Toxicology Summary

1. Resuscitation
   A, B
   C - Fluid bolus, may need inotropes; beware pulm oedema in Ca antagonist OD
   D - Detect & Correct: Hypoglycaemia, Seizures, Hyper/Hypo-thermia
   E - Emergency decontamination: paraquat, OP’s
       Emergency antidote: digibind, calcium, cyanide

2. Risk Assessment
   Agent, Dose, Time, Coingestants, Clinical features, Patient factors, Suicide risk

3. Supportive care and monitoring
   Document a comprehensive management plan - Expected clinical course, Potential complications
   Fluids, pressure area care, ventilatory support
   Invasive lines - CVC, art line, IDC<NGTA, pacing wire
   Inform next of kin/gain collateral history
   Initiate psych care ?guard, psych review when stable
   Consider NAI/neglect

4. Investigations
   Screening: ECG, paracetamol, glucose, VBG
   Specific: Levels, markers of toxicity (U+E, CK, lactate)

5. Decontamination
   Charcoal - doesn't bind alcohols, acids/alkalis, metals, hydrocarbons. 50g or 1g/kg
   Complications: vomiting, aspiration, impaired absorption subsequent oral antidotes, obstruction
   CI: decr LOC, seizures, bowel obstruction, corrosives
   Whole bowel irrigation - ties up staff, aspiration risk. For SR preps or don't bind charcoal
   For life-threatening: verapamil, diltiazem XR, iron >60mg/kg, K >2.5mmol/kg, arsenic, lead, packers
   Complications: N+V, NAGMA, aspiration, abdo cramps, rectal irritation
   Technique: NGT, charcoal, PEG 2L/hr, metoclopramide, on commode, continue until clear effluent
   Ipecac and gastric lavage - not recommended
   Endoscopy/surgery - specific indications

6. Enhanced Elimination
   MDAC - Interrupts enterohepatic circulation, GI dialysis. 1g/kg then 0.5g/kg q2h
   Risk charcoal bezoar, aspiration. Need: small molecule, small Vd, low PB
   Aminophylline/aspirin
   Barbiturates
   Carbamazepine
   Dapsone
   Mushrooms
   Quinine

   Urinary alkalinisation
   Indications: phenobarb coma, aspirin, methotrexate, rhabdo
   Technique: 1-2 mmol/kg bicarb bolus, infusion 100mmol in 1L 5% dex at 250mL/hour
   Check HCO3 and K Q4hrly; aim urine pH >7.5/serum pH 7.5-7.55
   CI: fluid overload, hypoK, renal failure
   Complications: alkaemia, hypoK, hypoCa, vol overload, pH shifts
Haemodialysis/filtration
Need small molecule, small Vd, rapid redistribution from tissues, slow endogenous elimination
  CI: CV instability (fluid shift, electrolyte imbalance), very small children, profound bleeding
  Lithium
  Metformin lactic acidosis
  Potassium
  Salicylates
  Theophylline
  Toxic alcohols
  Valproate/CBZ

7. Antidotes

8. Disposition

Criteria for admission to Emergency Observation:
Ongoing cardiac monitoring not required
Adequate sedation achieved
Clinical deterioration not anticipated.

Criteria for admission to ICU:
Airway control
Ventilation
Prolonged or invasive haemodynamic monitoring or support
Haemodialysis

In paeds
2 tabs can kill: amphetamines, CCB, chloroquine, opioids, propanolol, sulfonylureas, theophylline, TCA
A sip can kill: OP’s, paraquat, HC’s, camphor, mothball
2 tabs is fine: paracetamol, Fe, colchicine, digoxin, rodenticide

Hyperlacticaemia - Euglycaemia Therapy
CCB, BB OD - Improves myocardial metabolism, BP, contractility and PVR
50ml of 50% dextrose + 50IU insulin
End point: cardiovascular stability
Check BSL q 30min, Maintain normokalaemia
Complications: Hypoglycaemia (hyperglycaemia with CCB OD), Hypokalaemia

Lipid partitioning therapy
Indication: LA’s, propanolol, TCA, verapamil; life-threatening OD lipid-soluble drug where trt failed
Dose: 1ml/kg 20% intralipid over 1min (max 100ml) - rpt if needed - 10ml/hr infusion

NaHCO3
1. Hydrofluoric acid toxicity
2. Correction of severe metabolic acidosis
3. Cardiotoxicity secondary to fast Na channel blockade
   100ml IV, rpt. 100mmol in 1L N saline at 250ml/hr; aim pH 7.5-7.55
   TCA; Type 1a/1c antiarrhythmics: flecainide, quinine; Chloroquine; Propanolol
4. Urinary alkalinisation
5. Prevention of drug redistribution to CNS – incr unionized salicylate
Contraindications: HypoK, hypoCa, alkalosis, acute pulm oedema, renal failure, severe hyperNa
**SS vs NMS**
Both present with:
- Altered mental status
- Fever
- Muscle rigidity and elevated CK
Untreated both can progress to:
- Severe hyperthermia
- Rhabdo
- Renal failure + metabolic acidosis
- DIC/MOF/death

<table>
<thead>
<tr>
<th>SS</th>
<th>NMS</th>
</tr>
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<tbody>
<tr>
<td><strong>Mechanism</strong></td>
<td>Excess serotonin</td>
</tr>
<tr>
<td><strong>Dose related?</strong></td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Onset</strong></td>
<td>Hours</td>
</tr>
<tr>
<td><strong>Mental state</strong></td>
<td>Agitation, anxiety, seizures</td>
</tr>
<tr>
<td><strong>Neuromuscular</strong></td>
<td>R rigidity (lower&gt;upper), clonus, hyper-reflexia, akathisia</td>
</tr>
<tr>
<td><strong>Autonomic</strong></td>
<td>HTN, tachycardia, sweating, mydriasis</td>
</tr>
<tr>
<td><strong>Rhabdo</strong></td>
<td>Only in severe</td>
</tr>
<tr>
<td><strong>Labs</strong></td>
<td>Low Na in MDMA</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Benzos, stop drugs, cooling, fluids, cyproheptadine, intubate/paralyse if severe hyperthermia</td>
</tr>
<tr>
<td><strong>Disposition</strong></td>
<td>ICU unless mild</td>
</tr>
<tr>
<td><strong>Duration</strong></td>
<td>Days</td>
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</tbody>
</table>

**Goals**: early recognition, withdrawal of precipitants
Aggressive supportive care - cooling, IVF, treat rhabdo, monitor electrolytes, cardiovascular support
Bromocriptine: 2.5mg PO TDS - incr to max 5mg Q4h; dopamine agonist; in mod/severe cases
Cyproheptadine: 8mg PO - 4mg PO Q4h; 5-HT receptor antagonist

**Malignant Hyperthermia**
Disorder of skeletal muscle - increased free Ca2+ ions in muscle cells
**Causes**: Sux, Inhaled General Anaesthetics (not NO), Amide Local Anaesthetics (lignocaine, bupivacaine)
**Symptoms**
- Fever: >38.8, Muscle rigidity, decr reflexes, Autonomic changes, Altered LOC
- Resp acidosis and metabolic acidosis; rhabdo
- CK >20,000, incr Ca/K/phos/Mg/BSL/Ur/Cr/coags
- 2-3x incr ETCO2 (early sign)
- Late decr BSL/phos
- Early met acidosis - late resp acidosis
- Urine: myoglobin (+ive peroxidase test)
- Muscle biopsy
**Management**
- Cease Anaesthetic, 100% O2
- If unable to cease switch to N20/opiates/benzos/propofol
- Use non-depolarising NMJ blocker
- Cooling
- Correct electrolytes; IVF
**Dantrolene**: 1mg/kg bolus, Then 3mg/kg, Then 1-2mg/kg 6 hourly for 24-48hrs
**Cholinergic Toxidrome**
Mushrooms (inocybe, clitocybe), organophosphates, funnel web venom, betel nut, pilocarpine

