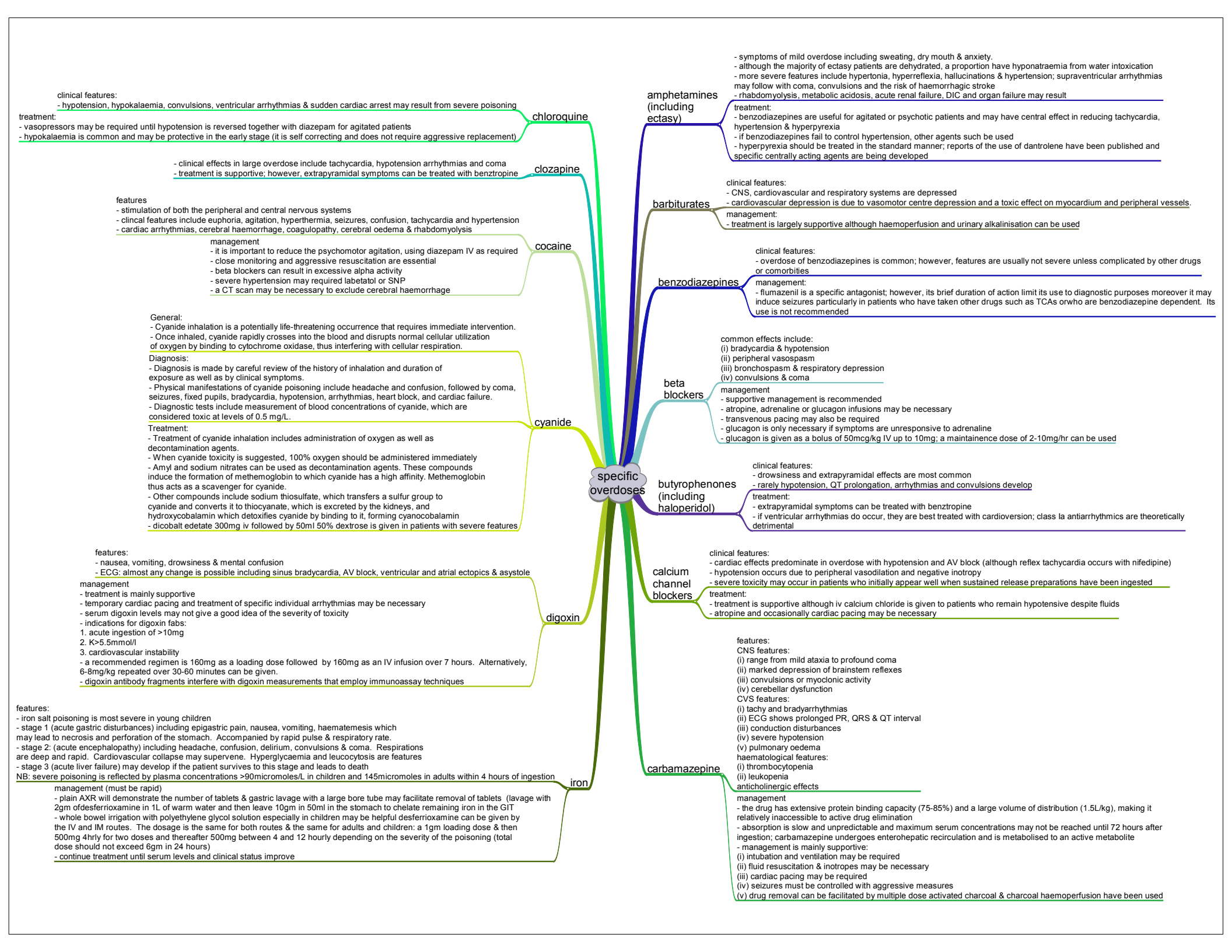
specific overdoses
chloroquine
clinical features:
- hypotension, hypokalaemia, convulsions, ventricular arrhythmias & sudden cardiac arrest may result from severe poisoning
treatment:
- vasopressors may be required until hypotension is reversed together with diazepam for agitated patients
- hypokalaemia is common and may be protective in the early stage (it is self correcting and does not require aggressive replacement)
clozapine
- clinical effects in large overdose include tachycardia, hypotension arrhythmias and coma
- treatment is supportive; however, extrapyramidal symptoms can be treated with benztropine
cocaine
features
- stimulation of both the peripheral and central nervous systems
- clincal features include euphoria, agitation, hyperthermia, seizures, confusion, tachycardia and hypertension
- cardiac arrhythmias, cerebral haemorrhage, coagulopathy, cerebral oedema & rhabdomyolysis
management
- it is important to reduce the psychomotor agitation, using diazepam IV as required
- close monitoring and aggressive resuscitation are essential
- beta blockers can result in excessive alpha activity
- severe hypertension may required labetatol or SNP
- a CT scan may be necessary to exclude cerebral haemorrhage
cyanide
General:
- Cyanide inhalation is a potentially life-threatening occurrence that requires immediate intervention.
