3):140-6. https://www.ncbi.nlm.nih.gov/pubmed/23418938
[xx] Activated charcoal (multi-dose): Position statement and practice guidelines on the use of multi-dose activated charcoal in the treatment of acute poisoning. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. J ToxicolClinToxicol. 1999;37(6):731-51.http://www.ncbi.nlm.nih.gov/pubmed/10584586
[xxi] Activated charcoal (single dose): National Department of Health: Affordable Medicines, EDP-Adult Hospital level. Medicine Review: Single dose activated charcoal for poisonings, May 2019. http://www.health.gov.za/
Activated charcoal (single dose): Chyka PA, Seger D; American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Position statement: single-dose activated charcoal. J Toxicol Clin Toxicol 1997;35(7):721-41. https://www.ncbi.nlm.nih.gov/pubmed/15822758
Activated charcoal (single dose): Rosenberg J, Benowitz NL, Pond S. Pharmacokinetics of drug overdose. Clin Pharmacokinet 1981; 6:161– 192. https://www.ncbi.nlm.nih.gov/pubmed/7016383
Activated charcoal (single dose): Yeates PJA, Thomas SHL. Effectiveness of delayed activated charcoal administration in simulated paracetamol (acetaminophen) overdose. Br J Clin Pharmacol 2000; 49:11–14. https://www.ncbi.nlm.nih.gov/pubmed/7016383
Activated charcoal (single dose): Laine K, Kivisto¨ KT, Pelttari S, Neuvonen PJ. The effect of activated charcoal on the absorption of fluoxetine, with special reference to delayed charcoal administration. Pharmacol Toxicol 1996; 79:270– 273. https://www.ncbi.nlm.nih.gov/pubmed/8936562
Activated charcoal (single dose): Laine K, Kivisto¨ KT, Neuvonen PJ. Effect of delayed administration of activated charcoal on the absorption of conventional and slow-release verapamil. J Toxicol Clin Toxicol 1997; 35:263–268. https://www.ncbi.nlm.nih.gov/pubmed/9140320
Activated charcoal (single dose): Laine K, Kivisto¨ KT, Ojala-Karlsson P, Neuvonen PJ. Effect of activated charcoal on the pharmacokinetics of pholcodine, with special reference to delayed charcoal ingestion. Ther Drug Monit 1997; 19:46– 50. https://www.ncbi.nlm.nih.gov/pubmed/9029746
Activated charcoal (single dose): Green R, Grierson R, Sitar DS, Tenenbein M. How long after drug ingestion is activated charcoal still effective? J Toxicol Clin Toxicol 2001; 39:601– 605. https://www.ncbi.nlm.nih.gov/pubmed/11762668
[xxii] Whole bowel irrigation (with polyethylene glycol): Thanacoody R, Caravati EM, Troutman B, Hojer J, Benson B, Hoppu K et al. Position paper update: whole bowel irrigation for gastrointestinal decontamination of overdose patients. Clin Toxicol (Phila) 2015;53:5-12. https://www.ncbi.nlm.nih.gov/pubmed/25511637
[xxiii]Paracetamol overdosing – repeated subtherapeutic ingestion: Chiew AL, Reith D, Pomerleau A, Wong A, Isoardi KZ, Soderstrom J, Buckley NA. Updated guidelines for the management of paracetamol poisoning in Australia and New Zealand. Med J Aust. 2020 Mar;212(4):175-183. https://pubmed.ncbi.nlm.nih.gov/31786822/
[xxiv]Paracetamol nomogram: Chiew AL, Reith D, Pomerleau A, Wong A, Isoardi KZ, Soderstrom J, Buckley NA. Updated guidelines for the management of paracetamol poisoning in Australia and New Zealand. Med J Aust. 2020 Mar;212(4):175-183. https://pubmed.ncbi.nlm.nih.gov/31786822/
Paracetamol nomogram: 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.http://www.ncbi.nlm.nih.gov/pubmed/18312195
[xxv] N-Acetycysteine,IV (dosing regimen): Chiew AL, Reith D, Pomerleau A, Wong A, Isoardi KZ, Soderstrom J, Buckley NA. Updated guidelines for the management of paracetamol poisoning in Australia and New Zealand. Med J Aust. 2020 Mar;212(4):175-183. https://pubmed.ncbi.nlm.nih.gov/31786822/
N-Acetycysteine,IV (dosing regimen): Wong A, Graudins A. Simplification of the standard three-bag intravenous acetylcysteine regimen for paracetamol poisoning results in a lower incidence of adverse drug reactions. Clin Toxicol (Phila). 2016;54(2):115-9. https://www.ncbi.nlm.nih.gov/pubmed/26594846
[xxvi] N-acetylcysteine, oral (dosing for paracetamol poisoning): Yarema MC, Johnson DW, Berlin RJ, Sivilotti ML, Nettel-Aguirre A, Brant RF, et al. Comparison of the 20-hour intravenous and 72-hour oral acetylcysteine protocols for the treatment of acute acetaminophen poisoning. Ann Emerg Med. 2009 Oct;54(4):606-14. https://www.ncbi.nlm.nih.gov/pubmed/19556028
N-acetylcysteine, oral (dosing for paracetamol poisoning): Williamson K, Wahl MS, Mycyk MB. Direct comparison of 20-hour IV, 36-hour oral, and 72-hour oral acetylcysteine for treatment of acute acetaminophen poisoning. Am J Ther. 2013 Jan;20(1):37-40. https://www.ncbi.nlm.nih.gov/pubmed/23299230
N-acetylcysteine, oral (dosing for paracetamol poisoning): Rumack and Bateman. Acetaminophen and acetylcysteine dose and duration: past, present and future. Clin Toxicol (Phila) 2012;50(2):91-98. https://www.ncbi.nlm.nih.gov/pubmed/22320209

[xxvii] N-acetylcysteine, oral (adverse effects): South African Medicines Formulary. 14th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.
[xxviii] Activated charcoal:Bradberry SM, Vale JA. Multiple-dose activated charcoal: a review of relevant clinical studies. J ToxicolClinToxicol. 1995;33(5):407-16. Review. http://www.ncbi.nlm.nih.gov/pubmed/7650765
Whole bowel irrigation:Mayer AL, Sitar DS, Tenenbein M. Multiple-dose charcoal and whole-bowel irrigation do not increase clearance of absorbed salicylate. Arch Intern Med. 1992 Feb;152(2):393-6. http://www.ncbi.nlm.nih.gov/pubmed/1739372
[xxix] Activated charcoal:Bradberry SM, Vale JA. Multiple-dose activated charcoal: a review of relevant clinical studies. J ToxicolClinToxicol. 1995;33(5):407-16. Review.http://www.ncbi.nlm.nih.gov/pubmed/7650765
Whole bowel irrigation:Mayer AL, Sitar DS, Tenenbein M. Multiple-dose charcoal and whole-bowel irrigation do not increase clearance of absorbed salicylate. Arch Intern Med. 1992 Feb;152(2):393-6. http://www.ncbi.nlm.nih.gov/pubmed/1739372
[xxx] Dextrose (salicylate poisoning): Kuzak N, Brubacher JR, Kennedy JR. Reversal of salicylate-induced euglycemic delirium with dextrose. Clin Toxicol (Phila). 2007 Jun-Aug;45(5):526-9. https://pubmed.ncbi.nlm.nih.gov/17503260/
[xxxi] Sodium bicarbonate (salicylate poisoning): Prescott LF, Balali-Mood M, Critchley JA, Johnstone AF, Proudfoot AT. Diuresis or urinary alkalinisation for salicylate poisoning? Br Med J (Clin Res Ed). 1982 Nov 13;285(6352):1383-6. https://pubmed.ncbi.nlm.nih.gov/6291695/
[xxxii] Haemodialysis (salicylate poisoning): McCabe DJ, Lu JJ. The association of hemodialysis and survival in intubated salicylate-poisoned patients. Am J Emerg Med. 2017 Apr 10. pii: S0735-6757(17)30280-2. https://www.ncbi.nlm.nih.gov/pubmed/28438446
Haemodialysis (salicylate poisoning): Juurlink DN, Gosselin S, Kielstein JT, Ghannoum M, Lavergne V, Nolin TD, Hoffman RS; EXTRIP Workgroup. Extracorporeal Treatment for Salicylate Poisoning: Systematic Review and Recommendations From the EXTRIP Workgroup. Ann Emerg Med. 2015 Aug;66(2):165-81. https://pubmed.ncbi.nlm.nih.gov/25986310/
[xxxiii] Naloxone: South African Medicines Formulary. 14th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.
Naloxone: FDA approved package insert – Hospira Inc.: Naloxone Hydrochloride, 2015. http://medlibrary.org/lib/rx/meds/naloxone-hydrochloride-8/
[xxxiv] TCAs - toxicity: David M. Taylor, Thomas R. E. Barnes, Allan H. Young. The Maudsley prescribing guidelines in psychiatry.13th edition. Hoboken, NJ : Wiley, 2019. (page 770).
[xxxv] Sodium bicarbonate, IV (tricyclic antidepressant poisoning): Bruccoleri RE, Burns MM. A Literature Review of the Use of Sodium Bicarbonate for the Treatment of QRS Widening. J Med Toxicol. 2016 Mar;12(1):121-9. https://www.ncbi.nlm.nih.gov/pubmed/26159649
[xxxvi] Phenytoin (tricyclic antidepressant poisoning): South African Medicines Formulary. 14th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.
[xxxvii] Flumazenil (tricyclic antidepressant poisoning): Penninga EI, Graudal N, Ladekarl MB, Jürgens G. Adverse Events Associated with Flumazenil Treatment for the Management of Suspected Benzodiazepine Intoxication - A Systematic Review with Meta-Analyses of Randomised Trials. Basic ClinPharmacolToxicol. 2016 Jan;118(1):37-44. http://www.ncbi.nlm.nih.gov/pubmed/26096314
[xxxviii] Desferrioxamine, IV: Tenenbein M. Benefits of parenteral deferoxamine for acute iron poisoning. J ToxicolClinToxicol. 1996;34(5):485-9. http://www.ncbi.nlm.nih.gov/pubmed/8800185
Desferrioxamine, IV: Tenenbein M, Kowalski S, Sienko A, Bowden DH, Adamson IY. Pulmonary toxic effects of continuous desferrioxamine administration in acute iron poisoning. Lancet. 1992 Mar 21;339(8795):699-701. http://www.ncbi.nlm.nih.gov/pubmed/1347583
Desferrioxamine, IV: South African Medicines Formulary. 14th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.
[xxxix] Deferoxamine (IV admin) Drug Monograph. Deferoxamine. Clinical Key accessed 3 February 2023. https://www.clinicalkey.com/#!/content/6-s2.0-170?scrollTo=%23top
Package Insert. Desferal. Novartis 10 Aug 2006.
[xl]Desferrioxamine, IV (pregnancy): Piccioni MG, Capone C, Vena F, Del Negro V, Schiavi MC, D'Ambrosio V, Giancotti A, Smacchia MP, Brunelli R. Use of deferoxamine (DFO) in transfusion-dependent β-thalassemia during pregnancy: A retrospective study. Taiwan J Obstet Gynecol. 2020 Jan;59(1):120-122. https://pubmed.ncbi.nlm.nih.gov/32039778/
[xli]Activated charcoal (multi-dose): Ghannoum M, Wiegand TJ, Liu KD, Calello DP, Godin M, Lavergne V, Gosselin S, Nolin TD, Hoffman RS; EXTRIP workgroup. Extracorporeal treatment for theophylline poisoning: systematic review and recommendations from the EXTRIP workgroup. Clin Toxicol (Phila). 2015 May;53(4):215-29. https://www.ncbi.nlm.nih.gov/pubmed/25715736
[xlii]Potassium chloride, IV: Ghannoum M, Wiegand TJ, Liu KD, Calello DP, Godin M, Lavergne V, Gosselin S, Nolin TD, Hoffman RS; EXTRIP workgroup. Extracorporeal treatment for theophylline poisoning: systematic review and recommendations from the EXTRIP workgroup. Clin Toxicol (Phila). 2015 May;53(4):215-29. https://www.ncbi.nlm.nih.gov/pubmed/25715736
[xliii] Phenytoin (seizures secondary to poisonings): South African Medicines Formulary. 14th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.
[xliv] Haemodialysis: Activated charcoal (multi-dose): Ghannoum M, Wiegand TJ, Liu KD, Calello DP, Godin M, Lavergne V, Gosselin S, Nolin TD, Hoffman RS; EXTRIP workgroup. Extracorporeal treatment for theophylline poisoning: systematic review and recommendations from the EXTRIP workgroup. Clin Toxicol (Phila). 2015 May;53(4):215-29. https://www.ncbi.nlm.nih.gov/pubmed/25715736
[xlv] Flumazenil: Penninga EI, Graudal N, Ladekarl MB, Jürgens G. Adverse Events Associated with Flumazenil Treatment for the Management of Suspected Benzodiazepine Intoxication - A Systematic Review with Meta-

