

FExNet 2026

Collated SAQs: Toxicology

ANSWER BOOKLET

Model answers for the 35 toxicology SAQs. References cited at the foot of each question. Items marked [core] are required for full marks. Antidote doses current to 2025; consult Australian Poisons Information Centre 13 11 26 for case-specific advice.

35 questions • 482 marks

ACEM-aligned format. Sub-questions labelled a) b) c). Mark allocations indicative; references cited in the Answer Booklet. Question and answer indexes are clickable.

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Click any entry to jump to its answer. Marks shown right.

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Question 1 | Carbamazepine Overdose (16 marks)

A 30-year-old, 90 kg woman presents to ED having taken a polypharmacy overdose 2 hours ago, including paracetamol/codeine, carbamazepine and temazepam. She becomes drowsy, tachycardic (120 bpm) and hypotensive (85/43 mmHg). Further history reveals up to 9 g of carbamazepine.

a) List 4 general contraindications to activated charcoal. (4 marks)

- Reduced level of consciousness with unprotected airway [core]
- Caustic / corrosive ingestion (alkali, acid) [core]
- Hydrocarbons (aspiration risk)
- Patient refusal / not cooperative
- Substance not adsorbed by charcoal (alcohols, lithium, iron, heavy metals)
- Bowel obstruction / perforation; ileus

b) List 4 investigations for this patient. (4 marks)

- 12-lead ECG (TCA-like Na-channel blockade — wide QRS, prolonged QT)
- VBG with electrolytes and lactate
- Paracetamol level (4-h post-ingestion, plus baseline due to staggered ingestion risk)
- Carbamazepine level (peak 2–8 h immediate-release; later for SR / massive ingestion)
- Bedside glucose; urea & creatinine; LFT

c) List 4 clinical features of significant carbamazepine overdose. (4 marks)

- CNS depression: drowsiness, ataxia, dysarthria, coma
- Anticholinergic: dry mouth, urinary retention, mydriasis, tachycardia
- Cardiovascular: sinus tachycardia, conduction abnormalities (wide QRS, AV block), hypotension
- Seizures (especially in massive ingestion)
- Hyponatraemia (SIADH, especially in chronic toxicity)
- Respiratory depression
- Movement disorders (dyskinesias, nystagmus, myoclonus)

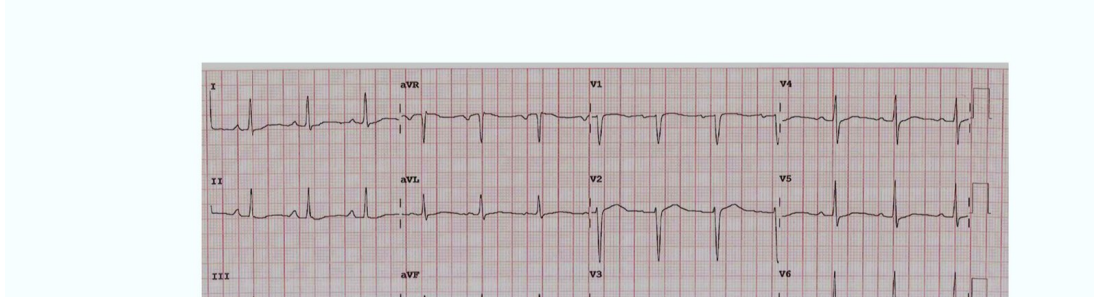
d) List 4 conditions to exclude when this patient has a tonic-clonic seizure. (4 marks)

- Hypoxia / airway obstruction
- Hypoglycaemia (bedside BSL — co-ingestants, sepsis)
- Hyponatraemia (carbamazepine SIADH; check Na)
- Other ingestions (TCA, theophylline, isoniazid, amphetamine)
- Intracranial pathology (head injury from collapse)
- Withdrawal seizures (alcohol, benzodiazepine)

References: Murray's Toxicology Handbook 4e • Tintinalli's Emergency Medicine 9e Ch 198 (Antiepileptics) • Australian Poisons Information Centre 13 11 26

Question 2 | Citalopram Overdose (14 marks)

A 36-year-old man with schizoaffective disorder presents after a large citalopram overdose. ECG shows QTc 540 ms. He is conscious, GCS 15, BP 110/70.



ECG — 12-lead ECG

a) Analyse the ECG: state rate, rhythm, axis. (3 marks)

Marker's note — depends on ECG. Typical: rate ~80 bpm, sinus rhythm, normal axis. Key abnormality is **prolonged QTc (540 ms)**.

b) Name the main ECG abnormality and the cellular mechanism. (2 marks)

Prolonged QTc due to inhibition of the rapid delayed rectifier potassium current (I_{Kr} / hERG channel) → delayed phase 3 repolarisation → substrate for early afterdepolarisations and Torsades de Pointes.

c) List 3 other agents that prolong QT. (3 marks)

- Other SSRIs (escitalopram), SNRIs (venlafaxine)
- Antipsychotics — haloperidol, quetiapine, ziprasidone, thioridazine
- Antiarrhythmics — sotalol, amiodarone, procainamide
- Macrolides / fluoroquinolones (erythromycin, moxifloxacin)
- Methadone
- TCAs (amitriptyline, nortriptyline)
- Antiemetics — ondansetron (high-dose IV), domperidone, droperidol

d) List 6 examination features consistent with significant citalopram poisoning. (6 marks)

- CNS depression / drowsiness / coma
- Seizures (citalopram is the most pro-convulsant SSRI)
- Tachycardia
- QTc prolongation on ECG
- Serotonin syndrome features: tremor, clonus, hyperreflexia, rigidity, hyperthermia (Hunter criteria)
- Hyponatraemia (SIADH)
- Mydriasis
- Diaphoresis

References: Murray's Toxicology Handbook 4e • Tintinalli's Emergency Medicine 9e Ch 191 (Antidepressants) • Hunter Serotonin Toxicity Criteria

Question 3 | Acute Digoxin Overdose (13 marks)

An 18-year-old woman, 65 kg, presents 1 hour after ingesting 200×62.5 mcg digoxin tablets. She has no significant past medical history. BP 110/65, HR 90.

a) Calculate the ingested dose and state the toxicity threshold. (1 mark)

Ingested dose = 200×62.5 mcg = **12.5 mg**. Lethal dose (untreated) is >10 mg in adults or >4 mg in children — this is potentially lethal.

b) List 3 clinical features of ACUTE digoxin toxicity. (3 marks)

- Hyperkalaemia (predicts mortality; $K^+ > 5.0$ indicates serious toxicity) [core]
- Nausea, vomiting, abdominal pain (early)
- Bradyarrhythmia (sinus brady, junctional, AV block) and / or ventricular arrhythmia
- Confusion / lethargy
- Visual disturbance (yellow-green halos — more chronic)

c) List 3 relevant investigations. (3 marks)

- Serum digoxin level at ≥ 6 h post-ingestion (early levels before distribution can mislead)
- 12-lead ECG and continuous monitoring (look for AV block, atrial tachycardia with block, bidirectional VT)
- UEC — particularly K^+ (most important prognostic marker)
- VBG — acid-base status
- Paracetamol level (any deliberate self-poisoning)

d) List 5 indications for digoxin-specific Fab fragments. (5 marks)

- Life-threatening arrhythmia (VT, VF, haemodynamically significant bradycardia) [core]
- Serum $K^+ > 5.0$ mmol/L in acute ingestion [core]
- Cardiac arrest attributed to digoxin
- Ingested dose >10 mg adult / >4 mg child
- Serum digoxin level >15 nmol/L (acute) or >6.4 (chronic)
- Co-ingestion of beta-blocker / calcium channel blocker

e) Calculate the dose of digoxin-specific Fab for this patient. (1 mark)

Dose (vials) = (Ingested dose in mg $\times 0.8$) / 0.5. For 12.5 mg: $(12.5 \times 0.8) / 0.5 =$ **20 vials**. Empirical alternative: 10 vials acute / 5 vials chronic toxicity, titrate to clinical response.

References: Murray's Toxicology Handbook 4e Ch on digoxin • Tintinalli's Emergency Medicine 9e Ch 187 (Digitalis) • Australian Poisons Information Centre 13 11 26

Question 4 | Diltiazem (CCB) Overdose (16 marks)

A 60-year-old man presents 5 hours after ingesting 25 × diltiazem XR 180 mg tablets (4.5 g elemental). He is alert. HR 60, BP 120/67, BSL 6.4. He is admitted for observation.

a) List 4 factors associated with significant CCB toxicity. (4 marks)

- Slow-release / sustained-release preparation **[core]**
- Large ingestion dose (>3 g of diltiazem; >10 tablets verapamil SR) **[core]**
- Co-ingestion of β -blocker, digoxin, other negative inotropes
- Pre-existing heart disease / reduced LV function
- Elderly patient
- Renal failure (delayed clearance)

b) List 4 clinical features of significant CCB toxicity. (4 marks)

- Bradycardia and hypotension (cardiogenic shock)
- AV block (1° → 3°), sinus arrest, junctional rhythm
- Hyperglycaemia (CCB inhibits pancreatic insulin release)
- Pulmonary oedema (especially dihydropyridines)
- Reduced level of consciousness (cerebral hypoperfusion)
- Lactic acidosis (poor perfusion)
- Mesenteric ischaemia (peripheral vasodilation)

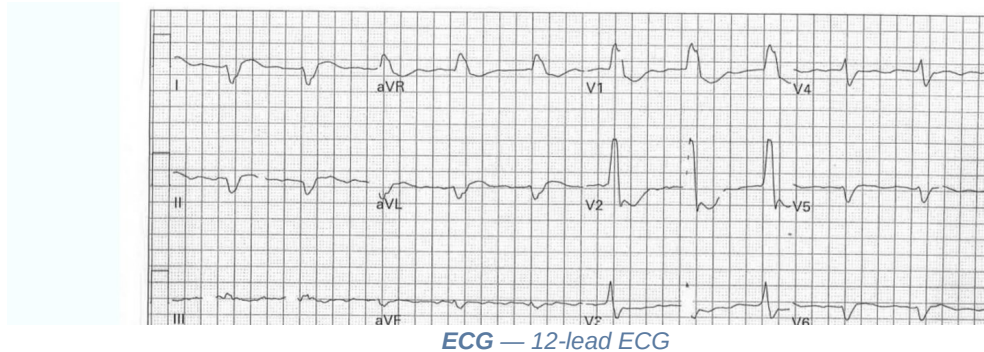
c) List 8 step-wise management priorities for the deteriorating patient (BP 87/40, HR 40, SpO₂ 93% RA, GCS 15). (8 marks)

