What is poisoning?
Poisoning is the exposure to a toxic chemical or substance thus causing physical harm to a person. The exposure may be through a variety of routes including:
- Oral
- Inhalational
- Dermal/subcutaneous
- Ocular
- Mucosal
- Intravenous

Reasons for the exposure may be:
- Accidental
- Deliberate (either self-harm or poisoned by someone else)
- Therapeutic misadventure, eg. excess paracetamol for toothache
- Recreational, eg. drug abuse
- Industrial/occupational
- Disaster/terrorist activity
- Envenomation, eg. spider bite, snake bite
- Iatrogenic, i.e. during the course of medical treatment

Whatever the reason for the toxicologic presentation, it is a symptom of the underlying social, psychological or medical stressor.

Paediatric Poisoning
Poisoning in children is usually accidental, particularly in the under 6 age group. Deliberate self poisoning may become apparent as they mature into teenage years. Non-accidental poisoning (either deliberate or due to neglect) should be considered and excluded. There should be a low threshold of suspicion to refer a poisoned child to the Child Protection Unit in your hospital or DOCS. Poisoning in children manifests clinically in a similar manner to adults. The management of poisoning in children is also similar.
2. **ASSESSMENT** of the poisoned patient

Poisoned patients should be assessed by a standard process which includes history, examination and appropriate investigations. The following outlines a typical assessment of a poisoned patient; there are situations where additional history, examination or investigations are appropriate.

**Taking a history**

- **Background medical problems**
- **Previous psychiatric history or contact**
- **Known depression**
- **Previous overdose or deliberate self-harm episodes**
- **Medications (including drugs to which the patient has access)**
- **Allergies**
- **Drug use – recreational and illicit**
- **Social history – home situation, type of work**
- **History of the poisoning**
- What did they get exposed to?
- In which form (powder, liquid etc.)?
- Through which route (oral, intravenous, etc.)?
- How much?
- When?
- Was it all in one go or staggered over a period of time?
- Where/from whom did they get access to this medication?
- Where did the overdose happen (home, office, park etc.)?
- Why (self-harm, accidental, recreational, etc.)?
- Did they vomit after the overdose? Were there any tablets present in the vomitus?
- Do they have any abdominal pain?

**Examination**

As in any patient presenting to the ED, the priorities in poisoned patients begin with ABC.

1° survey
- assess patency of airway (ensure C-spine protection if trauma is suspected)
- assess adequacy of ventilation and oxygenation
- assess haemodynamic status
- check vital signs
- assess GCS and neurological exam (incl. pupils, tone, reflexes, clonus)
- check BSL in all patients with altered level of consciousness

2° survey
- full head-to-toe examination
- look for external signs of trauma
- does the patient have secretions or an odour? (e.g. ethanol, cyanide, organophosphate)
- important systems to examine include the cardiovascular, respiratory, gastrointestinal, haematological and neurological systems
- look for pressure areas, bleeding/petechiae/ecchymoses, track marks, bite sites

**Investigations**

Blood glucose level should be performed on all patients with altered level of consciousness.
ECG should be performed on all poisoned patients presenting to the ED
Beta-HCG should be performed on all poisoned females of reproductive age
Paracetamol level as a screening test should be performed on all poisoned patients
Salicylate levels are not routinely indicated and should not be performed without senior consultation
Urine drug screens are usually not necessary nor are they likely alter management – they may be useful in detecting amphetamines in psychotic patients
CT brain is usually unnecessary unless the patient has a history or signs of head injury and a history of poisoning is not definite
Specific drug levels are useful in certain circumstances – consult senior advice

3. TOXIDROMES

Toxidromes are syndromes consisting of a cluster of symptoms and signs which characterise a particular type of poisoning. The main toxidromes to recognise are:

Sympathomimetic
Sympathetic or catecholamine overdrive
Symptoms & signs – hyperalertness, agitation, tremor, seizures, hyperreflexia, tachycardia
Seen in overdose of amphetamines, methylphenidate, cocaine, beta-agonists, theophylline