**Defecation**
**Urination**
**Meiosis**
**Bronchorrhea**
**Bradycardia**
**Emesis**
**Lacrimation**
**Salivation**

**Staff protection**

**Decontamination**

**ABC**: start at same time as decontamination; avoid sux (paralysis hrs-days); high flow O2; diazepam 5mg iv (prevents seizures, reduces resp depression)

**Antidotes:**
Atropine 1-2mg (0.05mg/kg in children) Q5min until drying of secretions, resolution of HR and good AE
Glycopyrolate: use if atropine run out, 0.05mg/kg IV
Pralidoxime: best given within few hrs (before aging) Indications: resistant to atropine
1-2g slow IV in 200ml 5% dex - INF 1g/hr

**Anticholinergic Toxidrome**

**M1** – red, hot, dry, retention, constipation, mydriasis, confusion, seizures, hallucinations, MOF, rhabdo

**H1** - Incr HR, hypotension, muscle weakness, postural hypotension, resp paralysis, sedation
Benztropine, antiparkinsons, atropine, hyoscine, glycopyrolate, antihistamines, TCA, CBZ, amanita muscaria

**Decontamination**: Charcoal, MDAC

**Supportive**: Supportive, benzo’s, treat hyperT; NaHCO3 if wide complex tachy

**Antidote**: **Physostigmine** (acetylcholinesterase inhibitor)

Indication: if severe CNS toxicity esp if not responding to benzos/requiring physical restraint
Dose: 0.1mg IV - rpt Q5min to 2mg max; on cardiac monitor

**Alcohol**

**Withdrawal**

Onset 6-24hrs, length 2-7 days
Tremor, agitation, sweating, incr HR, incr BP, N+V, hyperthermia
Hyperreflexia, generalised TC seizures, nightmares, hallucinations

**Delirium tremens**

Mortality 8%
Pcks at 3-4/7
Sx: above + T 40deg, mydriasis, delirium, resp/CV collapse (usually late and assoc with other illnesses)
Supportive management: 5-20mg PO diazepam Q2h until AWS <10, then Q6h; quiet; thiamine

**Wernicke’s encephalopathy**

Medical emergency, Due to thiamine deficit
Nystagmus, disorder of conjugate gaze (paresis of lateral gaze, bilaterally), ataxia, confusion/decr LOC
Decr/incr T, CV instability
Treatment: thiamine 500mg IV over 30mins TDS

**Amiodarone**

Acute toxicity rare, chronic common – pulm/hepatic toxicity, brady, AVB, TdP, hypotension, thyroid
Mostly III (K blockade); also I, II, IV; large VOD
Carbamazepine
Blocks: Na channel, NMDA; antimuscarinic/nicotinic; Increases: NE (decr re-uptake)
Peaks 2-8hrs; 24-96hrs if CR
1x 400mg tablet can cause significant toxicity in paeds - 20mg/kg observe 8 hours
Symptoms
Mild: dizzy, ataxia, mild confusion
Mod (<50mg/kg): choreoathetoid movements, decr GCS, tachy, nystagmus, dysarthria, ataxia, delirium, mydriasis/miosis, opthalmoplegia
Severe (>50mg/kg): seizures, GCS 3-5; arreflexia, anticholinergic sx
Hypotension, HypoNa, incr BSL
Investigations
Levels. ECG: 1st deg HB and wide QRS, long QTc, VT/VF/asystole (Na channel blockade)
Management
Difficult to eliminate as highly protein bound, large Vd, slow absorption, enterohepatic recirculation
Hypotension - IVF; Seizures - benzos
NaHCO3 if: decr BP despite IVF, QRS widening, significant arrhythmias
Charcoal <1hr, MDAC yes
Haemodialysis/filtration if severe toxicity, prolonged coma with rising levels at 48hrs or CV instability
Sodium valproate
Increases: GABA
Peak 4-17hrs
400-1000mg/kg = significant CNS depression; >1g/kg potentially fatal
Symptoms
May be delayed up to 12hrs
Lethargy, coma (>200mg/kg), Seizures, Respiratory depression, Decr BP, incr HR
Decr platelets, AGMA (lactate), hyperNH, decr WBC, metHb, hyperNa, decr BSL, incr LFT's, hypoCa/phos
Cerebral oedema, BM suppression
Management
Levels correlate well with symptoms
Charcoal if >400mg/kg, consider ETT 1st; can do rpt dose at 3-4hrs; MDAC/WBI yes if CR
Haemodialysis/perfusion if life-threatening
Phenytoin
Blocks: Na channels; K channels at high doses
Symptoms
Cerebellar: ataxia, dysarthria, nystagmus; Tremor, involuntary mvmts, opthalmoplegia, N+V
No cardiac problems if oral
If IV: decr HR, hypotension, asystole, V arrhythmia, AVN depression, incr PR, wide QRS, altered ST and T
Management
Levels (correlate with toxicity; coma >50mg/L; nystagmus >20mg/L)
Supportive: Charcoal if <4hrs; MDAC; benzos for seizures; if IV, may need atropine/pacing
Antihistamines
Sedating (1st generation): Block H1, M1, α, 5-HT, cardiac Na + K, Ca channels. Cross BBB (lipophilic)
Non-sedating (2nd generation): Block peripheral H1, cardiac K channels. Don’t cross CNS
Management
Low BP responds to IVF, α1 agonist (NAd)
Wide QRS/VF/VT: NaHCO3
QT prolongation/TdP: MgSO4 )
Antipsychotics

Olanzapine: 40-100mg = mild/mod, >300mg = coma
Quetiapine: <3g mild-mod, >3g severe
ECG: prolonged QRS and QTc, RAD, STD, TWI, TdP, incr PR
Decr BP: IVF + inotrope
Cardiotoxicity: NaHCO3 if incr QRS. MgSO4 and overdrive pacing if TdP
Seizures: benzos
EPSE: benztropine 1-2mg IV (1mg PO BD-QDS)

Aspirin

<100mg/kg – minimal Sx
>300mg/kg – severe
>500mg/kg – potentially lethal
<1.5 mmol/L = therapeutic
>2 mmol/L = toxic
>4 mmol/L = potentially lethal

Salicylism

N+V, Tinnitus, vertigo, seizures, hyperthermia, dehydration, coma, CV collapse

Investigations

Paracetamol level, often in same formulation
ABG – mixed lactic acidosis and resp alkalosis, AGMA
U+E (renal failure, hypoK)
FBC and coags (mild coagulopathy)
CXR – pulmonary oedema
Plasma salicylate level at 4hrs - poor correlation between levels and severity of toxicity; serial levels

Management

Hyperventilate, CPAP for pulmonary oedema
IVF for GI losses and to maintain high UO
K replacement; correct hypoglycaemia; treat seizure
Charcoal if: >150mg/kg and <8hrs; MDAC if significant tox
WBI: if SR prep

Urinary alkalinisation:

Incr urinary pH - drug ionised - cannot be reabsorbed - incr excretion
Indication: symptomatic; level >2.2mmol/L; pH <7.1
Endpoint: no symptoms; level <2.2mmol/L; acidosis resolved. SE: hypoK
Dose: 1-2mmol/kg HCO3 IV bolus - 100mmol/hr infusion if severe; aim urine pH >7.5

Haemodialysis if:

ARF
Acidosis refractory to UA
Severely toxic
Salicylate >4 despite treatment or salicylate >4 in chronic or salicylate >6-9 in acute

Beta-Blockers

Sotalol and propanolol dangerous - In paeds: Any dose propanolol or sotalol bad
Na channel blockade - propanolol (prolonged QRS, VF, VT, seizures)
K channel blockade - sotalol (prolonged QTc, VT, VF)
Alpha blockade - labetalol (worsened hypotension)
Highly lipid soluble - propanolol - worsened CNS Sx

