approach to poisoning

initial assessment

- the possibility of poisoning should be considered in all unconscious patients
- first line treatment involves securing the airway, giving oxygen & supporting the breathing & circulation
- consider the possibility of hyperglycaemia and give thiamine to all known alcholics to prevent Wernicke's

Airway:
- if there is doubt about the patency of the airway, rapidly intubate the patient with preoxygenation & cricoid pressure
- do not attempt to elicit a gag reflex as the patient may aspirate stomach contents

Breathing:
- many drugs (eg narcotics, sedatives & TCAs) can cause hypoventilation, hypercarbia & respiratory acidosis
- monitor the respiratory rate and check arterial blood gases frequently

Circulation:
- many of these patients are hypotensive on admission which can be due to:
(i) vasodilatory action of drugs (most common)
(ii) direct cardiac toxicity
(iii) hypovolaemia due to decreased fluids or fluid loss (eg vomiting)

investigations

general:
- most patients need only monitoring and basic investigations
- chest X-ray: may show aspiration or atelectasis
- oximetry: is required for continuous monitoring of oxygenation
- biochemistry: CK may be increased because of rhabdomyolysis; theophylline & tricyclics can cause low K
- ABG: may reveal unexplained acidosis due to salicylates, CO, methanol or ethylene glycol
- osmolar gap may be sueful in methanol or ethylene glycol poisoning

drug assays:
- specific serum or plasma levels are useful for dealing with the following drugs:
(i) paracetamol
(ii) iron
(iii) lithium
(iv) salicylates
(v) theophylline
(vi) digoxin
(vii) anticonvulsant agents
(viii) ethanol
(ix) ethylene glycol
(x) methanol

clinical effects of common poisons

Bullae	Barbiturates, tricyclics
Sweating	Salicylates, organophosphates, amphetamines, cocaine
Pupils	
Constricted	Opioids, organophosphates
Dilated	Hypoxia, hypothermia, tricyclics, phenothiazines, anticholinergics
Convulsions	Tricyclics, isoniazid, lithium, amphetamines, theophylline, carbon monoxide, phenothiazines, cocaine
Temperature	
Pyrexia	Anticholinergics, tricyclics, salicylates, amphetamine, cocaine
Hypothermia	Barbiturates, alcohol, opioids
Cardiac rhythm	
Bradycardia	Digoxin, β-blockers, organophosphates
Tachycardia	Salicylates, theophylline, anticholinergics
Arrhythmias	Digoxin, phenothiazines, tricyclics, anticholinergics

Anticholinergic syndromes:
- common manifestations are delirium, tachycardia, dry & flushed skin, dilated pupils, myoclonus, slightly elevated temperature, urinary retention & decreased bowel sounds. Seizures & dysrhythmias may occur in severe cases
- common causes include antihistamines, antiparkinsonian medications, atropine, scopolamine, antispasmodic agents, mydriatic agents, skeletal muscle relaxants and many plants (notably jimson weed and Amanita muscaria)

Sympathomimetic syndromes:
- common manifestations include delusions, paranoia, tachycardia (or bradycardia if the drug is a pure alpha agonist), hypertension, hyperpyrexia, diaphoresis, piloerection, mydriasis & hyperreflexia. Seizures, hypotension & dysrhythmia can occur in severe cases
- common causes include cocaine, amphetamines & theophylline

Opiate, sedative or ethanol intoxication:
- common manifestations include coma, respiratory depression, miosis, hypotension, bradycardia, hypothermia, pulmonary oedema, decreased bowel sounds, hyporeflexia & needle marks. Seizures can occur after overdoses of some narcotics (notably propoxyphene)
- common causes include narcotics, barbiturates, benzodiazepines, ethanol & clonidine

Cholinergic syndromes:
- common manifestations include confusion, CNS depression, weakness, salivation, lacrimation, urinary & faecal incontinence, gastrointestinal cramping, emesis, diaphoresis, muscle fasciculations, pulmonary oedema, miosis, bradycardia or tachycardia and seizures
- common causes include organophosphate and carbamate insecticides, physostigmine, edrophonium & some mushrooms

techniques to decrease drug absorption

induced emesis:
- used primarily for small children soon after ingestion; however, it seems to be falling out of favour even in that group

gastric lavage:
- exact role has not been established; however, it may be useful within 4 hours of ingestion of potentially lethal quantities of a drug
- it should not be given when ingestion of corrosives, caustics, acids or petroleum derivatives is suspected
- technique involves placing the patient in the semiprone position with the head dependent. A large bore nasogastric tube is inserted to aspirate the stomach. Water is inserted (1ml/kg) at body temperature and that amount is then recovered before more is instilled. Cycles are repeated until the return water is clear

charcoal:
- if given early enough, activated charcoal (1gm/kg) can reduce the gastrointestinal absorption of many drugs including aspirin, paracetamol, phenobarbitone, digoxin, carbamazepine, theophylline & phenytoin
- there may be added advantage of clearance from systemic circulation by gastrointestinal dialysis where charcoal is adsorbed onto the charcoal
- it is of little value for acids, alkalis, arsenic, bromide, cyanide, DDT, ethanol, ethylene glycol, heavy metals, hydrocarbons, iodide, iron, lithium, methanol, potassium, tobramycin