- Resuscitation — IV access × 2, O₂, monitoring, defibrillator/pacer to bedside, 12-lead ECG **[core]**
- Decontamination — single dose activated charcoal if airway protected and ingestion recent; consider whole-bowel irrigation with PEG (1.5–2 L/h) for SR preparations **[core]**
- IV fluid bolus 10–20 mL/kg crystalloid (caution if pulmonary oedema)
- Calcium chloride 10 mL 10% IV bolus (or calcium gluconate 30 mL 10% IV) — repeat to total 3 g
- Atropine 600 mcg IV for symptomatic bradycardia (often ineffective in significant CCB toxicity)
- **High-dose insulin euglycaemic therapy (HIET):** actrapid 1 U/kg IV bolus then infusion 0.5–1 U/kg/h titrated up to 10 U/kg/h with glucose infusion to maintain BSL 5–10 mmol/L **[core]**
- Vasopressor / inotrope: noradrenaline 0.05–0.5 mcg/kg/min ± adrenaline; consider isoprenaline for chronotropy
- Glucagon 5–10 mg IV bolus, then 1–5 mg/h infusion (less effective in CCB than in β -blocker but consider)
- Lipid emulsion 20% (Intralipid) 1.5 mL/kg bolus then 0.25 mL/kg/min for cardiotoxic CCB overdose with refractory shock
- Transvenous pacing if pharmacological therapy fails; VA-ECMO referral for refractory shock
- Toxicology consultation (Australian PIC 13 11 26)

References: Murray's Toxicology Handbook 4e Ch on Calcium Channel Blockers • Tintinalli's Emergency Medicine 9e Ch 189
• Australian Poisons Information Centre 13 11 26 • eTG Toxicology

Question 5 | Chronic Digoxin Toxicity (13 marks)

An 83-year-old man with AF and CCF presents with 2 weeks of lethargy, anorexia and dehydration. ECG shows AF with slow ventricular response (40 bpm) and 'reverse-tick' ST changes. Bloods: K^+ 6.4, Cr 200, digoxin 3.0 nmol/L. BP drops to 100/SBP, HR 30.



a) List 3 ECG features. (3 marks)

- Atrial fibrillation with slow ventricular response (~40 bpm)
- Sagging ('reverse-tick' / 'scooped') ST depression — digoxin effect
- T-wave inversion / flattening
- Short QT interval
- Possible AV block, atrial tachycardia with AV block (highly suggestive)

b) List 3 possible causes of these ECG abnormalities. (3 marks)

- Chronic digoxin toxicity (most likely — level 3.0, renal failure, dehydration) **[core]**
- Hyperkalaemia (peaked T waves, prolonged PR, wide QRS)
- Acute coronary ischaemia (RCA → AV block)
- Hypothyroidism (bradycardia, low voltage)
- Sick sinus syndrome / degenerative conduction disease

c) List 5 treatment steps. (5 marks)

- Cease digoxin; volume resuscitation cautiously (elderly, CCF) **[core]**
- Treat hyperkalaemia: insulin/dextrose (10 U + 50 mL 50%); salbutamol 10–20 mg neb; bicarbonate if acidotic **[core]**
- **Digoxin-specific Fab** 5–10 vials IV (chronic toxicity dosing; titrate to clinical response) **[core]**
- Atropine 600 mcg IV for symptomatic bradycardia
- Transcutaneous pacing if pharmacological therapy fails
- ICU / cardiology admission; renal replacement therapy consideration if Fab unavailable

d) Briefly explain the controversy around IV calcium in this setting. (2 marks)

Historical 'stone heart' theory suggested IV calcium in digoxin-induced hyperkalaemia could precipitate ventricular fibrillation due to massive intracellular calcium load. Recent observational evidence (Levine et al., 2011) does NOT support this association. **Current Australasian practice:** calcium can be given for life-threatening hyperkalaemia, but priority is digoxin-specific Fab; calcium should not be withheld if ECG shows membrane instability and Fab is not immediately available.

References: Murray's Toxicology Handbook 4e Ch on digoxin • Tintinalli's Emergency Medicine 9e Ch 187 • Levine M et al. Effects of calcium for hyperkalaemia in digoxin toxicity. J Emerg Med 2011

Question 6 | Eucalyptus Oil — Paediatric Ingestion (12 marks)

A 4-year-old girl presents to ED 30 minutes after being found beside an empty 100% eucalyptus oil vaporiser bottle. Vital signs: HR 120, BP 98/60, RR 26, SpO₂ 94% RA, T 36.1°C. She is drowsy.

a) List 3 sequelae of eucalyptus oil poisoning. (3 marks)

- CNS depression / coma (GCS may drop rapidly, especially <5 mL in young child)
- Aspiration pneumonitis (hydrocarbon — chemical pneumonia)
- Seizures
- Vomiting → aspiration risk
- Gastrointestinal irritation

b) List 4 priorities in acute management. (4 marks)

- Airway protection — assess for aspiration risk; intubate if GCS ≤8 or airway compromise **[core]**
- Supplemental O₂; CXR for aspiration
- **Do NOT induce emesis or perform gastric lavage** (hydrocarbon — aspiration risk outweighs benefit; charcoal not effective)
- IV access; supportive care (fluids, antiemetics if no contraindication)
- Continuous monitoring; early discussion with toxicology
- Disposition to paediatric ICU if symptomatic

c) List 5 components of a general toxicology risk assessment. (5 marks)

- **Agent(s)** — what, how many, formulation (SR/IR), strength **[core]**
- **Dose** — total mg/kg ingested; calculate from missing pills × strength × weight **[core]**
- **Time of ingestion** — peak effects, decontamination window **[core]**
- **Patient factors** — age, weight, comorbidities, organ function (renal, hepatic), pregnancy
- **Clinical features** — current presentation; predicted trajectory based on toxic dose

References: Murray's Toxicology Handbook 4e Ch on essential oils • Australian Poisons Information Centre 13 11 26 • Royal Children's Hospital Melbourne — Clinical Practice Guideline

Question 7 | GHB Overdose (12 marks)

A 14-year-old male presents 1 hour after ingesting GHB (γ -hydroxybutyrate). No co-ingestants reported. VBG normal. Initially GCS 8 (E2 V2 M4).

a) List 4 important complications of GHB overdose. (4 marks)

- Coma with rapid swings in conscious level (classic 'agitated, then unrousable' pattern)
- Respiratory depression / apnoea
- Aspiration of vomit
- Bradycardia, hypotension
- Hypothermia
- Withdrawal syndrome on cessation (chronic users)
- Co-ingestion potentiation (alcohol, opioids → fatal respiratory depression)

b) List 4 indications for intubation. (4 marks)

- Inability to protect airway — absent gag, drooling, vomiting [**core**]
- Respiratory depression / failure (rising EtCO₂, ↓SpO₂)
- Persistently low GCS <8 not awakening
- Need for procedure (e.g. CT) requiring airway control
- Aspiration evident on examination / CXR
- Concern for co-ingestion with prolonged sedation

c) List 4 sequential interventions for the patient (10 h post-ingestion, GCS 14, mobilising towards exit, wishes to leave). (4 marks)

- **Capacity assessment** — minor (14 yo); cannot consent / refuse alone; involve parents and DEMENT
- De-escalation / safety: calm interaction; senior + security to prevent harm; document interaction
- Mental Health Act order if criteria met (mental illness, harm risk, lacks capacity, no less restrictive option)
- Physical restraint if absolutely required; prefer chemical (droperidol 5–10 mg IM or olanzapine 10 mg IM) per departmental ABD protocol
- Mandatory child protection notification; mental health liaison

Marker's note: Marker's note — patient is a minor; legal framework for minors and capacity differs across Australian jurisdictions. Avoid 'detain at all costs'.

References: Murray's Toxicology Handbook 4e Ch on GHB • Tintinalli's Emergency Medicine 9e Ch 184 • ACEM Acute Behavioural Disturbance Position Statement

Question 8 | Insulin Overdose (10 marks)

A 25-year-old man with type 1 diabetes presents 30 minutes after a deliberate overdose of his insulin. He is alert; BSL 2.1 mmol/L.

a) List 4 features of your toxicology history. For each state the relevance. (8 marks)

History feature	Relevance
Type and amount of insulin (rapid / short / intermediate / long-acting)	Determines duration of monitoring; long-acting (glargine, detemir) requires >24 h observation
Time of injection	Predicts peak hypoglycaemia (rapid 30–90 min, regular 2–4 h, NPH 4–10 h, glargine flat over 24 h)
Site of injection / depot effect	Subcutaneous depot → delayed and prolonged release; consider local excision in massive intentional overdose
Co-ingestion (alcohol, oral hypoglycaemics)	Synergistic / prolonged hypoglycaemia; affects duration of monitoring

b) List 2 management steps with end-points after he responds to 25 mL 50% dextrose. (2 marks)

- Continuous 10% dextrose infusion (start 100 mL/h; titrate to keep BSL 5–10 mmol/L); end-point: stable BSL ≥6 hours off infusion for short-acting; ≥24 h for long-acting
- Frequent hourly BSL monitoring; admission to monitored bed; nutrition (oral / NG carbohydrate); end-point: patient awake, eating, BSL stable without dextrose

References: Murray's Toxicology Handbook 4e Ch on insulin / oral hypoglycaemics • Tintinalli's Emergency Medicine 9e Ch 220 • Australian Poisons Information Centre 13 11 26

Question 9 | Iron Ingestion — Paediatric (Risk Assessment) (12 marks)

A 23-month-old (12 kg) is brought in 30 min after his mother found him next to her iron tablets (ferrous sulfate 325 mg = 65 mg elemental iron each). Three tablets remain from a previously half-full bottle of 100.

a) Complete the table for dose-related risk of iron in paediatric ingestion. (4 marks)

Elemental iron (mg/kg)	Effect
<20	Asymptomatic; no intervention needed
20–60	Mild GI symptoms; observation
60–120	Significant systemic toxicity expected; admit / treat
>120	Potentially lethal; aggressive management and chelation

b) Complete the table for 4 investigations and the result indicating significant ingestion. (4 marks)

Investigation	Significant result
Serum iron level (4–6 h post-ingestion)	>90 µmol/L — significant; >180 µmol/L — severe
Abdominal X-ray	Radio-opaque tablets in stomach / small bowel (especially intact SR)
VBG with lactate	Anion-gap metabolic acidosis with elevated lactate — systemic toxicity
UEC + LFTs + coagulation	Hepatic injury (transaminases ↑↑); coagulopathy

c) Name 2 methods of decontamination in iron overdose and when to use each. (2 marks)

- **Whole-bowel irrigation with polyethylene glycol** 1500–2000 mL/h until rectal effluent clear — used when tablets visible on AXR or large ingestion (charcoal does NOT bind iron)
- **Endoscopic removal** — used for large tablet bezoars or perforation risk

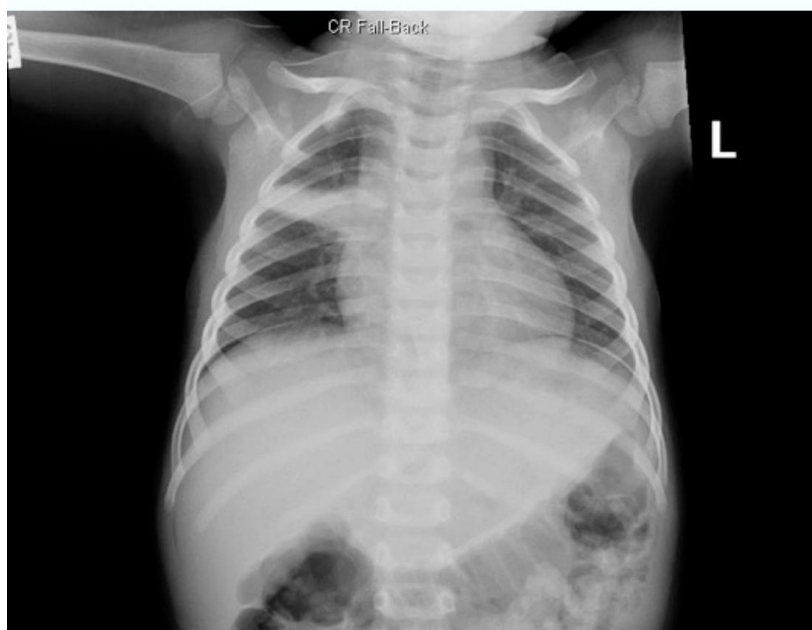
d) State the chelation therapy used in significant iron toxicity, with dose. (2 marks)

Desferrioxamine (deferoxamine) 15 mg/kg/h IV infusion (max 80 mg/kg/24 h). Indicated for: serum iron >90 µmol/L, severe symptoms (shock, altered conscious state), or evidence of systemic toxicity (acidosis). Stop when symptoms resolved, acidosis cleared, urine 'vin rosé' colour disappears.