Cholinergic
from excess acetylcholine action at nerve terminals
Central effects – confusion, convulsions, coma
Muscarinic effects – diarrhoea, urination, miosis, bradycardia, bronchorrhoea, emesis, lacrimation, salivation
Nicotinic effects – mydriasis, tachycardia, fever, weakness, hypertension, fasciculations
Seen in organophosphate insecticide exposure
A similar syndrome can be seen in funnel-web spiderbite

Anticholinergic
From antagonism at muscarinic receptors
Symptoms & signs – delirium, dry mucous membranes, blurred vision, fever, mydriasis, urinary retention, tachycardia
“Blind as a bat, red as a beet, dry as a bone, hot as a hare, mad as a hatter.”
Seen in poisoning from tricyclic antidepressants, neuroleptics, antihistamines, certain plants

Opiate
Excess stimulation of opioid receptors
Symptoms & signs: drowsiness/coma, apnoea, hypotension, miosis (pin-point pupils)
Seen in heroin overdose, excess morphine administration, and other opioid poisoning
Reversed with naloxone

Serotonergic
From excess stimulation of 5-HT receptors
Triad of:
Altered mental status – agitation, confusion
Neuromuscular dysfunction – clonus, hyperreflexia, rigidity
Autonomic instability – diaphoresis, fever, flushing, labile blood pressure
Seen in SSRI antidepressant overdose and multiple drug interactions (eg. between
SSRIs, MAOIs, ecstasy and/or some opioids

In any individual patient, toxidromes may not fully manifest with the classic symptoms and signs. The likely diagnosis is made on available history and signs elicited.

4. PRINCIPLES OF MANAGEMENT
General management of poisoned patients should follow these principles:

Resuscitation
Ensure patent airway and oxygenate patient (may require intubation if GCS < 9)
Support ventilation
Obtain intravenous access and support circulation
Maintain normothermia & euglycaemia

Decontamination, for example:
Remove patient from the source of toxicity into a well ventilated area
Remove contaminated clothing; irrigate skin/eyes/mucosa
Activated charcoal
Whole bowel irrigation
Note that inducing emesis (such as with syrup of ipecac) and gastric lavage are NOT recommended because of a risk of aspiration and oesophageal trauma

Antidotes
There are many specific antidotes for particular poisonings (e.g. N-acetylcysteine for paracetamol poisoning, bicarbonate for tricyclic antidepressant poisoning)
See table in ‘Antidotes’ section below

Supportive treatment, including:
IV rehydration
Correction of electrolyte abnormalities
Analgesia and anti-emetics
Anticoagulation
Blood products
Thiamine
Treat complications such as seizures, aspiration, ARDS, arrhythmias, delirium

Enhance elimination – consult Toxicologist before embarking in these therapies:
Multidose activated charcoal (indicated for a small number of overdoses)
Alkaline diuresis
Haemodialysis
Charcoal haemoperfusion

Consultation – may need to consult with:
Toxicologist
Psychiatrist
Social worker
Drug & Alcohol team
Intensive Care
Others (eg. Dentist: paracetamol OD for toothache)

Disposition
Admission or discharge (with appropriate follow-up)
If admitted, select level of care required in-hospital – eg. ICU, HDU, general ward, special nurse

Each poison has its own specific management and set of tailored interventions. However, many poisonings are managed with supportive measures and no specific interventions exist. Advice on individual cases is best sought from experienced Toxicologists.

5. **ANTIDOTES**
Selected antidotes used in Toxicology.

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<tr>
<td>Snake/spider envenoming</td>
<td>Antivenom</td>
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</table>
Activated charcoal is a synthetic preparation containing charcoal particles in colloidal suspension.

Available as 50g/250mL preparation for oral administration

**Dose:** 1g/kg orally or via gastric tube

**Indications** for use in overdose are:

- Drug ingested has significant potential for toxicity, **AND**
- Ingested drug is adsorbed by charcoal, **AND**
- Charcoal can be administered within 2 hours of ingestion, **AND**
- Patient is alert enough to drink charcoal (or via gastric tube in intubated patients).

