The primary aim in the treatment of poisoned patients is to reduce mortality, and early and late morbidity.

The secondary aim is to reduce hospital stay and use resources efficiently.

In the case of deliberate self harm a psychological evaluation must be done and the child admitted to hospital for review by CAHMS.

Child protection issues must also be considered, especially in younger children.

**General Management**

**History**

Substance ingested including amount

Timing of ingestion

**Examination**

A B C

AVPU pupils gag

HR rhythm RR BP

Temp BM
Check TOXBASE for advice on management and possible side effects.

Most children require observation only

Gastric emptying should not be performed routinely on any patient

Oral administration of activated charcoal should replace gastric emptying in most cases of acute poisoning.

Syrup of Ipecac should NOT be used

Gastric lavage is the method of choice for emptying the stomach. Its use should only be considered if:

A life threatening amount of a poison has been ingested

The patient presents within 1 hour of ingestion

The airway can be protected in children in grades P and U in the AVPU scale

Gastric lavage is of particular value if the poison is not adsorbed by activated charcoal:

Iron salts

Fluorides

Potassium salts

Lithium preparations

Methanol

Ethylene glycol
Gastric lavage is contraindicated after ingestion of:

- Strong alkalis (e.g. sodium hydroxide)
- Strong acids (Sulphuric and hydrochloric acid)
- Hydrocarbons

**Activated charcoal.**

Potentially, the administration of activated charcoal can reduce drug absorption and increase elimination.

Single doses are usually used.

- Multiple doses have been used but there is no controlled study which proves that this reduces morbidity and mortality.

There is no evidence to support late administration of charcoal for sustained release preparations, although it is given in some circumstances.

Activated charcoal is indicated for poisonings that fulfil the following criteria:

- Drug ingested has significant potential for toxicity
  
  And
  
- Time since ingestion is less than 1 hour
  Or
  
- The drug has significant enterohepatic circulation
  Or
The drug delays gastric emptying and time since ingestion is less than 4 hours

And the drug is adsorbed by charcoal

The dose of activated charcoal is 1g/kg

It is administered orally or by nasogastric tube if there is a risk of aspiration

You are advised to consider the indications for use and evidence of efficacy before giving charcoal.

Ref: www.spib.axl.co.uk
     http://ianwhyte.idl.com.au

Erin Dawson
Staff Grade Paediatric A&E
17th June 2005
Potentially harmful poisons.

**Alcohol**

(Accidental in toddlers, experimental in older children. NB child protection issues especially in younger children)

Can cause hypoglycaemia. Blood glucose must be monitored and IV glucose given if necessary.

**Acids and alkalis**

Strong alkalis more likely to damage stomach with ulceration, perforation and mediastinitis. May also cause metabolic acidosis, collapse, hypotension, acute renal failure and DIC.

Do **not** attempt gastric lavage or give neutralising chemicals as heat produced may cause further damage.

Check FBC U&E

CXR if perforation suspected

Opiate analgesia may be required

Airway management may be required in severely affected patients

Early endoscopy is recommended following acid ingestion but delayed endoscopy at 12-24hrs for alkali ingestion
**Bleach**

Problems seldom arise in children, but may get local lesions in mouth. Milk or water may be given orally.

**Digoxin**

Can cause arrhythmias and hyperkalaemia.

Consider activated charcoal.

Measure U&E and creatinine and digoxin levels.

Monitor HR, BP and cardiac rhythm.

Do 12 lead ECG.

Purified specific Fab antibody binding fragments are available for life threatening toxicity.

Observe for at least 36hrs

**Diphenoxylate (Lomotil)**

Can cause depressed respiration and atropine like effects.
Iron

An estimation of the amount of elemental iron ingested can predict the likelihood of toxicity.

<20mg/kg low risk

20-60mg/kg moderate risk

>60mg/kg high risk

The serum iron taken at 4 hours after ingestion is the best laboratory measure of severity.

Blood glucose, LFT, clotting and WCC should also be measured, and AXR to count number of tablets.

Early features (< 6hrs post ingestion) include nausea, vomiting, abdominal pain and diarrhoea.

Less common early effects in severe poisoning include haematemesis and rectal bleeding due to GI corrosion, drowsiness, convulsions and metabolic acidosis

6-12hrs post ingestion

Mild cases improve but more serious cases may have persistent metabolic effects

>12hrs post ingestion

Evidence of hepatocellular necrosis with jaundice, bleeding, hypoglycaemia, encephalopathy and metabolic acidosis. Hypotension may recur.
2-5 weeks post ingestion

Rarely, gastric stricture and/or pyloric stenosis may cause obstruction.

**Treatment**

Early decontamination of gut (gastric lavage/whole gut irrigation), desferrioxamine infusion, and aggressive management of shock and organ failure are the mainstay of treatment.

**Paracetamol**

Symptoms include gastric irritation and liver failure after 3-5 days

Check plasma concentration 4hrs post ingestion.

If >150 mg/kg paracetamol ingested or plasma concentration high, start IV acetylcysteine.

Monitor LFT, PTT and creatinine.

**Petroleum distillates** (paraffin, kerosene, white spirit)

Aspiration can cause pneumonitis

Usually no treatment indicated. May need oxygen and intensive care for aspiration.
Salicylates

Features include vomiting, dehydration, tinnitus, vertigo, deafness, and sweating, warm extremities with bounding pulses, increased respiratory rate and hyperventilation.

Some degree of acid-base disturbance is present in most cases.

Management

NB Salicylate poisoning is potentially fatal.

Activated charcoal if < 1hr post ingestion

Salicylate levels should be taken at 4 hours (2 hours in symptomatic patients) and repeated every 2 hours till levels falling.

Check U&E, INR/PTR, Glucose and ABG

Correct acidosis and hypokalaemia.

Haemodialysis may be required for severe poisoning.
Solvents

May present with acute intoxication, chronic intoxication or withdrawal symptoms.

History should include social background, school performance/decline, and ingestion of other substances.

Examination-conscious state/mental state, smell of breath, glue round face and mouth, erosions and inflammation of nasal and oral mucosa, nasal discharge, tremor, ataxia, neuropathy, chest (aspiration), liver (failure), anaemia (bone marrow suppression).

Investigations- blood for toxicology, LFTs, U&Es, FBC, Glucose Urine, protein, toxicology CXR ECG

Management- check specific treatment with Toxbase, admit for observation, treat withdrawal symptoms with long acting sedative, most hepatic, renal, haemopoeitic, pulmonary and CNS damage resolves with abstinence and time. Support from CAMHS may be required.

Tricyclic antidepressants

Peripheral features: sinus tachycardia, hot dry skin, dry mouth and tongue, dilated pupils and urinary retention.

ECG includes prolonged PR and QRS intervals.

Central features: ataxia, nystagmus, drowsiness, coma and respiratory depression

Increased tone, hyperreflexia, extensor plantar reflexes.

Divergent squint, hypotension, hypothermia.
Fits in 5%.

During recovery confusion, agitation and visual hallucinations may occur.

Management

Do not give flumazenil if a benzodiazepine has also been taken as it may unmask underlying seizure activity

Give activated charcoal

Check Blood gases

Correct hypotension, hypoxia, and acidosis

Control convulsions with IV Lorazepam as per seizure guidelines and delirium with oral diazepam

Observe for 6 hours if asymptomatic.

Ref:  [www.spib.axl.co.uk](http://www.spib.axl.co.uk)  
[www.indianpediatrics.net](http://www.indianpediatrics.net)

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