whole bowel irrigation:
- some agents such polyethylene glycol electrolyte solutions can decrease drug adsorption by decreasing the time for the drug to transit the gut. They can be useful for purging intact tablets from the gut (eg in cases of iron poisoning)
- it is suited for conscious patients who have ingested tablets that do not bind well to charcoal and can be identified on a plain radiograph
- because polyethylene will bind to charcoal, the two should probably not be used together
- come as commercially available water soluble powders (eg Go-lytely) to be dissolved in about 4L of water. For adults 1-4L/hr should be given until the patient passes clear fluid from the bowel (usually after 3-5L)

techniques to increase drug excretion

forced alkaline diuresis:
- Urinary alkalinization is the administration of intravenous sodium bicarbonate to produce urine with a pH of 7.5 or higher.
- theoretically attractive because it encourages ion trapping in the renal tubules
- can cause dehydration, hypokalaemia & pulmonary oedema
- Urinary alkalinization should be considered as first-line treatment in patients with moderately severe salicylate poisoning who do not meet the criteria for hemodialysis and in those with severe 2,4-dichlorophenoxyacetic acid or mecoprop (MCPP) poisoning

multiple dose activated charcoal:
- Multiple-dose activated charcoal is the repeated oral administration of activated charcoal to enhance drug elimination.
- If the drug concentration in the gut is lower than that in the blood, the drug will passively diffuse back into the gut. The concentration gradient, intestinal surface area, permeability, and blood flow determine the degree of passive diffusion. As the drug passes continuously into the gut, it is adsorbed to charcoal, a process called "gastrointestinal dialysis."
- Multiple-dose activated charcoal also interrupts the enterohepatic and enterogastric circulation of drugs.
- Drugs with a prolonged elimination half-life, a small volume of distribution (less than 1 L/kg), and little protein binding are the most amenable.
- should be considered if a patient has ingested a life-threatening amount of carbamazepine, dapsone, phenobarbital, quinine, or theophylline. With all of these drugs, data confirm enhanced elimination, although no controlled studies have demonstrated clinical benefit
- The initial dose of charcoal is 50 to 100 g, and this treatment is followed every 1, 2, or 4 hours by a dose equivalent to 12.5 g/hour.
- Addition of a cathartic (e.g., sorbitol) may be considered for the initial one or two doses.
- Continuous use of a cathartic can cause diarrhea and fluid and electrolyte imbalance.
- Multiple-dose activated charcoal may be continued until the patient improves clinically

Elimination increased in experimental and clinical studies	Elimination increased in volunteer studies	Elimination not increased in experimental or clinical studies
Carbamazepine	Amitryptyline	Astemizole
Dapsone	Dextropropoxyphene	Chlorpropamide
Phenobarbital	Digitoxin	Doxepin
Quinine	Digoxin	Imipramine
Theophylline	Disopyramide	Meprobamate
	Nadolol	Methotrexate
	Phenylbutazone	Phenytoin
	Phenytoin	Sodium valproate
	Piroxicam	Tobramycin
	Sotalol	Vancomycin

haemodialysis:
- is effective mainly for low molecular weight drugs that are not effectively bound to plasma proteins and have a small volume of distribution
- can be useful for potentially lethal doses of specific drugs such as lithium, ethylene glycol and salicylates

haemoperfusion:
- haemoperfusion involves passing the patient's blood through a device containing charcoal or adsorbent particles such as resin columns
- no trials demonstrate a benefit for haemoperfusion; however it is useful in serious overdoses of:
(i) theophylline (acute >440mcmol/L, chronic >330mcmol/L; lower threshold if age >60, IHD, seizure)
(ii) barbiturates
(iii) phenytoin
(iv) carbamazepine

antidotes

special problems

hypothermia:
- hypothermia is common after poisoning but it rarely requires active measures; it is a marker of increased risk for rhabdomyolysis & aspiration as a result of coma

hyperthermia:
- hyperthermia is uncommon and sometimes associated with TCAs, antipsychotics, antihistamines, amphetamines, cocaine, phencyclidine & salicylates; it may occur as a result of infection due to aspiration

seizures:
- seizures can occur as a direct result of poison and may be difficult to control
- seizures can occur in association with anticonvulsants, phenothiazines, antihistamines, TCAs & theophylline. Seizures can also occur as an indirect result of the poison (eg hypoglycaemia, hypoxia or as a result of global ischaemia)

rhabdomyolysis:
- rhabomyolysis usually occurs in association with pressure necrosis. It can complicate narcotic & cocaine abuse without coma; however, it should always be suspected a patient with prolonged coma

atelectasis & aspiration:
- a chest X-ray should be obtained to detect aspiration due to coma and depressed reflexes & to detect atelectasis