

FExNet 2026

Collated SAQs: Endocrine Emergencies

ANSWER BOOKLET

Model answers for the 22 endocrine SAQs. References cited at the foot of each question. Items marked [core] are required for full marks. Drug doses and protocols confirmed with current ADS / JBDS / ISPAD diabetes guidelines, American and European Thyroid Association guidelines, Endocrine Society guidelines, and ESE Hyponatraemia Guideline.

22 questions • 225 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.

Answer Index

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

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Question 1 | Paediatric Diabetic Ketoacidosis and Cerebral Oedema (11 marks)

A 5-year-old boy weighing 18 kilograms is brought by his mother to your emergency department with a two-day history of fever, vomiting, and progressive lethargy. There is no prior history of diabetes mellitus. On arrival: temperature 38.8 °C, heart rate 145 beats per minute, blood pressure 90/40 millimetres of mercury, respiratory rate 40 breaths per minute, peripheral oxygen saturation 91 percent on room air, Glasgow Coma Score 13 (eye 3, verbal 4, motor 6). A venous blood gas shows pH 7.18, partial pressure of carbon dioxide 21 millimetres of mercury, bicarbonate 8 millimoles per litre, base excess -18.5, sodium 140, potassium 5.7, chloride 98, glucose 38 millimoles per litre, ketones (beta-hydroxybutyrate) 5.8 millimoles per litre.

a) State the diagnosis AND give the 2 biochemical criteria from ISPAD that confirm it (3 items). (3 marks)

- **New-onset Type 1 diabetes mellitus presenting in diabetic ketoacidosis (DKA)** — likely precipitated by an intercurrent febrile illness (? pneumonia given fever) **[core]**
- **Hyperglycaemia** — venous/capillary blood glucose >11 mmol/L (here 38) **[core]**
- **Metabolic acidosis with ketosis** — venous pH <7.30 OR bicarbonate <15 mmol/L (here pH 7.18, HCO₃ 8) AND ketonaemia (β-OHB ≥3 mmol/L) or moderate-to-large ketonuria **[core]**
- Severity: **severe DKA** (pH <7.1 OR bicarbonate <5); moderate (pH 7.1-7.2 or HCO₃ 5-10); mild (pH 7.2-7.3 or HCO₃ 10-15)

b) Outline 5 priorities in the first-hour management of this child per ISPAD / APLS guidelines, including specific fluid and insulin doses. (5 marks)

- **Resuscitation if shocked:** 10 mL/kg 0.9% sodium chloride bolus over 20-30 min (here 180 mL); reassess and repeat once if persistent shock. Do **NOT** give rapid large boluses unless truly shocked — rapid fluid is a risk factor for cerebral oedema **[core]**
- **Maintenance + deficit fluid:** assume 5-7% dehydration; replace deficit evenly over 48 hours with 0.9% NaCl + 20-40 mmol/L potassium (target K 4-5). Total rate (maintenance + deficit) — typically 5 mL/kg/h initial; subtract any boluses from deficit **[core]**
- **Delay insulin for 1 hour** after starting IV fluids — then commence **insulin infusion at 0.05-0.1 units/kg/hour** (here 0.9-1.8 units/h). **No bolus.** Aim glucose fall of 4-5 mmol/L/h **[core]**
- **Potassium replacement:** K appears high (5.7) but total body K is depleted; add KCl 40 mmol/L to fluids once insulin started or urine output established. Monitor 2-hourly initially **[core]**
- **Identify and treat precipitant:** septic workup (CXR, urine, blood cultures), empiric antibiotics if suspected pneumonia; treat underlying infection. Continuous monitoring with hourly neurological observations, hourly BSL, 2-hourly electrolytes, strict fluid balance **[core]**
- Add **5% dextrose** to fluids when BSL falls to 14-17 mmol/L (do NOT stop insulin)
- Bicarbonate **NOT routinely indicated** — associated with cerebral oedema and paradoxical CSF acidosis (consider only if pH <6.9 and refractory shock)

c) Two hours into treatment the child becomes drowsy with a Glasgow Coma Score of 9, develops a headache, and his heart rate drops from 140 to 65 beats per minute. State the diagnosis AND name 2 specific treatments. (3 marks)

- **Cerebral oedema** — most feared complication of paediatric DKA (mortality 20-25%); Cushing reflex (bradycardia, hypertension) with deteriorating GCS **[core]**
- **Hypertonic 3% saline 2.5-5 mL/kg over 10-15 minutes** (here ≈ 45-90 mL) — first-line per ISPAD; OR mannitol 0.5-1 g/kg IV over 15 minutes **[core]**

- **Reduce IV fluid rate by one third**, elevate head of bed 30°, intubate and ventilate if GCS continues to fall (maintain normocapnia — avoid hyperventilation), urgent CT head and PICU/retrieval **[core]**
- Risk factors for cerebral oedema: **young age, new diagnosis, severe acidosis, low pCO₂, high urea, bicarbonate use, rapid fluid administration, failure of corrected sodium to rise during treatment**

References: *ISPAD Clinical Practice Consensus Guidelines (current)* • *Advanced Paediatric Life Support (APLS)* — current edition • *eTG (Therapeutic Guidelines, current)* • *Tintinalli's Emergency Medicine 9e Ch 224* • *RCH Clinical Practice Guideline — DKA*

Question 2 | Adult Severe Diabetic Ketoacidosis (11 marks)

A 28-year-old man with known Type 1 diabetes mellitus presents to your emergency department with two days of vomiting, polyuria, polydipsia, and abdominal pain. He has stopped his insulin because he was vomiting. On arrival: Glasgow Coma Score 14, heart rate 130 beats per minute, blood pressure 95/60 millimetres of mercury, respiratory rate 28 breaths per minute (deep), temperature 37.0 °C. Venous blood gas: pH 7.06, partial pressure of carbon dioxide 14 millimetres of mercury, bicarbonate 4 millimoles per litre, base excess -25, sodium 130, potassium 5.6, chloride 95, glucose 32 millimoles per litre, beta-hydroxybutyrate 6.8 millimoles per litre, creatinine 180 micromoles per litre, lactate 2.4.

a) Name 3 features that classify this episode as severe DKA (ADS / JBDS criteria). (3 marks)

- **pH <7.0** (here 7.06; severe per JBDS/ADS) AND bicarbonate <5 (here 4) **[core]**
- **β-hydroxybutyrate >6 mmol/L** (here 6.8) — marker of severity **[core]**
- **Haemodynamic compromise / hypotension** (SBP <90, HR >100) — here BP 95/60, HR 130; OR GCS <12; OR potassium <3.5 on admission; OR oxygen saturation <92%; OR anion gap >16 **[core]**
- Anion gap $[Na - (Cl + HCO_3)] = 130 - (95 + 4) = 31 \text{ mmol/L}$ — markedly elevated

b) Outline your fluid and insulin management in the first 2 hours per ADS Australian Diabetes Society 2024 guidance (5 items). (5 marks)