Analyses of Randomised Trials. Basic ClinPharmacolToxicol. 2016 Jan;118(1):37-44.http://www.ncbi.nlm.nih.gov/pubmed/26096314
[xlvi]Whole bowel irrigation (with polyethylene glycol): Thanacoody R, Caravati EM, Troutman B, Hojer J, Benson B, Hoppu K et al. Position paper update: whole bowel irrigation for gastrointestinal decontamination of overdose patients. Clin Toxicol (Phila) 2015;53:5-12. https://www.ncbi.nlm.nih.gov/pubmed/25511637
[xlvii] Phenytoin (seizures secondary to poisonings): South African Medicines Formulary. 12th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.
[xlviii] Haemodialysis (early referral): Decker BS, Goldfarb DS, Dargan PI, Friesen M, Gosselin S, Hoffman RS, Lavergne V, Nolin TD, Ghannoum M; EXTRIP Workgroup. Extracorporeal Treatment for Lithium Poisoning: Systematic Review and Recommendations from the EXTRIP Workgroup. Clin J Am Soc Nephrol. 2015 May 7;10(5):875-87. https://www.ncbi.nlm.nih.gov/pubmed/25583292
[xlix]Pyridoxine, oral: Lheureux P, Penaloza A, Gris M. Pyridoxine in clinical toxicology: a review. Eur J Emerg Med. 2005 Apr;12(2):78-85. http://www.ncbi.nlm.nih.gov/pubmed/15756083
Pyridoxine, oral: Dilrukshi M, Ratnayake C, Gnanathasan C. Oral pyridoxine can substitute for intravenous pyridoxine in managing patients with severe poisoning with isoniazid and rifampicin fixed dose combination tablets: a case report. BMC Res Notes 2017;10:370. https://www.ncbi.nlm.nih.gov/pubmed/28789699
[l] Benzodiazepines (seizures in INH toxicity): Hoffman RS, Howlands MA, Lewin Na, Nelson LS, Goldfrank LR. Goldfrank's Toxicologic Emergencies. 10th ed. China: McGraw-Hill Education; 2015.
[li] Phenytoin (avoid in isoniazid poisoning): Hoffman RS, Howlands MA, Lewin Na, Nelson LS, Goldfrank LR. Goldfrank's Toxicologic Emergencies. 10th ed. China: McGraw-Hill Education; 2015.
[lii]Calcium channel blocker poisonings and beta-blocker poisonings (similar management): Graudins A, Lee HM, Druda D. Calcium channel antagonist and beta-blocker overdose: antidotes and adjunct therapies. Br J Clin Pharmacol. 2016 Mar;81(3):453-61. https://www.ncbi.nlm.nih.gov/pubmed/26344579
[liii]Gut decontamination (Gastric lavage, activated charcoal administration or whole bowel irrigation): St-Onge M, Anseeuw K, Cantrell FL, Gilchrist IC, Hantson P, Bailey B, Lavergne V, Gosselin S, Kerns W 2nd, Laliberté M, Lavonas EJ, Juurlink DN, Muscedere J, Yang CC, Sinuff T, Rieder M, Mégarbane B. Experts Consensus Recommendations for the Management of Calcium Channel Blocker Poisoning in Adults. Crit Care Med. 2017 Mar;45(3):e306-e315. https://www.ncbi.nlm.nih.gov/pubmed/27749343
[liv]Atropine, IV: Proano L, Chiang WK, Wang RY. Calcium channel blocker overdose. Am J Emerg Med. 1995 Jul;13(4):444-50. http://www.ncbi.nlm.nih.gov/pubmed/7605536
[lv]Sodium chloride, 0.9%, IV: Proano L, Chiang WK, Wang RY. Calcium channel blocker overdose. Am J Emerg Med. 1995 Jul;13(4):444-50.http://www.ncbi.nlm.nih.gov/pubmed/7605536
[lvi]Calcium gluconate 10%, IV (calcium channel blocker toxicity): Proano L, Chiang WK, Wang RY. Calcium channel blocker overdose. Am J Emerg Med. 1995 Jul;13(4):444-50. http://www.ncbi.nlm.nih.gov/pubmed/7605536
Calcium gluconate 10%, IV (calcium channel blocker toxicity): Graudins A, Lee HM, Druda D. Calcium channel antagonist and beta-blocker overdose: antidotes and adjunct therapies. Br J Clin Pharmacol. 2016 Mar;81(3):453-61. https://www.ncbi.nlm.nih.gov/pubmed/26344579
[lvii] Phenytoin (seizures secondary to poisonings): South African Medicines Formulary. 14th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.
[lviii] Phenytoin (seizures secondary to poisonings): South African Medicines Formulary. 14th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.
[lix] Endoscopic evaluation (within 24-48 hours): Millar AJ, Cox SG. Caustic injury of the oesophagus. Pediatr Surg Int. 2015 Feb;31(2):111-21. https://www.ncbi.nlm.nih.gov/pubmed/25432099
[lx] Ethanol (indications for antidote): McMartin K, Jacobsen D, Hovda KE. Antidotes for poisoning by alcohols that form toxic metabolites. Br J Clin Pharmacol. 2016 Mar;81(3):505-15. https://www.ncbi.nlm.nih.gov/pubmed/26551875
[lxi] Ethanol (dosing of antidote): McMartin K, Jacobsen D, Hovda KE. Antidotes for poisoning by alcohols that form toxic metabolites. Br J Clin Pharmacol. 2016 Mar;81(3):505-15. https://www.ncbi.nlm.nih.gov/pubmed/26551875
[lxii] Co-factor therapy (ethylene glycol poisoning): Frommer JP, Ayus JC. Acute ethylene glycol intoxication. Am J Nephrol. 1982;2(1):1-5. https://pubmed.ncbi.nlm.nih.gov/7180899/
[lxiii] Metabolic acidosis cautions: McMartin K, Jacobsen D, Hovda KE. Antidotes for poisoning by alcohols that form toxic metabolites. Br J Clin Pharmacol. 2016 Mar;81(3):505-15. https://www.ncbi.nlm.nih.gov/pubmed/26551875
[lxiv] Ethanol antidote and haemodialysis (methanol poisoning): Jacobsen D, McMartin KE. Antidotes for methanol and ethylene glycol poisoning. J Toxicol Clin Toxicol. 1997;35(2):127-43. https://www.ncbi.nlm.nih.gov/pubmed/9120880
Ethanol antidote and haemodialysis (methanol poisoning): Roberts DM, Yates C, Megarbane B, Winchester JF, Maclaren R, Gosselin S, Nolin TD, Lavergne V, Hoffman RS, Ghannoum M; EXTRIP Work Group. Recommendations for the role of extracorporeal treatments in the management of acute methanol poisoning: a systematic review and consensus statement. Crit Care Med. 2015 Feb;43(2):461-72. https://www.ncbi.nlm.nih.gov/pubmed/25493973
[lxv] Phenytoin (seizures secondary to poisonings): South African Medicines Formulary. 14th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.
[lxvi] Intermediate syndrome in organophosphate poisoning: Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet. 2008 Feb 16;371(9612):597-607. https://www.ncbi.nlm.nih.gov/pubmed/17706760
[lxvii] Organophosphate poisoning – healthcare worker protection: Little M, Murray L; Poison Information Centres of New South Wales, Western Australia, Queensland, New Zealand, and the Australian Capital Territory. Consensus statement: risk of nosocomial organophosphate poisoning in emergency departments. Emerg Med Australas. 2004 Oct-Dec;16(5-6):456-8. https://www.ncbi.nlm.nih.gov/pubmed/15537409
Organophosphate poisoning – healthcare worker protection: Centers for Disease Control and Prevention (CDC). Nosocomial poisoning associated with emergency department treatment of organophosphate toxicity--Georgia, 2000. MMWR Morb Mortal Wkly Rep. 2001 Jan 5;49(51-52):1156-8. https://www.ncbi.nlm.nih.gov/pubmed/11198947

