

FExNet 2026

Collated SAQs: Clinical Chemistry and Acid-Base

ANSWER BOOKLET

Model answers for the 20 clinical chemistry SAQs. References cited at the foot of each question.

Items marked [core] are required for full marks. Calculations independently verified for mathematical accuracy. Drug doses confirmed with eTG Therapeutic Guidelines and ANZICS / Australian Diabetes Society guidelines.

20 questions • 230 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 | Mixed Acid-Base in the Alcoholic with Pancreatitis and Hyperglycaemia (14 marks)

A 41-year-old man with a long history of alcohol use disorder is brought to your emergency department with severe epigastric abdominal pain, intractable vomiting, and progressive lethargy. He last drank alcohol 36 hours ago. On arrival his vital signs are: heart rate 118 beats per minute, blood pressure 100/65 millimetres of mercury, respiratory rate 28 breaths per minute (deep), peripheral oxygen saturation 96 percent on room air, temperature 36.8 degrees Celsius. His arterial blood gas (room air, fraction of inspired oxygen 0.21) and biochemistry are: pH 7.17 (7.35-7.45); pCO₂ 30 mmHg (35-45); pO₂ 98 mmHg; bicarbonate 11 mmol/L (22-32); sodium 117 mmol/L (135-145); potassium 3.5 mmol/L; chloride 64 mmol/L (98-108); glucose 34.2 mmol/L; lactate 8.1 mmol/L (0.5-2.2); ionised calcium 0.74 mmol/L (1.15-1.32); albumin 28 g/L.

a) Calculate the anion gap, expected pCO₂ (Winter's formula), delta ratio, and glucose-corrected sodium AND state the overall acid-base interpretation. (6 marks)

- **Anion gap** = Na – (Cl + HCO₃) = 117 – (64 + 11) = 42 mmol/L (markedly elevated; normal 8-12) [core]
- **Albumin-corrected anion gap** = 42 + 0.25 × (40 – 28) = 42 + 3 = 45 mmol/L (severe HAGMA) [core]
- **Expected pCO₂ (Winter's)**: 1.5 × HCO₃ + 8 = 1.5 × 11 + 8 = 24.5 mmHg (± 2). Actual pCO₂ 30 — HIGHER than expected → co-existing respiratory acidosis (relative under-compensation) [core]
- **Delta ratio** = (AG – 12) / (24 – HCO₃) = (42 – 12) / (24 – 11) = 30 / 13 = 2.31 — >2.0 indicates HAGMA WITH co-existing metabolic alkalosis [core]
- **Corrected sodium for hyperglycaemia**: Na + (glucose – 5.5) × 0.3 = 117 + (34.2 – 5.5) × 0.3 = 117 + 8.6 = ~126 mmol/L (still mildly hyponatraemic after correction) [core]
- **Overall interpretation: TRIPLE acid-base disorder** — high anion-gap metabolic acidosis (HAGMA) + metabolic alkalosis (vomiting / hypochloraemia) + relative respiratory acidosis (inadequate compensation) [core]

b) List 4 contributing causes for the metabolic derangement. (4 marks)

- **Alcoholic ketoacidosis** — beta-hydroxybutyrate predominant; starvation, alcohol metabolism shifts NADH/NAD ratio [core]
- **Diabetic ketoacidosis** — glucose 34.2 supports new or precipitated diabetes; starvation and stress-induced insulinopenia [core]
- **Lactic acidosis** — lactate 8.1 from hypoperfusion (vomiting / dehydration), alcohol-induced lactate generation, thiamine deficiency [core]
- **Acute pancreatitis with metabolic alkalosis component** from vomiting (loss of hydrochloric acid) — explains the hypochloraemia (Cl 64) and elevated delta ratio [core]
- Toxic alcohol ingestion (methanol, ethylene glycol) — consider given alcohol history and severe HAGMA
- Hypocalcaemia — due to acute pancreatitis (saponification of calcium in fat necrosis)

c) State 4 immediate management priorities (drugs, doses, end-points). (4 marks)

- **Intravenous thiamine 300-500 mg IV** BEFORE any glucose-containing fluid (prevents Wernicke's encephalopathy in alcoholic patients) [core]
- **Resuscitation with crystalloid** — 0.9 percent saline 10-20 mL/kg bolus, titrate to MAP >65; transition to 5 percent dextrose with potassium supplementation once perfusion restored [core]
- **Fixed-rate insulin infusion 0.1 unit/kg/h IV** for diabetic component once potassium ≥3.5 mmol/L (PER ANZICS DKA guideline 2023); add 5 percent dextrose when glucose <14 to allow continued insulin [core]
- **Calcium replacement** — calcium gluconate 10% 10-20 mL slow IV (over 10 min); recheck ionised calcium [core]

- Correct hypomagnesaemia (likely): magnesium sulfate 10 mmol IV over 30 min
- Investigate pancreatitis (lipase, computed tomography); tetanus update; broad-spectrum antibiotics if septic; alcohol withdrawal monitoring

References: ANZICS / Australian Diabetes Society DKA guideline 2023 • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 117 • ACEM clinical policies and statements

Question 2 | Severe Combined Metabolic and Respiratory Acidosis (12 marks)

A 17-year-old female is brought to your emergency department by ambulance, found drowsy and agitated by her friends. She was last seen well 4 hours ago. Her Glasgow Coma Score is 8. Her vital signs are: heart rate 130 beats per minute, blood pressure 95/55 millimetres of mercury, respiratory rate 14 breaths per minute (shallow), peripheral oxygen saturation 92 percent on room air, temperature 37.5 degrees Celsius. Her electrocardiogram shows sinus tachycardia with right axis deviation and a QRS duration of 140 milliseconds. Her arterial blood gas (room air) and biochemistry: pH 6.90 (7.35-7.45); pCO₂ 70 mmHg (35-45); pO₂ 60 mmHg; bicarbonate 8 mmol/L (22-32); sodium 136 mmol/L; potassium 4.0 mmol/L; chloride 90 mmol/L; lactate 9 mmol/L; glucose 5.5 mmol/L.

a) Calculate the anion gap, expected pCO₂, AND interpret the acid-base disorder. (4 marks)

- **Anion gap** = $136 - (90 + 8) = 38$ mmol/L (severely elevated) [core]
- **Expected pCO₂ (Winter's):** $1.5 \times 8 + 8 = 20$ mmHg. Actual 70 — MARKEDLY higher than expected (50 mmHg elevation) [core]
- **Combined severe high anion-gap metabolic acidosis AND severe respiratory acidosis** (co-existing primary disorders) [core]
- **Delta ratio** = $(38 - 12) / (24 - 8) = 26 / 16 = 1.6$ — pure HAGMA picture (consistent with lactic acidosis pattern) [core]

b) List 4 toxicological causes that could produce this picture in a 17-year-old female. (4 marks)

- **Tricyclic antidepressant overdose** — anticholinergic, sodium-channel blockade (QRS >100 ms is the hallmark), respiratory depression, hypotension; lactate from seizures and decreased-perfusion state [core]
- **Aspirin (salicylate) overdose** — severe HAGMA, tachypnoea (early — note: this patient has DEPRESSED breathing — less consistent unless very late presentation) [core]
- **Methanol or ethylene glycol** — high anion gap and high osmolar gap; check osmolar gap (calculated osmolality = $2 \times \text{Na} + \text{glucose} + \text{urea}$) [core]
- **Toxic alcohol or recreational drug overdose** — propofol infusion syndrome, isoniazid (with seizures), iron, metformin [core]
- Cyanide (very rare; profound metabolic acidosis with normal SpO₂)

c) State 4 management priorities specific to tricyclic antidepressant toxicity. (4 marks)

- **Sodium bicarbonate 1-2 mmol/kg IV bolus** (= 50-100 mmol of 8.4 percent solution) — for QRS >100 ms or haemodynamic instability; target serum pH 7.45-7.55, repeat boluses as needed; subsequent infusion 100-150 mmol in 1 L 5 percent dextrose at 100-200 mL/h [core]
- **Endotracheal intubation and mechanical ventilation** with hyperventilation to pH 7.45-7.55 (combination of bicarbonate AND alkalisation by hyperventilation) [core]
- **Cardiovascular support** — noradrenaline first-line for vasoplegia (NOT adrenaline — beta-1 worsens tachycardia); avoid Class 1A and 1C antiarrhythmics (worsen sodium-channel blockade); consider intralipid 20 percent 1.5 mL/kg bolus then 0.25 mL/kg/min for refractory cardiotoxicity [core]
- **Avoid physostigmine, flumazenil** (precipitates seizures); single-dose activated charcoal 1 g/kg if presents within 1-2 hours and airway protected [core]
- Treat seizures with benzodiazepines (NOT phenytoin — exacerbates sodium channel blockade)
- Monitor in ICU for at least 6 hours after the QRS normalises and ECG abnormalities resolve

References: eTG (Therapeutic Guidelines, current) • Murray's Toxicology Handbook 4e • Tintinalli's Emergency Medicine 9e Ch 192 • Australian Poisons Information Centre 13 11 26

Question 3 | Severe Hypokalaemic Normal Anion Gap Metabolic Acidosis (11 marks)

A 30-year-old man presents to your emergency department with a 24-hour history of progressive weakness affecting all limbs. He has had no preceding illness, no diarrhoea, and no other symptoms. His past medical history is unremarkable. On examination, he has flaccid quadriparesis (Medical Research Council 2/5) with preserved cranial nerves and reflexes. Vital signs: heart rate 110 beats per minute (frequent ectopy), blood pressure 110/70 millimetres of mercury, respiratory rate 26 breaths per minute. His arterial blood gas and electrolytes are: pH 7.26 (7.35-7.45); pCO₂ 29 mmHg; pO₂ 101 mmHg; bicarbonate 13 mmol/L (22-32); sodium 137 mmol/L; potassium 1.8 mmol/L (3.5-5.0); chloride 116 mmol/L; creatinine 86 µmol/L; creatine kinase 1975 U/L.