- **0.9% sodium chloride 1 L IV stat** (if SBP <90 — bolus 500 mL repeated to achieve SBP >90), then 0.9% NaCl 1 L over 2 hours, 1 L over 4 hours; total deficit 4-6 L over first 24 h **[core]**
- **Fixed-rate intravenous insulin infusion 0.1 units/kg/hour** — for 70 kg, 7 units/hour of actrapid; **no bolus**. Continue patient's usual long-acting basal insulin (glargine/degludec) if previously prescribed **[core]**
- **Potassium replacement**: as soon as K <5.5 add 40 mmol KCl per 1 L 0.9% NaCl (hold insulin if K <3.3 until K replaced); monitor K 1-2 hourly **[core]**
- **Add 10% dextrose 125 mL/hour** when BSL <14 mmol/L; **continue insulin infusion** until ketones <0.6 AND venous pH >7.3 AND bicarbonate >18 — ketones, not glucose, drive duration **[core]**
- **Monitoring**: hourly BSL and ketones, 2-hourly VBG and U&E for 6 hours, strict fluid balance, IDC if anuric or obtunded, telemetry. Aim ketone fall $\geq 0.5 \text{ mmol/L/hour}$ OR HCO_3^- rise $\geq 3 \text{ mmol/L/hour}$ **[core]**
- Bicarbonate **NOT recommended** in adult DKA
- Search for and treat precipitant: infection (CXR, urine, blood cultures), MI (ECG, troponin), missed insulin (most common), pregnancy

c) List 3 complications of DKA treatment to anticipate and prevent. (3 marks)

- **Hypokalaemia** — insulin drives K intracellularly; severe hypoK risks arrhythmia and respiratory weakness. Mandatory K monitoring + replacement **[core]**
- **Hypoglycaemia** — from continued insulin once glucose normalised; add dextrose when BSL <14, monitor hourly **[core]**
- **Cerebral oedema** (rare in adults but described, particularly young adults) — deteriorating GCS during treatment; manage as paediatric DKA **[core]**
- Hyperchloraemic acidosis from large-volume 0.9% NaCl resuscitation — typically self-limiting
- Venous thromboembolism — DKA is pro-thrombotic; consider mechanical and chemical thromboprophylaxis
- Aspiration pneumonitis (gastroparesis, obtunded)

References: Australian Diabetes Society (ADS) Guidelines • JBDS Inpatient Care Group 2023 DKA • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 223 • ACEM clinical policies and statements

Question 3 | Hyperosmolar Hyperglycaemic State (12 marks)

A 78-year-old woman from a residential aged-care facility is brought to your emergency department with a three-day history of progressive lethargy, confusion and reduced oral intake. Past history: Type 2 diabetes mellitus on metformin and gliclazide, hypertension, mild dementia, recent urinary tract infection treated 1 week ago. On arrival: Glasgow Coma Score 10, heart rate 120 beats per minute, blood pressure 90/55 millimetres of mercury, respiratory rate 22 breaths per minute, temperature 38.3 °C, mucous membranes dry, skin turgor poor. Venous blood gas: pH 7.34, partial pressure of carbon dioxide 36, bicarbonate 20, sodium 152, potassium 4.8, chloride 110, glucose 48 millimoles per litre, ketones 0.4, creatinine 210 micromoles per litre, urea 22, lactate 2.1.

a) Define hyperosmolar hyperglycaemic state per JBDS / ADS criteria (4 biochemical features) AND calculate the effective serum osmolality. (4 marks)

- **Severe hyperglycaemia** — glucose ≥ 30 mmol/L (here 48); typically higher than DKA [core]
- **Hyperosmolality** — effective osmolality > 320 mOsm/kg $[2(\text{Na}) + \text{glucose} = 2(152) + 48 = 352 \text{ mOsm/kg}]$ [core]
- **Absent or minimal ketosis** — $\beta\text{-OHB} < 3$ mmol/L (here 0.4); pH ≥ 7.3 and $\text{HCO}_3^- \geq 15$ (here 7.34 and 20) [core]
- **Hypovolaemia** — fluid deficit 100-220 mL/kg (typically 8-12 L vs DKA's 5-7 L); pre-renal AKI common (here Cr 210, urea 22) [core]
- Corrected sodium = measured Na + $2.4 \times ((\text{glucose} - 5.5) / 5.5) = 152 + 2.4 \times (42.5/5.5) \approx 171 \text{ mmol/L}$ — true sodium markedly elevated

b) Outline 5 differences in management from diabetic ketoacidosis. (5 marks)

- **Slower fluid resuscitation** — 0.9% NaCl 1 L over 1 hour then 0.5-1 L/hour for 2-3 hours; aim to replace 50% deficit in first 12 hours, 50% over next 12. **No more than 2 L 0.9% saline in the first hour** unless shocked [core]
- **Delayed insulin** — fluid alone often lowers BSL by 5 mmol/L/hour; commence **insulin 0.05 units/kg/hour** (half DKA dose) only after fluid resuscitation AND if BSL not falling, or if significant ketonaemia present [core]
- **Slower glucose correction**: aim fall 4-6 mmol/L/hour (faster risks cerebral oedema and ODS); use 0.45% NaCl if osmolality not falling despite adequate fluid [core]
- **Cautious sodium correction** — limit fall in corrected sodium to ≤ 10 mmol/L/24 hours (osmotic demyelination risk if too rapid in chronic state) [core]
- **Active VTE prophylaxis** — low-molecular-weight heparin (enoxaparin 40 mg) for ALL patients with HHS; markedly elevated risk of thrombosis (arterial and venous) from hyperviscosity. Aggressive treatment of precipitant [core]
- K replacement: usually less depleted than DKA but still add KCl once K < 5.5 and renal function adequate
- Aim resolution: osmolality < 300 , normalised haemodynamics, conscious level recovered, eating and drinking

c) List 3 likely precipitants of HHS in this patient. (3 marks)

- **Infection (sepsis)** — recent treated UTI may have recurred; pneumonia (check CXR), cellulitis, foot infection [core]
- **Reduced oral intake** from dementia/illness with inadequate fluids in residential care [core]
- **Medications** — corticosteroids, diuretics, antipsychotics, tacrolimus/ciclosporin; new diabetes diagnosis (undertreated) [core]

- Acute MI, CVA, pancreatitis, pulmonary embolism, thyrotoxicosis

References: JBDS Inpatient Care 2022 HHS guideline • Australian Diabetes Society (ADS) Guidelines • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 223

Question 4 | Thyroid Storm (12 marks)

A 48-year-old woman with known Graves' disease on carbimazole presents to your emergency department with fever, agitation and palpitations 2 days after extraction of an infected molar. She has missed her carbimazole for the past 2 weeks. On arrival: Glasgow Coma Score 13 (agitated, confused), temperature 39.8 °C, heart rate 168 beats per minute (atrial fibrillation), blood pressure 100/55 millimetres of mercury, respiratory rate 28, peripheral oxygen saturation 93 percent on room air. Examination: lid lag, diffuse goitre with bruit, fine tremor, brisk reflexes, bibasal crepitations.