- Once inhaled, cyanide rapidly crosses into the blood and disrupts normal cellular utilization of oxygen by binding to cytochrome oxidase, thus interfering with cellular respiration.
Diagnosis:
- Diagnosis is made by careful review of the history of inhalation and duration of exposure as well as by clinical symptoms.
- Physical manifestations of cyanide poisoning include headache and confusion, followed by coma, seizures, fixed pupils, bradycardia, hypotension, arrhythmias, heart block, and cardiac failure.
- Diagnostic tests include measurement of blood concentrations of cyanide, which are considered toxic at levels of 0.5 mg/L.
Treatment:
- Treatment of cyanide inhalation includes administration of oxygen as well as decontamination agents.
- When cyanide toxicity is suggested, 100% oxygen should be administered immediately
- Amyl and sodium nitrates can be used as decontamination agents. These compounds induce the formation of methemoglobin to which cyanide has a high affinity. Methemoglobin thus acts as a scavenger for cyanide.
- Other compounds include sodium thiosulfate, which transfers a sulfur group to cyanide and converts it to thiocyanate, which is excreted by the kidneys, and hydroxycobalamin which detoxifies cyanide by binding to it, forming cyanocobalamin
- dicobalt edetate 300mg iv followed by 50ml 50% dextrose is given in patients with severe features
digoxin
features:
- nausea, vomiting, drowsiness & mental confusion
- ECG: almost any change is possible including sinus bradycardia, AV block, ventricular and atrial ectopics & asystole
management
- treatment is mainly supportive
- temporary cardiac pacing and treatment of specific individual arrhythmias may be necessary
- serum digoxin levels may not give a good idea of the severity of toxicity
- indications for digoxin fabs:
1. acute ingestion of >10mg
2. K>5.5mmol/l
3. cardiovascular instability
- a recommended regimen is 160mg as a loading dose followed by 160mg as an IV infusion over 7 hours. Alternatively, 6-8mg/kg repeated over 30-60 minutes can be given.
- digoxin antibody fragments interfere with digoxin measurements that employ immunoassay techniques
iron
features:
- iron salt poisoning is most severe in young children
- stage 1 (acute gastric disturbances) including epigastric pain, nausea, vomiting, haematemesis which may lead to necrosis and perforation of the stomach. Accompanied by rapid pulse & respiratory rate.
- stage 2: (acute encephalopathy) including headache, confusion, delirium, convulsions & coma. Respirations are deep and rapid. Cardiovascular collapse may supervene. Hyperglycaemia and leucocytosis are features
- stage 3 (acute liver failure) may develop if the patient survives to this stage and leads to death
NB: severe poisoning is reflected by plasma concentrations >90micromoles/L in children and 145micromoles in adults within 4 hours of ingestion
management (must be rapid)
- plain AXR will demonstrate the number of tablets & gastric lavage with a large bore tube may facilitate removal of tablets (lavage with 2gm ofdesferrioxamine in 1L of warm water and then leave 10gm in 50ml in the stomach to chelate remaining iron in the GIT
- whole bowel irrigation with polyethylene glycol solution especially in children may be helpful desferrioxamine can be given by the IV and IM routes. The dosage is the same for both routes & the same for adults and children: a 1gm loading dose & then 500mg 4hrly for two doses and thereafter 500mg between 4 and 12 hourly depending on the severity of the poisoning (total dose should not exceed 6gm in 24 hours)
- continue treatment until serum levels and clinical status improve
amphetamines (including ectasy)
- symptoms of mild overdose including sweating, dry mouth & anxiety.