Symptoms

Onset 1-4hrs (>6hrs if SR)
CV: decr BP, decr HR, conduction delays (VT, VF, asystole)
RS: pulmonary oedema, bronchospasm
Met: hypoG, hyperK
CNS: altered LOC, bronchospasm
**Investigations**
ECG: bradycardia, AV block, long PR, wide QRS (propanolol), long QTc (sotalol), VT, TdP, RBBB
Bloods: monitor electrolytes and glucose

**Management**
Propanolol: treat like TCA OD
Bradycardia and hypotension: IVF, NAdr, Atropine
NaHCO3: if wide QRS
CaGlu: if refractory to other treatment
If TdP : MgSO4, overdrive pacing
Charcoal: give if <2hrs or after all SR’s
MDAC: if significant sotalol OD
WBI: consider if SR prep
Dialysis/Charcoal haemoperfusion: can help in atenolol OD
Dextrose/insulin: propanolol OD with CV compromise
Glucagon: 5-10mg IV bolus - 2-5mg/hr in 5% dex
Intralipid: life-threatening OD propanolol

**Disposition**
Observe 4-6hrs, Sotalol 12hrs
Admit ICU: if any signs of toxicity
Cardiac arrest = prolonged CPR ie 4-8hrs ie put them on ecmo

**Calcium Channel Blockers**
>15mg/kg verapamil
>2mg/kg nifedipine
In paeds: 2+ of any SR verapamil/diltiazem potentially lethal

**Signs of toxicity**
CVS: bradycardia, hypotension, 1st deg block
Metabolic: hyperglycaemia, lactic acidosis, AGMA, hypokalaemia
ECG: Prolonged PR, AV dissoc and block, ST changes (ischaemia), Sinus arrest, asystole
Reflex sinus tachy (if not verapamil or diltiazem)/sinus brady; junctional and ventricular escape rhythms

**Management**
Rapidly escalating plan to manage hypotension - CVL and art line early
IVF: 10-20ml/kg (or up to 2L)
Calcium gluconate 60ml 10%, rpt 2-3 times
Inotropes: if not responding to IVF or Ca
Atropine: unlikely to be successful but can try 10-30mcg/kg to max 3mg
Pacing: ventricular; to bypass AVB. ECMO.
NaHCO3: give if QRS wide or for metabolic acidosis
Cardiac arrest: CPR, intralipid, bypass
Monitor gluc and temp
Charcoal: if <1hr (4hrs if SR)
WBI: if >10 tabs SR verapamil/diltiazem, presents <4hrs, and evidence of toxicity
Glucagon: 5mg IV stat - 1-5mg/hr; if resistant to Ca
Dextrose/insulin: if severe/resistant; has +ive inotrope action, incr EF; continue until CV toxicity resolved; aim to maintain normoG (monitor BSL hourly), may need KCl
Intralipid: consider if life-threatening OD

**Carbon Monoxide**
T1/2 depends on pO2. In room air: 4hr, 100% O2: 90min, hyperbaric O2 at 3atm: 23min.
CNS: headache, N&V, dizziness, confusion, cerebellar signs, seizures, syncope, coma
CVS: 1HR, 1BP, ischaemic ECG or MI, dysrhythmias, 1BP
NCPO, lactic acidosis, rhabdo, 1BSL, rhabdo, ARF, DIC

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Hyperthermia, Cherry red skin

If metabolic acidosis - suspect cyanide

**COHb levels** (do not correlate well with Sx):
- <10% in smokers
- 10%: Asymptomatic, headache
- 20%: Dizziness, nausea, SOB, weakness, decr cognitive function
- 30%: Vertigo, ataxia, visual disturbance
- 40%: Confusion, coma, seizures
- 50%: CV and RS failure, arrhythmias, death

CT head if symptoms not resolving
Neuropsychiatric testing at 3-12/12

**Management**

O2 via NRB

HBO Indications:
- Coma/decr LOC/neuro sx
- Ongoing sx after 100% O2 for 4hrs
- Myocardial ischaemia
- Acidosis
- Pregnant

**Cyanide**

Potentially life-threatening - immediate intervention

Histotoxic hypoxia: Binds Fe3+ (ferric) in cytochrome oxidase system - inhibits aerobic metabolism

**Symptoms**

Life threats: coma, seizures, shock, profound lactic acidosis

**Investigation**

Strongly suspect if altered LOC, lactate >10 - suggests cyanide >40, AGMA after smoke inhalation

**ABG**

Cyanide levels - lethal >100mmol/L; toxic >40mmol/L; symptomatic >20mmol/L

SaO2 measure high on pulse oximeter, high pO2 on VBG (decr cellular uptake), no cyanosis – but profoundly hypoxic due to cyanoHb

ECG: ST/T wave changes

**Management**

TIME CRITICAL

Staff PPE

Resuscitation takes priority over decontamination

ABC: high flow O2; HBO if assoc with CO poisoning; intubation/ventilation; correct acidosis

Antidotes: use immediately if severely poisoned (altered LOC, seizures, decr BP, significant lactic acidosis)

Endpoint: improved LOC, CV stability, improved AGMA

**Na thiosulphate**: transfers sulphur group to cyanide → thiocyanate: excreted by kidneys

Pros: fewer SE’s than nitrates; good in cases where diagnosis is in doubt

Indication: mild/mod severe cases can be used alone; otherwise in conjunction with below

Dose: 50ml 25% solution IV given after hydroxycobalamin or EDTA - can rpt at 30mins

SE: mild; N+V, decr BP, headache, AP

**Hydroxycobalamin (Vit B12)**: stable compound with cyanide (cyanocobalamin) - excreted in urine

Pros: safe and non-toxic; treatment of choice

Cons: falsely elevates COHb and bil; not widely available in Aussie

Dose: 5g (70mg/kg in children) IV - rpt if no response at 15mins

SE: minor hypotension, decr/incr HR; orange-red discoloration of skin/MM/urine for 12-48hrs

**Dicobalt EDTA**: forms stable compound with cyanide (greater affinity than MetHb) - excreted in urine

Pros: most widely available in Aussie

Cons: severe SE esp if not poisoned

Dose: 300mg (7.5mg/kg) in 20ml dextrose over 1-5mins; rpt Q5mins if needed
SE: common/severe; hypotension, V, incr HR, anaphylaxis, seizures, facial oedema, CP, SOB

**Amyl nitrite**: forms MetHb which cyanide has a high affinity for
Cons: CI in CO poisoning as will decr O₂ carrying capacity
Dose: INH via crushing under nose - MetHb levels 5%

**Na nitrite**: forms MetHb
Cons: CI in CO poisoning as will decr O₂ carrying capacity
Dose: 10ml 3% solution (=300mg; 10mg/kg in children) over 2-3mins - metHb levels 25%

<table>
<thead>
<tr>
<th></th>
<th>SaO₂</th>
<th>pO₂</th>
<th>Cyanosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyanide</td>
<td>High</td>
<td>High</td>
<td>Yet profound cellular hypoxia</td>
</tr>
<tr>
<td>Met-Hb</td>
<td>Lower</td>
<td>Normal</td>
<td>Yes</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Unresponsive to O₂</td>
</tr>
<tr>
<td>CO</td>
<td>Higher</td>
<td>Normal</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Yet profound cellular hypoxia</td>
</tr>
</tbody>
</table>

**Digoxin**

**Potentially lethal**: K >5, Dose >10g, level >15 nmol/L

**Symptoms**
N+V, AP, ECG changes, lethargy, confusion, weakness
Life threats: K >5.5, decr BP, arrhythmia, cardiac arrest
Chronic OD - usually asymptomatic (yellow vision, decr VA, chromatopsia, xanthopsia)

**Investigations**
ECG: Worsened by hypoK/Mg, hyperCa

**Digoxin effect:**
- Scooped ST segment depression; reverse tick
- Inverted/biphasic T waves, short QT, long PR, prominent U waves, J point depression