a) Name 3 components of the Burch-Wartofsky Point Scale that support a diagnosis of thyroid storm. (3 marks)

- **Thermoregulatory dysfunction:** temperature 39.8 °C scores 25 points (≥ 39.4) — high **[core]**
- **CNS dysfunction:** severe — agitated, confused → 20 points; cardiovascular dysfunction tachycardia 168 → 25 points; AF → 10; congestive HF (crackles) → 10 **[core]**
- **GI/hepatic dysfunction:** nausea, vomiting, diarrhoea, abdominal pain or jaundice → 10 points; presence of **precipitating event** (recent dental infection, withdrawal of antithyroid) scores 10 points **[core]**
- Total ≥ 45 = highly suggestive of thyroid storm; 25-44 = impending storm; < 25 = unlikely. (This patient's score > 75)

b) Outline 6 specific pharmacological treatments in the correct sequence with doses, including the rationale for any specific timing. (6 marks)

- **Propranolol 60-80 mg PO 4-hourly OR 1-2 mg IV slowly** (or esmolol infusion) — β -blockade to control HR and reduce peripheral T4→T3 conversion; use with caution if cardiac failure **[core]**
- **Propylthiouracil (PTU) 500-1000 mg loading PO/NG, then 250 mg 4-hourly** — preferred over carbimazole in storm because PTU also blocks peripheral T4→T3 conversion (alternative: carbimazole 60-80 mg/day if PTU unavailable) **[core]**
- **Lugol's iodine (potassium iodide) 5-7 drops PO 6-hourly OR sodium iodide 0.5-1 g IV** — given **at least 1 hour AFTER PTU/carbimazole** to prevent iodine providing substrate for further hormone synthesis (Wolff-Chaikoff effect) **[core]**
- **Hydrocortisone 100 mg IV 6-hourly** — blocks peripheral T4→T3 conversion AND treats potential coexisting adrenal insufficiency (autoimmune polyglandular) **[core]**
- **Active cooling:** tepid sponging, fans, ice packs to groin/axillae, cold IV fluids; **paracetamol 1 g PO/IV** — **AVOID aspirin** (displaces T4 from thyroid-binding globulin and worsens thyrotoxicosis) **[core]**
- **Treat precipitant:** empiric IV antibiotics for dental source (amoxicillin-clavulanate 1.2 g IV + metronidazole 500 mg IV); rate control AF (β -blocker first; digoxin second-line); fluid resuscitation **[core]**
- Refractory: **plasmapheresis** or therapeutic plasma exchange — last-line for failed medical therapy; thyroidectomy considered after biochemical control
- Avoid **radio-iodine** in storm (worsens thyrotoxicosis); avoid amiodarone, sympathomimetics, salicylates

c) List 3 differential diagnoses to consider. (3 marks)

- **Sepsis / septic shock** with delirium — fever, tachycardia, confusion; thyroid storm can co-exist with sepsis as a precipitant **[core]**
- **Sympathomimetic toxidrome** — cocaine, amphetamines, MDMA; or serotonin syndrome / NMS **[core]**
- **Phaeochromocytoma crisis;** alcohol/sedative withdrawal; heat stroke; anticholinergic toxicity **[core]**
- Malignant hyperthermia; severe DKA; non-thyroid-storm hyperthyroidism (without decompensation)

References: Thyroid Society of Australia and NZ position statement • Burch & Wartofsky 1993; American Thyroid Association Hyperthyroidism Guideline 2016 • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 226

Question 5 | Myxoedema Coma (11 marks)

A 75-year-old woman is brought to your emergency department in winter after being found at home obtunded by her family. Past history: long-standing hypothyroidism on thyroxine which she has stopped taking for 'several months'. On arrival: Glasgow Coma Score 9, temperature 32.1 °C, heart rate 48 beats per minute, blood pressure 85/50 millimetres of mercury, respiratory rate 8 breaths per minute, peripheral oxygen saturation 89 percent on room air. Examination: dry coarse skin, periorbital puffiness, hoarse voice, delayed reflex relaxation, non-pitting oedema. Venous blood gas: pH 7.28, partial pressure of carbon dioxide 65, bicarbonate 30, sodium 122, potassium 4.2, glucose 2.8 millimoles per litre.

a) Name 4 clinical or biochemical features that support a diagnosis of myxoedema coma. (4 marks)

- **Altered mental status** (GCS 9; obtundation; possible seizure or psychosis — 'myxoedema madness') **[core]**
- **Hypothermia** (32.1 °C) — frequently the trigger; correlates with mortality. **Hypoventilation** with type II respiratory failure (PaCO_2 65) from reduced ventilatory response to hypoxia/hypercapnia **[core]**
- **Bradycardia, hypotension**; cardiomegaly with pericardial effusion may be seen on imaging; QT prolongation, low voltage QRS, T-wave inversion on ECG **[core]**
- **Hyponatraemia** (Na 122 — SIADH-like; reduced free water excretion), **hypoglycaemia** (2.8), elevated creatine kinase, normocytic anaemia, raised cholesterol **[core]**
- Precipitating event: **medication non-compliance**, cold exposure, infection, MI, stroke, surgery, sedatives, contrast
- Confirm with TFT: **TSH markedly elevated, free T4 very low** (primary); central hypothyroidism has low TSH and low T4

b) Outline 5 priorities in initial management. (5 marks)

- **Airway and ventilation**: intubate and mechanically ventilate for type II respiratory failure (PaCO_2 65, GCS 9) — caution with sedatives (delayed metabolism); apnoeic oxygenation, etomidate or ketamine RSI **[core]**
- **Hydrocortisone 100 mg IV 6-hourly** BEFORE thyroid hormone — coexisting adrenal insufficiency in 5-10% and thyroid replacement can precipitate adrenal crisis **[core]**
- **Levothyroxine (T4) 200-400 micrograms IV loading**, then 50-100 micrograms IV daily ± **liothyronine (T3) 10-20 micrograms IV 8-hourly** (peripheral T4→T3 conversion impaired in severe illness) **[core]**
- **Passive rewarming** (warm blankets, warm IV fluids 38-42 °C, warmed humidified oxygen). **AVOID active external rewarming** — vasodilation precipitates circulatory collapse **[core]**
- **Cautious fluid resuscitation** with 0.9% NaCl + dextrose if hypoglycaemic; **correct hyponatraemia slowly** (≤ 8 -10 mmol/L/24 h to avoid ODS); empiric antibiotics if infection suspected; treat precipitant; ICU admission **[core]**
- Inotropic support if hypotensive after fluids — noradrenaline (but limited response until thyroid hormone given)

c) List 2 differential diagnoses other than myxoedema coma. (2 marks)

- **Severe primary hypothermia** (without thyroid disease), septic shock with hypothermia, drug overdose (opioid/sedative — slow metabolism) **[core]**
- **Addisonian crisis**, hepatic encephalopathy, hypoglycaemic coma, intracranial event (stroke, subdural haematoma), severe hyponatraemia from another cause **[core]**
- CO_2 narcosis from another aetiology (severe COPD); Wernicke's encephalopathy