Organophosphate poisoning – healthcare worker protection: Stacey R, Morfey D, Payne S. Secondary contamination in organophosphate poisoning: analysis of an incident. QJM. 2004 Feb;97(2):75-80. https://www.ncbi.nlm.nih.gov/pubmed/14747621

[lxviii] Atropine, IV (bolus dose): Abedin MJ, Sayeed AA, Basher A, Maude RJ, Hoque G, Faiz MA. Open-label randomized clinical trial of atropine bolus injection versus incremental boluses plus infusion for organophosphate poisoning in Bangladesh. J Med Toxicol. 2012 Jun;8(2):108-17. http://www.ncbi.nlm.nih.gov/pubmed/22351300

[lxix] Atropine, IV (protocol): Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet. 2008 Feb 16;371(9612):597-607. https://www.ncbi.nlm.nih.gov/pubmed/17706760

Atropine, IV (protocol): Eddleston M, Dawson A, Karalliedde L, Dissanayake W, Hittarage A, Azher S, Buckley NA. Early management after self-poisoning with an organophosphorus or carbamate pesticide - a treatment protocol for junior doctors. Crit Care. 2004 Dec;8(6):R391-7. https://www.ncbi.nlm.nih.gov/pubmed/15566582

[lxx] Diazepam, IV (seizures in organophosphate poisoning): Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet. 2008 Feb 16;371(9612):597-607. https://www.ncbi.nlm.nih.gov/pubmed/17706760

[lxxi]Freshfrozen plasma: Ansell J, Hirsh J, Hylek E, Jacobson A, Crowther M, Palareti G; American College of Chest Physicians. Pharmacology and management of the vitamin K antagonists: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). Chest. 2008 Jun;133(6 Suppl):160S-198S.http://www.ncbi.nlm.nih.gov/pubmed/18574265

[lxxii] Watt BE et al. Anticoagulant rodenticides. Toxicol Rev 2005:24(4); 259-269

Toxinz and Toxbase – June 2022 printouts

Oxford Desk Reference Toxicology 2014 p311-312.

Goldfrank Toxicologic Emergencies 10th ed. P820 and 836-838.

[lxxiii] Phenytoin (seizures secondary to poisonings): South African Medicines Formulary. 14th Edition. Division of Clinical Pharmacology. University of Cape Town, 2022.

[lxxiv]Heavy metal poisoning (flu-like syndrome): Brenner BE, Keyes D. Metal Fume Fever. [Updated 2023 Aug 21]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK583199/.

Nelson LS, Odujebe OA. Simple Asphyxiants and Pulmonary Irritants. In: Nelson LS, Howland M, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS. eds. Goldfrank's Toxicologic Emergencies, 11e. McGraw-Hill Education; 2019. Accessed April 16,

2024. https://accesspharmacy.mhmedical.com/content.aspx?bookid=2569&sectionid=210264279

https://www.msdmanuals.com/professional/special-subjects/occupational-and-environmental-medicine/metal-fume-fever-and-polymer-fume-fever?query=metal%20fume%20fever%20and%20polymer%20fume%20fever

[lxxv]Heavy metal poisoning (signs and symptoms): AfriTox 2019. [Accessed September 2019]. www.afritox.co.za

SOUTH AFRICAN ADULT HOSPITAL LEVEL ESSENTIAL MEDICINES LIST
CHAPTER 19: POISONINGS
NEMLC RECOMMEDATIONS FOR MEDICINE AMENDMENTS (2020-4 REVIEW CYCLE)

Medicine amendment recommendations, with supporting evidence and rationale are listed below.
Kindly review the medicine amendments in the context of the respective standard treatment guideline (STG) and supporting documents.

A: AMENDMENTS

SECTION	MEDICINE/MANAGEMENT	ADDED/DELETED/AMENDED
19. Contact Information	SAVP contact details	Amended
19.1 Insect bites and stings	Paracetamol dose	Amended
19.2 Snakebites	General measures	Editorial amendment
	Tetanus toxoid vaccine	Guidance added
	Severe pain	Editorial amendment
19.2.1 Cytotoxic and neurotoxic snakebite	Polyvalent snake antivenom	Guidance amended
19.2.3 Snake venom in the eye	Local anaesthetic, ophthalmic drops	Added as a therapeutic class
	Tetracaine 1%, ophthalmic drops	Retained as an example of class in the STG
	Oxybuprocaine 0.4%, ophthalmic drops	Added to TI database
19.3 Scorpion envenomation	Paracetamol	Indication amended
	Tetanus toxoid vaccine	Guidance added
	Calcium gluconate 10%	Retained
19.4 Spider envenomation	General measures	Amended
	Tetanus toxoid vaccine	Guidance added
	Calcium gluconate 10%	Retained
19.5.1 Paracetamol poisoning *- Repeated supratherapeutic ingestion (RSTI)*	RSTI toxic doses	Amended
- N-acetylcysteine IV unavailable	N-acetylcysteine, oral	Retained
- iatrogenic poisoning of paracetamol, IV formulations	Guidance	Not added
19.5.2 Salicylate poisoning	Dextrose 50%, IV	Added
19.5.3 Opioid poisoning	Naloxone	Guidance amended
19.6.1 Tricyclic antidepressant poisoning	Description	Editorial amendment
19.7 Iron poisoning	Deferoxamine (Desferrioxamine) – IM administration	Added
	Deferoxamine (Desferrioxamine) – IV administration	Guidance clarified
19.11 Calcium channel blocker and Beta blocker poisoning	High-dose Insulin Euglycaemic Therapy (HIET):	Not added
19.14 illicit drugs		
19.14.1 Cocaine Poisoning	Benzodiazepines	Editorial amendment
	Caution – beta-blockers	Amended
19.17.2 Ethylene glycol poisoning	Ethanol	Dose and directions for use amended
	Routine monitoring of ethanol levels	Not added
19.18.Pesticides and rodenticides 19.18.1 Amitraz poisoning	Notifiable condition	Guidance added
19.18.2 Organophosphate poisoning	Notifiable condition	Guidance added
	Atropine, bolus IV	Dose amended
	Atropine, IV infusion	Directions for use amended

	Atropine high strength formulation (Section 21)	Added
19.18.3 Paraquat poisoning	Notifiable condition	Guidance added
	Description	Editorial amendment
19.19 Anticoagulant (Warfarin and rodenticide superwarfarin) poisoning	Notifiable condition	Guidance added
	Medicine treatment	Guidance amended
-*Superwarfarin poisoning*	Vitamin K1	Guidance added
19.21 Heavy metal poisoning	Description	Editorial amendment

B: CLINICAL EDITORIAL AMENDMENTS

The associated EML chapter has been subject to clinical editorial review following NEMLC ratification of the chapter. These amendments have been incorporated at the end of the NEMLC report.

19. CONTACT INFORMATION

South African Vaccine Producers (SAVP): *amended*

Contact details for procurement of snake, spider and scorpion antivenom has been updated in consultation with the SAVP and Poisons information Centre.

19.1 INSECT BITES AND STINGS

Paracetamol dose: *Amended*

The dosing guidance for paracetamol for pain management has been aligned to guidance included in the PHC and AH Pain chapters. The chapter has been updated where relevant as tabulated below:

AMENDED FROM:

For pain:

- Paracetamol, oral, 1 g 4–6 hourly when required.
  - Maximum dose: 15 mg/kg/dose.
  - Maximum daily dose: 4 g in 24 hours.

AMENDED TO:

For 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.

19.2 SNAKE BITES

General measures: *editorial amendment*

The following statement was added to the STG to avoid unnecessary fasciotomies as true compartment syndrome is rare:

True compartment syndrome is extremely rare in cytotoxic snakebites, as swelling is localised to the subcutaneous tissues. Fasciotomy is seldom indicated.

Tetanus toxoid vaccine: *guidance added*

Guidance for the administration of tetanus toxoid vaccine has been added to align with the PHC Chp 13: Immunisation and PHC Chp 21: Emergencies and Injuries chapters, i.e. tetanus vaccination indicated if not previously immunised within the last 5 years, as tabulated below:

» Immunisation, primary or booster: (Z23.5)

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

Severe pain: *editorial amendments*

The STG has been amended editorially as tabulated below:

AMENDED FROM:

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).
  - Opioids should be used cautiously in neurotoxic snakebite.

LoE:IVb

Note: The use of NSAIDs is not recommended due to the antiplatelet effect and the potential danger of renal failure in a hypotensive patient.

AMENDED TO:

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).

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.