**Contraindications:**

- absent bowel sounds
- bowel obstruction or ileus
- unprotected airway
- charcoal does not adsorb the following poisons and should **not** be used for:
  - Metals e.g. lithium, iron, lead
  - Corrosives (acids & alkalis)
  - Alcohols (e.g. methanol, ethylene glycol)
  - Hydrocarbons (e.g. petrol, kerosene)

**Adverse effects:**

- black stools
- bowel obstruction
- potential for aspiration and pneumonitis

**Important notes:**

DO NOT insert a gastric tube in semi-conscious ( unintubated) patients for charcoal administration

Always check for the presence of bowel sounds prior to administering charcoal
If giving charcoal via a gastric tube it may need to be diluted with sterile water; always ensure proper position of the tube with a chest X-ray.

Charcoal administration beyond the 2-hour threshold may be justified in cases where the drugs ingested delay gastric emptying or are sustained-release preparations.

Multi-dose activated charcoal is a controversial treatment of certain types of poisoning – consult with a Toxicologist or the Poisons Information Centre (13 11 26).

7. **PARACETAMOL**

Paracetamol poisoning can occur in the context of suicide attempt, therapeutic misadventure or accidental ingestion. In overdose, paracetamol is increasingly metabolised into a toxic metabolite, which causes hepatotoxicity. Paracetamol can also cause direct coagulopathy and renal impairment.

**Toxic Dose**
- Adults: >200mg/kg in a single ingestion
- Children: >200mg/kg in a single ingestion
- Chronic (repeated supratherapeutic ingestion) – discuss with Toxicologist

**Clinical Effects**
Hepatotoxicity is due to the formation of a toxic metabolite of paracetamol. Glutathione inactivates the toxic metabolite by conjugation, but in overdose glutathione stores are rapidly depleted leading to cellular damage. Hepatic necrosis has been seen in adults ingesting 6g of paracetamol, and death with 15g. The patient may initially appear well and signs of hepatic failure may take up to 72 hours to appear. N-acetylcysteine protects the liver by acting as a precursor for glutathione.

Other clinical effects include:
- Hypoglycaemia (in severe hepatotoxicity)
- Gastritis & vomiting
- Prolongation of prothrombin time
- Acute renal failure (hepatorenal syndrome)
- Metabolic acidosis.

**Paracetamol Level**
A paracetamol level should be performed in all patients who present with a history of overdose. If paracetamol is ingested, the level should be done 4 hours after the drug ingestion, or, at time of presentation if the time since ingestion is >4 hours. If paracetamol is not ingested, the level can be done as a screening test at presentation. The unexpected detection of paracetamol in a poisoned patient should prompt further inquiry and investigation with LFTs & coagulation screen – discuss with a Toxicologist.

**N-acetylcysteine**
N-acetylcysteine (NAC) [Parvolex, Mayne Pharma P/L.] is the antidote used in paracetamol poisoning. Parvolex is available in 10mL ampoules (200mg/mL).

NAC dosage regimen:
1st infusion – 150mg/kg in 200mL of 5% dextrose over 15 minutes, followed by
2nd infusion – 50 mg/kg in 500mL of 5% Dextrose over 4 hours, followed by 3rd infusion – 100 mg/kg in 1000mL of 5% Dextrose over 16 hours.

Note that NAC may need to be administered for longer than 24 hours in some cases of paracetamol-induced hepatotoxicity – consult Toxicologist.

Side effects of NAC therapy:
Gastrointestinal upset – nausea, vomiting
Anaphylactoid reactions – rash, itch, wheeze, SOB, hypotension
These should be treated symptomatically (e.g. anti-emetics, antihistamines, steroids). In the event of an anaphylactoid reaction, NAC therapy should be ceased for 1hr and may be recommenced at half the rate.

Management of paracetamol poisoning
Resuscitation
ABCs
Check BSL
  Paracetamol level (at least 4 hours post-ingestion), EUC, LFTs, coagulation screen

Decontamination with oral activated charcoal (1g/kg) if presents within 2 hours of ingestion

NAC therapy – indications include:
Single ingestion and paracetamol level (taken 4-16 hrs post-ingestion) is above treatment line on nomogram (see following page)
Single ingestion of >200mg/kg AND Patient presents >8hrs post-ingestion
OR
Paracetamol result will be delayed beyond 8hrs post-ingestion
  (start NAC as soon as a paracetamol level is taken; NAC may be ceased later if level is below nomogram treatment line)
Staggered ingestion of >200mg/kg over a 24hr period
Established hepatotoxicity (deranged transaminases or coagulations studies)
Discuss other presenting scenarios with Toxicologist