References: *European Thyroid Association Myxoedema Coma consensus 2019 • Thyroid Society of Australia and NZ position statement • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 226*

Question 6 | Primary Adrenal Crisis (Addisonian Crisis) (11 marks)

A 32-year-old man with known Addison's disease (primary autoimmune adrenal insufficiency) on hydrocortisone 20 mg / 10 mg / 10 mg and fludrocortisone 100 micrograms daily presents to your emergency department with 3 days of vomiting and diarrhoea. He has not taken his oral steroids due to vomiting. On arrival: Glasgow Coma Score 14, heart rate 130 beats per minute, blood pressure 78/40 millimetres of mercury, respiratory rate 24, temperature 38.5 °C, capillary refill 4 seconds. Initial bloods: sodium 124, potassium 6.2, glucose 2.6 millimoles per litre, creatinine 160, haemoglobin 168 grams per litre (haemoconcentration).

a) Name 4 biochemical or clinical features that support a diagnosis of adrenal crisis. (4 marks)

- **Refractory hypotension** (BP 78/40 — disproportionate to apparent fluid loss) **not responding to fluid alone** [core]
- **Hyponatraemia + hyperkalaemia** (Na 124, K 6.2) — combination strongly suggests **mineralocorticoid deficiency** (primary, not secondary) with sodium wasting and potassium retention [core]
- **Hypoglycaemia** (2.6) — cortisol needed for gluconeogenesis; also seen with secondary AI [core]
- **Pre-renal acute kidney injury** (Cr 160 with haemoconcentration); metabolic acidosis (compensated); fever from precipitating illness (GI infection) [core]
- Eosinophilia, mild hypercalcaemia, normocytic anaemia (if previously undiagnosed); skin hyperpigmentation in primary AI (high ACTH/MSH)

b) Outline 5 priorities of management. (5 marks)

- **Hydrocortisone 100 mg IV stat**, then 200 mg/24 h either by infusion or 50 mg IV/IM 6-hourly — **before any investigation that delays treatment**. If hydrocortisone unavailable: dexamethasone 4 mg IV (preserves subsequent short Synacthen test) [core]
- **0.9% NaCl 1 L IV stat**, then 1 L over 1-2 h, then 1 L over 4 h (typically 4-6 L in first 24 h); **5-10% dextrose** for hypoglycaemia (50 mL of 50% dextrose if symptomatic, then maintenance dextrose-containing fluid) [core]
- **Hyperkalaemia treatment**: 10 mL 10% calcium gluconate if ECG changes; insulin-dextrose; salbutamol nebulised — but K usually corrects with steroid + fluid alone within 4-6 h [core]
- **Search for and treat precipitant**: gastroenteritis, infection (CXR, urine, blood cultures, stool culture); empiric antibiotics if febrile; check thyroid function and ?coexisting autoimmune polyglandular syndrome [core]
- **Avoid fludrocortisone in acute phase** — high-dose hydrocortisone (>50 mg/24 h) has sufficient mineralocorticoid activity; reintroduce fludrocortisone when oral; **monitor** BSL, electrolytes, BP; ICU/HDU admission [core]
- Education and follow-up: sick day rules, MedicAlert bracelet, emergency hydrocortisone 100 mg IM ampoule for home use

c) State 2 sick day rules every patient with chronic adrenal insufficiency must follow. (2 marks)

- **Double or triple oral hydrocortisone dose** for fever >38 °C, vomiting <24 h, minor surgery; continue until recovery [core]
- **Hydrocortisone 100 mg IM injection** (carried at all times) followed by ED presentation for: persistent vomiting, trauma, major surgery, severe infection; MedicAlert bracelet identifying steroid dependence [core]
- Major surgery: hydrocortisone 100 mg IV at induction + 200 mg/24 h infusion until tolerating oral intake

References: Endocrine Society Primary Adrenal Insufficiency Clinical Practice Guideline 2016 • Australasian Endocrine Society Sick Day Rules • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 225

Question 7 | Severe Sulfonylurea-Induced Hypoglycaemia (10 marks)

A 72-year-old woman with Type 2 diabetes mellitus and chronic kidney disease (estimated glomerular filtration rate 28 millilitres per minute per 1.73 square metres) on glibenclamide 10 mg daily is brought to your emergency department after a witnessed seizure at home. Paramedics administered intramuscular glucagon 1 milligram followed by 10% dextrose 200 millilitres intravenously, with a rise in blood sugar level from 1.4 to 5.6 millimoles per litre. On arrival in your resuscitation bay she is rousable but drowsy (Glasgow Coma Score 13), her blood sugar level is now 3.2 millimoles per litre.

a) Name 4 features specific to sulfonylurea hypoglycaemia that influence management. (4 marks)

- **Long duration of action** — glibenclamide $t_{1/2}$ 10 h, active metabolites renally excreted (worse with low eGFR); hypoglycaemia typically persists or recurs for 24-72 hours **[core]**
- **Mechanism is endogenous insulin secretion** (closes pancreatic β -cell K-ATP channels) — bolus dextrose alone triggers further insulin release and rebound hypoglycaemia **[core]**
- **Renal impairment markedly prolongs effect** — glibenclamide and active metabolites are renally cleared; this patient (eGFR 28) at very high risk of prolonged hypoglycaemia **[core]**
- **Glucagon less effective** than in insulin hypoglycaemia (may transiently raise BSL but glycogen stores depleted, and triggers further insulin release); **octreotide** is the specific antidote **[core]**
- Older patients more susceptible (reduced counter-regulatory hormone response, polypharmacy, renal impairment)

b) Outline 4 ongoing management priorities. (4 marks)

- **Octreotide 50-100 micrograms subcutaneously every 6-12 hours for 24-48 hours** (or continuous infusion 25 micrograms/hour) — somatostatin analogue suppresses pancreatic insulin release; **specific antidote** to sulfonylurea **[core]**
- **10% dextrose IV infusion to maintain BSL 5-10 mmol/L** (e.g. 75-125 mL/hour) rather than bolus 50% dextrose; bolus 50 mL of 50% dextrose reserved for BSL <3 mmol/L with symptoms **[core]**
- **Frequent BSL monitoring** — hourly for 12 hours, then 2-hourly for 48-72 hours minimum; admit to monitored bed given recurrence risk **[core]**
- **Withhold sulfonylurea**, review and switch to safer alternative (DPP-4 inhibitor, SGLT2i contraindicated if eGFR <30, insulin); review CKD medication doses; carer / community support; deprescribing review; MedicAlert if recurrent **[core]**
- Discharge criteria: BSL maintained without intervention for ≥ 24 hours, sulfonylurea ceased, sensible carer follow-up; **do not discharge from ED** after a single resolved episode
- Consider non-accidental ingestion in atypical cases (eg paediatric, deliberate)

c) List 2 other ED differentials for refractory hypoglycaemia. (2 marks)