a) Calculate the anion gap, expected pCO₂ AND interpret the acid-base picture. (5 marks)

- Anion gap = $137 - (116 + 13) = 8$ mmol/L (NORMAL anion gap) [core]
- Expected pCO₂ (Winter's): $1.5 \times 13 + 8 = 27.5$ mmHg (± 2). Actual 29 — appropriate compensation [core]
- Pure normal anion-gap metabolic acidosis (NAGMA) with appropriate respiratory compensation [core]
- Severe hypokalaemia (K 1.8) with hyperchloraemia (Cl 116) are characteristic of this picture [core]
- Likely diagnosis: distal (Type 1) renal tubular acidosis — failure of distal tubular hydrogen-ion secretion with renal potassium wasting [core]

b) List 3 immediate management priorities for severe hypokalaemia. (3 marks)

- Continuous cardiac monitoring in a resuscitation bay with crash trolley — risk of polymorphic ventricular tachycardia (torsade de pointes) [core]
- Intravenous potassium replacement via central venous catheter — 20 mmol potassium chloride in 100 mL 0.9 percent saline over 1 hour (maximum 40 mmol/hour via central line; 10-20 mmol/hour via large peripheral vein) [core]
- Magnesium replacement (concurrent hypomagnesaemia common; potassium replacement is ineffective without magnesium correction) — magnesium sulfate 10-20 mmol IV over 30-60 minutes [core]
- Recheck potassium hourly with concurrent calcium and magnesium
- Treat underlying acidosis with bicarbonate supplementation (1-2 mmol/kg/day) — correcting acidosis often improves potassium homeostasis
- Look for and treat underlying causes — autoimmune (Sjögren's, lupus), drugs (lithium, amphotericin), toluene poisoning, hereditary

c) List 3 differential diagnoses for normal anion gap metabolic acidosis (USED CARP mnemonic). (3 marks)

- Renal tubular acidosis (types 1, 2, 4) — distal, proximal, hyperkalaemic [core]
- Severe diarrhoea / gastroenteritis (loss of bicarbonate-rich fluid) [core]
- Acetazolamide / topiramate (carbonic anhydrase inhibitors) [core]
- Ureteroenterostomy / ileal conduit
- Pancreatic / biliary fistula or drainage
- Massive 0.9 percent saline infusion (hyperchloraemic acidosis)
- Toluene / glue inhalation (often Type 1 RTA-like)
- Mineralocorticoid deficiency (Addison's disease)

References: eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements • Tintinalli's Emergency Medicine 9e Ch 117

Question 4 | Euglycaemic Diabetic Ketoacidosis on SGLT2 Inhibitor (12 marks)

A 78-year-old woman presents to your tertiary hospital emergency department with nausea, vomiting, loose bowel motions, and malaise for 48 hours. Her past medical history includes atrial fibrillation, hypertension, and type 2 diabetes mellitus. Her current medications include candesartan 16 mg daily, apixaban 2.5 mg twice daily, dapagliflozin 10 mg daily, lercanidipine, bisoprolol, simvastatin, and linagliptin-metformin 2.5 mg / 1 g twice daily. On arrival her vital signs are: heart rate 90 beats per minute (irregular), blood pressure 141/72 millimetres of mercury, respiratory rate 28 breaths per minute (deep), peripheral oxygen saturation 93 percent on room air, temperature 35.2 degrees Celsius. Her arterial blood gas and biochemistry: pH 7.35 (7.35-7.45); pCO₂ 24 mmHg (32-48); pO₂ 86 mmHg; bicarbonate 3 mmol/L; sodium 132 mmol/L; potassium 6.4 mmol/L; chloride 102 mmol/L; glucose 15.7 mmol/L (3.0-7.8); lactate 15.0 mmol/L (0.5-2.2); urea 14.6 mmol/L; creatinine 237 µmol/L (40-90).

a) Calculate the anion gap, expected pCO₂, delta ratio, AND interpret the acid-base disturbance. (5 marks)

- **Anion gap = 132 – (102 + 3) = 27 mmol/L** (elevated; normal 8-12) **[core]**
- **Expected pCO₂ (Winter's): 1.5 × 3 + 8 = 12.5 mmHg.** Actual 24 — much HIGHER than expected → co-existing respiratory acidosis (relative) **[core]**
- **Delta ratio = (27 – 12) / (24 – 3) = 15 / 21 = 0.71** — <1 indicates co-existing NAGMA (likely from diarrhoea / dehydration) **[core]**
- **Profound mixed metabolic acidosis: HAGMA** (lactate + ketones, exacerbated by SGLT2-induced ketosis) + **NAGMA** (diarrhoeal bicarbonate loss) + **relative respiratory acidosis** **[core]**
- **Diagnosis: euglycaemic diabetic ketoacidosis on dapagliflozin;** exacerbated by metformin-induced lactic acidosis from acute kidney injury (Cr 237) and dehydration **[core]**

b) Explain the mechanism of euglycaemic DKA in patients taking SGLT2 inhibitors. (3 marks)

- **SGLT2 inhibitors lower blood glucose** by promoting urinary glucose excretion, reducing the threshold for glycosuria **[core]**
- **Continued ketogenesis** occurs because endogenous insulin production is reduced, and glucagon-driven hepatic ketogenesis continues; SGLT2 inhibitors also reduce ketone clearance **[core]**
- **Result: ketoacidosis WITHOUT marked hyperglycaemia** — glucose may be normal or only mildly elevated (15.7 in this case), masking the diagnosis if not considered **[core]**
- Common precipitants: intercurrent illness, vomiting, dehydration, surgery, low-carbohydrate diet, alcohol, insulin reduction

c) State 4 immediate management priorities. (4 marks)

- **Cease SGLT2 inhibitor (dapagliflozin) AND metformin immediately;** do not restart until DKA resolved and creatinine normalised **[core]**
- **Aggressive intravenous fluid resuscitation** — 0.9 percent saline 1-2 L over the first hour, then targeted to restore intravascular volume; note this patient has hyperkalaemia and AKI (use balanced crystalloid such as Hartmann's once volume restored) **[core]**
- **Insulin infusion 0.1 unit/kg/h IV** with concurrent **5 percent dextrose to maintain blood glucose 8-14 mmol/L** — treats ketoacidosis WITHOUT causing hypoglycaemia; continue until ketones <0.6 mmol/L AND bicarbonate normalised **[core]**
- **Treat hyperkalaemia and acidosis** — calcium gluconate 10 mL of 10% IV for membrane stabilisation; insulin/dextrose (see above) shifts K intracellularly; consider sodium bicarbonate ONLY if pH <6.9 with

haemodynamic compromise **[core]**

- Anticoagulant — withhold apixaban given AKI; recheck creatinine before restarting
- Monitor closely in HDU/ICU; consider continuous renal replacement therapy if persistent severe acidosis with renal failure
- Investigate intercurrent illness — full septic screen, lipase (pancreatitis is a known SGLT2 association)

References: ANZICS / Australian Diabetes Society DKA guideline 2023 • eTG (Therapeutic Guidelines, current) • Australian Diabetes Society SGLT2 inhibitor guidance

Question 5 | Diabetic Ketoacidosis with Acute Respiratory Failure (14 marks)

Your registrar calls you to the resuscitation bay to help with an 18-year-old male (body weight 80 kilograms) who has been brought in by ambulance with a one-day history of abdominal pain, vomiting, and polyuria following a missed insulin dose. He had a 5-minute generalised tonic-clonic seizure on arrival and was intubated for airway protection. He is currently sedated and mechanically ventilated, awaiting intensive care unit review. His arterial blood gas (with fraction of inspired oxygen 1.0): pH 7.02 (7.35-7.45); pCO₂ 60 mmHg (35-45); pO₂ 200 mmHg; bicarbonate 11 mmol/L; sodium 125 mmol/L; potassium 6.5 mmol/L; chloride 86 mmol/L; glucose 35 mmol/L; lactate 2.5 mmol/L; haemoglobin 155 g/L.

a) Complete the table — for each variable, state the formula, calculate the result, AND give the clinical implication. (8 marks)

- **Anion gap:** $AG = Na - (Cl + HCO_3) = 125 - (86 + 11) = 28 \text{ mmol/L}$. **Implication:** severely elevated AG consistent with HAGMA (ketoacidosis) [core]
- **Expected pCO₂ (Winter's formula):** $1.5 \times HCO_3 + 8 = 1.5 \times 11 + 8 = 24.5 \text{ mmHg } (\pm 2)$. Actual 60 — markedly higher than expected → **severe respiratory acidosis** in addition to metabolic acidosis [core]
- **Delta ratio:** $(AG - 12) / (24 - HCO_3) = (28 - 12) / (24 - 11) = 16 / 13 = 1.23$. **Implication:** ratio 1-2 = pure HAGMA pattern (consistent with ketoacidosis without significant additional NAGMA) [core]
- **A-a gradient:** $PAO_2 = FiO_2 \times 713 - pCO_2 / 0.8 = 1.0 \times 713 - 60 / 0.8 = 713 - 75 = 638 \text{ mmHg}$. $A-a = 638 - 200 = 438 \text{ mmHg}$. **Implication:** severely elevated → underlying acute lung pathology (aspiration / pulmonary oedema / ARDS) [core]
- **Corrected sodium for hyperglycaemia:** $Na + (glucose - 5.5) \times 0.3 = 125 + (35 - 5.5) \times 0.3 = 125 + 8.85 = \sim 134 \text{ mmol/L}$. **Implication:** pseudohyponatraemia from hyperglycaemia; near-normal corrected sodium [core]

b) State the diagnosis AND list 2 likely causes for respiratory acidosis. (3 marks)