**Toxicity:**
- Due to incr automaticity
- AF with slow V response <60
- Blocks, VT/VF/TdP, V ectopics (most common)

**Bloods:**
HyperK (marker of severity, occurs early, if >5.5 = 100% mortality without digibind)
Dig level
Incr Ur and Cr; Mg (worse toxicity if low)

**Management**
Refactory to conventional resus in cardiac arrest – continue 30mins after digibind given
HyperK: insulin/dextrose, NaHCO₃; aim K <5; try not to use Ca (role unclear), salbutamol, frusenide
Arrhythmia: atropine for AVB, may need pacing; MgSO₄ may help in ventricular arrhythmia
If ventricular arrhythmia: lignocaine 1mg/kg IV over 2mins (or phenytoin)
Charcoal: if <1hr; MDAC: if significant toxicity

**Digibind Indications:**
- Refractory arrhythmia/cardiac arrest
- Refractory hyperK >5
- Level >15
- >10mg (4mg in child) ingested
- Acute: ingested dose (mg) x 0.8 x 2 = no. ampoules
- 5 ampoules if stable, 10 ampoules if unstable, 20 ampoules in cardiac arrest
- Chronic: (dig level x weight)/100 = no. ampoules
- Dilute in 100ml N saline, give over 30mins
- 40mg/ampoule = decr dig level by 1 = binds 500mcg dig

**Hydrocarbons**

**Symptoms**
RS: aspiration, pneumonitis, dry cough, NCPO, pleural effusions, wheeze, SOB, decr sats, haemoptysis
GU: RTA, ARF

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CNS: similar to ETOH – rapid onset CNS depression, ataxia, euphoria, coma, seizures

GI: D/V; haematemesis; hepatic toxicity
CV: sensitises myocardium to catecholamines - arrhythmia; hypotension
Skin: eye and skin irritation
BM: incr WCC, aplastic anaemia
Met: toluene - rhabdo

Investigation
CXR: changes may lag 6hrs; may take 2-4/52 to resolve

Management
Decontamination
Indications for gastric lavage: all patients <1hr with any grp III/IV, or >1ml/kg grp II
ETT before lavage in all patients
ABC: O2
Reduce dose of adrenaline if needed
Withhold inotropes if possible (hypersensitive myocardium)
Give 5mg IV metoprolol for arrhythmia
Dialysis: may be used in severe
Discharge: if asymptomatic and normal CXR at 6hrs

Metformin (biguanide)
>10g ingested (same as dig except g rather than mg)

Symptoms
Lactic acidosis = N+V+D
SOB, incr HR, decr BP, coma
Hypoglycaemia minor and easily treated
Ix: Lactate, ABG, U+E

Management
NaHCO3 for metabolic acidosis
Charcoal: if <2hrs + >10g
Haemodialysis: normal dose: any unwell pt with lactic acidosis; OD: worsening lactate and clinical status
Observe 8hrs if >10g (>1700mg children)

Sulfonylureas
Symptoms
Sweating, incr HR, confusion, coma; profound prolonged hypoglycaemia (several days)
Can be delayed 8hrs (longer if CR)

Management
Charcoal
Dextrose
Octreotide: 25-50mcg IV (1mcg/kg in children)
Infusion 25-50mcg/hr (1mcg/kg/hr for children) for 24hrs

Iron
>20mg/kg GI sx: abdo pain, N/V/D, GI bleed, hypovolaemia due to fluid loss
>60mg/kg MOF (direct cellular toxicity) - shock, lactic acidosis (HAGMA), liver failure, coagulopathy
>120mg/kg Potentially lethal
>60mmol/L Toxic
>90mmol/L High risk
Iron content: Actual amount ingested = (mg x elemental %)/weight (kg)
### Symptoms

**Phase 1:** 0-6hrs  
GI

**Phase 2:** 6-12hrs  
Quiescent

**Phase 3:** 12-48hrs  
Systemic Sx (increasing lactic acidosis and shock state)

**Phase 4:** 2-5/7  
Acute hepatic failure, coma, hypoG, coagulopathy

**Phase 5:** 2 weeks  
Scarring and stricture formation

### Investigation

- FBC (WCC, Hb)
- Glucose (initial incr, then decr)
- ABG (lactic metabolic acidosis if severe; AGMA; metabolic alkalosis from GI losses)
- U+E (ATN)
- LFT; coag (incr INR + APTT); XM; Ca
- Fe levels (do 4-6hrs post ingestion; falsely low if desferrioxamine)
- AXR: 50% sens for Fe in stomach
- Markers of toxicity: WCC >15, BSL >8, AGMA

### Management

- Support A + B
- C - Restore circulatory volume (10-20mL/Kg boluses), assess response  
  Ongoing fluid replacement and monitoring (GI & 3rd space losses). Monitor UO
- D - correct hypoglycaemia/electrolytes
- Decontamination: WBI (if >60mg/kg confirmed on AXR)
- Surgical or Endoscopic removal (if WBI unsuccessful/impractical)

#### Desferrioxamine chelation therapy

- Indications:
  1. Level >90 micromol/L at 4-6 hours post-ingestion
  2. Evidence of systemic toxicity - Shock, Metabolic acidosis, Altered mental status
  15 mg/kg/h
- Adverse effects: Hypersensitivity, Hypotension, ARDS, toxic retinopathy, Yersinia sepsis

End point: Patient clinically stable and serum iron level is <60 micromol/L

### Disposition

<20mg/kg: observe 6hrs; discharge if minimal GI symptoms + non-toxic levels + <60mg/kg + AXR negative

### Isoniazid

Rare but potentially fatal

Severe poisoning - rapid onset seizures, coma, severe AGMA

#### Symptoms

>1.5g: dizzy, blurred vision, photophobia, N+V, incr HR, mydriasis, ataxia, hyperreflexia, hyperG

>3g: confusion, decr LOC, refractory seizures, lactic acid acidosis, decr BP, decr RR, incr T

>10g: uniformly fatal

#### Management

- High dose benzos; aggressive supportive treatment
- Charcoal once tubed
- Haemodialysis in severe toxicity resistant to treatment
- IV pyridoxine 5g (70mg/kg) IV over 3-5mins - rpt Q10-15minly until seizures controlled
- If ingested dose known, use same dose of pyridoxine (1g for 1g)
- Give with benzos for synergistic effect
- SE: transiently worsens acidosis; incr RR; orthostatic hypotension

### Local Anaesthetic Toxicity

#### Max Doses

- Bupivacaine  
2mg/kg
Ropivacaine 3mg/kg
Lignocaine 5mg/kg
Prilocaine 7mg/kg

**Clinical Features**
Early: tinnitus, dizziness, anxiety, confusion, perioral numbness
CNS: seizures, coma
CVS: initial hypertension and tachycardia, then hypotension, sinus brady, blocks, vent arrhythmias, asystole
Resp: respiratory depression, apnoea
Bupivacaine more cardiotoxic due to prolonged myocardial binding

**Management**
Limit LA exposure - stop injection, call for help
Prolonged normal resuscitation
Prevention acidosis (hyperventilate, bicarb), Treat seizures, Lipid emulsion (20% intralipid)
End points: ROSC, stabilisation of haemodynamic parameters

**Lead**
**Symptoms**
Acute or subacute lead toxicity:
- AP, N/V, haemolytic anaemia, hepatitis
- metallic taste
- cerebral oedema, encephalopathy, seizures, coma
- clinical effects correlate with levels
Chronic lead toxicity: vague constitutional sx, teratogenic

**Investigations**
Whole blood lead level
FBC: normochromic, normocytic anaemia with basophilic strippling of erythrocytes; U+Es, LFTs
AXR for ingested FB