- **Insulin overdose** (deliberate or accidental — long-acting analogue with prolonged effect; injection-site fat depot) **[core]**
- **Insulinoma** (rare); adrenal insufficiency; severe liver failure; sepsis; alcohol-induced (impaired gluconeogenesis); β -blocker poisoning; quinine-induced; surreptitious insulin (Munchausen) **[core]**
- Reactive / post-bariatric hypoglycaemia (post-prandial)

References: Murray's Toxicology 2.5e — Sulfonylureas • Australian Diabetes Society (ADS) Guidelines • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 224

Question 8 | Pheochromocytoma Crisis (11 marks)

A 38-year-old man presents to your emergency department with sudden-onset severe headache, chest pain, palpitations, sweating and a sense of impending doom. He has had three similar but milder episodes over the past 6 months. On arrival: heart rate 130 beats per minute, blood pressure 240/130 millimetres of mercury, temperature 37.6 °C, respiratory rate 22, profuse diaphoresis, pallor. Electrocardiogram shows sinus tachycardia with lateral ST depression. Bedside echocardiogram demonstrates a moderately reduced left ventricular ejection fraction with anterior wall hypokinesis.

a) State the suspected diagnosis AND list 2 features that argue for it over essential hypertension. (3 marks)

- **Pheochromocytoma crisis with catecholaminergic cardiomyopathy** (takotsubo-like ventricular dysfunction and ischaemic ECG changes) [core]
- **Episodic 'classical triad'** — headache, palpitations, sweating (specificity ≈95%, sensitivity ≈90%) — with pallor (rather than flushing) and 'feeling of doom' [core]
- **Paroxysmal hypertension** with severe surges (often 240+/120+); **orthostatic hypotension between episodes** from intravascular volume contraction; young patient; associated MEN2/VHL/NF1/SDH mutations [core]
- Resistant hypertension despite ≥3 agents; secondary HTN screen abnormalities (24-hour urine or plasma metanephrines); incidental adrenal mass

b) Outline 5 immediate management steps for this crisis, with specific drug doses. (5 marks)

- **α-blockade FIRST: phentolamine 1-5 mg IV boluses** (short-acting α₁+α₂-blocker; titrated to BP) OR phenoxybenzamine; sodium nitroprusside infusion 0.5-3 micrograms/kg/min (caution cyanide toxicity) [core]
- **Never start β-blocker before α-blockade** — unopposed α-stimulation causes severe vasoconstriction, hypertensive crisis and pulmonary oedema. Once α-blocked, add β-blocker (esmolol infusion or metoprolol 5-10 mg IV) for HR >100 or tachydysrhythmia [core]
- **Magnesium sulphate 2-4 g IV** (useful adjunct — inhibits catecholamine release at adrenergic nerve terminals and direct vasodilator); reassess BP/HR continuously [core]
- **Volume resuscitation** with 0.9% NaCl — patients are typically volume contracted from catecholamine-induced natriuresis; titrated to BP after α-blockade. Aim diastolic 80-90 mmHg (not normotension — risk of hypoperfusion) [core]
- **Search for and treat triggers:** drugs (cocaine, sympathomimetics, monoamine oxidase inhibitors, metoclopramide, beta-blockers as monotherapy), abdominal palpation, anaesthesia/surgery, micturition; admit ICU/HDU; urgent endocrine and surgical referral; CT abdomen for tumour localisation; plasma/24-hour urine metanephrines [core]
- Treat associated complications: pulmonary oedema, MI (avoid β-blockers initially), CVA, lactic acidosis

c) List 3 syndromes associated with pheochromocytoma. (3 marks)

- **Multiple Endocrine Neoplasia type 2A and 2B (MEN2A, MEN2B)** — RET proto-oncogene mutation; associated with medullary thyroid carcinoma + hyperparathyroidism (2A) or marfanoid habitus and mucosal neuromas (2B) [core]
- **Von Hippel-Lindau syndrome** — VHL gene; renal cell carcinoma, haemangioblastomas, pancreatic cysts [core]
- **Neurofibromatosis type 1 (NF1)** — café-au-lait spots, neurofibromas, axillary freckling; **Succinate dehydrogenase (SDH) B/D mutations** — familial paraganglioma syndrome [core]
- Carney triad (paraganglioma + gastric stromal tumour + pulmonary chondroma)

References: *Endocrine Society Pheochromocytoma and Paraganglioma Clinical Practice Guideline 2014* • eTG (Therapeutic Guidelines, current) • *Tintinalli's Emergency Medicine 9e Ch 228*

Question 9 | Pituitary Apoplexy (10 marks)

A 55-year-old man presents to your emergency department with sudden-onset severe occipital headache, followed by vomiting and diplopia. He is on no regular medications. On arrival: Glasgow Coma Score 14, heart rate 110, blood pressure 85/55 millimetres of mercury, temperature 36.0 °C. Examination: bitemporal field defect on confrontation, right-sided ptosis with a dilated pupil that does not react to light, restricted right eye adduction and elevation, no neck stiffness. CT head shows a haemorrhage within an enlarged pituitary fossa with no subarachnoid blood.

a) State the diagnosis AND name 2 clinical features that support it. (3 marks)

- **Pituitary apoplexy** — haemorrhagic or ischaemic infarction of a pre-existing pituitary adenoma (often previously undiagnosed); endocrine and neurosurgical emergency **[core]**
- **Bitemporal hemianopia** from chiasmal compression; **oculomotor (CN III) palsy with mydriasis** from cavernous sinus involvement (CN III most often affected, also IV, VI, V1, V2) **[core]**
- **Sudden thunderclap headache** (differential SAH); **hypopituitarism** with hypotension out of proportion to volume status (acute ACTH deficiency → secondary adrenal insufficiency); MRI shows haemorrhagic pituitary mass **[core]**
- Pre-existing features of a pituitary mass (amenorrhoea, galactorrhoea, gigantism/acromegaly, Cushingoid)

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

- **Hydrocortisone 100 mg IV stat** followed by 50 mg 6-hourly or infusion 200 mg/24 h — empirical steroid replacement for assumed ACTH deficiency; **do not delay for biochemistry [core]**
- **Resuscitation:** 0.9% NaCl IV 1-2 L; correct any glucose (5-10% dextrose if low); careful fluid balance (risk of central diabetes insipidus OR SIADH) **[core]**
- **Urgent endocrine bloods:** cortisol, ACTH, TSH/free T4, prolactin, LH/FSH, GH/IGF-1, sodium (hyponatraemia common — SIADH or adrenal); paired plasma and urine osmolality if polyuric **[core]**
- **Urgent MRI pituitary** (gold standard — CT misses up to 30%) and **neurosurgical referral** — transsphenoidal decompression for reduced GCS, severe or progressive visual field defect, ophthalmoplegia not improving with steroids **[core]**
- **Formal visual fields, acuity, ophthalmology assessment;** analgesia; anti-emetics; **thyroid hormone replacement** only AFTER hydrocortisone started (if hypothyroid — precipitates crisis); admit to monitored bed **[core]**
- Avoid bromocriptine if prolactinoma — although may shrink tumour, can precipitate further infarction

c) Name 2 differential diagnoses for the presentation. (2 marks)