- **Severe diabetic ketoacidosis with secondary respiratory failure / ARDS** [core]
- **Aspiration pneumonia / aspiration pneumonitis** from vomiting + seizure + depressed conscious state [core]
- **Cerebral oedema** with brainstem involvement → reduced respiratory drive [core]
- Ventilator-induced under-ventilation (insufficient minute ventilation for the metabolic demand of DKA)
- Pulmonary oedema (cardiogenic or non-cardiogenic)
- Pneumothorax / barotrauma
- Sepsis with ARDS (DKA precipitant)

c) State 3 ventilation strategies to manage this patient with severe metabolic acidosis. (3 marks)

- **Increase minute ventilation to match the patient's pre-intubation respiratory drive;** this patient's calculated baseline pCO₂ for compensated DKA was ~24 mmHg — ventilator should target similar minute ventilation (large tidal volumes risk barotrauma; high respiratory rate is preferred) [core]
- **Lung-protective settings:** tidal volume 6 mL/kg ideal body weight, plateau pressure <30 cm H₂O, respiratory rate up to 30+ to achieve needed minute ventilation; positive end-expiratory pressure 5-10 cm H₂O [core]
- **Sedation and synchrony:** heavy sedation (propofol + fentanyl) and neuromuscular blockade if patient-ventilator dyssynchrony; allow patient to trigger if synchronised [core]
- Avoid bicarbonate in DKA-related acidosis (per ANZICS DKA guideline 2023) — rebound CSF acidosis, hypokalaemia, hypocalcaemia
- Investigate and treat lung pathology — chest radiograph, suction, antibiotics for aspiration

References: ANZICS / Australian Diabetes Society DKA guideline 2023 • eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements • Tintinalli's Emergency Medicine 9e Ch 117

Question 6 | Severe Hyperkalaemia in End-Stage Renal Disease (12 marks)

A 67-year-old man is brought to your emergency department by his family after he collapsed at home. His past medical history includes haemodialysis-dependent end-stage renal disease (secondary to diabetic nephropathy), hypertension, ischaemic heart disease, and congestive cardiac failure. He missed his dialysis session 4 days ago. On arrival his vital signs are: Glasgow Coma Score 15, blood pressure 80/50 millimetres of mercury, heart rate 80 beats per minute, respiratory rate 30 breaths per minute, peripheral oxygen saturation 90 percent on room air. Bedside venous blood gas: pH 7.20, pCO₂ 28, bicarbonate 11, sodium 140, potassium 7.8, chloride 105, glucose 6.5, lactate 4.0. Electrocardiogram shows tall peaked T waves with broad P-waves and prolonged PR interval.

a) List 4 electrocardiographic abnormalities evolving with progressive hyperkalaemia. (4 marks)

- **Tall, peaked, narrow T waves** (earliest and most reliable change; K typically >5.5 mmol/L) [core]
- **Prolonged PR interval and broad / flattened P waves** (K typically >6.5) [core]
- **QRS broadening** (K typically >7) — leads to sine-wave appearance with progressive widening [core]
- **Sine-wave / atrial standstill / ventricular tachycardia or fibrillation** (K typically >8-9; pre-arrest) [core]
- Bradycardia, AV block
- Right or left bundle branch block patterns

b) Complete the table — list 3 management options with mechanism, dose, and target endpoint. (6 marks)

- **Calcium gluconate 10 percent 10-20 mL intravenous over 10 minutes** (repeatable). **Mechanism:** stabilises the cardiac membrane (does NOT lower potassium). **Endpoint:** ECG normalisation (T waves narrow, QRS narrows); effect within 5 minutes, duration 30-60 minutes [core]
- **Insulin 10 units IV + 50 mL of 50 percent dextrose IV** (or 100 mL of 25 percent if available). **Mechanism:** shifts potassium intracellularly via the Na/K ATPase. **Endpoint:** lowers potassium by 0.5-1.2 mmol/L over 30-60 min; monitor BSL hourly for hypoglycaemia [core]
- **Salbutamol 10-20 mg nebulised** (or 5 µg/kg intravenous). **Mechanism:** β₂-agonist shifts potassium intracellularly. **Endpoint:** lowers potassium by 0.5-1.0 mmol/L over 60 minutes [core]
- **Haemodialysis** — definitive treatment for end-stage renal disease; lowers potassium by 1 mmol/L per hour (2-3 hours of dialysis usually required); coordinate urgently with renal team [core]
- **Sodium bicarbonate 1 mmol/kg IV** — has limited effect on isolated hyperkalaemia; useful if severe acidosis (pH <7.1) coexists
- **Resonium / sodium polystyrene sulfonate or calcium polystyrene** — slow-acting (hours), useful for ongoing management; rectal administration causes colonic necrosis (avoid)
- **Sodium zirconium cyclosilicate (Lokelma)** — newer agent with rapid potassium reduction (PBS subsidy varies)

c) List 2 immediate priorities for this haemodynamically unstable dialysis patient (other than potassium correction). (2 marks)

- **Resuscitation** — cautious volume challenge (250 mL 0.9 percent saline boluses with reassessment); avoid overload (no working kidneys); vasopressor support (noradrenaline) if persistent hypotension [core]
- **Investigate underlying cause of deterioration** — sepsis (lactate 4 supports), missed dialysis, cardiac event (troponin); examine dialysis access (infection, thrombosis); ECG and investigations for ischaemia [core]
- Avoid further potassium-raising agents (ACE-i, ARB, spironolactone, NSAIDs)

References: eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements • Tintinalli's Emergency Medicine 9e Ch 117 • Australian Resuscitation Council and NHF advanced life support

Question 7 | Severe Symptomatic Hyponatraemia with Seizure (12 marks)

A 24-year-old man is brought to your emergency department by friends after participating in a marathon endurance event. He reported drinking large quantities of water during the race. Following the event, he developed progressive confusion, headache, and a 2-minute generalised tonic-clonic seizure in the ED. His vital signs: heart rate 110 beats per minute, blood pressure 120/85 millimetres of mercury, respiratory rate 22 breaths per minute, peripheral oxygen saturation 98 percent on room air, temperature 36.0 degrees Celsius, Glasgow Coma Score 12 post-ictal. His urgent biochemistry shows: sodium 109 mmol/L (135-145), potassium 4.1 mmol/L, chloride 91 mmol/L, bicarbonate 22 mmol/L, glucose 10 mmol/L, urea 4.5 mmol/L, plasma osmolality 230 mOsm/kg (275-295).

a) Categorise this hyponatraemia (3 features) AND calculate calculated osmolality. (3 marks)

- **Hypotonic hyponatraemia** — confirmed by measured plasma osmolality 230 mOsm/kg (low) [core]
- **Severe** (Na <125 mmol/L) AND **symptomatic** (confusion, seizure) [core]
- **Acute (<48 hours onset)** — exercise-associated hyponatraemia from hypotonic fluid intake during prolonged exertion [core]
- **Calculated osmolality** = $2 \times \text{Na} + \text{glucose} + \text{urea} = 2 \times 109 + 10 + 4.5 = 232.5 \text{ mOsm/kg}$; osmolar gap = $230 - 232.5 = -2.5$ (essentially zero — no toxic alcohol or other osmole) [core]

b) State 5 immediate management steps with drug, dose, and end-point. (5 marks)

- **3 percent hypertonic saline 100-150 mL IV bolus over 10 minutes** (or 2 mL/kg for adults); this rapidly raises serum Na by approximately 2-3 mmol/L [core]
- **Repeat 100-150 mL bolus if seizure ongoing**; aim to raise Na by 4-6 mmol/L in the first hour to abort seizure [core]
- **Treat seizure pharmacologically** if ongoing — benzodiazepine (midazolam 5-10 mg IV/IM); levetiracetam 60 mg/kg IV second-line [core]
- **Strict rate of correction limit**: target Na rise of ≤ 8 mmol/L in any 24-hour period ($\leq 4-6$ mmol/L in chronic / high-risk patients) to avoid **osmotic demyelination syndrome**; relower with 5 percent dextrose if overshooting [core]
- **Monitor in critical care** — hourly serum sodium for first 4-6 hours, then 2-4 hourly; airway and circulatory support as needed [core]
- Once seizure controlled and Na >120 — switch to fluid restriction (1-1.5 L/day); manage underlying cause

c) List 4 risk factors for osmotic demyelination syndrome (ODS). (4 marks)

- **Chronic hyponatraemia** (≥ 48 hours duration) — brain has adapted by losing intracellular osmoles; rapid correction exposes neurons to osmotic stress [core]
- **Severe hyponatraemia** (Na <105 mmol/L) at presentation [core]
- **Concurrent hypokalaemia, hypomagnesaemia, or hypophosphataemia** [core]
- **Alcohol use disorder, malnutrition, liver disease** [core]
- Hyponatraemia post liver transplant
- Sjögren's syndrome
- Burns
- Acute on chronic — acute superimposed correction

References: eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements • Tintinalli's Emergency Medicine 9e Ch 117 • European Society of Endocrinology Hyponatraemia Guideline 2014

Question 8 | Paediatric Diabetic Ketoacidosis — Calculation Formulae (12 marks)