Supportive treatment – in particular, do NOT administer any paracetamol-containing compounds

Consultation (depending on context of overdose)
Toxicology
Psychiatry
Relevant specialty: e.g. Orthopaedics, Acute Pain Services or Dentist
Social worker

Disposition (from a Toxicological viewpoint)
Asymptomatic patients with single ingestions and paracetamol level below the treatment line may be discharged medically (Note: they still require referral to the appropriate service for the context of their overdose)
Admit patients under medical team who require NAC therapy.
Paracetamol Nomogram

The nomogram should only be used if:
History of single ingestion (i.e. not staggered over a period of time)
Time of ingestion is known
A paracetamol level is taken between 4 and 16 hours post-ingestion

Adapted from Prescott et al. BMJ, 1979; 2: 1097-1100.
8. BENZODIAZEPINES

The sequelae of sedative drug overdose include apnoea, coma, aspiration, hypothermia and rhabdomyolysis (from prolonged lying and local myonecrosis). Mortality can be prevented in these poisonings by the provision of supportive care.

Management of benzodiazepine poisoning:

Resuscitation
Oxygenate and monitor
Airway and ventilatory support
Prevent aspiration in obtunded patients
IV access and circulatory support
Prevent hypothermia and maintain euglycaemia

DON’T EVER FORGET GLUCOSE in any patient with altered level of consciousness

Decontamination
Activated charcoal for pure benzodiazepine ingestion is not recommended

Supportive treatment
Hourly neurological observations in patients with reduced levels of consciousness until GCS 15
Consultation (e.g. Psychiatry, Drug & Alcohol services)

Disposition
Admit non-alert patients to a monitored bed
Alert and asymptomatic patients who have been observed for >4 hours may be toxicologically cleared

Notes:
Some benzodiazepines (e.g. alprazolam) are longer acting and patients may need observation for longer periods.
The use of flumazenil in any overdose (including benzodiazepines) is not routinely recommended and advice from a Toxicologist should be sought. Unmasking of any proconvulsant co-ingestants could lead to seizures after flumazenil administration.
9. **OPIODS**

Drugs such as heroin, morphine sulfate, methadone and oxycodone produce the opioid toxidrome (as described above) causing primarily reduced level of consciousness, respiratory failure and cardiovascular collapse. Opioid poisoning can be rapidly reversed by naloxone administration (usually intramuscular, intravenous, or intranasal). Complications of opioid poisoning include hypoxia, shock, coma, aspiration, and non-cardiogenic pulmonary oedema.

**Management of opioid poisoning:**

**Resuscitation**

Oxygenate and monitor

Airway and ventilatory support

Prevent aspiration in obtunded patients (by intubation)

IV access and circulatory support

Prevent hypothermia and maintain euglycaemia

DON'T EVER FORGET GLUCOSE in any patient with altered level of consciousness

**Antidote**

Naloxone (IM, IV, IN); in the hospital setting, intravenous boluses of 100-400mcg with titration to clinical effect

Beware patients who may become agitated post-naloxone and abscond from the ED; these patients may re-sedate minutes later in an unsafe out-of-hospital setting.

Patient who ingest long-acting or slow-release opioids (e.g. methadone, MS-Contin) may require a naloxone infusion (consult Toxicologist).

**Decontamination**

Consider charcoal for large ingestions which present within 2 hours

**Supportive treatment**

Anti-emetics

Hourly neurological observations in patients with reduced levels of consciousness

CXR to look for non-cardiogenic pulmonary oedema

**Consultation** (e.g. Psychiatry, Drug & Alcohol services)

**Disposition**
Admit non-alert patients to a monitored bed

Alert and asymptomatic patients who have been observed for at least 2 hours post-overdose may be toxicologically cleared.
10 ANTI DEPRESSANTS

Tricyclic antidepressants
TCADs cause the following clinical effects in overdose:
Central: sedation, coma, seizures
Cardiovascular: hypotension, wide-complex arrhythmias
Anticholinergic effects – urinary retention, confusion, ileus, mydriasis, tachycardia