References: Murray's Toxicology Handbook 4e Ch on Iron • Tintinalli's Emergency Medicine 9e Ch 196 • Australian Poisons Information Centre 13 11 26 • Royal Children's Hospital Melbourne — Clinical Practice Guideline

Question 10 | Iron Toxicity — Stages & X-ray (16 marks)

A 2-year-old boy ingested 90 mg/kg elemental iron 1 hour ago. He is currently asymptomatic. AXR shows multiple radio-opaque tablets in the stomach.



AXR — Supine abdominal X-ray

a) Complete the table of dose-related effects. (4 marks)

Elemental iron	System / effects
<20 mg/kg	GI mucosa — minimal / asymptomatic
20–60 mg/kg	Local GI: vomiting, diarrhoea, abdominal pain
60–120 mg/kg	Systemic toxicity: acidosis, shock, hepatic / renal injury
>120 mg/kg	Severe systemic toxicity: lethal; multi-organ failure

b) Complete the table of classical stages of iron toxicity. (5 marks)

Time	Clinical features
0–6 h	GI: vomiting (often haematemesis), diarrhoea, abdominal pain
6–12 h	Apparent recovery / 'latent' phase — deceptive
12–48 h	Systemic toxicity: shock, metabolic acidosis, coagulopathy, renal failure
2–5 days	Hepatic necrosis, hepatic failure
2–6 weeks	Late: pyloric / intestinal stricture from scarring

c) List 2 relevant findings on AXR. (2 marks)

- Radio-opaque tablets visible in stomach / small bowel (intact SR formulations especially)
- Number / location guides decontamination strategy
- Free intraperitoneal gas if perforation

d) State how the X-ray alters management. (2 marks)

Visible tablets confirm ongoing absorption potential and support **whole-bowel irrigation** (polyethylene glycol PEG 1500–2000 mL/h) as the decontamination strategy. Re-image to confirm clearance. Activated charcoal is NOT useful (does not adsorb iron). Endoscopy if large bezoar or pyloric obstruction.

e) List 3 blood tests with reasoning. (3 marks)

- Serum iron level at 4–6 h — peak; >90 µmol/L significant
- VBG with lactate — anion-gap metabolic acidosis predicts systemic toxicity
- LFTs + INR / coagulation — hepatic injury surrogate; stage 4 progression
- UEC + creatinine — renal involvement
- Paracetamol level (deliberate ingestion) and FBC

References: Murray's Toxicology Handbook 4e Ch on Iron • Tintinalli's Emergency Medicine 9e Ch 196 • Royal Children's Hospital Melbourne — Clinical Practice Guideline

Question 11 | Iron Overdose — Adult (14 marks)

A 65-year-old woman has taken a deliberate overdose of iron tablets in a suicide attempt. She presents 2 hours later with vomiting and abdominal pain.

a) List 5 components of the toxicological risk assessment. (5 marks)

- Agent — formulation (ferrous sulfate, gluconate, fumarate); elemental iron content per tablet **[core]**
- Dose ingested in mg/kg elemental iron **[core]**
- Time since ingestion (peak 2–4 h; later for SR)
- Patient factors — weight, age, comorbidities, pregnancy (altered iron handling)
- Clinical features and trajectory (vomiting, haematemesis, acidosis)

b) Complete the table for assessment features of potentially lethal iron ingestion. (6 marks)

Domain	Finding
History	Ingested dose >120 mg/kg elemental iron
History	Persistent vomiting, haematemesis
Examination	Hypotension, altered mental state
Examination	Metabolic acidosis features (Kussmaul)
Investigation	Serum iron >180 µmol/L at 4–6 h
Investigation	Anion-gap metabolic acidosis with elevated lactate

c) List 2 decontamination methods for this patient. (2 marks)

- Whole-bowel irrigation with PEG 2000 mL/h until effluent clear (tablets on AXR or large ingestion)
- Endoscopic removal — large bezoar or pylorus-blocking tablets

d) Name the specific therapy for severe iron toxicity. (1 mark)

Desferrioxamine 15 mg/kg/h IV infusion (max 80 mg/kg/24 h). Indications: serum iron >90 µmol/L, severe symptoms / metabolic acidosis. Stop when symptoms and acidosis resolve and 'vin rosé' urine clears.

References: Murray's Toxicology Handbook 4e Ch on Iron • Australian Poisons Information Centre 13 11 26

Question 12 | Slow-Release Potassium Overdose (14 marks)

A 68-year-old woman presents 2 hours after ingesting 120 × 600 mg slow-release potassium tablets. She is vomiting, GCS 14. VBG: pH 7.32, lactate 5, K^+ 6.1, creatinine 110. ECG shows peaked T waves.

a) List 4 components of the risk assessment. (4 marks)

- Total potassium ingested ($120 \times 600 \text{ mg} = 72 \text{ g KCl} \approx 960 \text{ mmol K}^+$) — potentially lethal **[core]**
- SR formulation — ongoing absorption over hours; bezoar risk
- Pre-existing renal function (Cr 110 — borderline reduces excretion)
- Co-ingestion (ACEi / ARB / spironolactone exacerbate)
- Current K^+ 6.1 + ECG changes = significant hyperkalaemia trajectory

b) Name 2 specific toxicological interventions for this ingestion. (2 marks)

- Whole-bowel irrigation with polyethylene glycol (PEG) 1500–2000 mL/h via NG until effluent clear — for SR preparation tablet bezoar
- Endoscopy — direct removal if bezoar / obstruction; consider gastrostomy in massive ingestion

c) List 1 advantage and 1 disadvantage of each intervention. (4 marks)

Method	Advantage	Disadvantage
Whole-bowel irrigation	Non-invasive; effective for SR tablets	Slow; requires NG tube and cooperative patient; aspiration risk
Endoscopy	Direct visualisation and removal of bezoar	Invasive; requires sedation; not all hospitals 24/7

d) List 4 other management priorities. (4 marks)

- Treat hyperkalaemia: calcium gluconate 10 mL 10% IV; insulin 10 U + 50 mL 50% dextrose IV; salbutamol 10–20 mg neb; bicarbonate if acidotic **[core]**
- Continuous cardiac monitoring; serial 12-lead ECGs
- Consider urgent haemodialysis — definitive treatment for severe hyperkalaemia + ongoing absorption
- IV fluids — restore renal perfusion; cease K^+ -retaining drugs
- Antiemetic; airway protection if vomiting and GCS falling
- Toxicology consultation

References: Murray's Toxicology Handbook 4e Ch on Potassium • Tintinalli's Emergency Medicine 9e Ch on electrolytes • Australian Poisons Information Centre 13 11 26

Question 13 | Local Anaesthetic Systemic Toxicity (Bier's) (12 marks)

A 28-year-old man is having reduction of a fractured radius under Bier's block with 40 mL 0.5% prilocaine. Fifteen minutes in he becomes cyanotic, SpO₂ 85% on supplemental O₂, complaining of dyspnoea.

a) List 3 differential diagnoses for his deterioration. (3 marks)

- **Methaemoglobinaemia** (prilocaine — major ortho-toluidine metabolite) **[core]**
- Local anaesthetic systemic toxicity (LAST) — early CNS or cardiotoxic features
- Tourniquet failure / premature release with systemic absorption
- Anaphylaxis to local anaesthetic / additive
- Pulmonary embolism / aspiration / pneumothorax

b) List 2 investigations with justification. (2 marks)

Investigation	Justification
Co-oximetry / arterial blood gas with MetHb	Measures methaemoglobin directly; pulse oximeter saturates ~85% in MetHb
12-lead ECG	LAST-related arrhythmia; QRS widening, AV block

c) List 3 treatment options for the most likely diagnosis. (3 marks)

- **Methylene blue 1–2 mg/kg IV over 5 min** (repeat once if needed; max 7 mg/kg). Avoid in G6PD deficiency. **[core]**
- High-flow O₂; supportive care
- Cease offending agent (release tourniquet)
- Ascorbic acid 1–2 g IV (alternative if methylene blue contraindicated)
- Exchange transfusion / hyperbaric O₂ for refractory cases

d) List 4 contraindications to Bier's block. (4 marks)

- Severe hypertension / inability to tolerate tourniquet ischaemia
- Sickle cell disease (vaso-occlusive risk)
- Local infection at limb / systemic sepsis
- Open fracture / vascular compromise / limb ischaemia
- Allergy to local anaesthetic agent
- Patient unable to cooperate (paediatric, intellectual disability)
- Crush injury (rhabdomyolysis with reperfusion)

References: ANZCA / AAGBI Local Anaesthetic Systemic Toxicity guideline • Murray's Toxicology Handbook 4e Ch on local anaesthetics • ANZCA Bier's Block guidance

Question 14 | Paediatric LAST (16 marks)

An 8-year-old boy (30 kg) presents with a midshaft femur fracture. The senior registrar performs a US-guided femoral nerve block using 20 mL of 0.75% ropivacaine. The patient develops severe LAST with seizures and ventricular arrhythmia.

a) Calculate the maximum safe dose of ropivacaine for a 30 kg child. (1 mark)

Maximum dose ropivacaine = **3 mg/kg** (with adrenaline). $30 \text{ kg} \times 3 \text{ mg/kg} = \mathbf{90 \text{ mg}}$. The administered dose ($20 \text{ mL} \times 7.5 \text{ mg/mL} = 150 \text{ mg}$) **exceeded** the safe limit.

b) List 6 clinical features of LAST. (6 marks)