A 13-year-old girl (estimated weight 45 kilograms) presents to your emergency department with confusion and abdominal pain. She has a known background of type 1 diabetes mellitus requiring insulin treatment (missed insulin for 24 hours after gastroenteritis). She has had 3 days of vomiting and 2 days of diarrhoea. Her vital signs: heart rate 124 beats per minute, blood pressure 120/70 millimetres of mercury, respiratory rate 30 breaths per minute (deep, Kussmaul), peripheral oxygen saturation 99 percent on room air, central capillary refill time 4 seconds, temperature 37 degrees Celsius. Initial venous blood gas: pH 6.93 (7.35-7.45); pCO₂ 9 mmHg; pO₂ 44.9 mmHg; bicarbonate 2 mmol/L; base excess -25.5 mmol/L; sodium 124 mmol/L; potassium 6.2 mmol/L; chloride 79 mmol/L; glucose 43.5 mmol/L; haemoglobin 162 g/L.

a) Quote 6 relevant formulae for analysis of this blood gas WITH calculated values. (6 marks)

- **Anion gap** = $\text{Na} - (\text{Cl} + \text{HCO}_3) = 124 - (79 + 2) = 43 \text{ mmol/L}$ (severely elevated) [core]
- **Expected pCO₂ (Winter's)** = $1.5 \times \text{HCO}_3 + 8 = 1.5 \times 2 + 8 = 11 \text{ mmHg}$. Actual 9 — at lower limit of expected, indicating mild over-compensation (probably normal given resp rate of 30) [core]
- **Delta ratio** = $(\text{AG} - 12) / (24 - \text{HCO}_3) = (43 - 12) / (24 - 2) = 1.41$ — pure HAGMA (consistent with ketoacidosis pattern) [core]
- **Corrected sodium for hyperglycaemia** = $\text{Na} + (\text{glucose} - 5.5) \times 0.3 = 124 + (43.5 - 5.5) \times 0.3 = 124 + 11.4 = \sim 135 \text{ mmol/L}$ (normal — pseudohyponatraemia from hyperglycaemia) [core]
- **Calculated osmolality** = $2 \times \text{Na} + \text{glucose} + \text{urea} = 2 \times 124 + 43.5 + (\text{urea} \sim 5) = \sim 296 \text{ mOsm/kg}$; effective serum osmolality particularly important — monitor cerebral oedema risk [core]
- **Maintenance fluid calculation (4-2-1 rule)**: 4 mL/kg/h for first 10 kg + 2 mL/kg/h for next 10 kg + 1 mL/kg/h for remainder = 40 + 20 + 25 = **85 mL/h** for a 45 kg child [core]
- Fluid deficit = 5-7 percent $\times$ weight (kg) $\times$ 10 for moderate dehydration = $5 \times 45 \times 10 = \sim 2.25 \text{ L}$ over 48 hours

b) State 6 principles of fluid and electrolyte treatment with rationale per current ANZICS / ISPAD paediatric DKA guidance. (6 marks)

- **Cautious fluid resuscitation**: 0.9 percent saline 10 mL/kg over 30-60 minutes only if shocked; avoid >40 mL/kg in first 4 hours (cerebral oedema risk in paediatric DKA) [core]
- **Calculate and replace deficit over 48 hours** (not 24) — slow correction minimises cerebral oedema risk; total fluid = deficit + maintenance over 48 hours [core]
- **Insulin infusion 0.05-0.1 unit/kg/h IV STARTED 1 HOUR AFTER** fluids commenced (do NOT bolus insulin in paediatric DKA — abrupt osmolar shift) [core]
- **Add 5 percent dextrose to fluids when blood glucose <14-17 mmol/L**; do NOT stop insulin (continues to suppress ketogenesis) [core]
- **Potassium replacement** — add 40 mmol/L K to fluid once urine output established (insulin will rapidly drop K); monitor every 2-4 hours [core]
- **AVOID sodium bicarbonate** in paediatric DKA — associated with worse outcomes (cerebral oedema, prolonged acidosis); even at pH <6.9, prefer optimising fluid and insulin [core]
- Monitor for cerebral oedema — hourly Glasgow Coma Score, headache, behavioural change, papilloedema; treat with mannitol 0.5-1 g/kg IV or 3 percent saline 5 mL/kg
- Identify and treat precipitating illness (gastroenteritis here)

References: ISPAD/APEG paediatric DKA guideline 2022 • ANZICS / Australian Diabetes Society DKA guideline 2023 • ACEM clinical policies and statements

Question 9 | Salicylate Overdose — Mixed Acid-Base Disorder (12 marks)

A 35-year-old woman is brought to your emergency department by ambulance after her husband found her confused with several empty packets of aspirin. He estimates she may have ingested up to 30 grams of aspirin approximately 6 hours ago. On arrival, she is diaphoretic, tinnitus-affected, with a respiratory rate of 36 breaths per minute (deep). Her vital signs: heart rate 130 beats per minute, blood pressure 110/68 millimetres of mercury, peripheral oxygen saturation 99 percent on room air, temperature 38.5 degrees Celsius. Her arterial blood gas: pH 7.38 (7.35-7.45); pCO₂ 16 mmHg; bicarbonate 9 mmol/L; sodium 138 mmol/L; potassium 3.8 mmol/L; chloride 100 mmol/L; lactate 3.0 mmol/L; salicylate level 870 mg/L (toxic >300, lethal >700).

a) Calculate the anion gap, expected pCO₂, AND interpret the acid-base. (4 marks)

- **Anion gap** = $138 - (100 + 9) = 29$ mmol/L (elevated) [core]
- **Expected pCO₂ if pure HAGMA** = $1.5 \times 9 + 8 = 21.5$ mmHg; actual 16 — LOWER than expected → also primary respiratory alkalosis [core]
- **Mixed disorder: HAGMA (lactate, ketones) + primary respiratory alkalosis** — pH essentially normal but profoundly abnormal underlying physiology [core]
- **Classic salicylate toxicity** — direct stimulation of medullary respiratory centre causes early respiratory alkalosis; uncoupling of oxidative phosphorylation produces metabolic acidosis (lactate, ketones, salicylate anion) [core]

b) State 4 management priorities specific to salicylate toxicity. (4 marks)

- **Activated charcoal 50 g orally** if presents within 1-2 hours of ingestion AND airway protected; multiple-dose activated charcoal (50 g every 4 hours for 12-24 hours) for ongoing absorption [core]
- **Urinary alkalinisation with sodium bicarbonate** — 1.26 percent (or 8.4 percent diluted) infused at 100-150 mL/h; target urine pH >7.5 (enhances renal salicylate elimination); aim plasma pH 7.45-7.55 [core]
- **Haemodialysis** for severe toxicity — indications include salicylate level >700 mg/L (this patient), pulmonary oedema, persistent acidosis, altered mental state, AKI, hyperthermia [core]
- **AVOID intubation if at all possible** — induced sedation reduces minute ventilation; if intubation essential, set ventilator to match the patient's high minute ventilation (otherwise rapid deterioration into severe acidosis) [core]
- Treat hypoglycaemia (CNS hypoglycaemia despite normal serum glucose) — empirical 50 mL of 50 percent dextrose IV
- Correct hypokalaemia (urine alkalinisation requires K replacement), hypothermia, dehydration
- Consult Australian Poisons Information Centre 13 11 26

c) List 4 features of severe salicylate toxicity. (4 marks)

- **Mixed acid-base disorder** — primary respiratory alkalosis + HAGMA; serum pH may be normal initially [core]
- **Tinnitus, deafness** (cochlear involvement; this patient symptomatic) [core]
- **Hyperthermia** from uncoupled oxidative phosphorylation; tachypnoea / deep respirations (Kussmaul) [core]
- **Cerebral oedema, seizures, coma** in severe toxicity; pulmonary oedema (non-cardiogenic; capillary leak) [core]
- Tachycardia, hypotension, gastrointestinal symptoms
- Dehydration; hypokalaemia (urinary loss); hypoglycaemia (cerebral)
- Coagulopathy — bleeding from platelet dysfunction

References: eTG (Therapeutic Guidelines, current) • Murray's Toxicology Handbook 4e • Australian Poisons Information Centre

Question 10 | Toxic Alcohol — Osmolar Gap and Anion Gap (11 marks)

A 45-year-old man with a long history of alcohol use disorder is brought to your emergency department by his partner after she found him confused, vomiting, and complaining of blurred vision. He had no access to ethanol over the past day but was found with an open container of windshield wash fluid. His vital signs: heart rate 120 beats per minute, blood pressure 105/65 millimetres of mercury, respiratory rate 28 breaths per minute, temperature 36.5 degrees Celsius. His arterial blood gas and biochemistry: pH 7.20 (7.35-7.45); pCO₂ 22 mmHg; bicarbonate 8 mmol/L; sodium 142 mmol/L; potassium 4.5 mmol/L; chloride 100 mmol/L; glucose 6 mmol/L; urea 5 mmol/L; lactate 1.2 mmol/L; ethanol <0.05 (undetectable). Measured serum osmolality 360 mOsm/kg.

a) Calculate the anion gap, calculated osmolality, AND osmolar gap. (4 marks)

- **Anion gap** = $142 - (100 + 8) = 34$ mmol/L (markedly elevated) [core]
- **Calculated osmolality** = $2 \times \text{Na} + \text{glucose} + \text{urea} = 2 \times 142 + 6 + 5 = 295$ mOsm/kg [core]
- **Osmolar gap** = measured – calculated = $360 - 295 = 65$ mOsm/kg (markedly elevated; normal <10) [core]
- **Combination of HAGMA + raised osmolar gap** in this clinical context is diagnostic of toxic alcohol ingestion (methanol or ethylene glycol) [core]
- Note: ethanol can cause raised osmolar gap but this patient's level is undetectable; the windshield fluid suggests methanol; visual symptoms support methanol
- Each 100 mg/dL of methanol or ethylene glycol raises osmolar gap by ~30-35 mOsm/kg

b) List 4 management priorities for suspected methanol or ethylene glycol toxicity. (4 marks)