**Management**
Mannitol 1g/kg + dexamethasone 10mg for cerebral oedema
Endoscopy if above GO junction, whole bowel irrigation if below and symptomatic
Chelation if symptomatic
Sodium calcium EDTA iv for acute encephalopathy
Succimer (DMSA) po if no encephalopathy or asymptomatic but high levels
Consider others exposed - notifiable; Identify source

**Arsenic**
>1mg/kg potentially lethal
Severe gastroenteritis with MOF - Rapid onset severe watery diarrhoea, vomiting, abdo pain, GI bleed
Encephalopathy, seizures, cardiovascular collapse
Hypersalivation, Garlic odour, Acute cardiomyopathy, prolonged QT, arrhythmias
ARDS, renal failure, hepatic injury, bone marrow suppression (max 2-3/52)
Spot urinary arsenic level or 24 hour urinary arsenic excretion

**Management**
ABC. Immediate life threats: hypovolaemia and shock due to GI losses
Cooperative patients, + XR - whole bowel irrigation
Chelation when acute, severe poisoning - Succimer po
Dimercaprol im if unable to give orally due to GI symptoms

**Mercury**
Inhaled elemental mercury aerosol or vapour: pneumonitis, NCPO, neurological injury; H/N/V, metal taste, salivation, visual disturbance
Ingestion inorganic mercury salts: haemorrhagic gastroenteritis, ARF, shock
Organic mercury ingestion/inhalation/skin contact: GI sx, dermatitis, ARF, delayed neurologic injury

**Investigations**
Whole blood or urinary mercury level - confirms recent exposure but not total body burden
XR - radio-opaque; Endoscopy

**Management**
Inhalational - close monitoring, supportive
Ingestion - aggressive fluid resus, supportive care for MOF.
Environmental - remove contaminated clothes, don’t vacuum (aerosols)
Whole bowel irrigation for massive elemental mercury, Charcoal for organic mercury
Chelation if unwell - dimercaprol (not for elemental), penicillamine or succimer

**Dimercaprol**
Rarely used, toxic, im chelator for severe poisoning from lead, inorganic arsenic, mercury.
If possible use succimer - orally-active analogue of dimercaprol

**Lithium**
Therapeutic levels: 0.6-1.2mmol/L. Low therapeutic index; renal clearance; suitable for dialysis

**Acute Toxicity**
>2500mg (>40mg/kg) - GI Sx
CV Sx (HB, prolonged QTc; usually not assoc with significant CV effects)
Neuro Sx uncommon
Levels correlate poorly with toxicity
Indications for GI decontamination: Acute overdose + >40mg/kg ingested + within 1-2hrs ingestion

**Management**
Maintain hydration and sodium repletion with iv normal saline. Urine output >1 mL/kg/hour
Monitor fluid/electrolytes, renal function, serum lithium and clinical features of neurotoxicity
Haemodialysis if severe and renal failure with neurotoxicity

**Disposition**
Discharge if no evidence neurotoxicity, level <2.5 mmol/L and falling

**Chronic Toxicity**
More severe Sx at lower levels. >1.5mmol/L = toxicity

**Effects of chronic use:** nephrogenic DI, hypothyroidism. Tremor, hyperreflexia, ataxia, seizures, coma
Neurotoxicity more common; may be permanent.

**Investigations**
Li level, U+E (decr K, low AG, decr/incr Na, acidosis), FBC (chronic Li use - neutrophilia, WBC 10-15)
ECG (chronic Li use - T wave flattening and inversion; toxicity - long PR, QRS, QTc), AXR

**Indications for dialysis**
Li level >6mmol/L (acute), >2.5mmol/L (chronic)
Severe neuro Sx with high level; ARF even if lower level
Decr BP not responding to fluids

**Methaemoglobinaemia**

**Cellular hypoxia**
Presence of oxidised iron (ferric, Fe3+) in Hb - met-Hb - doesn’t carry O2 - Shifts curve to L
Symptomatic: 20-50%; Potentially lethal: >70%
Causes: Congenital, aniline dyes, chloroquine, dapsone, lignocaine, metoclopramide, nitroglycerin, sulphonamides;
Recluse spider

**Symptoms**
Level 25-40% - chocolate brown blood, dark chocolate colour lips and tongue
Cyanosis out of proportion to resp distress and unresponsive to O2
Falsely decreased Sats but normal PaO2
Headache, weakness, anxiety, syncope, incr HR, SOB
Level 45-55% - decr LOC
Level 55-70% - coma, seizures, arrhythmias

**Investigation**
ABG (co-oximetry)

**Management**
High flow O2, HBO
Avoid/cease precipitants
Antidote: Methylene blue 1-2mg/kg over 5 mins, may need repeat
Decontamination: exchange transfusion if fails to respond to methylene blue

**NSAIDs**

**Ibuprofen**
- <100mg/kg – asymptomatic
- 100-300mg/kg – mild GI and CNS Sx
- >300mg/kg – risk of MOF – rapid onset shock, coma, seizure, ARF, AG metabolic acidosis

**Symptoms**
Often asymptomatic. Mild N+V+AP within 4hrs; mild drowsiness
Less severe metabolic/coag/thermal complications than aspirin
Massive (>300mg/kg) - shock, seizures, coma, ARF, met acidosis, headache, nystagmus, hyperK, hypoCa

**Colchicine**
Uncommon but potentially lethal. Toxicity characterised by GI/T symptoms and delayed MOF
- <0.5mg/kg GI Sx
- 0.5-0.8mg/kg Systemic toxicity, BM dep, 10% mortality (due to myelosupp)
- >0.8mg/kg CV collapse, coagulopathy, ARF; nearly 100% mortality

**Symptoms**
2-24hrs: N/V/D/AP, large GI fluid loss - hypotension. Neutrophilia
2-7/7: MOF: BM suppression; Rhabdo, ARF, haematuria, metabolic acidosis, DIC, ARDS, arrhythmias
>7/7: Incr WBC, alopecia - recovery

**Management**
If presents early, decontamination>resus
Early ICU and ventilatory/cardiovascular supportive care if >0.5mg/kg ingested
IVF++ (maintain high UO)
Charcoal asap if >0.5mg/kg, MDAC
Admit all, observe 24hrs - discharge is asymptomatic and normal WBC

**Opiates**

**Tramadol**: toxic dose >10mg/kg or >1.5g
µ/M/5-HT/NAD
Mild sedation (coma unusual), seizures, agitation, mydriasis, anaphylactoid reactions
Only partially antagonised by naloxone

**Management**
Charcoal yes, maybe in tramadol; MDAC in dextropropoxyphene, SR
Serum alkalinisation: in dextropropoxyphene
Naloxone if GCS <12, RR <6, SaO2 <90%
Onset: 1-2mins, DOA: 20-90mins. 100mcg IV (10mcg/kg in children); 400mcg IM bolus/800mcg SC/2mg IN

**Dependence/withdrawal**
Within hr, peaks at 36-72hrs: anxiety, yawning, craving, lacrimation, rhinorrhoea, diaphoresis, AP+N+V+D
Management: supportive; IVF; antiemetics, antidiarrhoeal; clonidine/benzos
Admit if: severe withdrawal, significant complications/intercurrent illness/psych prob
**Organophosphates**

Rapidly absorbed by dermal, oral and pulmonary routes  
Inactivate acetylcholinesterase (AChE) - incr ACh at muscarinic/nicotinic receptors

**Aging:** After binding, the OP-AChE bond ‘ages’, making complex irreversibly bound (not carbamates)

Nerve gases (1-3mins); dimethyl compounds (2-9hrs), diethyl compounds (36-58hrs)

**Symptoms**

Life threats: coma, decr BP, seizures, resp failure  
**4 Typical clinical syndromes**

1. Acute intoxication - Cholinergic/Muscarinic effects: DUMBELLS, Bradycardia and hypotension
2. Intermediate syndrome - Delayed paralysis (2-4 days)
3. Delayed - Organophosphate-induced delayed neuropathy
4. Chronic organophosphate-induced neuropsychiatric disorder