- **Subarachnoid haemorrhage** — thunderclap headache, CN palsy (particularly posterior communicating artery aneurysm with CN III palsy) **[core]**
- **Bacterial / viral meningitis or encephalitis; cavernous sinus thrombosis; internal carotid artery dissection;** brainstem infarction; basilar artery aneurysm **[core]**
- Severe migraine; sphenoid sinusitis; Tolosa-Hunt syndrome

References: UK Pituitary Apoplexy Guideline (Society for Endocrinology) • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 226 • ACEM clinical policies and statements

Question 10 | Severe Hypercalcaemia of Malignancy (11 marks)

A 68-year-old woman with metastatic breast carcinoma presents to your emergency department with 4 days of progressive lethargy, confusion, constipation and polyuria. Past history: bony metastases on letrozole and denosumab (which she stopped 3 months ago). On arrival: Glasgow Coma Score 12, heart rate 110, blood pressure 95/55 millimetres of mercury, respiratory rate 18, temperature 36.7 °C, mucous membranes very dry, urine output 50 millilitres per hour. Initial bloods: corrected calcium 3.85 millimoles per litre (reference 2.10-2.60), albumin 28 grams per litre, phosphate 1.6, creatinine 220 micromoles per litre, urea 18, potassium 3.2.

a) Calculate the corrected calcium AND name 2 ECG features of severe hypercalcaemia. (3 marks)

- **Corrected Ca = measured Ca + 0.02 × (40 – albumin).** Here total Ca not given, but corrected = 3.85 mmol/L is stated — **severe hypercalcaemic crisis (>3.5 mmol/L)**; alternative formula: $\text{Ca} + 0.02 \times (40 - 28) \rightarrow +0.24$ [core]
- **Short QT interval** (shortened ST segment) — characteristic; **bradyarrhythmias, heart blocks, broadened T waves; Osborn (J) waves** with severe hypercalcaemia may appear similar to hypothermia [core]
- **Ventricular ectopy or VT/VF** at very high levels; digoxin sensitivity increased (avoid loop diuretics + digoxin combination if possible) [core]
- Symptoms: **'stones, bones, abdominal groans, psychic moans, polyuric tones'** — renal calculi, bone pain, constipation/pancreatitis, confusion/depression, nephrogenic DI

b) Outline 6 specific treatments with doses and expected time to onset. (6 marks)

- **0.9% sodium chloride 1-2 L IV over 1-2 hours**, then 200-300 mL/hour to maintain urine output ≥ 2 mL/kg/hour (total 4-6 L in first 24 h) — restores GFR, dilutes serum Ca, promotes calciuresis. **Onset: hours; reduces Ca by 0.3-0.5 mmol/L** [core]
- **Furosemide** (e.g. 20-40 mg IV) only **after** volume replete — promotes calciuresis but causes hypokalaemia and hypomagnesaemia; **not routine** per current guidance (only if fluid overload) [core]
- **Zoledronic acid 4 mg IV over 15 minutes** (single dose; reduce if eGFR <60) OR pamidronate 60-90 mg IV over 2-4 hours — bisphosphonate inhibits osteoclast bone resorption. **Onset 24-72 hours; peak 4-7 days; duration weeks** [core]
- **Calcitonin (salmon) 4 IU/kg IM/SC 12-hourly for 24-48 hours** — rapid onset (hours) reduces Ca by 0.3-0.5 mmol/L; **tachyphylaxis after 48 hours** — bridge therapy until bisphosphonate effective [core]
- **Denosumab 120 mg SC** (RANK-ligand monoclonal antibody) — alternative if renal impairment or bisphosphonate failure; **onset 4-10 days**; previously used by this patient; **note rebound hypercalcaemia** on cessation [core]
- **Hydrocortisone 100 mg IV 6-hourly OR prednisolone 40 mg PO daily** — effective for vitamin-D-mediated hypercalcaemia (lymphoma, myeloma, sarcoidosis, vitamin D toxicity); **haemodialysis with low-Ca dialysate** if Ca >4.5 mmol/L, renal failure, refractory to medical therapy, severe symptoms [core]
- Treat underlying malignancy; cinacalcet for parathyroid carcinoma or PTH-mediated; avoid thiazides, lithium, calcium supplements, vitamin D

c) List 2 mechanisms by which malignancy causes hypercalcaemia. (2 marks)

- **Humoral hypercalcaemia of malignancy (PTHrP-mediated)** — squamous cell lung, breast, head & neck, renal cell — PTHrP secretion mimics PTH (suppressed endogenous PTH on testing) [core]
- **Osteolytic bone metastases / local cytokine release** — breast cancer (this patient), multiple myeloma, lymphoma; or **ectopic calcitriol (1,25-OH-vit-D) production** by lymphoma, sarcoidosis,

granulomatous disease **[core]**

- Rebound hypercalcaemia after withdrawal of denosumab (relevant to this patient)

References: Endocrine Society Hypercalcaemia of Malignancy Guideline • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 228 • BMJ Best Practice — Hypercalcaemia 2024

Question 11 | Severe Hypocalcaemia Post-Thyroidectomy (10 marks)

A 42-year-old woman presents to your emergency department on day 2 after a total thyroidectomy for multinodular goitre. She describes peri-oral tingling, paraesthesia in the hands and feet, muscle cramps, and one brief generalised tonic-clonic seizure en route. On arrival: Glasgow Coma Score 15, heart rate 95, blood pressure 130/80 millimetres of mercury, temperature 37.0 °C. Carpopedal spasm is demonstrated after blood pressure cuff inflation for 2 minutes. Bloods: total calcium 1.40 millimoles per litre (corrected 1.50), ionised calcium 0.71 millimoles per litre, magnesium 0.42, phosphate 2.10, parathyroid hormone <0.3 picomoles per litre.

a) Name 3 clinical signs or ECG features of severe hypocalcaemia. (3 marks)

- **Trousseau sign** — carpopedal spasm with BP cuff inflated above SBP for 3 minutes (demonstrated here); **Chvostek sign** — facial twitch on tapping facial nerve **[core]**
- **Seizures** (generalised or focal); **laryngospasm and stridor** (medical emergency); paraesthesia (peri-oral and digits); tetany; muscle cramps **[core]**
- **Prolonged QT interval** on ECG (>500 ms — torsades risk); broad T waves; reduced cardiac contractility with hypotension; refractory cardiac failure **[core]**
- Confusion, delirium, depression, anxiety; basal ganglia calcification on chronic (Fahr's)

b) Outline 5 management priorities including drug doses. (5 marks)