- **Fomepizole (4-methylpyrazole) 15 mg/kg IV bolus over 30 minutes**, then 10 mg/kg every 12 hours for 4 doses (then 15 mg/kg every 12 hours) — alcohol dehydrogenase inhibitor; first-line if available [core]
- **OR ethanol IV / oral** (if fomepizole unavailable) — competitively inhibits alcohol dehydrogenase; loading dose 0.6 g/kg followed by 100 mg/kg/h infusion; target serum ethanol level 1-2 g/L; less preferred (sedation, hypoglycaemia) [core]
- **Folinic acid (leucovorin) 50 mg IV every 6 hours** for methanol; **thiamine and pyridoxine** for ethylene glycol — accelerate metabolism of formate / glyoxylate [core]
- **Haemodialysis** — removes parent alcohol AND toxic metabolites; indications: severe acidosis (pH <7.25), renal failure, visual symptoms, haemodynamic instability, methanol level >500 mg/L or ethylene glycol level >500 mg/L [core]
- Sodium bicarbonate 1-2 mmol/kg IV for severe acidosis (avoid in volume-overloaded patients)
- Investigate for visual symptoms (methanol — blindness from optic nerve injury), oxalate crystals in urine (ethylene glycol)
- Australian Poisons Information Centre 13 11 26 — specialist toxicology advice

c) List 3 features distinguishing methanol from ethylene glycol poisoning. (3 marks)

- **Methanol**: visual symptoms (blurred vision, blindness — optic nerve injury from formate); 'snowfield blindness'; **ethylene glycol**: NO visual symptoms [core]
- **Methanol**: NO calcium oxalate crystals; **ethylene glycol**: **calcium oxalate crystals in urine** (monohydrate / dihydrate) [core]
- **Ethylene glycol**: hypocalcaemia (precipitates with oxalate as crystals); renal failure; **methanol**: less frequent renal involvement [core]
- Both produce HAGMA + raised osmolar gap
- Both treated with fomepizole / ethanol + dialysis

- Methanol intoxication classically described in people accidentally drinking windshield washer fluid; ethylene glycol from antifreeze

References: *eTG (Therapeutic Guidelines, current)* • *Murray's Toxicology Handbook 4e* • *Australian Poisons Information Centre 13 11 26*

Question 11 | Acute Liver Failure — Paracetamol Toxicity (12 marks)

A 26-year-old woman is brought to your emergency department after being found unconscious in her bed at home. She has not been seen for 2 days. Her biochemistry on arrival is: Sodium 135 mmol/L; potassium 4.5 mmol/L; chloride 100 mmol/L; bicarbonate 2 mmol/L; urea 5.8 mmol/L; creatinine 160 μ mol/L (60-110); total protein 50 g/L; albumin 30 g/L; globulin 20 g/L. Liver function tests: bilirubin 77 μ mol/L (<20); ALP 143 U/L; gamma GT 328 U/L (<55); ALT 8050 U/L (<45); AST 7560 U/L (<35). INR 5.8 (unmeasurable). Lactate 7.5 mmol/L. Glucose 2.5 mmol/L. Paracetamol level pending.

a) State 4 abnormalities AND their pathophysiological basis. (4 marks)

- **Anion gap = 135 – (100 + 2) = 33 mmol/L** (severe HAGMA with HCO_3^- 2); lactic acidosis from hepatic dysfunction and shock **[core]**
- **Profoundly elevated transaminases (ALT 8050, AST 7560)** — hepatocellular injury of massive degree; AST:ALT ratio <1 (toxic / viral pattern); ALT >1000 in <5 percent of liver disease — narrow differential **[core]**
- **Severe coagulopathy** (INR 5.8) and synthetic dysfunction (low albumin) — hepatic failure **[core]**
- **Acute kidney injury** (creatinine 160) — hepatorenal syndrome OR direct nephrotoxicity (paracetamol-induced tubular necrosis); hypoglycaemia (gluconeogenesis failure) **[core]**
- Hyperbilirubinaemia and pattern suggest acute hepatocellular failure

b) List 4 differential diagnoses for ALT >5000 U/L. (4 marks)

- **Paracetamol (acetaminophen) toxicity** — most common cause; classic biochemical picture **[core]**
- **Ischaemic / hypoxic hepatitis** (shock liver) — sepsis, cardiac arrest, acute heart failure **[core]**
- **Viral hepatitis** — fulminant hepatitis A, B, E (especially in pregnancy), or non-A-E **[core]**
- **Autoimmune hepatitis** **[core]**
- Wilson's disease (young patients)
- Drug-induced (NSAID, isoniazid, valproate, amiodarone, illicit/herbal medicines)
- Vascular (Budd-Chiari syndrome, hepatic artery thrombosis post-transplant)

c) State 4 management priorities including specific antidote. (4 marks)

- **N-acetylcysteine (NAC) intravenous infusion** — start empirically without waiting for paracetamol level given the severity; **200 mg/kg over 4 hours, then 100 mg/kg over 16 hours (Australian 20-hour protocol)**; continue until improvement **[core]**
- **Resuscitation and supportive care** — ICU admission; intravenous fluids (0.9 percent saline cautiously given volume status); empirical sepsis screening; treat hypoglycaemia with 10 percent dextrose 100 mL bolus then infusion **[core]**
- **Coagulopathy management** — vitamin K 10 mg IV; fresh frozen plasma 10-15 mL/kg ONLY if active bleeding (FFP masks INR as prognostic marker); consider 4-factor PCC if life-threatening haemorrhage **[core]**
- **Liver transplant unit referral** (King's College criteria for acetaminophen-induced acute liver failure: pH <7.30, OR all of: INR >6.5, creatinine >300, hepatic encephalopathy grade III-IV) **[core]**
- Avoid hepatotoxic medications; lactulose 30-45 mL PO three times daily for hepatic encephalopathy
- Investigate cause (toxicology screen, paracetamol/salicylate level, blood alcohol, viral serology, autoimmune serology)

References: eTG (Therapeutic Guidelines, current) • Murray's Toxicology Handbook 4e • King's College criteria

Question 12 | Triple Acid-Base Disorder — Pancreatitis with Vomiting and Hypoperfusion (12 marks)

A 45-year-old man with a long history of alcohol use presents to your emergency department with intractable vomiting for 36 hours. He last drank alcohol 18 hours ago and reports severe epigastric pain radiating to his back. On examination he is diaphoretic with epigastric tenderness. Vital signs: heart rate 105 beats per minute, blood pressure 105/65 millimetres of mercury, respiratory rate 28 breaths per minute, peripheral oxygen saturation 98 percent on room air, temperature 37.5 degrees Celsius. His venous blood gas: pH 7.61 (7.35-7.45); pCO₂ 19 mmHg; bicarbonate 18 mmol/L; sodium 137 mmol/L; potassium 3.7 mmol/L; chloride 98 mmol/L; ionised calcium 1.03 mmol/L (1.15-1.32); glucose 4.8 mmol/L; lactate 8.5 mmol/L.

a) Calculate the anion gap and expected pCO₂ (considering all 3 disorders) AND interpret. (5 marks)

- **Anion gap = 137 – (98 + 18) = 21 mmol/L** (elevated; consistent with lactic acidosis 8.5 + ketones) **[core]**
- **Delta gap analysis:** $\Delta AG = 21 - 12 = 9$; $\Delta HCO_3 = 24 - 18 = 6 \rightarrow \text{delta-delta} = 9 / 6 = 1.5 \rightarrow \text{HAGMA}$ with co-existing metabolic alkalosis (otherwise HCO₃ would be lower) **[core]**
- **Expected pCO₂ for primary metabolic alkalosis:** $0.7 \times HCO_3 + 21 = 0.7 \times 18 + 21 = 33.6 \text{ mmHg}$. Actual 19 — much lower → primary respiratory alkalosis also present **[core]**
- **TRIPLE acid-base disorder:** (1) HAGMA from lactic acidosis + alcoholic ketosis; (2) metabolic alkalosis from vomiting (loss of HCl); (3) primary respiratory alkalosis from pain / sepsis / hyperventilation **[core]**
- **Likely diagnosis:** acute pancreatitis with vomiting, ketosis, lactic acidosis, and hyperventilation; lipase will be markedly raised; supports hypocalcaemia (saponification of fat necrosis) **[core]**

b) List 4 likely contributors to the lactic acidosis. (4 marks)

- **Hypoperfusion / hypovolaemia** from vomiting → dehydration → tissue hypoxia (Type A lactic acidosis) **[core]**
- **Thiamine deficiency** (alcoholic) — impaired pyruvate to acetyl-CoA conversion (Type B); requires intravenous thiamine 300-500 mg before any glucose **[core]**
- **Alcohol-induced metabolic shift** (NADH accumulation favours pyruvate to lactate) **[core]**
- **Sepsis** from infected pancreatitis or another source **[core]**
- Bowel ischaemia (consider if HAGMA out of proportion to clinical picture)
- Mitochondrial toxicity (other drugs, metformin)

c) List 3 management priorities specific to this presentation. (3 marks)

- **Aggressive intravenous fluid resuscitation** with balanced crystalloid (Hartmann's) — high-volume fluid loss; monitor urine output and lactate **[core]**
- **Intravenous thiamine 300-500 mg IV BEFORE any glucose** (Wernicke's encephalopathy prevention in alcoholic) **[core]**
- **Electrolyte replacement** — calcium (saponification), magnesium, potassium; investigate pancreatitis (lipase, computed tomography) **[core]**
- Analgesia (opioid titrated), anti-emetics, nasogastric tube if persistent vomiting
- Manage alcohol withdrawal, complete history including all medications and toxin exposure
- Monitor for sepsis / infected pancreatitis — blood cultures, broad-spectrum antibiotics if septic