**Investigation**

ECG (prolonged QTc, STE, TWI, prolonged PR, tachy, brady, AF, VF)  
RBC acetylcholinesterase – indicates severity of poisoning and response to trt; result will take >24hrs  
Plasma pseudocholinesterase – measure of acute exposure, but does not tell severity

**Management**

Staff protection: gloves, clothing, masks, eye shields, resp filter if INH  
Decontamination; charcoal  
ABC: start at same time as decontamination

Sux may cause paralysis for hrs-days; relative resistance to non-depolarising; atracurium good alternative  
High flow O2; diazepam (prevents seizures, may improve survival, reduces resp depression; 5-10mg IV)  
Atropine: 1-2mg (0.05mg/kg in children) Q5min until drying of secretions, resolution of HR and good AE  
Glycopyrolate: reverses cholinergic Sx (not CNS); use if atropine run out; 0.05mg/kg IV  
Pralidoxime: best given within few hrs (before aging)

Reverses some CNS toxicity (may initially worsen paralysis, but should reverse NM blockade)  
Indications: severe Sx, resistant to atropine  
Dose: 1-2g slow IV in 200ml 5% dex (25-50mg/kg in children) - INF 0.5 – 1g/hr 24-48hrs  
**FFP:** increases plasma pseudocholinesterase levels; give 2iu/day until atropine no longer needed

**Strychnine Poisoning**

>15mg (accidental taste) may be fatal in children, >50mg may be fatal in adults, >100mg death common  
**Source**

Rodenticides; adulterant of street drugs

**Symptoms**

*Like tetanus*  
Life threats: muscle rigidity, resp failure, hyperthermia, rhabdomyolysis  
Normal LOC until metabolic acidosis, resp failure, conjugate gaze palsy, mydriasis

**Management**

Time Critical

Decontaminate: give activated charcoal after airway secured  
Other: avoid sensory stimulation; treat spasms (diazepam 5mg QS-10min; paralysis); supportive

**Paracetamol**

| Toxic dose | Adult: 150mg/kg or >10g | Child: 200mg/kg |
| Chronic: | >200mg/kg/day | or >10g/day |
| | >150mg/kg/day for 48hrs | or >6g/day |
| | >100mg/kg/day for 72hrs | or >4g/day |
Toxic levels:
4hrs - 150mg/L 1000mcmol/L
8hrs - 75mg/L 500mcmol/L
12hrs - 38 mg/L 250mcmol/L
16hrs - 19 mg/L 125 mcmol/L

Criteria for liver transplant

HE CRASH
Hypoglycaemia
Encephalopathy
Coagulopathy (INR >3.0 at 48hrs)
Renal failure
Acidaemia (pH <7.3)
Severe thrombocytopenia
Hypotension (BP<80)

Risk factors
Decr GSH: malnutrition, HIV, chronic hepatic diseases
Induction of cP450: ETOH, anticonvulsants

Symptoms
Phase 1 (<24hrs): mild N+V, anorexia, sweating; hypoK correlates with high 4hr lvl
Phase 2 (1-3/7): RUQ pain; ALT/AST peak at 48-72hrs (toxicity if >1000); incr PT, INR, bil; ARF
Phase 3 (3-4/7): fulminant hepatic failure, coagulopathy, encephalopathy, MOF, met acidosis, lactate, ARF
Phase 4 (4/7-2/52): recovery phase; complete resolution of hepatic dysfunction by 1-3/12

Investigations
Aussie/NZ Nomogram - valid for single ingestion, known time of ingestion, non-SR, non-rapid release
LFT’s: toxicity = AST/ALT >1000 (>24hrs); also incr LDH, ALT good in risk assessment
Coag: INR and plt good at predicting risk of death from hepatic failure
Others: hypoG, lactic acidosis; ECG (ST/T changes); hypoK; ATN; decr Ur:Cr (due to hepatic necrosis)

Management
Acute OD Presents >8hrs: do LFTs (ALT) + paracetamol
If reported dose >200mg/kg/Sx of toxicity (AP+N+V), commence NAC immediately
- if normal, stop
- if abnormal continue + add on INR and plt - commence NAC if not already
Repeat bloods after 20hrs - if improving, OK, stop NAC
If not, continue infusion at 100mg/kg/16hrs and recheck ALT/AST Q12-24hrs until decreasing

Acute OD Presents >24hrs: do LFTs/INR/paracetamol/U+E/glu/ABG
If reported dose >200mg/kg/Sx of toxicity, commence NAC immediately
- if normal, stop
- if +ive level or abnormal LFT’s/coag, continue NAC and trt as above

Acute OD Presents ?time: do LFTs/INR/paracetamol/U+E/glu/ABG
If reported dose >200mg/kg/Sx toxicity, commence NAC, continue 20hrs regardless 1st bloods
Repeat bloods after 20hrs
- if normal AST/ALT stop NAC; if abnormal, continue infusion
If SR: start NAC immediately if >200mg/kg or 10g ingested - do 4hr lvl
- if 4hr level +ive, continue treat
- if 4hr level below, rpt level 4hrs later

Chronic OD (supratherapeutic/staggered OD >8hr period)
Essentially treated as >8hr grp
If reported dose toxic levels as above/Sx of toxicity, commence NAC immediately
Several ingestions at known time: take as having occurred at earliest time and use nomogram Several
ingestions at unknown time:
ALT/AST <50 + paracetamol <120mmol/L: no treatment
ALT/AST >50 / paracetamol >120: NAC as above and stop when ALT normalises

**N-acetylcysteine**
Indication: plasma levels as above, half life >4hrs, history large OD and delay to levels, signs/Sx liver damage regardless of paracetamol level
Side effects: Anaphylactoid reaction, Fever, N+V
150mg/kg in 200ml 5% dex over 15mins
50mg/kg  in 500ml 5% dex over 4hrs
100mg/kg in 1000ml 5% dex over 16hrs, repeat until LFTs improve

**Paraquat**
One of most lethal poisons known to man
Denatured when contact with earth
Concentrated in lung (type 2 cells) - late and irreversible pulmonary fibrosis
Excretion: renal - get ATN shortly after ingestion - delayed excretion

**Symptoms**
Immediate: N+V+D
Hours: skin and eye irritation; oral burns; metabolic acidosis
<48hrs: acidosis, hypotension, arrhythmia, ATN, liver necrosis, cough, haemoptysis, NCPO
>48hrs: NCPO, pul fibrosis (late), dysphagia, perf, mediastinitis, pancreatitis, coma, seizures
<10ml 20% or <30mg/kg - mild-mod GI effects, full recovery
10-18ml 20% or 30-50mg/kg - GI corrosive inj, MOF, pul fibrosis
>18ml 20% or >50mg/kg - MOF, alveolitis, metabolic acidosis, death

**Investigation**
Bloods: paraquat levels; urine dithionate test turns blue if exposure; CXR

**Management**
TIME CRITICAL
Staff protection. Decontamination priority over resus - aim to decr dose that reaches lungs
At scene, give food/soil ASAP
Fuller’s earth (1000ml 15-30%) or Charcoal (1-2g/kg or 50g)
Cathartics (200ml 20% mannitol/MgSO4/sorbitol)
Lavage: <2hrs ingestion
Charcoal haemoperfusion: <2-4hrs ingestion
ABC: avoid O2 (worsens toxicity, aim SaO2 90-91%)
IVF, analgesia; consider NAC
Ingestion >6g - all patients die in 1-5/7; CV collapse, NS toxicity
Ingestion 3-6g - all patients die in several weeks; pulm, renal, hepatic toxicity
Ingestion 0.5-2g - may survive

**Amanita phalloides**
Death cap. Contains Amatoxin: not inactivated by cooking; single mushroom can cause death
Early aggressive treatment: mortality 10%; treatment delay >48hrs: mortality 75%