- **Calcium gluconate 10% 10 mL (1 ampoule = 2.2 mmol Ca) IV over 10 minutes** — bolus for symptomatic hypocalcaemia (seizure, tetany, laryngospasm, prolonged QT); may repeat **[core]**
- **Calcium gluconate infusion:** 10 ampoules (100 mL) of 10% calcium gluconate diluted in 1 L of 5% dextrose or 0.9% NaCl, infused at 50-100 mL/hour titrated to ionised calcium 1.0-1.2 mmol/L. Use **calcium gluconate not chloride** peripherally (chloride is irritant; if used must be central) **[core]**
- **Replace magnesium:** magnesium sulphate 2 g (8 mmol) IV over 20 minutes — hypomagnesaemia (here 0.42) impairs PTH secretion and end-organ response to PTH; **hypocalcaemia will not correct until magnesium replete [core]**
- **Activated vitamin D (calcitriol) 0.5-1 microgram PO/IV 12-hourly** AND oral calcium carbonate 1-2 g 8-hourly — for ongoing management of post-surgical hypoparathyroidism (no PTH; this patient PTH <0.3) **[core]**
- **Cardiac monitoring** (QT interval); frequent ionised Ca, Mg, PO₄, K monitoring (2-4 hourly); admit monitored bed; surgical / endocrine review; outpatient endocrine follow-up for likely permanent hypoparathyroidism **[core]**
- Avoid loop diuretics; monitor for hyperphosphataemia (common in hypoparathyroidism); rule out haematoma at surgical site if also airway compromise

c) List 2 other ED causes of severe hypocalcaemia. (2 marks)

- **Massive blood transfusion** (citrate chelation); **acute pancreatitis** (saponification with free fatty acids); **rhabdomyolysis** (early — Ca deposition in injured muscle); tumour lysis syndrome **[core]**
- **Hypomagnesaemia** (PPI, alcoholism, diuretics); **vitamin D deficiency** (housebound elderly); **chronic kidney disease** (secondary hyperparathyroidism with low calcitriol); **hungry bone syndrome** after parathyroidectomy; pseudohypoparathyroidism **[core]**
- Drug-induced: bisphosphonates, denosumab, phenytoin, ethylene glycol (oxalate Ca chelation), hydrofluoric acid

References: eTG (Therapeutic Guidelines, current) • ESE Hypoparathyroidism Guideline 2015 • Tintinalli's Emergency Medicine 9e Ch 228 • ACEM clinical policies and statements

Question 12 | Syndrome of Inappropriate Antidiuretic Hormone (10 marks)

A 68-year-old man with a 50 pack-year smoking history presents to your emergency department with a 1-week history of confusion, lethargy, and nausea. On arrival: Glasgow Coma Score 13, heart rate 78, blood pressure 130/80 millimetres of mercury, temperature 36.8 °C. Examination: euvolaemic (mucous membranes moist, no oedema, JVP not raised, normal skin turgor). Bloods: sodium 118 millimoles per litre, potassium 4.0, urea 2.5 millimoles per litre, creatinine 60, serum osmolality 248 milliosmoles per kilogram, urine osmolality 480 milliosmoles per kilogram, urine sodium 65 millimoles per litre, TSH and morning cortisol normal.

a) Name 4 diagnostic criteria for SIADH (Bartter & Schwartz). (4 marks)

- **Euvolaemic hyponatraemia** (Na <135) with **true hypo-osmolality** (plasma osmolality <275 mOsm/kg) — here 118 and 248 **[core]**
- **Inappropriately concentrated urine** (urine osmolality >100 mOsm/kg in the face of plasma hypo-osmolality) — here 480 **[core]**
- **Urinary sodium >30 mmol/L** on normal salt and water intake (here 65) **[core]**
- **Exclusion** of other causes of hyponatraemia: euthyroid (TSH normal), normal cortisol (no adrenal insufficiency), no diuretics, normal renal function, no clinical hypovolaemia or oedematous state **[core]**
- Low serum uric acid (<0.24 mmol/L) and low urea support diagnosis; both rise with salt-water restriction

b) Outline 4 management priorities for this patient. (4 marks)

- **Symptomatic severe hyponatraemia** (Na <125 with seizure, coma, or severe neurological compromise): **hypertonic 3% saline 150 mL IV over 10-20 minutes**, repeat up to 3 times until 5 mmol/L rise in sodium and symptom improvement. This patient confused — moderate symptoms; cautious 3% saline may still be appropriate **[core]**
- **Cap correction rate** at ≤8-10 mmol/L in first 24 hours, ≤16-18 mmol/L over 48 hours — to avoid **osmotic demyelination syndrome**; frequent sodium monitoring 1-2 hourly during 3% saline, then 4-6 hourly **[core]**
- **Fluid restriction** 500-1000 mL/24 h as first-line for chronic SIADH management; **tolvaptan (V2 antagonist) 15 mg PO** for resistant cases — initiate in hospital with cap on sodium rise **[core]**
- **Investigate underlying cause** (critical in this smoker): **chest X-ray and CT chest** for small cell lung carcinoma (most common cause); CT brain for intracranial pathology; drug review (SSRIs, carbamazepine, MDMA, opiates); imaging for occult malignancy **[core]**
- If correction too fast: re-lower with 5% dextrose ± desmopressin 2-4 micrograms IV/SC

c) List 2 categories of causes of SIADH. (2 marks)

- **Malignancy** — particularly small cell lung carcinoma (paraneoplastic); also head and neck, breast, GI, GU, thymoma, mesothelioma **[core]**
- **CNS disorders** (stroke, haemorrhage, infection, trauma, tumour); **pulmonary disease** (pneumonia, TB, asthma, CF); **drugs** (SSRIs, carbamazepine, MDMA, vincristine, cyclophosphamide, desmopressin, oxytocin, opioids); **postoperative** (especially transsphenoidal); HIV; idiopathic **[core]**
- Hereditary nephrogenic syndrome of inappropriate antidiuresis (NSIAD) — gain-of-function mutation of V2 receptor

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

Question 13 | Diabetes Insipidus (10 marks)

A 35-year-old woman who underwent transsphenoidal resection of a non-functioning pituitary adenoma 2 days ago is referred to your emergency department with profound thirst and polyuria. She reports drinking and passing approximately 8 litres of urine over the last 24 hours. On arrival: Glasgow Coma Score 15, heart rate 110, blood pressure 100/65 millimetres of mercury, dry mucous membranes. Bloods: sodium 156 millimoles per litre, potassium 4.2, creatinine 90, glucose 5.5 millimoles per litre, serum osmolality 320 milliosmoles per kilogram, urine osmolality 95 milliosmoles per kilogram, urine specific gravity 1.001.

a) Name 3 features that confirm diabetes insipidus AND specify whether central or nephrogenic. (3 marks)

- **Hypotonic polyuria** — >3 L/24 h of dilute urine (here 8 L) with urine osmolality <300 mOsm/kg (here 95) and **inappropriately low urine osmolality relative to plasma osmolality** [core]
- **Hypernatraemia with elevated serum osmolality** (Na 156, osm 320 — i.e. ADH should be maximally stimulated; dilute urine confirms ADH problem) [core]
- **Central diabetes insipidus** in this patient (recent pituitary surgery — common cause, classic triphasic pattern: polyuria → SIADH 5-10 days → permanent DI). Distinguish: **response to desmopressin (DDAVP)** — urine osmolality rises >50% (central); <10% (nephrogenic) [core]
- Definitive test: water deprivation test (rarely performed in ED) or measurement of copeptin

b) Outline 5 management priorities for this patient. (5 marks)