References: eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements • Tintinalli's Emergency Medicine 9e Ch 117

Question 13 | Severe Uraemic Acidosis (11 marks)

A 32-year-old Aboriginal woman presents to your emergency department with a 2-week history of lethargy, nausea, and decreased urine output. She is previously well and has not been seen by medical services for 10 years. Her vital signs: heart rate 95 beats per minute, blood pressure 165/100 millimetres of mercury, respiratory rate 26 breaths per minute, peripheral oxygen saturation 96 percent on room air. Her venous blood gas and biochemistry: pH 7.20 (7.35-7.45); pCO₂ 28 mmHg; bicarbonate 11 mmol/L; sodium 132 mmol/L; potassium 6.5 mmol/L; chloride 96 mmol/L; urea 62 mmol/L; creatinine 1842 µmol/L (40-90); haemoglobin 76 g/L; calcium 1.6 mmol/L; phosphate 3.5 mmol/L.

a) Calculate the anion gap, expected pCO₂, AND interpret the acid-base. (5 marks)

- **Anion gap** = 132 – (96 + 11) = **25 mmol/L** (elevated) [core]
- **Expected pCO₂ (Winter's)** = 1.5 × 11 + 8 = **24.5 mmHg** (± 2). Actual 28 — close to expected (mild respiratory acidosis component) [core]
- **HAGMA from uraemia** — accumulation of organic acids (sulphate, phosphate, urate) from impaired renal excretion [core]
- **Severely elevated urea and creatinine** (creatinine 1842 → eGFR <5 mL/min/1.73 m²) consistent with end-stage renal disease [core]
- **Concurrent abnormalities**: hyperkalaemia (K 6.5), hypocalcaemia (Ca 1.6), hyperphosphataemia (3.5), anaemia (Hb 76 — chronic from erythropoietin deficiency); supports **chronic kidney disease at end stage** [core]

b) List 4 likely causes of chronic kidney disease in this 32-year-old Aboriginal woman. (4 marks)

- **Diabetic nephropathy** (high prevalence in Indigenous Australians; type 2 diabetes often undiagnosed) [core]
- **Hypertensive nephropathy** (BP 165/100 this admission; often longstanding) [core]
- **Glomerulonephritis** — IgA nephropathy (common in Indigenous populations); post-infectious / post-streptococcal glomerulonephritis [core]
- **Reflux nephropathy** with chronic pyelonephritis [core]
- Drug nephropathy — analgesic / NSAID use
- Polycystic kidney disease (autosomal dominant)
- Chronic pyelonephritis from recurrent urinary tract infections

c) State 2 immediate management priorities (with drug, dose). (2 marks)

- **Treat hyperkalaemia** — calcium gluconate 10 mL of 10% IV (membrane stabilisation); insulin 10 units + 50 mL 50 percent dextrose IV (cellular K shift); salbutamol 10-20 mg nebulised; sodium bicarbonate 50 mmol if pH <7.1 [core]
- **Urgent renal team consultation for haemodialysis** — definitive treatment given end-stage renal disease with metabolic / electrolyte derangement [core]
- Volume status assessment — diuretics if pulmonary oedema; cautious fluid replacement
- Cultural safety — engage Aboriginal Liaison Officer; family / community involvement; trauma-informed care given prior medical avoidance

References: eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements • RACGP Indigenous Health Guidelines • Tintinalli's Emergency Medicine 9e Ch 95

Question 14 | Severe Metabolic Alkalosis with Hypokalaemia (10 marks)

A 40-year-old man with a developmentally delayed background is brought to your emergency department by his carers with vomiting. He had a percutaneous endoscopic gastrostomy (PEG) inserted 4 weeks ago for feeding; ongoing reflux and vomiting have led to estimated 3-week persistent vomiting. He weighs approximately 40 kilograms. On arrival his Glasgow Coma Score is 13 (his baseline per carers). Vital signs: heart rate 90 beats per minute, blood pressure 140/70 millimetres of mercury, respiratory rate 13 breaths per minute, peripheral oxygen saturation 95 percent on room air. His venous blood gas and biochemistry: pH 7.55 (7.35-7.45); pCO₂ 50 mmHg; bicarbonate 42 mmol/L; sodium 145 mmol/L; potassium 2.0 mmol/L (3.5-5.0); chloride 88 mmol/L; ionised calcium 1.02 mmol/L; glucose 7.4 mmol/L.

a) Calculate expected pCO₂ for primary metabolic alkalosis AND interpret the acid-base. (4 marks)

- **Expected pCO₂ for primary metabolic alkalosis** = $0.7 \times \text{HCO}_3^- + 21 = 0.7 \times 42 + 21 = 50.4$ mmHg. Actual 50 — appropriate compensation **[core]**
- **Pure metabolic alkalosis with appropriate respiratory compensation** **[core]**
- **Anion gap** = $145 - (88 + 42) = 15$ mmol/L (mildly elevated; possibly albumin-related) **[core]**
- **Cause:** prolonged vomiting / nasogastric losses (loss of hydrochloric acid → both H⁺ and Cl⁻ lost); secondary hyperaldosteronism perpetuates with K loss **[core]**
- Hypochloraemia (Cl 88) is characteristic; low urinary chloride supports the diagnosis (saline-responsive)

b) Describe the relationship between alkalosis and hypokalaemia AND quantify expected K shift with alkalosis correction. (3 marks)

- **Alkalosis shifts potassium intracellularly** in exchange for hydrogen ion (cellular buffering); also promotes renal potassium loss via aldosterone-mediated potassium excretion **[core]**
- **Approximate rule:** K decreases by ~0.6 mmol/L for every 0.1 increase in pH; this patient's pH is 7.55 (0.15 above normal 7.40) → ~0.9 mmol/L of K reduction is alkalosis-related **[core]**
- **True total body K deficit is much greater** than the serum K suggests — may approach 5-10 mmol/kg in longstanding vomiting (200-400 mmol deficit in this patient) **[core]**

c) State 3 management priorities with drug, dose, and monitoring. (3 marks)

- **0.9 percent saline 1-2 L IV bolus** (saline-responsive metabolic alkalosis); restores chloride and corrects intravascular volume → cessation of secondary hyperaldosteronism **[core]**
- **Potassium replacement** — 40 mmol/L potassium chloride in fluid (max 40 mmol/h via central line, 10-20 mmol/h via large peripheral); concurrent magnesium replacement (10-20 mmol IV); monitor with hourly K levels **[core]**
- **Continuous cardiac monitoring** (severe hypokalaemia 2.0 — arrhythmia / torsade risk); 12-lead ECG looking for U waves, prolonged QT, T-wave flattening **[core]**
- Investigate underlying cause and address (reflux / aspiration risk; consider gastrointestinal review and PEG management)
- Anti-emetic (metoclopramide if no contraindication; ondansetron 4-8 mg IV alternative)
- Avoid loop diuretics (worsen alkalosis)

References: eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements • Tintinalli's Emergency Medicine 9e Ch 117

Question 15 | Diabetic Ketoacidosis with Hyperosmolar Features (13 marks)

A 60-year-old man with a past history of type 2 diabetes mellitus is brought to your emergency department by his elderly mother who found him collapsed on the floor. He has been unwell with vomiting for the past 3 weeks and has not been eating. His vital signs: blood pressure 65/30 millimetres of mercury, heart rate 120 beats per minute (regular), respiratory rate 36 breaths per minute (deep, Kussmaul), peripheral oxygen saturation 100 percent on 15 litres oxygen via non-rebreather mask, Glasgow Coma Score 13. His venous blood gas: pH 6.91 (7.35-7.45); pCO₂ 13 mmHg; bicarbonate 4 mmol/L; sodium 106 mmol/L; potassium 6.3 mmol/L; chloride 73 mmol/L; glucose 82 mmol/L (3-7); lactate 3.1 mmol/L; urea 32 mmol/L; creatinine 280 µmol/L.

a) Calculate anion gap, corrected sodium, expected pCO₂, AND calculated osmolality. (5 marks)

- **Anion gap** = $106 - (73 + 4) = 29$ mmol/L (severely elevated) [core]
- **Corrected sodium for hyperglycaemia** = $106 + (82 - 5.5) \times 0.3 = 106 + 23 = \sim 129$ mmol/L (still mildly low — may reflect water shifts, vomiting losses, or true hyponatraemia) [core]
- **Expected pCO₂ (Winter's)** = $1.5 \times 4 + 8 = 14$ mmHg. Actual 13 — at lower limit of expected range (maximal compensation for severe metabolic acidosis) [core]
- **Calculated osmolality** = $2 \times \text{Na} + \text{glucose} + \text{urea} = 2 \times 106 + 82 + 32 = 326$ mOsm/kg (HIGH — hyperosmolar state superimposed on DKA) [core]
- **Effective serum osmolality** (excludes urea) = $2 \times 106 + 82 = 294$ mOsm/kg; this patient has features of mixed DKA and hyperosmolar hyperglycaemic state (HHS) [core]

b) List 5 immediate management priorities. (5 marks)

- **Aggressive intravenous fluid resuscitation** — 0.9 percent saline 1-2 L IV bolus over the first hour, guided by perfusion (target MAP >65); anticipate large total fluid requirements (7-10 L over first 24 hours) [core]
- **Insulin infusion 0.1 unit/kg/h IV (approximately 7 units/h for 70 kg)** — do NOT bolus; commenced AFTER initial fluid resuscitation; adjust based on glucose drop (target 3-4 mmol/L per hour) [core]
- **Potassium replacement** — current K 6.3 (despite cellular shifts); insulin will rapidly lower K → add K to fluid (typically 20-40 mmol/L) ONCE serum K <5 mmol/L [core]
- **Treat hyperkalaemia and shock** — calcium gluconate 10 mL 10% IV for ECG changes; vasopressor (noradrenaline infusion) if hypotension persists despite fluid [core]
- **Identify and treat precipitant** — infection (full septic screen, broad-spectrum antibiotics empirically), acute coronary syndrome (ECG, troponin), cerebrovascular event, drug error [core]
- Monitor in HDU/ICU; consider ICU admission given severity
- AVOID rapid sodium correction (hyperosmolar state) — rate ≤8 mmol/L per 24 hours to prevent osmotic demyelination
- Deep vein thrombosis prophylaxis once stable

c) State 3 ventilation considerations if intubation becomes necessary. (3 marks)