Phase	Features
Early (CNS excitation)	Perioral paraesthesia, tinnitus, metallic taste, anxiety, dysarthria
Early (CNS excitation)	Tremor, muscle twitching, restlessness
Severe — CNS	Seizures (generalised tonic-clonic)
Severe — CNS	CNS depression, coma, respiratory arrest
Severe — Cardiac	Bradycardia, AV block, asystole
Severe — Cardiac	Wide-complex tachycardia, ventricular fibrillation

c) List 5 treatment options for severe LAST with reason for each. (5 marks)

Treatment	Reason
Stop injection; call for help; airway / 100% O ₂	Cease ongoing absorption; oxygenation reduces myocardial irritability
20% Lipid Emulsion (Intralipid) bolus 1.5 mL/kg, then 0.25 mL/kg/min infusion (max 12 mL/kg total)	Specific antidote for LAST — 'lipid sink' removes lipophilic LA from cardiac tissue
Benzodiazepine for seizures (midazolam 0.1 mg/kg IV)	Cease seizure activity; reduce metabolic / cardiac demand
AVOID standard ACLS drugs (adrenaline, vasopressin, lidocaine, β -blockers, CCBs); if adrenaline used → small doses ($\leq 1 \text{ mcg/kg}$)	Standard doses worsen LAST cardiac toxicity per ANZCA / ASRA guidance
VA-ECMO / cardiopulmonary bypass for refractory cardiac arrest	Buys time for lipid sink redistribution

d) List 4 measures to improve safety of nerve blocks. (4 marks)

- Dose check — calculate maximum mg/kg before drawing up; verbal cross-check with assistant
- Use lowest effective concentration and volume; aspirate every 3–5 mL; inject in fractional doses
- Ultrasound guidance; nerve stimulator confirmation; intermittent aspiration
- 20% Intralipid kit immediately available; LAST poster / checklist on wall
- Department training and simulation; mandatory monitoring (ECG, NIBP, SpO₂) for all major blocks; trained second person present

References: ANZCA / AAGBI Local Anaesthetic Systemic Toxicity guideline (current) • ASRA Practice Advisory on LAST 2020

Question 15 | Chronic Lithium Toxicity (14 marks)

A 50-year-old female with bipolar disorder on long-term lithium presents with confusion and tremor. She has had a recent infection with reduced oral intake. Lithium level 3.1 mmol/L; creatinine 180 μ mol/L.

a) State the volume of distribution of lithium. (1 mark)

0.7–0.9 L/kg — small Vd; distributes in total body water; no protein binding; excreted unchanged renally — amenable to haemodialysis.

b) List 6 signs / symptoms of chronic lithium toxicity. (6 marks)

- Coarse tremor, ataxia, dysarthria
- Hyperreflexia, clonus, myoclonus
- Confusion, agitation, drowsiness, seizures
- Nephrogenic diabetes insipidus (polyuria, hypernatraemia)
- Renal failure (acute on chronic)
- Cardiac: T-wave inversion, prolonged QT, AV block
- Thyroid dysfunction (hypothyroidism)
- GI: nausea, diarrhoea (early)

c) List 5 principles of management. (5 marks)

- Cease lithium; cease NSAIDs / ACEi / diuretics potentiating toxicity [**core**]
- IV crystalloid 0.9% Saline at 1.5–2 $\times$ maintenance to enhance renal excretion (caution if SIADH / renal failure) [**core**]
- Treat seizures (benzodiazepines), ICU monitoring
- **Haemodialysis** — indicated for: severe symptoms / neurological features, renal failure, level >4 mmol/L acute or >2.5 chronic with symptoms
- Continuous monitoring; serial lithium levels post-dialysis (rebound from intracellular compartment)
- Treat precipitating factor (sepsis, dehydration, drug interaction)

d) List 2 major differences between acute and chronic lithium toxicity. (2 marks)

Acute	Chronic
Predominantly GI symptoms (nausea, vomiting, diarrhoea)	Predominantly neurological symptoms (tremor, ataxia, confusion, seizures)
Severity correlates poorly with serum level (intracellular distribution slow)	Severity correlates with serum level (steady-state tissue concentration)

References: Murray's Toxicology Handbook 4e Ch on Lithium • Tintinalli's Emergency Medicine 9e Ch 191

Question 16 | Methaemoglobinaemia (10 marks)

A 21-year-old man at a music festival is brought in complaining of dyspnoea and chest pain. His lips and oral mucosa appear cyanosed (chocolate-brown). T 36.2, P 121, BP 130/88, RR 34, SpO₂ 85% on 10 L O₂.

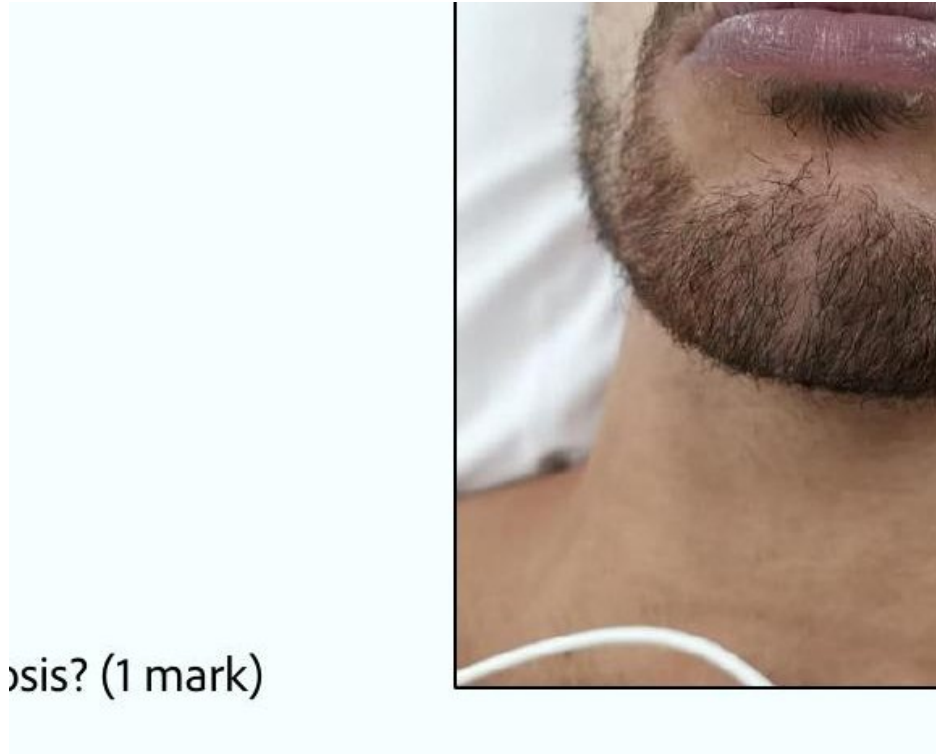


PHOTO — Clinical photo — cyanotic lips

a) State the most likely diagnosis. (1 mark)

Methaemoglobinaemia — chocolate-brown cyanosis unresponsive to supplemental O₂, with fixed SpO₂ ~85%.

b) State the pathogenesis. (2 marks)

Oxidation of haemoglobin iron from Fe²⁺ (ferrous) to Fe³⁺ (ferric) creates methaemoglobin, which cannot bind O₂. This shifts the O₂-Hb dissociation curve LEFT, reducing tissue oxygen delivery. NADH-cytochrome b₅ reductase normally reduces small amounts; oxidative stress or congenital deficiency overwhelms this.

c) Name 4 agents that can cause methaemoglobinaemia. (4 marks)

- Sodium nitrite (recreational use — 'Rush'; food preservative)
- Local anaesthetics — prilocaine, benzocaine, lidocaine
- Dapsone, sulfa drugs (sulfasalazine, sulfamethoxazole)
- Antimalarials — primaquine, chloroquine
- Industrial — aniline dyes, nitrobenzene
- Inhaled NO at high concentration

d) Outline management priorities. (3 marks)

- 100% O₂ via NRB; supportive ABC; remove ongoing exposure **[core]**
- **Methylene blue 1–2 mg/kg IV over 5 min** (0.1–0.2 mL/kg of 1% solution); repeat in 30–60 min if MetHb >30%. Avoid in G6PD deficiency (haemolysis) **[core]**

- Co-oximetry to confirm and trend MetHb level
- Ascorbic acid 1–2 g IV alternative if methylene blue contraindicated
- Exchange transfusion / hyperbaric O₂ for refractory or G6PD-deficient cases

References: *Murray's Toxicology Handbook 4e Ch on Methaemoglobinaemia* • *Tintinalli's Emergency Medicine 9e Ch on environmental and chemical toxicology*

Question 17 | Methamphetamine — Severe Toxicity (17 marks)

A 28-year-old man with known methamphetamine use is brought in pre-sedated with droperidol. T 38.4, HR 128, BP 158/80, GCS 9 (E2 V2 M5), Na^+ 116.

a) List 5 possible causes of his presentation. (5 marks)

- Acute methamphetamine intoxication with sympathomimetic toxidrome
- Serotonin syndrome (if SSRI co-ingestion)
- Sedation effect (droperidol) layered on intoxication
- Hyponatraemia (water intoxication common in MDMA / amph use) → cerebral oedema
- Intracranial pathology (haemorrhage, ischaemic stroke, trauma from collapse)
- Sepsis / meningitis
- Hyperthermic crisis / NMS

b) List 5 investigations and reasoning. (5 marks)

Investigation	Reasoning
VBG, electrolytes, lactate	Confirm hyponatraemia, acidosis, lactate from sympathomimetic / agitation
Bedside glucose	Hypoglycaemia as alternate cause of ↓GCS
12-lead ECG and troponin	Sympathomimetic-induced ACS / arrhythmia; QT effects
CT head (non-contrast)	Exclude intracranial haemorrhage, stroke, trauma from collapse
Creatine kinase	Rhabdomyolysis from agitation, hyperthermia
Urine drug screen	Confirm exposure; identify co-ingestants

c) List 4 steps in immediate management of his tonic-clonic seizure (drug doses). (4 marks)

- Airway: position, suction; high-flow O_2 ; low threshold for intubation if persistent ↓GCS / aspiration [**core**]
- **Midazolam 0.1–0.2 mg/kg IV** (5–10 mg) — first-line; repeat after 5 min if seizure continues [**core**]
- Levetiracetam 60 mg/kg IV (max 4500 mg) if seizures persist or recurrent; or sodium valproate 40 mg/kg IV
- Treat hyperthermia: external cooling; aggressive cooling if $T > 39.5^\circ C$ — paralyse and ventilate; consider dantrolene 1 mg/kg IV
- Correct hyponatraemia cautiously (see (d))

d) Outline the initial management of hyponatraemia (Na^+ 116) and 24-h plan. (3 marks)

Acute symptomatic hyponatraemia with seizures = emergency. Hypertonic saline 3% 100–150 mL IV bolus over 10 min; repeat × 2 if seizures persist. Aim to raise Na^+ by 4–6 mmol/L acutely to abort seizures.