**Symptoms**
Amatoxin suggested if delayed onset (>6hrs) - N+V. Latent phase after 1-2/7
After 3-4/7 centrilobular hepatic necrosis, coagulopathy, GI bleeding, hepatic encephalopathy, renal failure

**Investigation**
Meixner test (on mushroom or GI contents; highly sens, poorly spec); amatoxin assay (on blood, urine, gastric contents); LFT, U+E, coag

**Management**
Admit all; get expert to identify mushroom
Decontamination: ipecac if <4hrs since ingestion; charcoal if <36hrs since ingestion + MDAC
Enhanced elimination: IVF; forced diuresis; charcoal haemoperfusion
Supportive care: supplemental glucose; treat complications; liver transplant
Antidotes: NAC, silibinin, penicillin Gm thioctic acid

**Benzos**
Incr GABA activity via incr frequency of opening of channels  
**Interactions:** diazepam incr metabolism of ETOH and phenytoin  
**Sx:** hypotonia, nystagmus, forced downward asymmetric movement with caloric testing; aspiration pneumonia, hypothermia, DVT, rhabdo  
**Charcoal:** if significant toxicity (not usually required)  
**Flumazenil:** antagonist; max effect 5 mins; may cause withdrawal/seizures; 0.1-0.2mg/min to max 2mg

**Barbiturates**
Incr GABA activity via incr duration of opening of channels  
**Sx:** Miosis, vertigo, nystagmus, decr tone, mimic brain death (unreactive pupils, loss dolls eye, arreflexia)  
Decr RR/ BP/T/BSL, ARDS, decr bowel sounds  
**Ix:** levels correlate well with CNS depression  
**Management:** Charcoal, MDAC if significant; Haemodialysis/perfusion/filtration if severe; ETT early if decreasing LOC  
**Disposition:** observe 6hrs

**GHB**
25mg/kg - sleep, 50mg/kg - coma  
**Sx:** cycling agitation and coma; vomiting; seizures; hypotonia and decr reflexes; nonreactive pupils/miosis; myoclonic movements; bradycardia; U waves on ECG; resp depression; hypothermia; loss of airway reflexes; Sx last 4-6hrs with sudden recovery characterised by delirium and vomiting  
**Management:** ventilation may be needed for 3-6hrs; prognosis good

**SSRIs**
Much less toxic than TCA's  
>1000mg usually significant (>5mg/kg in children)  
Citalopram: >500mg significant; >4.5g cardiotoxicity (like TCA)  
**Symptoms**  
Begin 4hrs, peak 6-8hrs, resolve by 12hrs  
Seizures uncommon, Incr HR; drowsiness; tremor; N+V, dizziness, euphoria, headache, BBB  
Serotonin syndrome  
Citalopram: drowsy, V, seizures, tremor, prolonged QTc and QRS; TdP rare  
**Investigations**  
Include CK if SS  
**Management**  
Benzos for seizures  
Manage serotonin syndrome  
Charcoal: if >600mg citalopram <4hrs; otherwise not usually needed

**Venlafaxine (SNRI)**  
Peak levels 6-8hrs  
Potentially life threatening  
<1.5g = <5% seizures  
<3g = 10% seizures  
>3g = >30% seizures  
>4.5g = 100% seizures, decr BP, minor QRS and QTc changes  
>7g = decr BP, arrhythmias  
**Management**  
Early ETT if >7g  
NaHCO3 for broad complex tachy; benzos for seizures
Manage serotonergic syndrome

Charcoal if <2hrs and >4.5g ingested; not later as risk of seizures
Observe 16hrs due to risk of delayed onset Sx; ECG monitoring 12hrs if >4.5g ingested, 6hrs otherwise

**Monoamine Oxidase Inhibitors**

Produces a hyperadrenergic syndrome from inability to inactivate noradrenaline

**Symptoms**

Mydriasis, flushing, diaphoresis, tachycardia, hypertension, hyperthermia, muscular rigidity, delirium, seizure

Then hypotension from adrenergic depletion

**Management**

Consider gastric lavage and activated charcoal if present within 1 hour

May require ETT

Seizures - benzos

Hypertension - phentolamine

Hypotension - fluids +/- NAdr

Hyperthermia - cool

**Sympathomimetics**

Withdrawal states, amphetamine, cocaine, theophylline, BZP, hypermetabolic syndromes (MH, NMS), MAOI

**Symptoms**

CV: Incr HR, incr BP, cardiomyopathy, arrhythmias, aortic dissection, long QTc/QRS, sudden death

Hyperadrenergic cardiac failure

Myocardial ischaemia/ACS: 50% due to thrombosis, 50% from vasospasm

NS: Mydriasis, nystagmus, hyperreflexia, muscle pains, myoclonic movements, seizures, ICH, CVA

GI/GU: AP, D, urinary retetion, hepatitis, NCPO, ischaemic colitis, GI ulceration

Met: Hyperthermia, hypoNa, metabolic acidosis, rhabdo, DIC, ARF, coagulopathy

RS: pulm haem, barotrauma, pneumonitis, asthma, NCPO

Amphetamine induced psychosis - Delusions, hallucinations; resolves within days

**Investigations**

ECG; U+E, CK, Trop, coags; CXR (dissection); CT head if LOC, seizure or headache

**Management**

Charcoal effective (but not advised as risk of seizures)

Benzos for incr HR/incr BP/seizures/agitation

Antihypertensives (GTN, nitroprusside, labetalol, hydralazine, phentolamine)

Benzos/phenobarb for seizures

iv fluids if rhabdo

Cooling

Arrhythmias - MgSO4 or NaHCO3 if wide QRS

Hypertonic saline if Na <120 + altered LOC / seizures (4ml/kg of 3% over 30mins aiming Na >120)

**TCAs**

>5mg/kg = toxic

>10mg/kg = potentially major

>30mg/kg = severe, coma

In paeds >10mg/kg potentially lethal. Dothiepin: 1 tablet fatal (NS Sx)

**Symptoms**

Peak level 1-2hrs - rapid onset and rapid deterioration

Coma and resp depression, Seizures (QRS >100-120), Arrhythmias (QRS >160), Decr BP

Anticholinergic Sx: mad, blind, hot, dry; bowel and bladder paralysis

**Investigations**

ECG: tachycardia; bradycardia = severe toxicity

long QTc (K), long PR, QRS> 100 (Na) in limb leads
RAD, RBBB; large R waves >3mm in aVR, RS ratio >0.7 in aVR; R rabbit ear taller

Brugada type pattern in severe
ABG: acidosis enhances binding of drug so increases toxicity

**Management**

ABC: early ETT (GCS <12/wide QRS)

Hyperventilate to pCO2 <40 and pH 7.5-7.55

Hypotension: IVF; NAD 0.1-1mcg/kg/min

Seizures: benzos; if occur, expect CV toxicity

Arrhythmias: NaHCO3 100mmol, Rpt Q5mins to max 300mmol in 1st hr aim pH >7.5/narrow QRS

MgSO4 if resistant to above/TdP

Overdrive pacing; defibrillation unlikely to be effective

Charcoal: if >10mg/kg ingested; MDAC: for significant amitrip/nortrip OD

Charcoal haemoperfusion: in very severe refractory OD; less helpful though due to very large VOD

**Disposition**

Admit all symptomatic patients

Admit ICU if: GCS <8, QRS >100 in limb leads, seizures, hypotension, significant arrhythmia

Discharge if: 6hrs observation + HR <100, QRS <100, normal LOC, no complications

**Theophylline**

Causes beta-adrenergic toxidrome (like Irukanji syndrome). Life threatening

**5-10mg/kg:** therapeutic loading dose

**>10mg/kg:** Toxic. Anxiety, N+V, tremor, headache, agitation, confusion, incr HR

**>50mg/kg:** Life threatening. Arrhythmia (SVT, AF, flutter, VT), refractory hypotension, seizures, coma, hyperthermia, rhabdo, severe hypoK/Ph/Mg, hyperG/Ca