- **Match minute ventilation to patient's compensation:** this patient is spontaneously generating very high minute ventilation (RR 36, deep Kussmaul); intubation rapidly reduces minute ventilation → dramatic worsening of acidosis. Set ventilator to high RR (20-25) and adequate tidal volume to match pre-intubation [core]
- **Avoid sedation that depresses respiratory drive excessively;** ketamine (1-2 mg/kg) may be preferred induction agent over propofol given haemodynamic-sparing properties [core]
- **Risk of cardiovascular collapse on induction** — pre-load with crystalloid; have noradrenaline drawn up; rapid sequence intubation by senior operator; potassium replacement before high-dose rocuronium /

suxamethonium **[core]**

- Anticipate persistent acidosis post-intubation; frequent gas measurement; ICU support

References: ANZICS / Australian Diabetes Society DKA guideline 2023 • eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements

Question 16 | Severe Hypocalcaemia in Chronic Pancreatitis (10 marks)

A 62-year-old male with a long history of chronic alcohol-related pancreatitis presents to your emergency department with several days of nausea, vomiting, and intermittent muscle spasms. On examination he has positive Chvostek's and Trousseau's signs, peri-oral tingling, and intermittent carpopedal spasm. He is otherwise haemodynamically stable. His biochemistry: Total calcium 1.50 mmol/L (2.10-2.55); ionised calcium 0.78 mmol/L (1.15-1.32); albumin 28 g/L; magnesium 0.42 mmol/L (0.7-1.0); phosphate 2.0 mmol/L; potassium 3.0 mmol/L; ALT 60; lipase 850 U/L. Electrocardiogram shows prolonged QT interval (corrected QT 510 ms).

a) Calculate the albumin-corrected calcium AND comment on the difference from ionised calcium. (3 marks)

- **Albumin-corrected total calcium** = total Ca + 0.02 × (40 – albumin g/L) = 1.50 + 0.02 × (40 – 28) = 1.50 + 0.24 = **1.74 mmol/L [core]**
- **Ionised calcium 0.78** remains severely low (normal 1.15-1.32) — albumin correction does not normalise; the reduction in IONISED calcium is physiologically active and accounts for the symptoms **[core]**
- **True hypocalcaemia confirmed** (ionised calcium is more reliable than total calcium in critical illness, albumin disturbance, acid-base shifts) **[core]**

b) List 4 clinical features of severe hypocalcaemia. (4 marks)

- **Tetany** — peri-oral tingling, carpopedal spasm, laryngospasm **[core]**
- **Chvostek's sign** (facial twitch on tapping facial nerve) and **Trousseau's sign** (carpal spasm with blood pressure cuff inflation) **[core]**
- **Cardiovascular** — prolonged QT interval, polymorphic ventricular tachycardia (torsades de pointes), hypotension, heart failure **[core]**
- **Neurological** — seizures, paraesthesia, depression, confusion, coma; raised intracranial pressure with papilloedema (chronic) **[core]**
- Stridor / laryngospasm
- Hyperreflexia

c) State 3 management priorities including drug, dose, and end-points. (3 marks)

- **Calcium gluconate 10 percent 10-20 mL (2.2-4.4 mmol Ca) IV slowly over 10 minutes**; can repeat; for severe tetany / arrhythmia commence infusion (100 mL of 10 percent in 1 L 0.9 percent saline at 50-100 mL/h, titrated to ionised calcium 1.10-1.20 mmol/L) **[core]**
- **Magnesium sulfate 10-20 mmol IV over 30-60 minutes** — concurrent hypomagnesaemia must be corrected; calcium replacement is INEFFECTIVE without magnesium correction **[core]**
- **Continuous cardiac monitoring** (prolonged QT — torsade risk); 12-lead ECG; treat any arrhythmia per ALS guidelines (avoid digoxin in hypocalcaemia) **[core]**
- Identify and treat underlying cause — chronic pancreatitis with malabsorption (refer gastroenterology); investigate hypoparathyroidism (parathyroid hormone level)
- Vitamin D status (25-hydroxyvitamin D); replacement if deficient
- Avoid bicarbonate (lowers ionised calcium further)

References: eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements • Tintinalli's Emergency Medicine 9e Ch 117

Question 17 | Acute Adrenal Insufficiency (10 marks)

A 45-year-old man with known autoimmune Addison's disease (taking long-term hydrocortisone 20 mg orally daily) presents to your emergency department with 5 days of vomiting, severe abdominal pain, and lethargy. He stopped taking his hydrocortisone 3 days ago because he was unable to keep oral intake. His vital signs: heart rate 130 beats per minute, blood pressure 70/40 millimetres of mercury, respiratory rate 28 breaths per minute, peripheral oxygen saturation 95 percent on room air, temperature 35.5 degrees Celsius. His venous blood gas and biochemistry: pH 7.30 (7.35-7.45); pCO₂ 26 mmHg; bicarbonate 12 mmol/L; sodium 122 mmol/L; potassium 6.4 mmol/L; chloride 96 mmol/L; glucose 2.8 mmol/L; lactate 5.2 mmol/L; calcium 2.6 mmol/L (raised).

a) List 4 biochemical abnormalities with explanation. (4 marks)

- **Hyponatraemia (Na 122)** — combined loss of sodium (mineralocorticoid deficiency, vomiting) and water retention (cortisol deficiency increases antidiuretic hormone); pseudohyponatraemia from cortisol deficiency [core]
- **Hyperkalaemia (K 6.4)** — mineralocorticoid (aldosterone) deficiency → reduced renal potassium excretion [core]
- **Hypoglycaemia (glucose 2.8)** — cortisol deficiency reduces gluconeogenesis; particularly in vomiting / fasting state [core]
- **HAGMA (HCO₃ 12, lactate 5.2)** — hypoperfusion and lactic acidosis; mild metabolic acidosis from mineralocorticoid deficiency (Type 4 RTA-like) [core]
- Hypercalcaemia (mild) — uncommon but described in adrenal crisis
- Eosinophilia, raised urea / creatinine

b) State 4 immediate management priorities. (4 marks)

- **Hydrocortisone 100 mg intravenous STAT**, then 100 mg every 6 hours (provides both glucocorticoid and mineralocorticoid effect at high dose); fludrocortisone NOT needed acutely [core]
- **Aggressive fluid resuscitation** — 0.9 percent saline + 5 percent dextrose 1-2 L bolus to correct shock and hypoglycaemia; cautious sodium correction (target rise <10 mmol/L in 24 hours to prevent osmotic demyelination) [core]
- **Treat hyperkalaemia** — calcium gluconate 10 mL 10% IV for ECG changes; insulin / dextrose if persistent (will also help hypoglycaemia); salbutamol; but resolution of glucocorticoid / mineralocorticoid deficiency usually corrects within hours [core]
- **Identify and treat precipitating stress / illness** — sepsis, vomiting illness, surgery, trauma; full septic screen with broad-spectrum antibiotics if septic [core]
- Investigate cause of adrenal crisis — non-compliance, intercurrent illness, drug interaction (rifampicin, phenytoin)
- Continue stress-dose steroids until stable; transition to oral when patient eating reliably

c) List 2 sick-day rules for patients with Addison's disease. (2 marks)

- **Double the usual hydrocortisone dose** during minor illness with fever; **triple the dose** for severe illness, vomiting, or surgical procedures [core]
- **Use intramuscular hydrocortisone (100 mg)** at home if unable to keep oral intake (vomiting); attend hospital immediately if symptoms continue [core]
- Carry a steroid emergency card AND medic-alert bracelet
- Educate family / carers in IM injection technique

References: eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements • Tintinalli's Emergency Medicine 9e Ch 117

Question 18 | Metformin-Associated Lactic Acidosis (10 marks)

A 75-year-old man with type 2 diabetes mellitus (treated with metformin 1 g twice daily and gliclazide), chronic kidney disease (estimated glomerular filtration rate 30 millilitres per minute), and chronic heart failure presents with 3 days of vomiting, diarrhoea, and progressive lethargy. His vital signs: heart rate 110 beats per minute, blood pressure 95/55 millimetres of mercury, respiratory rate 28 breaths per minute, peripheral oxygen saturation 95 percent on room air, temperature 36.0 degrees Celsius. His biochemistry: pH 7.10 (7.35-7.45); pCO₂ 25 mmHg; bicarbonate 10 mmol/L; sodium 138 mmol/L; potassium 5.5 mmol/L; chloride 102 mmol/L; glucose 6.5 mmol/L; lactate 12.5 mmol/L; urea 22 mmol/L; creatinine 350 µmol/L.

a) Calculate the anion gap, expected pCO₂, AND interpret the acid-base. (4 marks)