24-hour limit: ≤8 mmol/L rise in chronic; ≤10 mmol/L in acute (avoid osmotic demyelination). After acute correction, fluid restrict and monitor every 2 hours. ICU admission; senior endocrine / nephrology input.

References: Murray's Toxicology Handbook 4e Ch on Sympathomimetics • Tintinalli's Emergency Medicine 9e Ch 184 • Australian National Drug & Alcohol Research Centre

Question 18 | Paracetamol Overdose — Risk Assessment (12 marks)

A 21-year-old woman is brought to ED after an intentional paracetamol overdose. The exact dose and timing are unclear from history.

a) List 4 key history factors influencing risk assessment. (4 marks)

- Total dose (mg/kg) — ≥ 200 mg/kg or ≥ 10 g (whichever less) is the threshold for risk **[core]**
- Time since ingestion — guides nomogram use, NAC timing **[core]**
- Single vs staggered ingestion (over >8 h — nomogram unreliable)
- Formulation — immediate-release vs slow-release
- Risk modifiers — chronic alcohol, malnutrition, CYP inducers (rifampicin, phenytoin, isoniazid)
- Prior liver disease
- Co-ingestants (especially anticholinergics, opioids — delay gastric emptying)

b) List 3 indications for activated charcoal in this patient. (3 marks)

- Massive ingestion (≥ 30 g paracetamol or ≥ 500 mg/kg) — give within 4 hours
- Slow-release paracetamol — extended absorption window; give up to 4 hours post-ingestion
- Co-ingestion of an agent that will benefit (TCA, carbamazepine, theophylline)

c) List 5 King's College criteria for liver-transplant referral in paracetamol-induced ALF. (5 marks)

- Arterial pH <7.30 (or $H^+ >50$ nmol/L) after resuscitation **[core]**
- OR all 3 of: INR >6.5 , creatinine >300 $\mu\text{mol/L}$, grade 3–4 encephalopathy **[core]**
- Lactate >3.5 mmol/L 4 h after fluid resuscitation, OR >3.0 mmol/L at 12 h
- Hypoglycaemia not responding to dextrose
- Coagulopathy worsening despite NAC
- Hepatic encephalopathy

References: Murray's Toxicology Handbook 4e Ch on Paracetamol • Australasian Paracetamol Poisoning Statement (Daly et al 2024 update) • Australian Poisons Information Centre 13 11 26

Question 19 | Paediatric Paracetamol — Liquid (12 marks)

A 3-year-old boy (14 kg) is brought in by his mother 30 minutes after she found him with an empty 200 mL bottle of paracetamol liquid containing 10 g total (originally three-quarters full = 7.5 g).

a) State your approach to decontamination. (1 mark)

Liquid paracetamol is rapidly absorbed (peak 30 min); decontamination is generally NOT useful at 30 min post-ingestion. Activated charcoal is rarely indicated for liquid paracetamol unless massive co-ingestion.

b) State the threshold paracetamol dose (mg/kg) at which a level should be measured. (1 mark)

≥200 mg/kg (or ≥10 g) in a single acute ingestion. Calculated dose: 7.5 g / 14 kg = 535 mg/kg — well above threshold.

c) When (post-ingestion) is the paracetamol level checked? (1 mark)

≥4 hours post-ingestion (or 2 h post-ingestion if liquid IR — earlier sampling acceptable). Plot on the Australian paracetamol nomogram.

d) List 4 medications that may be life-threatening if a small child ingests 1–2 tablets ('one-pill killers'). (4 marks)

- Calcium channel blockers (verapamil, diltiazem)
- Tricyclic antidepressants (amitriptyline)
- Opioids (methadone, buprenorphine, oxycodone)
- Sulfonylureas (glibenclamide, glipizide)
- Antimalarials (chloroquine, quinine)
- Theophylline
- Camphor / eucalyptus / tea tree oils
- Clonidine
- Beta-blockers (propranolol)

e) List 5 immediate steps if NAC infusion is followed by anaphylactoid reaction with flushing and wheeze. (5 marks)

- Stop the NAC infusion immediately [**core**]
- Maintain airway / 100% O₂; salbutamol neb
- IV fluids if hypotensive
- Antihistamine (promethazine 0.25 mg/kg IV; max 12.5 mg) and / or hydrocortisone 4 mg/kg IV
- If severe / anaphylactic: adrenaline 10 mcg/kg IM (1:1000)
- Once symptoms settle, restart NAC at half rate; usually tolerated. Continue full course.

References: Murray's Toxicology Handbook 4e Ch on Paracetamol • Daly FFS et al. Paracetamol poisoning in Australia and New Zealand. MJA 2024 • Australian Poisons Information Centre 13 11 26

Question 20 | Slow-Release Paracetamol Overdose (10 marks)

A 30-year-old man presents 4 hours after intentional overdose of slow-release paracetamol (Panadol Osteo). His 4-h paracetamol level is plotted on the nomogram and is above the treatment line.

a) List 2 specific indications for activated charcoal in SR paracetamol. (2 marks)

- Massive ingestion (≥ 30 g or ≥ 500 mg/kg) up to 4 hours post-ingestion (extended window for SR formulation)
- Massive ingestion with co-ingestant that delays gastric emptying (anticholinergic, opioid)

b) List 3 criteria for cessation of NAC. (3 marks)

- Total course completed (21 h IV) AND ALT not rising AND paracetamol level < 10 mg/L ($66 \mu\text{mol/L}$) [core]
- INR < 2.0 and falling
- Patient asymptomatic with normal LFTs / coagulation
- Repeat 4-h paracetamol level after SR formulation as absorption continues — extended NAC may be needed

c) List 5 King's College criteria for liver transplant referral. (5 marks)

- Arterial pH < 7.30 after resuscitation
- Lactate > 3.0 mmol/L at 12 h despite NAC and resuscitation
- INR > 6.5
- Creatinine $> 300 \mu\text{mol/L}$
- Grade 3–4 hepatic encephalopathy
- Hypoglycaemia unresponsive to dextrose

References: Murray's Toxicology Handbook 4e Ch on Paracetamol • Australasian SR paracetamol management 2020 update

Question 21 | Polypharmacy Overdose — Activated Charcoal (19 marks)

A 30-year-old (90 kg) woman presents having taken Panadeine Forte, carbamazepine and temazepam. She is stable initially. Subsequently she becomes drowsy, tachycardic (120 bpm) and hypotensive (85/43 mmHg). Carbamazepine ingested = 9 g.

a) State the dose of activated charcoal you would administer. (1 mark)

1 g/kg PO / NG (typical adult dose 50 g). Considered if airway is protected and ingestion is recent (<1–2 h for IR; <4 h for SR / massive).

b) List 5 general contraindications to activated charcoal. (5 marks)

- Reduced level of consciousness without protected airway [core]
- Caustic / corrosive ingestion (alkali / acid)
- Hydrocarbons (aspiration risk)
- Substance not adsorbed (alcohols, lithium, iron, heavy metals)
- Bowel obstruction / perforation; ileus
- Patient refusal / unable to cooperate

c) List 4 investigations. (4 marks)

- 12-lead ECG (Na-channel blockade — wide QRS, prolonged QT)
- VBG with electrolytes and lactate
- Paracetamol level (4 h; staggered ingestion)
- Carbamazepine level (peak 2–8 h IR; later SR / massive)
- Bedside glucose; UEC; LFTs

d) List 5 clinical features of significant carbamazepine toxicity. (5 marks)

- CNS depression: drowsiness, ataxia, dysarthria, coma
- Anticholinergic features: dry mouth, urinary retention, mydriasis, tachycardia
- Cardiotoxicity: wide QRS, AV block, hypotension
- Seizures (especially in massive ingestion)
- Hyponatraemia (SIADH — chronic)
- Movement disorders (dyskinesia, nystagmus, myoclonus)

e) List 4 differential causes for tonic-clonic seizure in this context. (4 marks)

- Hypoxia / airway obstruction
- Hypoglycaemia (BSL bedside)
- Hyponatraemia (carbamazepine SIADH)
- Other ingestion (TCA, theophylline, isoniazid, amphetamine)
- Intracranial pathology (head injury from collapse)
- Withdrawal seizure

References: Murray's Toxicology Handbook 4e Ch on Carbamazepine and AC • Australian Poisons Information Centre 13 11 26

Question 22 | Polypharmacy + Propranolol Overdose (16 marks)

A 54-year-old woman presents with apparent alcohol intoxication. 2 hours later she is increasingly drowsy: GCS 9, BP 102/64, HR 75. ECG shows wide QRS with right axis deviation. She had access to her partner's propranolol.



ECG — 12-lead ECG

a) List 5 differential diagnoses for her deteriorating consciousness. (5 marks)

- Drug overdose (β -blocker, TCA, sedative, opioid)
- Hypoglycaemia
- Hypoxia / hypercapnia
- Intracranial pathology (head injury from collapse, ICH)
- Hyponatraemia / metabolic derangement
- Sepsis / meningitis
- Postictal state

b) List 3 relevant ECG features in propranolol toxicity. (3 marks)

- Sinus bradycardia / junctional escape
- Wide QRS (Na-channel blocking effect of propranolol)
- Prolonged PR (1° AV block) progressing to higher-degree block
- Right axis deviation / terminal R wave in aVR (Na-channel blockade)
- Long QT possible

c) List 4 immediate life-threats in propranolol overdose. Name a specific treatment for each. (8 marks)

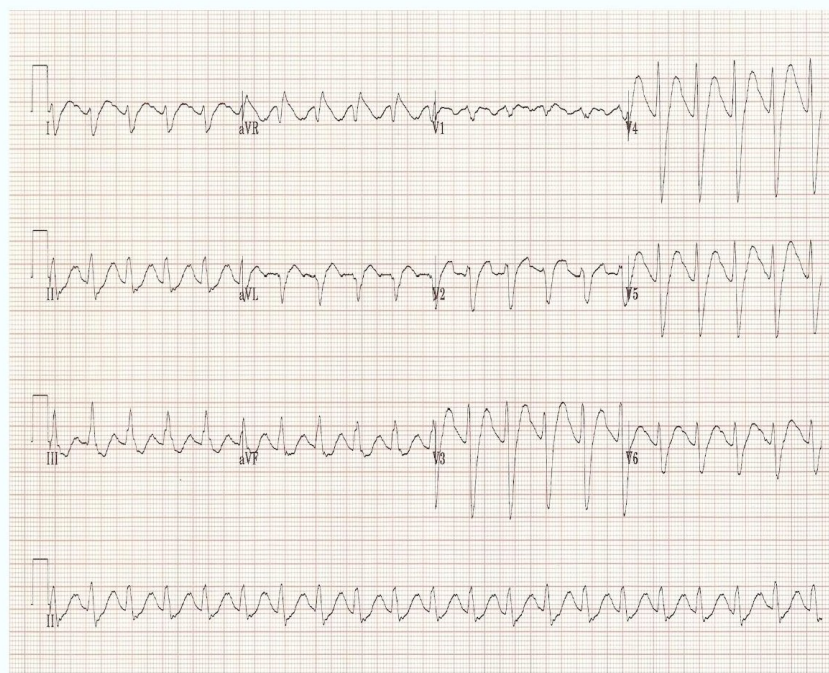
Life-threat	Specific treatment
Bradycardia / hypotension	Glucagon 5–10 mg IV bolus, then 2–10 mg/h infusion (bypasses β -receptor — activates cAMP); atropine, fluids
Refractory cardiogenic shock	High-dose insulin euglycaemic therapy (1 U/kg bolus then 0.5–1 U/kg/h)
Wide-complex arrhythmia / Na-channel blockade	Sodium bicarbonate 1–2 mmol/kg IV bolus

Life-threat	Specific treatment
Seizures (propranolol — Na-channel + lipophilic CNS penetration)	Benzodiazepines (midazolam 0.1 mg/kg IV); intubation; lipid emulsion 1.5 mL/kg bolus, then 0.25 mL/kg/min for refractory cardiotoxicity

References: Murray's Toxicology Handbook 4e Ch on β -blockers • Australian Poisons Information Centre 13 11 26

Question 23 | Polypharmacy Overdose — TCA-like ECG (19 marks)

A 34-year-old man is brought to ED after intentional polypharmacy overdose. He is unconscious, GCS 8, RR 8 with obstructed pattern, SpO₂ 90% on room air.