19.2.1 CYTOTOXIC AND NEUROTOXIC SNAKEBITE

Polyvalent antivenom: *guidance amended*

Guidance on the use of polyvalent antivenom has been aligned to the updated National Snakebite Advisory Group (NSAG) and the South African Snakebite Symposium (SASS) guidelines.[1] Guidance on the reconstitution of antivenom has been amended i.e. 200mL of sodium chloride 0.9% is recommended instead of 100mL in view of the viscosity of the antivenom concentrate.

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 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 ~~>2.5cm per hour~~ >5 cm per hour
- discolouration of the skin / necrosis at the bite site
- a threatened airway due to swelling

LoE:IVb

- evidence of complication e.g. compartment syndrome
- Additional features of severe systemic cytotoxicity include:
  - haematological abnormalities: Hb <8 g/dL, thrombocytopaenia, (<100 x 109/L), raised INR or abnormal thromboelastography (if available)
  - arrhythmias (rare).
  - Shock

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.

[1] Hardcastle TC et al (progressive cytotoxic envenomation). Approach to the diagnosis and management of snakebite envenomation in South Africa in humans: The hospital phase – emergency unit general principles. SAMJ June 2023 Vol 113, No6.

Administration and polyvalent antivenom dose:
Pre-treat with adrenaline (epinephrine), SC, 0.25 mL of 1:1000 solution.
Note: (Contraindicated in patients with IHD, stroke, uncontrolled hypertension, and tachyarrhythmia).

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.
- 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).
- ~~Mozambique spitting cobras, give 100 mL initially, preferably within 6 hours of the bite.~~
- 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.
- Administer IV, over 30 minutes.
- Reassess once the infusion is completed. A repeat dose may be given if there is ongoing neurotoxicity or cytotoxicity

19.2.3 SNAKE VENOM IN THE EYE

Venom in the eyes

Local anaesthetic, ophthalmic drops: *added as a therapeutic class*
Tetracaine 1%, ophthalmic drops: *retained as an example of class in the STG*
Oxybuprocaine hydrochloride 0.4%, ophthalmic drops: *Added to TI database*

Added as an example of class to the therapeutic interchange database. Aligned with section 18.8: Surgical and diagnostic products of chapter 18: Eye conditions of the Adult Hospital Level STGs and EML, 2019 edition and PHC Chp 18 Eye conditions.

19.3 SCORPION ENVENOMATION

Analgesia

Paracetamol: *indication amended*

Guidance provided for mild pain management with paracetamol and management with severe local pain with lidocaine infiltration. The STG text was amended as follows

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 local pain:

Application of ice, if tolerated.

- Lidocaine 1–2%, 2 mL: infiltrate affected area as a local anaesthetic.

Tetanus toxoid vaccine: *guidance added*

Guidance for the administration of tetanus toxoid vaccine has been added to align with the PHC Chp 13: Immunisation and PHC Chp 21: Emergencies and Injuries chapters, i.e. tetanus vaccination indicated if not previously immunised within the last 5 years, as tabulated below:

» Immunisation, primary or booster: (Z23.5)

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

Calcium gluconate 10%: *retained*

Guidance on the use of calcium gluconate 10% for the management of severe muscle pain and cramps following scorpion envenomation has been retained and a supporting reference has been added.[2] Calcium chloride is retained

[2] Muller, G J et al. Scorpion sting in southern Africa: diagnosis and management. Continuing Medical Education, [S.l.], v. 30, n. 10, p. 356-361, sep. 2012. ISSN 2078-5143. Available at: <http://www.cmej.org.za/index.php/cmej/article/view/2545/2580>. Date accessed: 18 Apr. 2023.

in the therapeutic interchange database although the dosing guidance is not equivalent to calcium gluconate 10%. Furthermore, calcium chloride is associated with local irritation and may cause extravasation when used intravenously, so use is generally limited to situations where IV lines to larger vessels are accessible. A maximum recommended dose of 2 grams[3,4] has been added to the EML. Amendments to the EML are as tabulated below:

AMENDED FROM:

Severe muscle pain and cramps:

- Calcium gluconate 10%, bolus IV infusion, 10 mL over 10 minutes.
  - Repeat if needed, only once

Note: Effect may only last for 20–30 minutes and there is a limited amount that can be given.

AMENDED TO:

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.

19.4 SPIDER ENVENOMATION

General measures: *Amended*

The PHC/Adult Hospital Level Committee was of the opinion that asymptomatic individuals who are well after at most 12 hours, should be able to go home, whilst individuals with neurotoxicity, should be observed for at least 24 hours. The STG text was amended as follows:

Observe all cases of potential neurotoxic spider bite for at least ~~12~~ 24 hours.

Level of Evidence: Expert opinion

Tetanus toxoid vaccine: *guidance added*

Guidance for the administration of tetanus toxoid vaccine has been added to align with the PHC Chp 13: Immunisation and PHC Chp 21:Emergencies and Injuries chapters, i.e. tetanus vaccination indicated if not previously immunised within the last 5 years, as tabulated below:

» Immunisation, primary or booster: (Z23.5)

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

Calcium gluconate 10%: *retained*

Guidance on the use of calcium gluconate 10% for the management of severe muscle pain and cramps following spider envenomation has been retained and a supporting reference has been added.[5] Calcium chloride is retained in the therapeutic interchange database although the dosing guidance is not equivalent to calcium gluconate 10%. Furthermore, calcium chloride is associated with local irritation and may cause extravasation when used intravenously, so use is generally limited to situations where IV lines to larger vessels are accessible. A maximum recommended dose of 2 grams[6,7] has been added to the EML, as detailed in Section 19.3 Scorpion envenomation above.

19.5.1 PARACETAMOL POISONING

Repeated supratherapeutic ingestion (RSTI)

RSTI toxic doses: *amended*

[3] Package Insert: Calcium Gluconate Injection Fresenius solution for injection. Fresenius Kabi Manufacturing SA (Pty) Ltd. Last revised 7 February 2023.

[4] Shann F. Drug doses: 17th Edition. 2017.

[5] MULLER, G J et al. Spider bite in southern Africa: diagnosis and management. Continuing Medical Education, [S.l.], v. 30, n. 10, p. 382-391, sep. 2012. ISSN 2078-5143. Available at: <http://www.cmej.org.za/index.php/cmej/article/view/2547/2582>. Date accessed: 18 Apr. 2023.

[6] Package Insert: Calcium Gluconate Injection Fresenius solution for injection. Fresenius Kabi Manufacturing SA (Pty) Ltd. Last revised 7 February 2023.

[7] Shann F. Drug doses: 17th Edition. 2017.

The guidance defining RSTI toxic doses was updated, aligned with the updated guidelines for the management of paracetamol poisoning by Chieu et al (2020)[8]. STG text was amended from:

~~RSTI toxic doses are 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 have symptoms suggestive of liver injury.~~

To:

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.

AGREE 2 assessment of these updated guidelines scored a mean score of 5/7 (4/7 and 6/7 by respective reviewers). There is general overall agreement to recommend use of these guidelines in the South African setting, with modification. The rigour of development of these guidelines, however was questioned.
Level of Evidence: Low certainty, strong recommendation

If N-acetylcysteine IV is unavailable:
N-acetylcysteine, oral: *retained*
Oral dose of N-acetylcysteine was confirmed to be correct - NEMLC-approved in the previous review cycle of the Adult Hospital Level STGs and EML, 2019 – see NEMLC report below:

NEMLC report for the Adult Hospital Level Emergencies and injuries chapter (2017-9 review cycle):
N-acetylcysteine, oral: retained and dosing regimen added
Where parenteral formulation is unavailable, oral NAC recommended as a safe alternative where IV administration is not an option. Previous recommendation of oral NAC if IV formulation is unavailable has been expanded to include a dosing regimen, as follows:
If N-acetylcysteine, IV is unavailable:
- *N-acetylcysteine, oral, 140 mg/kg, followed by 70 mg/kg 4 hourly for seventeen doses.*

Note: Avoid giving activated charcoal if giving N-acetylcysteine orally as it will reduce the systemic absorption and thus negate the effect of oral N-acetylcysteine.
Level of Evidence: III Observational studies[9] [10] [11]
Recommendation: Guidance for oral NAC to be updated in the PHC STGs and EML.

Updates to the STG are as tabulated below:

AMENDED FROM:
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. In the presence of bronchospasm, stop the infusion.

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

If N-acetylcysteine, IV is unavailable:
- N-acetylcysteine, oral, 140 mg/kg, followed by 70 mg/kg 4 hourly for seventeen doses.

Note: Avoid giving activated charcoal if using oral N-acetylcysteine, as it will reduce systemic absorption of the antidote.

[8] Chiew AL, Reith D, Pomerleau A, Wong A, Isoardi KZ, Soderstrom J, Buckley NA. Updated guidelines for the management of paracetamol poisoning in Australia and New Zealand. Med J Aust. 2020 Mar;212(4):175-183. https://pubmed.ncbi.nlm.nih.gov/31786822/
[9] Yarema MC, Johnson DW, Berlin RJ, Sivilotti ML, Nettel-Aguirre A, Brant RF, Spyker DA, Bailey B, Chalut D, Lee JS, Plint AC, Purssell RA, Rutledge T, Seviour CA, Stiell IG, Thompson M, Tyberg J, Dart RC, Rumack BH. Comparison of the 20-hour intravenous and 72-hour oral acetylcysteine protocols for the treatment of acute acetaminophen poisoning. Ann Emerg Med. 2009 Oct;54(4):606-14. https://www.ncbi.nlm.nih.gov/pubmed/19556028
[10] Williamson K, Wahl MS, Mycyk MB. Direct comparison of 20-hour IV, 36-hour oral, and 72-hour oral acetylcysteine for treatment of acute acetaminophen poisoning. Am J Ther. 2013 Jan;20(1):37-40. https://www.ncbi.nlm.nih.gov/pubmed/23299230
[11] Rumack and Bateman. Acetaminophen and acetylcysteine dose and duration: past, present and future. Clin Toxicol (Phila) 2012;50(2):91-98. https://www.ncbi.nlm.nih.gov/pubmed/22320209

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

AMENDED TO:

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.