Management of TCAD poisoning
Resuscitation
Oxygenate & monitor (early ECG & blood gas)
Maintain and support airway and ventilation
IV access and circulatory support
Prevent hypothermia and maintain euglycaemia
DON’T EVER FORGET GLUCOSE in any patient with altered level of consciousness
Decontamination
Activated charcoal if administered within 2hrs of ingestion
Dencontaminate via gastric tube if the patient is intubated
Ensure presence of bowel sounds prior to charcoal administration
Antidote
Sodium bicarbonate (8.4% NaHCO₃) is used to treat severe TCAD poisoning
Indications include:
Seizures or coma
Intractable hypotension
Acidosis
Dose: IV boluses of 1-2mmol/kg at intervals of 3-5minutes
Endpoints: normal blood pressure, pH and QRS duration
Supportive treatment
Treat seizures with benzodiazepines
Hourly neurological observations in patients with reduced levels of consciousness
Correct electrolytes (esp. hypokalaemia)
Avoid type 1 anti-arrhythmics and phenytoin
May also need to consider inotropes for persistent hypotension
Performing a serum tricyclic level confirms exposure but does not alter management
Consultation
Toxicology
Psychiatry
Disposition
Admit patients with any of the following:
Arrhythmias or ECG abnormalities
Persistent tachycardia or hypotension
Altered mental status
Seizures
Anticholinergic signs
Monitor all patients for at least 6 hours post-ingestion
Asymptomatic patients with normal examination/ECG at 6 hours may be toxicologically cleared

11. SSRIs
Selective serotonin reuptake inhibitors (SSRIs) in overdose can cause serotonin toxicity
manifest by:
Altered mental status
Autonomic disturbance (labile BP/HR, diaphoresis, hyperthermia)
Neuromuscular dysfunction (hyperreflexia, clonus)

Interactions between several classes of drugs can also produce this toxidrome, including:
SSRIs
Selective noradrenalin reuptake inhibitors (SNRIs – venlafaxine)
Monoamine oxidase inhibitors (MAOIs)
Tricyclic antidepressants (TCADs)
Opioids (e.g. pethidine, fentanyl, tramadol)
MDMA (ecstasy)

The diagnosis of serotonin toxicity is not straightforward and can be difficult to differentiate from other conditions such as neuroleptic malignant syndrome – consult Toxicology advice.

Management of serotonin toxicity is largely supportive, involving cessation of the offending drug(s) and the use of serotonin antagonists (e.g. olanzapine, chlorpromazine & cyproheptadine). Severe toxicity may require invasive ventilation, sedation and paralysis.

SNake BITE

Australian elapid snakes are amongst the most venomous in the world. The primary effects of toxins in snake venom include:
Local effects – pain, redness, bleeding, swelling
Systemic effects – regional pain, lymphadenopathy, headache, nausea, vomiting
Neurotoxicity – ptosis, cranial nerve palsy, diplopia, limb weakness, respiratory paralysis
Myotoxicity – myalgia, rhabdomyolysis
Coagulopathy – both anticoagulant and consumptive forms are seen

Important questions to ask:
When and where was the bite?
Was it a pet snake? If so, what type of snake was it?
Was pressure-immobilisation bandage applied? When? How effective is it?
How were they transported to hospital?
Any bleeding sites (bite site, haematuria, haematemesis)?
Any symptoms (local or systemic) or collapse?

Management of snakebite:
Resuscitation (ABC)

First aid
If the patient presents under 4hrs, apply a pressure immobilisation bandage (PIB) – if it has already been applied, check that it has been correctly done
Apply a firm crepe (or similar) bandage over bite site and then over the entire limb
Immobilise limb with rigid splint
Do NOT remove PIB until patient is at a hospital with adequate laboratory facilities and antivenom supply

Further examination
Examine bite site & regional lymph nodes
Look for evidence of bleeding (skin, mucosa, urine) and muscle tenderness
Examine neurological system (in particular cranial nerves) looking for weakness

Investigations (dependent on type of snake bite and availability in your facility)
BSL, Urinalysis
Pathology: FBC, EUC, LFTs, Coagulation studies, D-Dimer, fibrinogen, CK
Snake venom detection kit: from bite site or urine (NOT from blood)
Spirometry and peak flow measurements of respiratory function
Radiology sometimes may be important (such as CXR or CT brain)
Urinary myoglobin can be done to confirm rhabdomyolysis

Consultation
In any patient with signs of systemic envenomation, speak to a Toxicologist

Antidotes
Check available stock of antivenom (AV) in your ED & Pharmacy
Appropriate antivenom should be administered to when signs of systemic envenomation are present; pre-medication (with steroids & adrenalin) is not routinely recommended.