ECG — 12-lead ECG

a) List 3 common conditions and their treatments to address in resuscitation post-overdose. (6 marks)

Condition	Treatment
Hypoxia / airway compromise	Airway management (head-tilt, jaw-thrust, suction); 100% O ₂ ; intubation if GCS ≤8 / inadequate ventilation
Hypoglycaemia	Bedside glucose; if <3.5 mmol/L → 50 mL 50% dextrose IV (or 2 mL/kg 25% in children)
Hypotension / shock	IV access × 2; crystalloid bolus 10–20 mL/kg; vasopressor (noradrenaline) if refractory

b) List 3 initial interventions for the low SpO₂. (3 marks)

- Reposition airway (head-tilt, jaw-thrust); suction; oropharyngeal airway
- 100% O₂ via NRB; bag-mask ventilation if inadequate
- Endotracheal intubation given GCS 8 with obstructed breathing — RSI with appropriate haemodynamic precautions

c) Analyse the ECG (assume wide QRS, terminal R in aVR, long QT). Interpret. (4 marks)

Sodium-channel blockade pattern, classically tricyclic antidepressant overdose. Features: QRS >100 ms; rightward terminal axis with prominent R wave (>3 mm) in aVR; long QT; sinus tachycardia. Differentials include propranolol, carbamazepine, cocaine, hydroxychloroquine, flecainide.

d) List 6 management priorities given GCS 4, BP 75/60, HR 100, SpO₂ 96% on Hudson. (6 marks)

- RSI and intubation — protect airway and facilitate ventilation; use ketamine or etomidate for haemodynamic stability **[core]**
- **Sodium bicarbonate 1–2 mmol/kg IV** bolus for QRS >100 ms; repeat to maintain pH 7.45–7.55 and narrow QRS **[core]**
- IV fluid resuscitation (crystalloid 20 mL/kg)
- Vasopressor: noradrenaline 0.05–0.5 mcg/kg/min for refractory hypotension
- Treat seizures with benzodiazepines (avoid phenytoin — Na-channel blocker)
- Lipid emulsion 1.5 mL/kg bolus then 0.25 mL/kg/min for refractory cardiotoxicity
- Consider VA-ECMO in cardiac arrest from massive TCA / Na-channel blocker

References: *Murray's Toxicology Handbook 4e Ch on TCAs* • *Tintinalli's Emergency Medicine 9e Ch 191*

Question 24 | Adolescent Polypharmacy — Anticholinergic / TCA (18 marks)

A 14-year-old (40 kg) presents after taking pill packets from her parents' medicine cabinet (all dispensed yesterday): 20 × Citalopram 20 mg, 20 × Quetiapine 300 mg, 50 × Amitriptyline 25 mg, 24 × Paracetamol/Codeine 500/30 mg. Initial vitals: HR 105, BP 105/60, GCS 13.

a) List 4 key aspects of risk assessment. (4 marks)

- Amitriptyline 1.25 g (~31 mg/kg) — >10 mg/kg is potentially lethal; sodium-channel blockade and seizures expected **[core]**
- Quetiapine 6 g (150 mg/kg) — significant sedation, tachycardia, prolonged QT
- Citalopram 400 mg (10 mg/kg) — QT prolongation, serotonin syndrome, seizures (highest pro-convulsant SSRI)
- Paracetamol 12 g (300 mg/kg) — >200 mg/kg threshold → NAC required
- Codeine 720 mg + adolescent → CNS / respiratory depression risk; opioid-naïve

b) List 3 key investigations and reasoning. (6 marks)

Investigation	Reasoning
12-lead ECG	TCA Na-channel blockade (wide QRS, R in aVR); QTc (citalopram, quetiapine); arrhythmia risk
Paracetamol level at 4 h	Confirm hepatotoxic exposure; plot on nomogram; duration of NAC
VBG + electrolytes + lactate	Acid-base, hypokalaemia / hypoMg ²⁺ from vomiting; lactate from systemic toxicity

c) Name the toxidrome to assess for. (1 mark)

Anticholinergic / TCA toxidrome (with overlapping serotonergic features from citalopram).

d) List 4 clinical features of the anticholinergic toxidrome. (4 marks)

- Tachycardia, hypertension
- Mydriasis with poor accommodation
- Dry skin and mucous membranes
- Hyperthermia
- Urinary retention; absent bowel sounds
- Confusion, agitation, hallucinations ('mad as a hatter')
- Seizures

e) List 4 key treatments for deterioration (GCS 9, BP 80/40). (4 marks)

- RSI / airway protection — ketamine 1 mg/kg + rocuronium 1.2 mg/kg **[core]**
- **Sodium bicarbonate 1–2 mmol/kg IV** bolus for QRS >100 ms; repeat to pH 7.45–7.55 **[core]**
- IV fluid 20 mL/kg crystalloid; noradrenaline if refractory hypotension
- Treat seizures with midazolam 0.1–0.2 mg/kg IV; avoid phenytoin
- Commence NAC 21-h regimen for paracetamol
- Naloxone 0.04–0.4 mg titrated for opioid component if respiratory depression

References: Murray's Toxicology Handbook 4e Ch on TCAs • Australian Poisons Information Centre 13 11 26 • Royal Children's Hospital Melbourne — Clinical Practice Guideline

Question 25 | Aeromedical Transfer — Polypharmacy (12 marks)

A 42-year-old woman requires aeromedical retrieval from a rural ED to a tertiary centre 400 km away (75-min flight). She ingested paracetamol 7.5 g, olanzapine 250 mg, diazepam 25 mg 90 minutes ago. Vitals: SpO₂ 92% RA, BP 100/60, HR 110, RR 22, GCS 10.

a) List 4 management decisions for safe transport with justification for each. (8 marks)

Decision	Justification
Endotracheal intubation prior to flight	GCS 10 with declining trajectory; deteriorating during transit unsafe — pre-emptive airway control
Commence IV NAC infusion before transfer	Paracetamol level pending; NAC effective within 8 h; transfer time may exceed window
IV access × 2; arterial line if available	Continuous BP monitoring during flight; vibration artefact on NIBP
Continuous capnography, ECG, SpO ₂ , temperature	Capnography most reliable in flight; cardiac monitoring for olanzapine QT effects
Pre-position drugs and equipment	Limited space; can't restock mid-flight — anticipate deterioration drugs (sedation, paralysis, vasopressor)

b) Complete the table for dose-related olanzapine effects. (4 marks)

Dose	Clinical features
<40 mg	Sedation, mild anticholinergic; usually mild course
40–100 mg	Significant sedation, miosis, tachycardia, mild hypotension
100–200 mg	Coma, respiratory depression, hypotension; QT prolongation
>200 mg	Severe CNS depression; intubation required; cardiotoxicity

References: Murray's Toxicology Handbook 4e Ch on Antipsychotics • Australian Poisons Information Centre 13 11 26 • RFDS / aeromedical retrieval guidelines

Question 26 | Quetiapine Overdose with Hyperkalaemia (12 marks)

A 69-year-old man is found unconscious. He is intubated on arrival. Bloods: Na 136, K 8.3, Cl 101, HCO_3^- 16, Urea 15, Cr 285, Glucose 7.3. Background: quetiapine and diazepam overdose.

a) List 3 abnormalities and likely causes. (6 marks)

Abnormality	Likely cause
Hyperkalaemia (8.3)	Acute kidney injury; rhabdomyolysis from prolonged immobilisation; metabolic acidosis
Acute kidney injury (Cr 285, Urea 15)	Pre-renal hypoperfusion + rhabdomyolysis pigment nephropathy
Metabolic acidosis (HCO_3^- 16)	Lactic acidosis from hypoperfusion / shock

b) State the most likely clinical scenario. (2 marks)

Prolonged immobilisation post-overdose with rhabdomyolysis and acute kidney injury. Quetiapine + diazepam → deep coma → pressure-related muscle damage → CK rise → AKI → hyperkalaemia and metabolic acidosis.

c) List 4 management steps for hyperkalaemia with doses. (4 marks)

- Calcium gluconate 10 mL 10% IV over 5 min (membrane stabilisation) — repeat if persistent ECG changes **[core]**
- Insulin 10 U IV + 50 mL 50% dextrose (intracellular shift)
- Salbutamol 10–20 mg neb
- Sodium bicarbonate 50–100 mEq IV (1–2 mmol/kg) if acidotic
- Urgent haemodialysis — definitive in severe hyperkalaemia with AKI
- Aggressive IV fluids (crystalloid 20 mL/kg) for rhabdomyolysis; treat the underlying cause

References: Murray's Toxicology Handbook 4e Ch on Antipsychotics • Tintinalli's Emergency Medicine 9e Ch on Rhabdomyolysis • eTG Toxicology

Question 27 | Salicylate Toxicity (10 marks)

A 42-year-old man presents 4 hours after ingesting an unknown quantity of aspirin. He is anxious, tachypnoeic, diaphoretic and complains of tinnitus.

a) List 5 clinical features of salicylate toxicity. (5 marks)

- Tinnitus / hearing loss
- Tachypnoea, hyperventilation (direct respiratory centre stimulation)
- Hyperthermia
- Mixed acid-base: respiratory alkalosis + anion-gap metabolic acidosis
- Vomiting; GI bleeding
- Confusion, lethargy, seizures (severe / late)
- Pulmonary oedema (non-cardiogenic)
- Hypoglycaemia (especially in children)

b) Complete the table for management options. (5 marks)