- **Anion gap** = $138 - (102 + 10) = 26$ mmol/L (markedly elevated) [core]
- **Expected pCO₂ (Winter's)** = $1.5 \times 10 + 8 = 23$ mmHg (± 2). Actual 25 — appropriate compensation [core]
- **Pure HAGMA from severe lactic acidosis** (lactate 12.5) [core]
- **Likely diagnosis: metformin-associated lactic acidosis** in setting of acute kidney injury (creatinine 350; eGFR very low) — metformin accumulates → mitochondrial inhibition → lactate accumulation; precipitated by dehydration from vomiting / diarrhoea [core]
- **Delta ratio** = $(26 - 12) / (24 - 10) = 14 / 14 = 1$ — pure HAGMA without other component

b) State 4 management priorities. (4 marks)

- **Cease metformin** and other lactate-generating drugs (linezolid, antiretrovirals) [core]
- **Aggressive fluid resuscitation** with balanced crystalloid (Hartmann's) — 0.9 percent saline 1-2 L bolus, then guided by haemodynamics; correct dehydration [core]
- **Renal replacement therapy (haemodialysis)** — definitive treatment for metformin-associated lactic acidosis (removes metformin AND corrects acidosis); indications: pH <7.0, lactate >15, persistent acidosis despite resuscitation, or AKI requiring dialysis [core]
- **Sodium bicarbonate** — controversial; consider if pH <7.0 with haemodynamic instability (BICAR-ICU trial); 1-2 mmol/kg IV bolus; aim pH 7.20 [core]
- Identify and treat precipitant (gastroenteritis, sepsis, acute coronary syndrome)
- Vasopressor support (noradrenaline) if hypotensive despite fluid
- ICU admission for monitoring

c) List 2 contraindications to metformin. (2 marks)

- **Severe renal impairment** — eGFR <30 mL/min/1.73 m² (relative); absolute <15 mL/min/1.73 m² [core]
- **Decompensated heart failure / acute myocardial infarction / shock** — any state with tissue hypoperfusion; respiratory failure with hypoxia [core]
- Severe hepatic dysfunction
- Active alcohol use disorder (lactate burden)
- Acute illness with risk of dehydration / decompensation
- Imaging with iodinated contrast — withhold metformin per RANZCR guidance if eGFR <30

References: eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements • Tintinalli's Emergency Medicine 9e Ch 117 • Australian Diabetes Society

Question 19 | Rhabdomyolysis (10 marks)

A 28-year-old man is brought to your emergency department after collapsing during the 35-kilometre mark of a marathon on a hot summer day (ambient temperature 38 degrees Celsius). He has been drinking water but feels thirsty. On arrival, his vital signs: heart rate 130 beats per minute, blood pressure 95/60 millimetres of mercury, respiratory rate 28 breaths per minute, peripheral oxygen saturation 96 percent on room air, temperature 40.5 degrees Celsius (rectal). He passes dark, tea-coloured urine on catheterisation. Biochemistry: Sodium 142 mmol/L; potassium 6.8 mmol/L; chloride 98 mmol/L; bicarbonate 14 mmol/L; urea 14 mmol/L; creatinine 220 µmol/L; calcium 1.8 mmol/L; phosphate 3.0 mmol/L; creatine kinase 75,000 U/L (<200); urinalysis: blood +++ (no red cells on microscopy — myoglobin).

a) Define rhabdomyolysis AND state 3 biochemical abnormalities expected. (3 marks)

- **Rhabdomyolysis** = breakdown of skeletal muscle with release of intracellular contents (myoglobin, creatine kinase, potassium, phosphate, myocyte enzymes) into the systemic circulation **[core]**
- **Hyperkalaemia, hyperphosphataemia, hypocalcaemia** (calcium binds to released phosphate and to damaged muscle) **[core]**
- **Markedly raised creatine kinase** ($>5 \times$ upper limit of normal — typically >1000 ; severe >5000); myoglobinuria (positive urine dipstick for blood with no red cells on microscopy) **[core]**
- Acute kidney injury (myoglobin precipitates in renal tubules)
- Metabolic acidosis (lactic, organic acid release)
- Disseminated intravascular coagulation
- Compartment syndrome (cause and effect)

b) State 4 immediate management priorities. (4 marks)

- **Aggressive intravenous fluid resuscitation** — 0.9 percent saline 1-2 L bolus, then 1-2 mL/kg/h continuous infusion; target urine output 2-3 mL/kg/hour to flush myoglobin **[core]**
- **Treat hyperkalaemia** — calcium gluconate 10 mL 10% IV (membrane stabilisation); insulin 10 units + 50 mL 50% dextrose IV; salbutamol 10-20 mg nebulised; consider haemodialysis if persistent **[core]**
- **Active cooling** for heat stroke (temperature 40.5) — cold-water immersion preferred; ice packs to neck, axillae, groin; cold IV fluids; cease cooling at core temperature 38 degrees Celsius **[core]**
- **Urinary alkalinisation** — sodium bicarbonate to maintain urine pH >6.5 (reduces myoglobin precipitation); evidence is limited but commonly recommended; AVOID if hypocalcaemia severe **[core]**
- Monitor for compartment syndrome — limb pain, swelling, neurovascular compromise
- Investigate underlying cause and treat (drug toxicity, statin, illicit drugs, viral myositis, trauma, prolonged immobility)
- ICU admission; renal replacement therapy if persistent oliguria, refractory hyperkalaemia, or severe acidosis

c) List 3 causes of rhabdomyolysis. (3 marks)

- **Trauma / crush injury**; prolonged immobility (alcohol intoxication, drug overdose with prolonged unconsciousness); **extreme exertion** (this case — marathon, military training) **[core]**
- **Drug-induced**: statins (especially with fibrate combination), colchicine, neuroleptic malignant syndrome, serotonin syndrome, cocaine, amphetamines **[core]**
- **Heat stroke** (this case), severe hyperthermia **[core]**
- Inherited muscle disorders (McArdle disease, muscular dystrophy)
- Severe infections (influenza, Legionella, sepsis)
- Electrolyte disturbance (severe hypokalaemia, hypophosphataemia)

- Status epilepticus

References: eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements • Tintinalli's Emergency Medicine 9e Ch 269 • Wilderness Medical Society

Question 20 | Rapid Arterial Blood Gas Interpretation Series (10 marks)

Below are 5 arterial blood gases drawn from 5 different patients in your emergency department. For each, calculate the anion gap (where applicable), expected $p\text{CO}_2$ (Winter's formula or appropriate compensation rule), AND state the most likely primary disorder.

a) Patient A: pH 7.10, $p\text{CO}_2$ 60, bicarbonate 18, Na 138, K 5.0, Cl 100. State the disorder. (2 marks)

- **Anion gap** = $138 - (100 + 18) = 20 \text{ mmol/L}$ (mildly elevated) **[core]**
- **Expected $p\text{CO}_2$ if pure HAGMA** = $1.5 \times 18 + 8 = 35 \text{ mmHg}$. Actual 60 — much higher → primary respiratory acidosis dominates with co-existing HAGMA. **Likely diagnosis: respiratory failure (pulmonary cause) with secondary lactic acidosis [core]**

b) Patient B: pH 7.55, $p\text{CO}_2$ 50, bicarbonate 42, Na 140, K 3.0, Cl 88. State the disorder. (2 marks)

- **Expected $p\text{CO}_2$ for pure metabolic alkalosis** = $0.7 \times 42 + 21 = 50.4 \text{ mmHg}$. Actual 50 — appropriate compensation **[core]**
- **Pure metabolic alkalosis with appropriate respiratory compensation**; low chloride, low potassium → consistent with vomiting / nasogastric losses **[core]**

c) Patient C: pH 7.35, $p\text{CO}_2$ 28, bicarbonate 14, Na 142, K 4.0, Cl 102, lactate 6. State the disorder. (2 marks)

- **Anion gap** = $142 - (102 + 14) = 26 \text{ mmol/L}$ (elevated) **[core]**
- **Expected $p\text{CO}_2$ (Winter's)** = $1.5 \times 14 + 8 = 29 \text{ mmHg}$. Actual 28 — appropriate. **HAGMA from lactic acidosis with appropriate respiratory compensation**; pH near-normal because compensated. Cause: hypoperfusion / sepsis **[core]**

d) Patient D: pH 7.42, $p\text{CO}_2$ 50, bicarbonate 32. State the disorder (consider chronic). (2 marks)

- **Acute respiratory acidosis**: expected $\Delta\text{HCO}_3^- = +1$ per $+10 p\text{CO}_2$ rise (above 40); $(50-40)/10 \times 1 = 1$; expected $\text{HCO}_3^- = 24 + 1 = 25$ — actual is 32, much higher than expected for acute **[core]**
- **Chronic respiratory acidosis with compensation**: expected $\Delta\text{HCO}_3^- = +4$ per $+10 p\text{CO}_2 = 4$; expected $\text{HCO}_3^- = 24 + 4 = 28$ — still lower than actual 32 → could be **chronic respiratory acidosis with added metabolic alkalosis** (e.g. diuretic use); pH near-normal (over-compensated) **[core]**

e) Patient E: pH 7.30, $p\text{CO}_2$ 30, bicarbonate 14, Na 134, K 4.5, Cl 113, glucose 22. State the disorder. (2 marks)

- **Anion gap** = $134 - (113 + 14) = 7 \text{ mmol/L}$ (NORMAL anion gap) **[core]**
- **Expected $p\text{CO}_2$ (Winter's)** = $1.5 \times 14 + 8 = 29 \text{ mmHg}$. Actual 30 — appropriate. **Pure NAGMA with appropriate respiratory compensation**; hyperchloraemia (Cl 113); raised glucose may suggest **diabetes with diabetic ketoacidosis recovery phase** (NAGMA from chloride / saline therapy) OR Type 4 RTA / hyperalimentation. Note: in established DKA with HAGMA, treatment with 0.9 percent saline causes hyperchloraemic NAGMA **[core]**

References: eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements • Tintinalli's Emergency Medicine 9e Ch 117 • Australian / NZ ICU acid-base teaching resources