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.

Note:

As anaphylactoid reactions to N-acetylcysteine do occur, the loading dose should preferably be administered in a monitored area.

Avoid giving oral N-acetylcysteine together with activated charcoal as systemic absorption and effect of N-acetylcysteine is reduced.

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.

Poisoning of paracetamol, IV formulations

Guidance on iatrogenic poisoning of paracetamol, IV formulations: *not added*

Guidance for iatrogenic poisoning of paracetamol, IV formulations was not included in the STG. At the time of review the AH Chp 19 Poisoning chapter, paracetamol, IV was not approved as an EML medicine.

19.5.2 SALICYLATE POISONING

Dextrose 50%, IV: *added*

Dextrose-containing fluids were included in the STG for hydration. Two case reports suggests that dextrose infusions with boluses may improve salicylate-induced delirium in euglycaemic patients.[12]

Level of Evidence: Case reports

The EML has been updated as tabulated below:

AMENDED FROM :
19.5.2 SALICYLATE POISONING

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

AMENDED TO :
19.5.2 SALICYLATE POISONING

Diagnosis:

Mild toxicity:

» Nausea, vomiting, tinnitus, fever, lethargy, dizziness

Moderate toxicity:

» Sweating, restlessness, dehydration, hyperventilation, respiratory alkalosis with or without metabolic acidosis

Severe toxicity:

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

GENERAL MEASURES

» Assess severity with history, clinical examination.

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

[12] Kuzak N, Brubacher JR, Kennedy JR. Reversal of salicylate-induced euglycemic delirium with dextrose. Clin Toxicol (Phila). 2007 Jun-Aug;45(5):526-9. https://pubmed.ncbi.nlm.nih.gov/17503260/

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.

For hydration:

- Dextrose 5%, IV.

Treat acidosis and enhance renal excretion:

- Sodium bicarbonate 8.4%, IV and urinary alkalinisation (blood pH 7.45-7.5 and urine pH 7.5–8.5) in consultation with specialist and arrange for transfer.

REFERRAL

Where acidosis does not respond to sodium bicarbonate, refer for haemodialysis.

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

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.
    - 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.

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.
- 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.

REFERRAL

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

19.5.3 OPIOID POISONING

Naloxone: *Amended*

Guidance on the administration of naloxone has been amended to include the following:

- 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).

19.6.1 TRICYCLIC ANTIDEPRESSANT POISONING

Description: Editorial a*mendnent*

Editorial updates to the description are as tabulated below:

AMENDED FROM:

DESCRIPTION

Patients can deteriorate rapidly.

AMENDED TO[13]:

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.

19.7 IRON POISONING

Deferoxamine (desferrioxamine) - IM administration: *Added*

Deferoxamine (desferrioxamine) is registered for both IM and IV administration, and can be used in clinical practice particularly for low risk patients.[14] The literature supports both IM and IV administration, except for patients in cardiogenic shock when IV administration is preferred.[15,16,17,18] The inclusion of the IM route of administration will empower clinicians, particularly when IV access is not readily available. IM administration will also avert the insertion of unnecessary IV lines.

Deferoxamine (desferrioxamine)-IV administration: *Guidance clarified*

Dosing guidance on the administration of IV Deferoxamine (desferrioxamine) has been clarified as tabulated below:

AMENDED FROM:

- Desferoxamine (deferoxamine), IV infusion, 15 mg/kg/hour to a total of 80 mg/kg, i.e. given over about 6 hours. Beware of hypotension.
  - **Note:** Prolonged use >24 hours of high doses is associated with acute lung injury and should be avoided. However, in severe poisonings, additional doses may be required.
  - Desferoxamine can be used in pregnant women.

AMENDED TO:

Deferoxamine (desferrioxamine), IV infusion, 80 mg/kg.

- 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.

19.11 CALCIUM CHANNEL BLOCKER AND BETA BLOCKER POISONING

High-dose Insulin Euglycaemic Therapy (HIET): *Not added*

[13] David M. Taylor, Thomas R. E. Barnes, Allan H. Young. The Maudsley prescribing guidelines in psychiatry.13th edition. Hoboken, NJ : Wiley, 2019. (page 770)
[14] Package Insert – Desferal. Novartis. 10 August 2006.
[15] Drug monograph – deferoxamine. Clinical Key. Last revised 3 October 2022. Date accessed 2 March 2023
[16] Drug monograph – deferoxamine. Micromedex. Last modified 22 November 2022. Date accessed 2 March 2023
[17] Drug monograph – deferoxamine. Statpearls. Last modified 29 May 2022. Date accessed 2 March 2023
[18] Drug monograph – deferoxamine. UptoDate. Date accessed 2 March 2023

The Committee noted that the evidence on the use of HIET as a first line therapy for the management of calcium channel blocker and/or beta blocker poisoning is evolving although much of this is limited to observational studies. Following extensive deliberation by the ERC and NEMLC, it was agreed that HIET not be included in the AH Chp 19: Poisonings chapter, as patients receiving HIET require management in a High Care/ICU setting. The use of HIET for managing calcium channel blocker and/or beta blocker poisoning to be considered for prioritisation during the next review cycle as part of the AH Chp 23 Critical Care review.

Updates to the STG are as tabulated below:

AMENDED FROM	AMENDED TO

19.11 CALCIUM CHANNEL BLOCKER AND BETA BLOCKER POISONING
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 euglycaemia therapy (HIET) and ionotrope and vasopressor infusions.
 	
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.
 	
Treat hypotension: I95.9 + (T46.1/X44.99/X64.99/Y14.99)
• Sodium chloride 0.9%, IV.
 	
If hypotension 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.
 	
Treat 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.
 	
Simultaneously use vasopressors and ionotropes as needed, e.g. adrenaline (epinephrine) infusion for persistent hypotension (section 20.1: Cardiac arrest) or dobutamine for bradycardia (section 20.1.5: Cardiogenic shock).
 	
REFERRAL
Refer for management with high dose insulin for resistant hypotension and bradycardia, in a high care or ICU setting.
 	
If glucose <10 mmol/L:
• Dextrose 50%, IV, 50 mL
Followed by:
• Insulin, short acting, IV, 1 unit/kg.
  ○ Followed by 0.5 unit/kg/hour.
  ○ Titrate dose up until hypotension is corrected, to maximum 10 units/kg/hour.
Monitor and correct potassium and glucose.
	19.11 CALCIUM CHANNEL BLOCKER AND BETA BLOCKER POISONING
	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.
	
	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.
	
	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.
	
	Hypotension: I95.9 + (T46.1/X44.99/X64.99/Y14.99)
	• Start with sodium chloride, 0.9%, 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.
	• 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.

19.14 ILLICIT DRUGS

19.14.1 COCAINE POISONING

Medicine treatment – benzodiazepines: *Editorial amendment*

The following statement has been added to the STG to highlight the therapeutic value of benzodiazepines which included sympathetic stimulation: "*Benzodiazepines play a key role in the management of sympathetic and psychomotor features of cocaine poisoning."*

Caution – beta-blockers: *Amended*

The caution with the use of beta-blockers has been amended as tabulated below:

AMENDED FROM:

ß–blockers should not be used.

AMENDED TO:

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

19.17.2 ETHYLENE GYCOL POISONING

Ethanol: *dose and directions for use amended*

Background: Errors were detected in the Adult Hospital Level STG s and EML, 2019 regarding management in this clinical setting. An erratum was subsequently circulated by NDoH:

AdultHospitalLevelS
TGErrata_EthyleneGl

Subsequent to the erratum that was issued, comments were received that the guidance on the dilution and administration of ethanol was not clear. Following a pilot of several alternatives, the guidance was amended as below for improved clarity. The NEMLC would like to encourages ongoing comment/suggestions from clinicians to improve clarity and eliminate any ambiguity with interpreting the STG.

Amended from (circular):

- Ethanol ~~95%~~ 96% BP, oral,
  - Loading dose 1 mL/kg.
  - Dilute the calculated ethanol volume to 20% (1 part ethanol to 4 parts juice or water) and give over 15-30 minutes.
  - Maintenance dose:
    - non-drinker: 0.125 mL/kg/hour
    - chronic drinker: 0.2 mL/kg/hour
  - Dilute the calculated ethanol volume to 20% (1 part ethanol to 4 parts juice or water)

OR

- Ethanol 40% v/v (gin, whiskey, vodka), oral
  - Loading dose: 2 mL/kg
  - Dilute the calculated ethanol volume to 20% (1 part ethanol to 1 part juice or water) and give over 15-30 minutes
  - Maintenance dose:
    - non-drinker: ~~25~~ 0.25 mL/kg/hour
    - chronic drinker: 0.5 mL/kg/hour
  - Dilute the calculated ethanol volume to 20% (1 part ethanol to 1 part juice or water)

Amended to:

Ethanol

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).

Monitoring of ethanol levels

Routine monitoring of ethanol levels: *not added (for tertiary level of care)*

It was reported that ethanol levels are not always readily available and thus clinical response generally guides management in practice. NHLS reported that currently only one tertiary facility requests this laboratory test. However, pre-analytical considerations are important as the blood sample needs to be spun down and sent frozen, as ethanol is oxidized.

Recommendation: The PHC/Adult Hospital Level Committee recommended that this test not be motivated for routine use at all tertiary level facilities as not pragmatic. However, the STG be expanded to include guidance be provided on clinical management of patients (in the absence of monitoring of ethanol levels).

Level of Evidence: Expert opinion

19.18 PESTICIDES AND RODENTICIDES

19.18.1 AMITRAZ POISONING

Notifiable condition: *guidance added*
Editorial guidance added to indicate that poisoning from all pesticides is a notifiable condition.