Management	Indication	Dosing
Activated charcoal	Recent ingestion (<2–4 h, especially massive); consider multi-dose for SR / large bezoar	1 g/kg PO (50 g adult); MDAC 25 g q4h
Urinary alkalinisation	Salicylate level >3.6 mmol/L (50 mg/dL) or symptomatic toxicity; urine pH <7.5	Sodium bicarbonate 1–2 mmol/kg IV bolus, then 150 mmol in 1 L 5% dextrose at 250 mL/h. Target urine pH 7.5–8.0; serum pH <7.55. Replace K ⁺ aggressively
Haemodialysis	Severe toxicity: level >7.2 mmol/L (100 mg/dL); altered conscious state; renal failure; pulmonary oedema; refractory acidosis; failed alkalinisation	Intermittent HD preferred; CVVHDF if unstable

References: Murray's Toxicology Handbook 4e Ch on Salicylates • Tintinalli's Emergency Medicine 9e Ch 196 • Australian Poisons Information Centre 13 11 26

Question 28 | Sodium Nitrite — Methaemoglobinaemia (10 marks)

A 72-year-old man presents after intentional ingestion of sodium nitrite tablets used for cleaning his dentures. Airway patent, lungs clear. SpO₂ 85% regardless of oxygen titration. Lips appear chocolate-brown.

a) List 3 immediate bedside investigations. (3 marks)

- Co-oximetry / arterial blood gas with methaemoglobin level
- 12-lead ECG (associated cardiac toxicity, hypotension)
- Bedside glucose; venous blood gas with lactate
- Visual blood inspection — chocolate-brown blood diagnostic

b) State the unifying diagnosis and 1 underlying condition that increases failure of treatment. (2 marks)

	Answer
Unifying diagnosis	Severe acquired methaemoglobinaemia from sodium nitrite ingestion
Underlying condition	G6PD deficiency — methylene blue can cause haemolysis and is contraindicated

c) State the treatment of choice. (1 mark)

Methylene blue 1–2 mg/kg IV over 5 minutes (0.1–0.2 mL/kg of 1% solution); may repeat once after 30–60 minutes. Total dose <7 mg/kg.

d) Name 1 additional therapy. (1 mark)

- Ascorbic acid 1–2 g IV (cofactor for endogenous reduction; alternative if methylene blue contraindicated)
- Exchange transfusion / hyperbaric O₂ for refractory cases or G6PD deficiency
- 100% O₂ via NRB

e) List 3 toxic exposures that can cause this condition. (3 marks)

- Sodium nitrite (recreational / suicidal; food preservative)
- Local anaesthetics — prilocaine, benzocaine, lidocaine
- Dapsone, sulfa antibiotics
- Antimalarials — primaquine, chloroquine
- Industrial: aniline dyes, nitrobenzene, well-water nitrates
- Inhaled nitric oxide / amyl nitrite ('poppers')

References: Murray's Toxicology Handbook 4e Ch on Methaemoglobinaemia • Tintinalli's Emergency Medicine 9e Ch 195 • Australian Poisons Information Centre 13 11 26

Question 29 | TCA (Amitriptyline) Overdose (14 marks)

A 23-year-old (60 kg) woman presents 1 hour after ingesting an empty box of 50 × 25 mg amitriptyline. She is GCS 15, BP 120/60, HR 110, BSL 6.6. ECG borderline QRS 100 ms.

a) State your initial risk assessment. (3 marks)

Ingested 1.25 g amitriptyline = 21 mg/kg. **>10 mg/kg is potentially lethal**; expect Na-channel blockade, anticholinergic toxidrome, seizures and ventricular arrhythmia. Peak effect 1–6 h post-ingestion. High-risk course expected over the next 4–6 hours.

b) Name 1 key investigation and 2 other investigations, with justification. (6 marks)

Investigation	Justification
12-lead ECG (KEY)	QRS >100 ms = significant Na-channel blockade; QRS >160 ms = high risk of seizures/VT; R wave in aVR >3 mm correlates with toxicity
Paracetamol level	Routine in deliberate self-harm — paracetamol co-ingestion screen
VBG + electrolytes	Acid-base; hypokalaemia / hypoMg ²⁺ exacerbate Na-channel blockade and arrhythmia

c) Following a tonic-clonic seizure (now GCS 3, HR 120, BP 90/50), list 5 immediate management priorities with endpoints. (5 marks)

- Airway: RSI with ketamine 1 mg/kg + rocuronium 1.2 mg/kg; endpoint — secured airway, adequate ventilation **[core]**
- **Sodium bicarbonate 1–2 mmol/kg IV** bolus, repeat as needed; endpoint — QRS narrows to <100 ms, pH 7.45–7.55 **[core]**
- Treat seizures: midazolam 0.1 mg/kg IV (avoid phenytoin); endpoint — seizures controlled
- IV crystalloid 20 mL/kg; vasopressor noradrenaline 0.05–0.5 mcg/kg/min; endpoint — MAP >65
- Lipid emulsion 1.5 mL/kg IV bolus then 0.25 mL/kg/min for refractory cardiotoxicity; endpoint — return of perfusion
- Activated charcoal via NG once airway secured (1 g/kg) if ingestion <2 h

References: Murray's Toxicology Handbook 4e Ch on TCAs • Tintinalli's Emergency Medicine 9e Ch 191 • Australian Poisons Information Centre 13 11 26

Question 30 | Toxidromes — Hyperthermic Patient (16 marks)

A 32-year-old man is brought in agitated and febrile (40°C) after celebrating with friends post-exam. Background: amitriptyline for migraine prophylaxis. HR 140, BP 163/92, diaphoretic, clonic features in lower limbs.

a) List 5 differential diagnoses, each in a different category. (5 marks)

- Toxicological — Serotonin syndrome (TCA + SSRI or stimulant; recent celebration) **[core]**
- Toxicological — Sympathomimetic (cocaine, amphetamine)
- Toxicological — Neuroleptic malignant syndrome (if recent antipsychotic)
- Infective — Sepsis / meningitis (rule out)
- Endocrine — Thyroid storm
- Neurological — Status epilepticus / encephalitis
- Environmental — Heatstroke (especially if outdoor / alcohol)

b) List the 5 components of the Hunter Serotonin Toxicity Criteria. (5 marks)

- Spontaneous clonus **[core]**
- Inducible clonus + agitation OR diaphoresis **[core]**
- Ocular clonus + agitation OR diaphoresis
- Tremor + hyperreflexia
- Hypertonia AND temperature >38°C AND ocular OR inducible clonus

Marker's note: Patient must be on a serotonergic agent. Sensitivity 84%, specificity 97% for serotonin toxicity.

c) Complete the toxidrome features table for the 6 named toxidromes. (6 marks)

Toxidrome	Mental status	Eyes	Pulse	Bowel sounds	Skin / Neuro
Sympathomimetic	Agitated, paranoid	Mydriasis	↑↑	Normal/↑	Diaphoretic; tremor; seizure
Cholinergic	Confused	Miosis	↓ (or ↑)	↑↑ (SLUDGE)	Wet (sweating); fasciculation
Anticholinergic	Confused, agitated	Mydriasis (poor accom.)	↑	↓ / absent	Dry, hot skin; urinary retention
Opioid withdrawal	Anxious, dysphoric	Mydriasis	↑	↑↑	Yawning, lacrimation, piloerection
NMS	Confused / coma	Normal	↑	Normal	Lead-pipe rigidity; hyperthermia
Serotonin syndrome	Agitated, confused	Mydriasis	↑	↑	Clonus, hyperreflexia, hyperthermia, diaphoresis

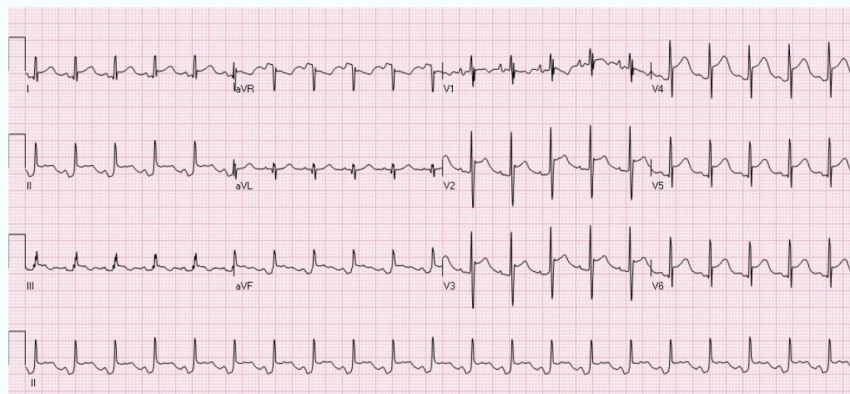
References: Murray's Toxicology Handbook 4e Ch on Toxidromes • Hunter Serotonin Toxicity Criteria (Dunkley et al)

Question 31 | Sympathomimetic — Cocaine / Amphetamine (12 marks)

A 22-year-old man presents after taking 'some pills' at a nightclub. He is diaphoretic, pale, with chest pain. ECG: sinus tachycardia at 120; ST/T-wave abnormalities.

Question 31 (Total 12 Marks)

A 22 year old male is brought to your emergency department following the ingestion of 'some pills' at a nightclub.
He is diaphoretic, pale and complaining of chest pain.



ECG — 12-lead ECG

a) List 3 main ECG features. (3 marks)

- Sinus tachycardia (~120 bpm)
- ST elevation / ischaemic ST changes (cocaine-induced vasospasm or thrombus)
- Q-T prolongation
- Wide QRS (Na-channel blockade by cocaine)
- Ventricular ectopy / arrhythmia

b) Name 2 likely drug classes and example drug. (4 marks)

Class	Drug
Sympathomimetic / cocaine derivatives	Cocaine, methamphetamine, MDMA
Designer stimulants / cathinones	Mephedrone, MDPV (bath salts)

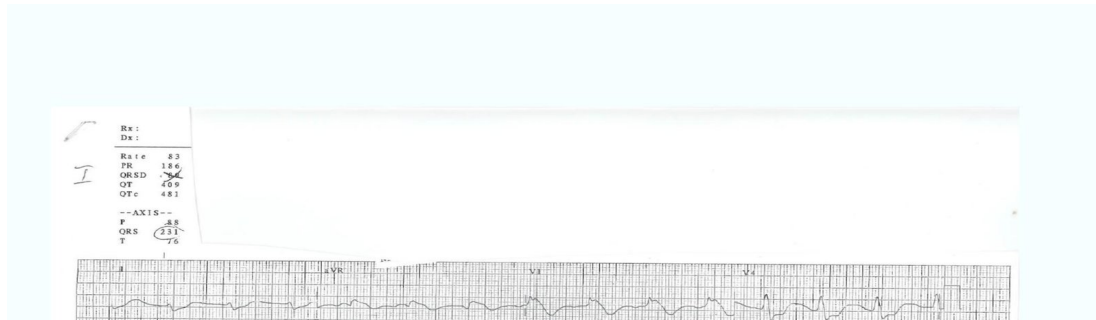
c) Outline cardiovascular management. (5 marks)

- **Benzodiazepine (diazepam 5–10 mg IV)** — first-line for cocaine-induced HTN/tachycardia/agitation; titrate to calm **[core]**
- GTN SL / IV for ongoing chest pain (caution if hypotension)
- Aspirin 300 mg PO if ACS suspected
- **AVOID β -blockers** (unopposed α -vasoconstriction worsens ischaemia / HTN)
- Sodium bicarbonate 1–2 mmol/kg IV if QRS >100 ms
- Active cooling for hyperthermia

References: *Murray's Toxicology Handbook 4e Ch on Cocaine / Sympathomimetics* • *Tintinalli's Emergency Medicine 9e Ch 184*

Question 32 | Unknown Polypharmacy with Wide QRS (14 marks)

A 35-year-old woman is brought to ED unconscious. She had written a suicide note. No empty pill packets found. GCS 8, BP 75/45, SpO₂ 99% on 15 L NRB. ECG shows wide QRS, terminal R wave in aVR, and right axis deviation.