- although the majority of ectasy patients are dehydrated, a proportion have hyponatraemia from water intoxication
- more severe features include hypertonia, hyperreflexia, hallucinations & hypertension; supraventricular arrhythmias may follow with coma, convulsions and the risk of haemorrhagic stroke
- rhabdomyolysis, metabolic acidosis, acute renal failure, DIC and organ failure may result
treatment:
- benzodiazepines are useful for agitated or psychotic patients and may have central effect in reducing tachycardia, hypertension & hyperpyrexia
- if benzodiazepines fail to control hypertension, other agents such be used
- hyperpyrexia should be treated in the standard manner; reports of the use of dantrolene have been published and specific centrally acting agents are being developed
barbiturates
clinical features:
- CNS, cardiovascular and respiratory systems are depressed
- cardiovascular depression is due to vasomotor centre depression and a toxic effect on myocardium and peripheral vessels.
management:
- treatment is largely supportive although haemoperfusion and urinary alkalinisation can be used
benzodiazepines
clinical features:
- overdose of benzodiazepines is common; however, features are usually not severe unless complicated by other drugs or comorbities
management:
- flumazenil is a specific antagonist; however, its brief duration of action limit its use to diagnostic purposes moreover it may induce seizures particularly in patients who have taken other drugs such as TCAs orwho are benzodiazepine dependent. Its use is not recommended
beta blockers
common effects include:
(i) bradycardia & hypotension
(ii) peripheral vasospasm
(iii) bronchospasm & respiratory depression
(iv) convulsions & coma
management
- supportive management is recommended
- atropine, adrenaline or glucagon infusions may be necessary
- transvenous pacing may also be required
- glucagon is only necessary if symptoms are unresponsive to adrenaline
- glucagon is given as a bolus of 50mcg/kg IV up to 10mg; a maintainence dose of 2-10mg/hr can be used
butyrophenones (including haloperidol)
clinical features:
- drowsiness and extrapyramidal effects are most common
- rarely hypotension, QT prolongation, arrhythmias and convulsions develop
treatment:
- extrapyramidal symptoms can be treated with benztropine
- if ventricular arrhythmias do occur, they are best treated with cardioversion; class Ia antiarrhythmics are theoretically detrimental
calcium channel blockers
clinical features:
- cardiac effects predominate in overdose with hypotension and AV block (although reflex tachycardia occurs with nifedipine)
- hypotension occurs due to peripheral vasodilation and negative inotropy
- severe toxicity may occur in patients who initially appear well when sustained release preparations have been ingested
treatment:
- treatment is supportive although iv calcium chloride is given to patients who remain hypotensive despite fluids
- atropine and occasionally cardiac pacing may be necessary
carbamazepine
features:
CNS features:
(i) range from mild ataxia to profound coma
(ii) marked depression of brainstem reflexes
(iii) convulsions or myoclonic activity
(iv) cerebellar dysfunction
CVS features:
(i) tachy and bradyarrhythmias
(ii) ECG shows prolonged PR, QRS & QT interval
(iii) conduction disturbances
(iv) severe hypotension
(v) pulmonary oedema
haematological features:
(i) thrombocytopenia
(ii) leukopenia
anticholinergic effects
management
- the drug has extensive protein binding capacity (75-85%) and a large volume of distribution (1.5L/kg), making it relatively inaccessible to active drug elimination
- absorption is slow and unpredictable and maximum serum concentrations may not be reached until 72 hours after ingestion; carbamazepine undergoes enterohepatic recirculation and is metabolised to an active metabolite
- management is mainly supportive:
(i) intubation and ventilation may be required
(ii) fluid resuscitation & inotropes may be necessary
(iii) cardiac pacing may be required
(iv) seizures must be controlled with aggressive measures
(v) drug removal can be facilitated by multiple dose activated charcoal & charcoal haemoperfusion have been used