19.18.2 ORGANOPHOSPHATE POISONING

Notifiable condition: *guidance added*
Editorial guidance added to indicate that poisoning from all pesticides is a notifiable condition.

Atropinisation
Atropine, bolus IV: *amended*
The dose of bolus IV atropine was simplified from a range, "2-5 mg" to "2mg".

Atropine, IV: *directions for use amended*
Expanded guidance on atropine infusion after atropinization was included.
An open-label RCT [19](n=156) compared conventional bolus dose atropine (group A, n=81) to rapidly incremental doses of atropine followed by infusion (group B, n=75). The mortality was 22.5% (18/80) vs 8% (6/75) ($p<0.05$) and the mean duration of atropinization was 151.74 min vs 23.90 min ($p<0.001$). More patients in group A (compared to group B) experienced atropine toxicity (28.4% vs 12.0%, $p<0.05$); intermediate syndrome (13.6% versus 4%, $p<0.05$), and required more respiratory support (24.7% versus 8%, $p<0.05$). Thus, improved mortality and morbidity survival has been demonstrated in patients with organophosphate poisoning who received rapid incremental dose atropinization followed by atropine infusions.
Level of Evidence: Low certainty, strong recommendation

Atropine high dose formulation (Section 21): *Added*
In response to external requests, NEMLC supported the recommendation to include a high strength formulation of atropine 100mg in 10mL ampoule (obtainable via Section 21), on the EML. A summary of the deliberations by NEMLC is detailed below:

EVIDENCE SUMMARY

A. Local prevalence

- Based on a 20-year review of death certificate recordings (1997-2016) in South Africa, poisonings from unspecified drugs (non-medicinal), organic solvents, gases/vapours and unspecified chemicals, was the third most common method of intentional self-harm, with hanging and firearm use being the first and second leading causes respectively.Deaths due to pesticide poisoning and drugs in the total population were 2.4% (208) and 8.7% (766), respectively. [20]
- The Poisons Information Helpline (PIH) 2019 annual report had 12 236 patient-related telephone enquiries. The second largest group of toxins were pesticides (n = 1930, 13.5%)[21].
- Other means of surveillance is via the notification of cases. Agricultural or stock remedy poisoning is a Category 2 notifiable medical condition (NMC)[22], requiring reporting either via paper- or electronic based mechanisms within 7 days of diagnosis. Since the Q4 2024 cases, it has been made a Category 1 condition, to be reported within 24 hours.

[19] Abedin MJ, Sayeed AA, Basher A, Maude RJ, Hoque G, Faiz MA. Open-label randomized clinical trial of atropine bolus injection versus incremental boluses plus infusion for organophosphate poisoning in Bangladesh. J Med Toxicol. 2012 Jun;8(2):108-17. https://pubmed.ncbi.nlm.nih.gov/22351300/
[20] Kootbodien, T.; Naicker, N.; Wilson, K.S.; Ramesar, R.; London, L. Trends in Suicide Mortality in South Africa, 1997 to 2016. *Int. J. Environ. Res. Public Health* **2020**, *17*, 1850. https://doi.org/10.3390/ijerph17061850
[21] https://health.uct.ac.za/sites/default/files/media/documents/health_uct_ac_za/547/poisons_information_helpline_annual_review_2019_1.pdf
[22] https://www.nicd.ac.za/nmc-overview/overview/

Recent data on *notified cases* as shared by the Western Cape PTC is included in Addendum B. However, under-reporting is always a concern[23].

B. Clinical severity

- Organophosphate poisoning has persistently shown a greater severity than other toxins.[24,25]

C. Atropine as an antidote in organophosphate poisoning

- While atropine is widely accepted as the mainstay of treatment in organophosphate poisoning, consensus on an appropriate dosing regimen was lacking[26]. Historically, two approaches were used. The first is bolus dose treatment with atropine initially every 5/10/15 minutes followed by lower doses at longer intervals until recovery. The second regimen is rapid atropinisation with incremental boluses followed by an atropine infusion calculated from the total bolus doses required and adjusted according to clinical response and tapered until recovery[27].
- Both inadequate and excessive doses of atropine have a bearing on patient outcomes, i.e. inadequate dosing and/or premature withdrawal of atropine can result in a rebound of symptoms while excessive doses of atropine can result in toxicity with agitation, confusion, headache, hyperthermia and severe tachycardia.
- Therefore, a call was made to health care providers to provide a recommended guideline and to perform trials investigating which protocol provided better outcomes.[11]
- Eddleston et al[28] complied guidelines for junior doctors in poorly resourced areas using the available evidence. Since then, there has been a notable trend in international guidelines towards the increased speed to atropinisation[29]. Observational evidence suggested that rapid atropinisation with dose doubling every 3 to 5 min is protective against mental status changes and decreases the case fatality rate in severe cases[30].
- Abedin et al performed an open-label RCT (n=156) that compared conventional bolus dose atropine (group A, n=81) to rapidly incremental bolus dose atropine followed by infusion (group B, n=75). Both groups of patients received the same dose of pralidoxime. Mortality was significantly higher for Group A (22.5% (18/80) versus 8% (6/75), p<0.05) with a shorter time to atropinisation for Group B (23.90 versus 151.74 min, p<0.001). More patients in group A (compared to group B) experienced atropine toxicity (28.4% vs 12.0%, p<0.05); intermediate syndrome (13.6% versus 4%, p<0.05), and required more respiratory support (24.7% versus 8%, p<0.05). Thus, improved mortality and morbidity has been demonstrated in patients with organophosphate poisoning who received rapid incremental bolus dose atropine followed by infusion. **Level of Evidence: Low certainty, strong recommendation.** Findings from this study informed the most recent STG recommendations.
- Another RCT conducted in China in 2014[31] enrolled 60 patients with severe organophosphate poisoning who were randomised into one of two treatment arms: experimental group: patients received a continuous micropump of atropine and pralidoxime chloride and control group: patients were given intermittent injections of atropine and pralidoxime chloride. The time to atropinisation and time to acetylcholinesterase recovery was shorter in the experimental group than the control group, leading to a lower total dose of

[23] Razwiedani LL, Rautenbach P. Epidemiology of organophosphate poisoning in the Tshwane District of South Africa. Environ Health Insights 2017;11 1178630217694149PMID: 28469445; PMCID:PMC5345965. doi:10.1177/1178630217694149.

[24] Khan M, Solomon F, Izu A, Bengura P, Okudo G, Maroane B, Lala N and Dangor Z (2023) The burden of poisoning in children hospitalised at a tertiary-level hospital in South Africa. Front. Public Health 11:1279036. doi: 10.3389/fpubh.2023.1279036

[25] Balme K, Stephen C. Streamlining referral decisions for childhood poisoning: a cross-sectional study from a tertiary children's hospital in Cape Town, South Africa. Afr J Emerg Med. 2022 Dec;12(4):432-437. doi: 10.1016/j.afjem.2022.10.008. Epub 2022 Oct 29. PMID: 36348737; PMCID: PMC9634013.

[26] Eddleston M, Buckley NA, Checketts H, Senarathna L, Mohamed F, Sheriff MH, Dawson A. Speed of initial atropinisation in significant organophosphorus pesticide poisoning--a systematic comparison of recommended regimens. J Toxicol Clin Toxicol. 2004;42(6):865-75. doi: 10.1081/clt-200035223. PMID: 15533026; PMCID: PMC1475556.

[27] Abedin MJ, Sayeed AA, Basher A, Maude RJ, Hoque G, Faiz MA. Open-label randomized clinical trial of atropine bolus injection versus incremental boluses plus infusion for organophosphate poisoning in Bangladesh. J Med Toxicol. 2012 Jun;8(2):108-17. https://pubmed.ncbi.nlm.nih.gov/22351300/.

[28] Eddleston M, Dawson A, Karalliedde L, Dissanayake W, Hittarage A, Azher S, Buckley NA. Early management after self-poisoning with an organophosphorus or carbamate pesticide - a treatment protocol for junior doctors. Crit Care. 2004 Dec;8(6):R391-7. doi: 10.1186/cc2953. Epub 2004 Sep 22. PMID: 15566582; PMCID: PMC1065055.

[29] Connors NJ, Harnett ZH, Hoffman RS. Comparison of current recommended regimens of atropinization in organophosphate poisoning. J Med Toxicol. 2014 Jun;10(2):143-7. doi: 10.1007/s13181-013-0324-9. PMID: 23900961; PMCID: PMC4057538.

[30] Perera PM, Shahmy S, Gawarammana I, Dawson AH (2008) Comparison of two commonly practiced atropinization regimens in acute organophosphorus and carbamate poisoning, doubling doses vs. ad hoc: a prospective observational study. Hum Exp Toxicol 27:513– 518

[31] Liu HX, Liu CF, Yang WH. Clinical study of continuous micropump infusion of atropine and pralidoxime chloride for treatment of severe acute organophosphorus insecticide poisoning. J Chin Med Assoc. 2015 Dec;78(12):709-13. doi: 10.1016/j.jcma.2015.08.006. Epub 2015 Oct 3. PMID: 26441220.

atropine being administered to the experimental group. The APACHE II score was significantly better in the experimental group relative to the control group with a lower case fatality rate in the experimental arm (10% vs 26.7%). The shorter duration to atropinisation as well as lower mortality rates with the infusion regimen is not completely consistent with findings from the Abedin study. However, limitations associated with their study as opposed to the Abedin RCT, are differing dosing regimens of both atropine and pralidoxime, and an inadequate sample size. They made no comments on the differences in atropine toxicity.