ECG — 12-lead ECG

a) List 3 main ECG abnormalities. (3 marks)

- QRS >100 ms (sodium-channel blockade)
- Terminal R wave >3 mm in aVR (highly suggestive)
- Right axis deviation of terminal QRS
- Long QT
- Sinus tachycardia

b) Name the drug group most likely responsible. (1 mark)

Tricyclic antidepressants (sodium-channel blocking agent group). Differentials: propranolol, carbamazepine, cocaine, hydroxychloroquine, flecainide.

c) List 5 management steps with drugs, doses and endpoints. (10 marks)

- RSI / intubation — ketamine 1 mg/kg + rocuronium 1.2 mg/kg; endpoint — secured airway **[core]**
- Sodium bicarbonate 1–2 mmol/kg IV bolus, repeat to maintain pH 7.45–7.55 and QRS <100 ms **[core]**
- IV crystalloid 20 mL/kg; vasopressor — noradrenaline 0.05–0.5 mcg/kg/min; endpoint — MAP ≥65 mmHg
- Treat seizures with midazolam 0.1 mg/kg IV; AVOID phenytoin (Na-channel blocker)
- Lipid emulsion 1.5 mL/kg IV bolus then 0.25 mL/kg/min for refractory cardiotoxicity
- Continuous monitoring; consider VA-ECMO if cardiac arrest from refractory cardiotoxicity

References: Murray's Toxicology Handbook 4e Ch on TCAs • Tintinalli's Emergency Medicine 9e Ch 191 • Australian Poisons Information Centre 13 11 26

Question 33 | Toxic Alcohol — Ethylene Glycol (18 marks)

A 23-year-old man presents at a rural ED. Family suspects an overdose. BSL normal. VBG: pH 7.34, HCO_3^- 16, BE -6, lactate 9.6, Na 136, Cl 104, K 4.2.

a) State the 2 primary acid-base abnormalities. (2 marks)

- High anion-gap metabolic acidosis (anion gap = $136 - (104 + 16) = 16$; raised)
- Compensatory respiratory alkalosis (pCO_2 31 mmHg)

b) Calculate the expected CO_2 . (1 mark)

Winters' formula: expected $\text{pCO}_2 = (1.5 \times \text{HCO}_3^-) + 8 \pm 2 = (1.5 \times 16) + 8 = \mathbf{32 \text{ mmHg}}$. Measured 31 mmHg = appropriate compensation.

c) List 3 most likely causes of this picture. (3 marks)

- Toxic alcohol ingestion (ethylene glycol, methanol, diethylene glycol)
- Lactic acidosis from another cause (sepsis, cyanide, metformin)
- Diabetic / starvation / alcoholic ketoacidosis
- Salicylate toxicity

d) List 2 features on blood gas that are MOST specific for toxic alcohol. (2 marks)

- Markedly elevated osmolal gap (osmolality measured – calculated $>10 \text{ mOsm/kg}$)
- High anion-gap metabolic acidosis

e) List 4 important investigations with justification. (8 marks)

Investigation	Justification
Serum osmolality (measured)	Calculate osmolal gap; elevated suggests unmeasured osmole (toxic alcohol)
Ethylene glycol level (or methanol / isopropanol)	Definitive — guides fomepizole / dialysis decision
UEC + creatinine + calcium	Hypocalcaemia from oxalate; renal failure from oxalate crystals
Urinalysis — calcium oxalate crystals; UV/Wood's lamp	Calcium oxalate crystals support EG; fluorescein additive in radiator coolant fluoresces under UV

f) List 3 management priorities (with route and dose). (6 marks)

Management	Justification
Fomepizole 15 mg/kg IV loading , then 10 mg/kg q12 h for 4 doses (then 15 mg/kg q12 h); or ethanol infusion if fomepizole unavailable	Inhibits alcohol dehydrogenase, preventing toxic metabolite formation
Sodium bicarbonate 1–2 mmol/kg IV; maintain pH >7.30	Buffer acidosis; reduces oxalate uptake into tissues; protects renal tubules
Urgent haemodialysis	Removes parent alcohol and metabolites; indicated for severe acidosis, renal failure, level $>500 \text{ mg/L}$ (EG)

Management	Justification
IV crystalloid; thiamine 100 mg IV; pyridoxine 50 mg IV (cofactors for safer EG metabolism)	Volume restoration; cofactors shunt EG metabolism to non-toxic glycolate / glycine

References: *Murray's Toxicology Handbook 4e Ch on Toxic Alcohols* • *Tintinalli's Emergency Medicine 9e Ch 184* • *Australian Poisons Information Centre 13 11 26*

Question 34 | Toxic Alcohol — Severe Acidosis (17 marks)

A 33-year-old man presents 4 hours after ingestion of an unknown substance. Vitals: HR 105, BP 142/79, RR 30, SpO₂ 94% on 6 L Hudson, GCS 9. ABG: pH 7.02, pCO₂ 20, pO₂ 85, HCO₃⁻ 8, lactate 8.2, glucose 8, urea 24, Cr 210, osmolality 299, Na 142, K 4.0, Cl 106, Ca 1.9, Mg 0.8.

a) Describe the acid-base disturbances. (2 marks)

Severe high anion-gap metabolic acidosis (anion gap = $142 - (106 + 8) = 28$; severely raised) with **partial respiratory compensation** (pCO₂ 20; expected = $1.5 \times 8 + 8 = 20$ — appropriate).

b) List 3 other abnormalities. State the likely diagnosis with reasoning. (5 marks)

- Acute kidney injury (Cr 210, urea 24)
- Hypocalcaemia (Ca 1.9) and hypomagnesaemia (Mg 0.8)
- Elevated osmolar gap = $299 - \text{calculated } (2 \times 142 + 8 + 24/2.8) = 299 - 300.6 \approx 0$ — paradoxically low/normal (late presentation, parent alcohol metabolised)

Likely diagnosis: ethylene glycol poisoning with established toxicity. Acidosis from glycolate / oxalate; renal failure from oxalate crystals; hypocalcaemia from calcium-oxalate precipitation. Late presentation explains low osmolar gap (parent alcohol already metabolised) but high anion gap and end-organ damage.

c) List 2 other investigations with rationale. (4 marks)

Investigation	Rationale
Ethylene glycol / methanol levels and urine calcium-oxalate crystals; UV fluorescence	Confirm diagnosis; calcium oxalate crystals in urine are pathognomonic
Lactate dehydrogenase, anion gap calculation, serum osmolality (measured)	Confirm metabolic acidosis pattern, exclude alternative causes

d) Following a tonic-clonic seizure (5 min), list 6 management steps. (6 marks)

- Airway / RSI as required; benzodiazepine (midazolam 0.1 mg/kg IV) for seizure **[core]**
- Calcium gluconate 10 mL 10% IV (or calcium chloride 5 mL 10%) for hypocalcaemia and membrane stabilisation **[core]**
- **Fomepizole 15 mg/kg IV loading**, then 10 mg/kg q12 h **[core]**
- Magnesium sulphate 2 g IV; correct other electrolytes
- **Urgent haemodialysis** — definitive treatment (severe acidosis, AKI, neurological features)
- Sodium bicarbonate 1–2 mmol/kg IV to maintain pH >7.30
- Pyridoxine 50 mg IV + thiamine 100 mg IV (cofactors)
- ICU admission and toxicology consultation

References: Murray's Toxicology Handbook 4e Ch on Toxic Alcohols • Tintinalli's Emergency Medicine 9e Ch 184 • Australian Poisons Information Centre 13 11 26

Question 35 | Verapamil SR Overdose (10 marks)

A 32-year-old female presents 6 hours after ingesting 20×240 mg SR verapamil. She is vomiting, listless. GCS 14, HR 40, BP 75/40, BSL 12 mmol/L.

a) State your risk assessment. (1 mark)

Ingested 4.8 g SR verapamil = potentially lethal (>1 g lethal in non-tolerant adult). SR formulation has extended absorption with delayed peak effect at 6–18 h. **Massive ingestion with cardiogenic shock and emerging AV block expected.**

b) State the pathotoxicological mechanisms. (2 marks)

Verapamil (non-dihydropyridine CCB): blocks **L-type calcium channels** in: (1) cardiac myocytes — negative inotropy and chronotropy; (2) AV node — block; (3) vascular smooth muscle — peripheral vasodilation. Also inhibits pancreatic insulin release → hyperglycaemia (metabolic clue to diagnosis).

c) State the significance of BSL 12 mmol/L. (1 mark)

Hyperglycaemia is a marker of severity in CCB toxicity (CCB inhibits insulin release; β -blocker overdose typically does not). Severity correlates with BSL and supports HIET as treatment.

d) List 6 management steps with doses (after deterioration to GCS 9, junctional rhythm 30 bpm). (6 marks)

- Resuscitation: IV access $\times 2$, O_2 , monitoring, defibrillator/pacer to bedside; consider RSI for declining GCS **[core]**
- Calcium chloride 10 mL 10% IV (or calcium gluconate 30 mL 10% IV); repeat to total 3 g **[core]**
- **High-dose insulin euglycaemic therapy**: insulin 1 U/kg IV bolus, then 0.5–1 U/kg/h infusion (titrate up to 10 U/kg/h); glucose infusion to maintain BSL 5–10 mmol/L **[core]**
- IV fluid 10–20 mL/kg crystalloid; vasopressor noradrenaline 0.05–0.5 mcg/kg/min $\pm$ adrenaline
- Atropine 600 mcg IV (likely ineffective in significant CCB toxicity); transcutaneous pacing if bradycardia persists
- Glucagon 5–10 mg IV bolus, then 1–5 mg/h infusion (less effective in CCB than β -blocker)
- Lipid emulsion 1.5 mL/kg IV bolus, then 0.25 mL/kg/min for refractory shock (lipophilic verapamil)
- Whole-bowel irrigation with PEG (SR formulation); VA-ECMO referral if refractory shock; toxicology consultation

References: Murray's Toxicology Handbook 4e Ch on CCB • Tintinalli's Emergency Medicine 9e Ch 189 • Australian Poisons Information Centre 13 11 26 • eTG Toxicology