Supportive treatment
Analgesia
Tetanus prophylaxis
IV rehydration
Treatment of any allergic reactions to antivenom (e.g. rash, wheeze, hypotension)

Disposition
Admit all patients with snake bite (real or suspected) for at least 12 hours or overnight
If the patient is not able to be treated at your facility, arrange transfer to another hospital with adequate laboratory capability, antivenom supply and medical expertise.

**SPIDER BITE**

The medically important spider bites in Australia are those of the funnel-web spider (FWS) and red-back spider (RBS). Clinical features and management of these two types of spider bite is very different.

*Funnel-web spider bite* (*Atrax* and *Hadronyche* genera)

All deaths due to FWS have been attributed to the male *Atrax* spp. prior to the availability of FWS antivenom; cause of death is due to pulmonary oedema or cardiovascular collapse.

**Clinical features**

Local pain
Perioral tingling, piloerection, fasciculations
Muscle spasm (potential for laryngospasm)
Nausea, vomiting, abdominal pain
Tachycardia, hypertension
Increased secretions (giving it an appearance similar to organophosphate poisoning)

**Management**

Support ABCs, oxygenate, monitor, IV access
Remove spider with care if still attached

**Apply pressure immobilisation bandage** (as for snake bite) – do NOT remove bandage until adequate antivenom supply is available
Consult with Toxicologist through PIC for FWS bites with signs of envenomation
If there are signs of systemic envenomation, administer 2 vials of FWS antivenom: given by IMI or slow intravenous infusion; pre-medication (with steroids & adrenalin) is not routinely recommended.
Administer analgesia and antiemetics as required
Check tetanus status
Look for and treat any signs of allergic reaction (e.g. rash, wheeze, hypotension)
Observe all patients with suspected FWS bite for at least 2 hours
If the patient is not able to be treated at your facility, arrange transfer to another hospital with adequate laboratory capability, antivenom supply and medical expertise.

**Red-back spider bite** (*Lactrodectus hasselti*)
Red back spider (RBS) envenomation (known as Latrodectism) is the commonest envenomation syndrome to present to hospital in Australia. RBS envenomation is not fatal and treatment is primarily symptomatic.

**Clinical features**
Local pain is the dominant feature
Local sweating, piloerection, fasciculations
Tachycardia, hypertension
Diaphoresis
Regional pain (e.g. pain over the entire limb)
Chest and abdominal pain, headache

Management
Support ABCs, apply oxygen & monitoring, IV access
Ice packs over bite site may improve symptoms
Do NOT apply compressive bandages or tourniquets
If there are signs consistent with envenomation, give 2 vials of RBS antivenom (diluted in 100mL saline) by slow IV infusion; pre-medication (with steroids & adrenalin) is not routinely recommended.
Administer analgesia and antiemetics as required
Check tetanus status
If you have any concerns with management, consult with Toxicology
Look for and treat any signs of allergic reaction (e.g. rash, wheeze, hypotension)
If the patient is not able to be treated at your facility, arrange transfer to another hospital with adequate laboratory capability, antivenom supply and medical expertise.

INFORMATION & HELP

When to call for HELP?
You can call for help regarding any patient presenting to the ED where poisoning is known or suspected.

Local expertise
Local Toxicology service
Senior ED staff (e.g. Emergency Physician)
NSW Poisons Information Centre 13 11 26

24-hour hotline for advice on the management of the poisoned patient
staffed by experienced Specialists in Poisons Information and Consultant Toxicologists

Websites

CIAP – Micromedex/Poisindex: HYPERLINK "http://


Textbooks