- Looking at higher dose atropine, according to a local study published in 2023[32] where researchers conducted a retrospective chart review of all patients presenting to the emergency centre at Thelle Mogoerane Regional Hospital in Vosloorus with symptoms suggestive of cholinergic toxidrome or a history of suspected organophosphorus exposure. Patients received atropine based on an adapted protocol; with administration of an initial 15mg atropine intravenous bolus, followed by incremental 15mg intravenous bolus administration every 3-5min until resolution of cholinergic respiratory symptoms. After rapid atropinisation, a maintenance atropine infusion, calculated at 20% of the total atropine dose was started. *The protocol recommended 10mg/ml atropine, in 10ml vials, produced by Dr Franz Kohler Chemie GmbH and imported from Germany (obtained via Section 21 application to the South Africa Health Products Regulation Authority).*
    - Of the 205 patient encounters identified over a 20 month period, 134 files were accessible for inclusion in the study.
    - The median atropinisation dose was 100mg (IQR 45-167) which was comparable to the Bangladesh RCT which reported the mean atropine bolus dose in each study arm as 109mg and 136mg[33].
    - The atropinisation dose was significantly higher for intubated patients (Median=140.0mg; IQR=90-219,5mg) compared to patients who were not intubated (Median=60mg; IQR=20.5-120mg) ($p < 0.05$).
    - There was a moderate positive correlation between atropinisation dose and length of stay (Correlation coefficient = 0.37, p=0.00).
    - The median length of stay was 8 nights (IQR 5-13 days) and the longest hospital stay was 37 days. The mortality rate was 13.4% with 81.3% patients having survived with no outcome recorded in 5.2% of cases.
    - Treatment complications were documented for 70 patients, of which 55 patients (78.6%) were documented adverse effects from atropine toxicity. The most common symptoms of atropine toxicity were confusion (70%), tachycardia (13.4%) hallucinations (5.7%), hyperthermia (4.3%) and seizures (2%).

ATROPINE FORMULATIONS AND IMPLICATIONS FOR LOCAL PRACTICE

- Formulations of atropine currently on tender include:

Product	Pack size	Ave price (MHPL Jan 2025)
Atropine 1mg/mL	1mL injection	R4.35
Atropine 500mcg/mL	1mL injection	R3.94

- There is a lack of predictability with stock holding due to patient-specific dose titration and the potentially large number of ampoules required for adequate dosing of atropine with formulations currently on tender. This presents logistic and access challenges not conducive to emergency management. Furthermore, the large number of ampoules are likely to present calculation and dosing errors as well as an onerous burden on staff time which may lead to treatment delays.
- Access to suitable atropine formulations is not limited to our local setting - alternative suggestions for reconstituting powdered atropine for injectable use, fortifying existing injectable atropine with pharmaceutical grade powder for IM administration[34] or alternatively sublingual administration of

[32] Khonje V, Hart J, Venter J, Deonarain S, Grossberg S. Acute organophosphorus toxicity in a regional hospital in Johannesburg, South Africa: A retrospective chart review. Afr J Emerg Med. 2023 Jun;13(2):104-108. doi: 10.1016/j.afjem.2023.04.002. Epub 2023 Apr 26. PMID: 37152660; PMCID: PMC10160343.

[33] Abedin MJ, Sayeed AA, Basher A, et al. Open-label randomized clinical trial of atropine bolus injection versus incremental boluses plus infusion for organophosphate poisoning in Bangladesh. J Med Toxicol 2012;8:108–17.

[34] Geller RJ, Lopez GP, Cutler S, Lin D, Bachman GF, Gorman SE. Atropine availability as an antidote for nerve agent casualties: validated rapid reformulation of high-concentration atropine from bulk powder. Ann Emerg Med. 2003 Apr;41(4):453-6. doi: 10.1067/mem.2003.103. PMID: 12658242.

atropine[35] are described in the literature. These alternatives are deemed less suitable than a commercial high strength injectable formulation (albeit not SAHPRA registered), for local needs.

Some PCTs have opted to obtain access to a S21 high dose formulation of atropine. Product details as supplied by Equity Pharmaceuticals as follows:

Atropinsulfat (Atropine Sulphate) 100mg/10ml (5 x 10ml amps) = R 646.00 excl. vat (NAPPI 3007898001)
VAT inclusive = R742.90 per pack of 5 ampoules
Cost per 100mg ampoule = R148.58

Comparative costs

Comparative costs for an adult requiring atropinisation as detailed below. Dose estimates as informed by local clinical expert:[36]

Bolus to atropinise adequately:
Generally a 30 - 60mg bolus in total depending on quantity/concentration ingested - the majority are on the low end.

Infusion to maintain:
3mg - 6mg / hour for 10 hours if accepted by TBH (Tygerberg Hospital)

If not accepted by TBH:
Then we would wean the atropine over another 24 hours so perhaps half the rate (1.5-3mg/hr x 24hr)

- This may be less depending on agent ingested.

If we go at the lower, mid, and high point of these then:
Low: 30mg bolus, 30mg for the 10hours, 36mg if not transferred = **96mg**
Mid: 45mg bolus, 45mg for the 10hours, 54mg if not transferred = **144mg**
High: 60mg bolus, 60mg for the 10hours, 72mg if not transferred = **192mg**

Formulation	Ave cost per amp (Rands)	Equivalent number of ampoules for each dose range			Range of treatment cost (Rands)	
		Low	Mid	High	Low	High
1mg ampoule	4.35	96	144	192	417.6	835.2
500mcg ampoule	3.94	192	288	384	756.48	1512.96
100mg ampoule*	148.58	1	2	2	148.58	297.16

*Note: Medicines imported through Section 21 approvals are not regulated by local medicine price controls and are subject to exchange rate volatilities.

The STG text was amended as tabulated below:

AMENDED FROM:

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.

[35] Rajpal S, Ali R, Bhatnagar A, et al. Clinical and bioavailability studies of sublingually administered atropine sulphate. Am J Emerg Med. 2010; 28:143-50.
[36] Motivation from Dr Luke Bush to the western Cape PTC.

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.

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.
- » 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:

- 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.

For severe agitation:

- Diazepam, IV, 5–10 mg, immediately.
  - Repeat after 30–60 minutes if needed.

REFERRAL

Refer if ventilatory support is unavailable.

AMENDED TO :

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.

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.
- 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:

STEP 1:

- Atropine bolus, IV
  - Administer 2mg atropine as an IV bolus.
  - 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

ATROPINE : SECTION 21

A high strength formulation (100mg in 10mL) of atropine may be available at select facilities. Confirm the strength available before preparing and administering to the patient.

STEPS TO FOLLOW	**ATROPINE FORMULATIONS**		
Select formulation of atropine available	**100mg/10mL ampoule SECTION 21**	**1mg/mL ampoule**	**500mcg/mL ampoule**
Prepare atropine syringe for IV bolus	Dilute 1mL atropine with 9mL diluent for a 1mg/mL solution.	Use undiluted	
Atropine doses	**Volume of atropine**		
	Diluted 1mg/mL solution SECTION 21	**1mg/mL ampoule**	**500mcg/mL ampoule**
2mg	2mL	2mL	4mL
4mg	4mL	4mL	8mL
8mg	8mL	8mL	16mL
16mg	16mL	16mL	32mL
32mg	32mL	32mL	64mL
etc			
Total bolus dose = e.g. 62mg			

STEP 2:

- Atropine IV infusion
  - Calculate the total dose of atropine given as boluses. Give 10% of this dose per hour. titrating up to 20% per hour based on clinical response.

Worked example: Total bolus dose = 62mg. Administer 10-20% (6-12mg) per hour by IV infusion.			
STEPS TO FOLLOW	**ATROPINE FORMULATIONS**		
Select formulation of atropine available	**Atropine 100mg/10mL ampoule (SECTION 21)**	**Atropine 1mg/mL ampoule**	**Atropine 500mcg/mL ampoule**
Prepare solution for IV infusion	200mg atropine = 20mL (2 amps) Add to 180mL diluent to prepare a 1mg/mL atropine solution	40mg atropine = 40mL (40 amps) Add to 160mL diluent to prepare a 0.2mg/mL solution	40mg atropine = 80mL (80 amps) Add to 120mL diluent to prepare a 0.2mg/mL solution
Calculate 10% of total bolus dose to be given per hour	6mg	6mg	6mg
Start infusion at rate equivalent to 10% of bolus dose	6mL/hour	30mL/hour	30mL/hour

- Titrate according to clinical response, by frequent reassessment and adjustments:
  - 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.

STEP 3 (May be required in some patients)

- Atropine bolus, IV
  - Some organophosphates are lipophilic in nature and cholinergic symptoms may recur even once the atropine infusion is in place. In such cases, repeat bolus doses, starting at 2mg, can be given in addition to the atropine infusion. Bolus doses may be increased incrementally as in Step 1 above.
  - Once the patient is restabilised, consider increasing the infusion rate, to 10% of the new total bolus doses i.e.

    Infusion rate = 0.1 X (STEP 1 total bolus dose + STEP 3 total bolus dose).
  - Titrate the infusion rate up to 20% based on clinical response.

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.

For severe agitation:

- Diazepam, IV, 5–10 mg, immediately.
  - Repeat after 30–60 minutes if needed.

REFERRAL

Refer if ventilatory support is unavailable.

19.18.3 PARAQUAT POISONING

Notifiable condition: *guidance added*

Editorial guidance added to indicate that poisoning from all pesticides is a notifiable condition.

Description: *Editorial amendment*

The following statement has been amended to indicate that paraquat is the most toxic herbicide known[37]:

Paraquat is the most toxic herbicide known, and toxicity causes multi-organ failure which is often fatal.

[37] SAMF, 14th edition: Paraquat, pg 646

19.19 ANTICOAGULANT (WARFARIN AND RODENTICIDE SUPERWARFARIN) POISONING

Notifiable condition: *guidance added*

Editorial guidance added to indicate that poisoning from all pesticides is a notifiable condition.

Medicine treatment: *guidance amended*

Guidance on the use of vitamin k for the management of warfarin poisoning was amended as tabulated below. The approved package insert for the injectable formulations of vitamin K available on tender, advise against the dilution of the IV formulation[38]. STG guidance has therefore been amended that vitamin k IV should be administered undiluted as a slow IV bolus injection.