**Investigations**

Levels correlate well with Sx in acute

10-20mg/L – therapeutic >100mg/L – usually fatal

Bloods: elects; mixed metabolic (upper GI loss)/resp alkalosis; met acidosis if seizure/hypoT; WCC; CK

ECG: arrhythmia; sinus tachycardia

**Management**

Death may occur despite all treatment

Intubation likely; IVF; may need norad

Beta-blockers for SVT, Control seizures (benzos), K replacement

Charcoal indicated even in delayed presentation. MDAC, WBI: if SR

Haemodialysis: level >100mg/L acute/>60mg/L chronic/arrhythmia, hypoT, seizures

Charcoal haemoperfusion: level >500mmol/L/severe toxicity

Pyridoxine for refractory seizures

Observe 12hrs if CR – which is common prep

**Ethylene Glycol**

**Toxicity**

100ml (1ml/kg) 100%

Toxic metabolites (glycolic acid, lactate) inhibit oxidative phosphorylation and protein synthesis - AGMA

Oxalate precipitates with Ca - crystals - widespread tissue damage renal tubules, myocardium, muscle, brain

ARF within 12-24hrs, hypoCa

**Symptoms**

Phase 1: 1-12hrs; CNS Sx similar to ETOH - absent reflexes, nystagmus, myoclonic jerks, seizures, coma

Phase 2: 12-24hrs; cardiorenal Sx (due to resp, vasc, CV deposition of crystals) - SOB, incr HR, HTN, CCF, APO, decr LOC, shock, coma, seizures, hypoCa - prolonged QTc, arrhythmias; most deaths here

Phase 3: 24-72hrs; renal Sx - AP, ATN, oliguric ARF

Phase 4: 5-20d - cranial neuropathies
Bloods: Incr osmolar gap then AGMA develops due to metabolites (with resp compensation)

Only ethylene glycol, meths and alcoholic ketoacidosis cause incr OG AND AG
Incr lactate, decr Ca, Incr Cr, ketones
Ethylene glycol level (rarely immediately available)
ECG: long QTc
Urine: Ca oxalate crystals in urine, renal epiT cells, protein, microscopic haematuria; urinary fluorescence

Treatment
Maintain hyperventilation; benzos for seizures; trt hypoG/hyperK/hypoMg
Pyridoxine: 100mg IV OD until AGMA resolved; helps convert toxic metabolites to non-toxic
Thiamine: 100mg IV OD until AGMA resolved; as above
NaHCO3: if pH <7.25; 1-2mmol/kg; correction of acidosis encourages metabolism by non-toxic pathways
Ca: if symptomatic of low Ca (eg. seizures, prolonged QTc)
Mg: helps conversion
Aggressive supportive care. Charcoal resistant

Haemodialysis indications:
OG >10
pH <7.25
Level >4-8mmol/L
Visual changes
Deteriorating vital signs despite
Endpoint: level <1.5-3mmol/L, correction of acidosis, OG <10

Antidote
Use until haemodialysis
ETOH: 1g/kg 10% ETOH IV in 5% dex - 150mg/kg/hr 10% ETOH, Aim conc 22-33mmol/L
Fomepizole: alcohol dehydrogenase inhibitor

Discharge
Child: well, bic >20, no OG, >4hrs
Adult: well, bic >20, no OG, no ETOH, >4hrs
Adult: symptoms - admit; ensure FU to make sure no CN probs develop

Methanol
>25ml 40%; lethal dose >1g/kg or >0.5-1ml/kg
Severe AGMA and direct cellular toxicity
1hr – like ETOH but N+V+AP
12-24hr (delayed even longer if ETOH co-ingested) – headache, dizzy, SOB; blurred vision, decr VA, photophobia, fixed dilated pupils, retinal oedema; coma and seizures; severe gastritis and pancreatitis, AP+N+V; oliguric ARF; CCF; pulm oedema

Investigations
Incr OG, AGMA (with resp compensation). Incr lactate, Meths level
CT head: >90% putamen hypodensity, 25% putamen haemorrhage, subcortical white matter haemorrhage

Management
Maintain hyperventilation; benzos for seizures; trt hypoG
NaHCO3: 1-2mmol/kg for urinary alkalinisation if pH <7.3
Folate: 50mg IV QID for 48hrs
Thiamine and pyridoxine and Mg
Haemodialysis: Indications: same as ethylene glycol except level >15mmol/L
Endpoints: meth level <6, correction of acidosis, OG <10,
ETOH or fomepizole: as above; continue until methanol level <6mmol/L
Folinic acid 2mg/kg IV Q6hrly helps

Disposition
Well, bic >20, no ETOH, >8hrs

**Isopropanol**
Augments GABAa receptor - CNS depression; causes ketonaemia; GI irritant; CV depression
As per ETOH but longer and more potent; onset in 30-60mins, peak in few hrs; smell ketosis; AP+N+V, haematemesis, haemorrhagic tracheobronchitis, ATN, haemolytic anaemia, myopathy, resp depression, decr BP; hypoG

**Treatment**
Supportive; thiamine. Haemodialysis: if profound coma, decr BP refractory to IVF, >65mmol/L

**Warfarin**

**Toxic dose**
>2mg/kg - significant incr in INR within 72hrs
If no therapeutic need: trt with Vit K and discharge; check INR in 48hrs as an OP
If therapeutic need: monitor INR Q6hrly

**Treatment**

**Normal INR and no therapeutic need**
If >0.5mg/kg ingested - give 10mg PO Vit K
Discharge; INR in 48hrs in adults, none in children

**INR <5**
Omit dose; if unintentional, consider 10% dose reduction

**INR >5**
If no therapeutic need
No bleeding: 10mg IV vit K - ?discharge, close FU
Active uncontrolled bleeding, clinically significant or major haemorrhage or INR >9
- give 150-300ml / 1-2iu / 10-15ml/kg FFP (works fastest)
- 50iu/kg PTX (contains II, IX, X; small vol, only takes few mins to give, doesn’t need to be thawed, blood grouping not needed; Cl’ed in active thrombosis and DIC; SE = allergy, thrombosis)
- 5-10mg Vit K IV over 2-3mins (risk anaphylaxis with IV vit K; rpt vit K BD if still incr INR; onset action 6-12hrs; XS vit K decreases effectiveness FFP and PTX, re-initiation warfarin difficult)

Endpoint: INR <1.4

If therapeutic need
Aim is to titrate Vit K; when trting, take into account risk categories
No bleeding: 1-2.5mg PO Vit K if INR 5-9
5mg PO if INR >9 - recheck INR in 6-12hr - give repeat doses until INR <5
stop warfarin 1-2/7 - restart at reduced dose once INR <5
start heparin if INR <2 if high risk
Life threatening bleeding: as above
High risk of bleeding (eg. active peptic ulcer, recent OT in 2/52, on aspirin, plt <50) (? If INR >9):
consider CF replacement (INR 2-4 = 25iu/kg PTX, INR 4-6 = 35iu/kg, INR >6 = 50iu/kg)

**Decontamination**
Charcoal if <1hr and patient usually on anticoagulants

**Antidote: Vit K**
Onset: 6-12hr PO, 3-6hrs IV (?1-3hrs)

**Monitoring**
Admit those usually on warfarin; can often give Vit K then discharge those not on warfarin

**Superwarfarins**
Long-acting anticoagulant rodenticides (e.g. brodifacoum)
Benign in single paediatric unintentional OD.
Repeated or massive deliberate OD → prolonged (weeks-months) effects
Serial INR (if normal @48h excludes toxicity)
Single accidental ingestion doesn’t cause significant anticoagulation.
Massive OD>0.1mg/kg of brodifacoum (>2g/kg of 0.005% bait in adult)
Charcoal if <12hr post-OD if deliberate. Vitamin K only if raised INR as otherwise may mask subsequent toxicity