Original STG text	2023 STG Amendments
T45.5 + (X44.99/X64.99/Y14.99) * Notifiable condition – rodenticide superwarfarin poisoning **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. Measure INR at baseline and 48 hours post ingestion, as the anticoagulant effect may be delayed by 1–2 days. 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. **MEDICINE TREATMENT** **For patients on warfarin therapy** INR 5 to 9 without bleeding: » Stop warfarin » Evaluate bleeding risk – High risk patients: (history of bleeding, stroke, renal insufficiency, anaemia, hypertension). • Vitamin K_1 oral, 1–2.5 mg, for 1–2 days and monitor INR. – Low risk patients: Monitor INR. INR >9 without bleeding: » Stop warfarin. • Vitamin K_1 oral, 2.5–5 mg, for 1–2 days and monitor INR (response usually in 24 to 48 hrs). » Resume warfarin therapy, at a lower dose. Vitamin K_1 is available as a parenteral preparation only, but is safest given orally in anticoagulant poisoning. *LoE:IIIa* Elevated INR with significant bleeding: R58 + (T45.5/X44.99/X64.99/Y14.99)	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 www.nicd.ac.za for further information. **DESCRIPTION** **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 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* • 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

[38] Package Insert. Konakion MM. Pharmaco Distribution (Pty) Ltd. Last revision: 30 September 2011, 20 July 2018

» Stop warfarin. • Lyophilised plasma, IV, 15 mL/kg. *LoE:IIIa* **OR** FFP 15 mL/kg. *LoE:IIIa* Followed by: • Vitamin K_1, IV,5 mg diluted in 100 mL sodium chloride 0.9% over 20 minutes and monitor for prophylaxis. *LoE:IIIa* **Note:** » In patients with prosthetic heart valves, high dose vitamin K is associated with increased resistance to warfarin and increased risk of thromboembolism. Treat as above, but monitor INR frequently to prevent overcorrection in consultation with a specialist. » In all patients on therapeutic warfarin, a major overdose or bleeding episode should prompt careful review of the need for anticoagulation » **If warfarin is indicated it should be re-instituted, once the INR is in the therapeutic range.** **Rodenticide ingestion - Superwarfarins** Do not given prophylactic vitamin K. INR >4 or the patient is actively bleeding: *LoE:IVb* • Vitamin K_1 oral, 10–25 mg, daily may be required. ○ Vitamin K1 is available as a parenteral preparation only, but is safest given orally in anticoagulant poisoning. ○ Treatment with vitamin K1 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. Note: » Treatment with vitamin K1 may need to be prolonged for several months as superwarfarins are very long acting.	therapy. ○ Decrease Vitamin K dose by half, i.e. Vitamin K1, IV, 5 mg. Administer as a slow IV injection. **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 K1 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 Where 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 hours later if the INR remains ≥ 8. **Note:** • 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. • 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 K1 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.

Superwarfarin poisoning

Vitamin K_1: *guidance added*

As treatment with vitamin K_1 may be prolonged for superwarfarin poisoning, guidance for consultation with the Poison Information Centre or haematologist was added to the STG text.

19.21 HEAVY METAL POISONING

Description: *Editorial amendments*

The description for heavy metal poisoning has been amended to include a flu-like syndrome,[39,40,41] that may present following occupational exposure. Updates to the STG are as tabulated below:

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.

B: CLINICAL EDITORIAL AMENDMENTS

The associated EML chapter has been subject to clinical editorial review following NEMLC ratification of the chapter. These amendments are detailed below.

19.2 SNAKE BITES

MEDICINE TREATMENT
AMENDED FROM: Antibiotics are seldom needed, except for secondary infection:
AMENDED TO: Antibiotics are seldom indicated unless there is evidence of secondary infection.

19.2.1 CYTOTOXIC AND NEUROTOXIC SNAKEBITE

NOTE:
AMENDED FROM: » 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.
AMENDED TO: 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.

[39] Brenner BE, Keyes D. Metal Fume Fever. [Updated 2023 Aug 21]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK583199/.

[40] Nelson LS, Odujebe OA. Simple Asphyxiants and Pulmonary Irritants. In: Nelson LS, Howland M, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS. eds. Goldfrank's Toxicologic Emergencies, 11e. McGraw-Hill Education; 2019. Accessed April 16, 2024. https://accesspharmacy.mhmedical.com/content.aspx?bookid=2569&sectionid=210264279

[41] https://www.msdmanuals.com/professional/special-subjects/occupational-and-environmental-medicine/metal-fume-fever-and-polymer-fume-fever?query=metal%20fume%20fever%20and%20polymer%20fume%20fever

19.2.2 BOOMSLANG SNAKEBITE

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

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.

AMENDED TO:
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.

Boomslang monovalent antivenom
AMENDED FROM:
- o Re-evaluate regularly, and if after 6 hours there is ongoing evidence of coagulopathy, a repeat dose of 10 mL may be considered.

AMENDED TO:
- o Re-evaluate regularly: Consider a repeat dose of 10 ml of antivenom if there is ongoing evidence of coagulopathy after 6 hours.

POISONING

DESCRIPTION
AMENDED FROM:
Suspect intentional ingestion in adults.

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

GENERAL MEASURES
AMENDED FROM:
It is very important to ascertain if a 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. A high index of suspicion is important.
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.

AMENDED TO:
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.

Other treatment modalities

AMENDED FROM:

Urinary alkalinisation (e.g. severe salicylate or tricyclic antidepressant poisoning)

AMENDED TO:

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.

Once medically stable:

AMENDED FROM:

Assess and manage a substance use disorder

» quantify the amount of substance used and related harms, e.g. with ASSIST (http://www.who.int/substance_abuse/activities/assist/en/) or DUDIT (https://paihdelinkki.fi/sites/default/files/duditmanual.pdf) rating scales and discuss with patient.
» provide brief intervention with motivational interview.
» refer for rehabilitation.

AMENDED TO:

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.

19.5.1 PARACETAMOL POISONING

Acute single ingestion <8 hours post-ingestion:

AMENDED FROM:

» Perform a serum paracetamol level 4 hours post-ingestion and ALT.
» 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.

AMENDED TO:

» 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.

MEDICINE TREATMENT

AMENDED FROM:

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. In the presence of bronchospasm, stop the infusion.

AMENDED TO:

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.

19.5.3 OPIOID POISONING

GENERAL MEASURES

AMENDED FROM:

Body packers/stuffers:
Patients may ingest packages of heroin, and are at great risk of life-threatening toxicity in the event of rupture.

AMENDED TO:

Body packers/stuffers:
Patients may ingest packages of illicit opioids and are at increased risk of life-threatening toxicity in the event of rupture.

MEDICINE TREATMENT

AMENDED FROM:

- 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; therefore repeated incremental doses (e.g.: 0.4 mg, 0.8 mg, 2 mg, 4 mg etc.) may be needed at 2 to 3 minute intervals, followed by a naloxone infusion of 0.4 mg/hour.
  - 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 questioned.

Consider intramuscular or subcutaneous administration if the intravenous route is not available

AMENDED TO:

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

19.6.1 TRICYCLIC ANTIDEPRESSANT POISONING

GENERAL MEASURES

AMENDED FROM:

» Discharge patients only when
  - asymptomatic, or
  - mild symptoms/signs of toxicity and ECG has normalised for 24 hours.

AMENDED TO:

Discharge patients only when:
- asymptomatic, or
- mild symptoms/signs of toxicity and ECG has normalised for at least 24 hours.

MEDICINE TREATMENT

AMENDED FROM:

Serum alkalinisation for all patients with:

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

- Sodium bicarbonate, IV 1–2 mEq/kg as an 8.4% solution, as bolus doses to achieve a pH of 7.45–7.55 (Specialist consultation).
  - Monitor acid-base status, serum potassium and sodium.
  - If sodium bicarbonate is unavailable or fluid restrictions limit intake, consider hyperventilation of intubated patients.

AMENDED TO:

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.

19.9.1 BENZODIAZEPINE POISONING

AMENDED FROM:

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, in the elderly, the danger of respiratory depression with overdose exists. Management is supportive and ventilation may be required

AMENDED TO:

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.

19.9.2 LITHIUM POISONING

MEDICINE TREATMENT

AMENDED FROM:

Correct electrolyte abnormalities: see section: 7.2 Major electrolyte abnormalities.
For seizures: R56.8 + (T43.9/X44.99/X69.99/Y14.99)
Treat with benzodiazepines - see section: 14.4.1 Status epilepticus.
Note: Phenytoin should be avoided (due to potential cardiotoxicity

AMENDED TO:

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

» 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).

19.10 ISONIAZID POISONING

DESCRIPTION

AMENDED FROM:

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

AMENDED TO:

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.

19.14.1 COCAINE POISONING

Body packers/stuffers:

AMENDED FROM:

Body packers/stuffers:

» Patients may ingest packages of cocaine, and are at great risk of life-threatening toxicity in the event of rupture.
» Abdominal X-rays or CT scan may show packages.

AMENDED TO:

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.

19.16 INGESTION OF CAUSTIC SUBSTANCES

DESCRIPTION

AMENDED FROM:

Depending on the concentration, caustic substances can cause necrosis of the gut mucosa and underlying tissue resulting in acute perforation especially with strong alkalis and possible strictures later with both acids and alkalis.

AMENDED TO:

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.

19.17.3 METHANOL POISONING

DESCRIPTION

AMENDED FROM:

No longer found in methylated spirits as methanol replaced with less toxic agents 10-20 years ago. Methanol may be found in stove or model fuels, or in antifreeze and windscreen washes.

AMENDED TO:

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.

19.18.2 ORGANOPHOSPHATE POISONING

DESCRIPTION
AMENDED FROM:
Absorption occurs through the skin, when the agent is taken orally, or by inhalation

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