- **Replace free water deficit:** Water deficit (L) = $0.6 \times \text{weight} \times ([\text{Na}/140] - 1)$ — for 60 kg patient: $0.6 \times 60 \times (156/140 - 1) \approx 4.1$ L. **Replace half in first 24 hours;** with 5% dextrose IV or oral water if alert. Do NOT lower sodium >10-12 mmol/L per 24 h [core]
- **Desmopressin (DDAVP) 0.5-2 micrograms IV/SC OR 60-240 micrograms intranasal OR 100-200 micrograms PO** — synthetic ADH analogue; titrated to control urine output. Risk of subsequent hyponatraemia if patient drinks normally + DDAVP given [core]
- **Match urine output mL-for-mL with hypotonic fluids** until DDAVP effect or in conscious patient encourage oral water; **strict fluid balance** and hourly urine output; **weigh daily** [core]
- **Frequent sodium monitoring** (2-4 hourly) — risk of overshoot hypernatraemia correction OR subsequent hyponatraemia after DDAVP with continued water intake [core]
- **Endocrine referral;** investigate / rule out coexisting anterior pituitary insufficiency from surgery (cortisol, TSH); avoid intravenous fluid with high sodium load; **MRI pituitary** to confirm post-surgical changes [core]
- Watch for the **triphasic response** after pituitary surgery: 1) polyuria (this patient) 2) transient SIADH days 5-10 — restrict fluid 3) permanent DI (if posterior pituitary destroyed)

c) List 2 causes of nephrogenic diabetes insipidus. (2 marks)

- **Lithium** — most common acquired (up to 40% of long-term users); typically irreversible after years [core]
- **Hypercalcaemia, hypokalaemia,** obstructive uropathy / post-obstructive diuresis, chronic kidney disease (medullary), demeclocycline, foscarnet, amphotericin, sickle cell, hereditary (AVPR2 mutation — X-linked) [core]
- Pregnancy-related (transient — placental vasopressinase) — responds to DDAVP not vasopressin

References: Endocrine Society Diabetes Insipidus Guideline • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 226 • ACEM clinical policies and statements

Question 14 | Hyperthyroidism Evaluation in the Emergency Department (10 marks)

A 45-year-old woman is referred to your emergency department by her general practitioner with newly abnormal thyroid function tests performed for investigation of palpitations, tremor and 6 kg unintentional weight loss over 3 months. She is afebrile and haemodynamically stable. Thyroid function tests: TSH <0.01 milliunits per litre (suppressed), free T4 56 picomoles per litre (elevated), free T3 22 picomoles per litre (elevated).

a) List 4 examination findings that would support thyrotoxicosis. (4 marks)

- **Sinus tachycardia** (resting HR >100 — or atrial fibrillation in 10-15%); **warm peripheries, fine tremor of outstretched hands, brisk reflexes** [core]
- **Goitre** (diffuse in Graves; nodular in toxic multinodular goitre or toxic adenoma); **thyroid bruit** (pathognomonic of Graves — high vascular flow) [core]
- **Graves ophthalmopathy**: exophthalmos (proptosis >20 mm), lid lag (von Graefe sign), lid retraction (Dalrymple sign), conjunctival injection, chemosis, restricted ocular motility [core]
- **Pretibial myxoedema** (Graves; non-pitting violaceous plaques anterior shin); **thyroid acropachy** (clubbing); proximal myopathy; muscle wasting; warm, moist skin; hair thinning; fine straight hair; oligo-/amenorrhoea [core]
- Onycholysis (Plummer's nails); palmar erythema; gynaecomastia

b) Complete the table — biochemical pattern for primary vs secondary hyperthyroidism, AND give 2 specific causes of each. (4 marks)

- **Primary hyperthyroidism**: free T4 high, **TSH suppressed (<0.01)** — thyroid gland over-producing hormone with feedback suppression of pituitary TSH [core]
- **Secondary hyperthyroidism**: free T4 high, **TSH inappropriately normal or elevated** — pituitary or hypothalamic source [core]
- **Causes of primary**: Graves' disease (autoimmune, TSH-receptor antibodies — bruit, eye signs, pretibial myxoedema); **toxic multinodular goitre** (Plummer's — multiple nodules); **toxic adenoma** (single hot nodule); thyroiditis (subacute / De Quervain's — painful, post-viral; post-partum; silent); factitious (T4 ingestion); iodine-induced (amiodarone, contrast) [core]
- **Causes of secondary**: **TSH-secreting pituitary adenoma** (very rare); **resistance to thyroid hormone** (Refetoff syndrome — TR- β mutation); β -hCG-mediated (hyperemesis gravidarum, gestational trophoblastic disease) [core]
- Struma ovarii (ovarian teratoma with ectopic thyroid tissue)

c) List 2 differentiating tests for the cause of primary hyperthyroidism. (2 marks)

- **TSH-receptor antibodies (TRAb) positive in >95% of Graves' disease** — first-line; anti-TPO antibodies also commonly positive but less specific (also Hashimoto's) [core]
- **Thyroid radionuclide uptake scan (99mTc-pertechnetate or 123I)**: diffuse increased uptake (Graves); patchy multiple nodules (TMNG); single hot focus (toxic adenoma); **absent uptake** (thyroiditis, factitious, iodine-induced — distinguishes release of stored hormone from increased synthesis) [core]
- Thyroid ultrasound (high vascular flow in Graves vs nodular in TMNG); serum thyroglobulin (low in factitious; high in destructive thyroiditis)

References: American Thyroid Association Hyperthyroidism Guideline 2016 • Thyroid Society of Australia and NZ position statement • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 226

Question 15 | Deliberate Insulin Overdose (10 marks)

A 22-year-old female health-care worker is brought to your emergency department by ambulance following a witnessed deliberate self-injection with an unknown amount of insulin glargine (long-acting analogue) approximately 1 hour ago. On arrival: Glasgow Coma Score 7, heart rate 110 beats per minute, blood pressure 110/70 millimetres of mercury, respiratory rate 14, temperature 36.4 °C, peripheral oxygen saturation 98 percent on room air. Bedside blood sugar level 1.6 millimoles per litre. Empty insulin pen and suicide note found at the scene.

a) Name 3 features of insulin overdose that differ from sulfonylurea poisoning. (3 marks)

- **Mechanism:** insulin acts directly on peripheral tissues — no requirement for endogenous insulin release (unlike sulfonylureas). C-peptide will be **low** (suppressed exogenous insulin) — useful in factitious cases **[core]**
- **Duration depends on insulin preparation:** ultra-rapid (lispro, aspart) 2-4 h; regular 5-8 h; intermediate (NPH) 12-24 h; long-acting (**glargine, detemir, degludec**) 20-42 h (degludec can be 42+ h). Massive doses form **SC depot** with prolonged absorption — duration much longer than expected **[core]**
- **Hypokalaemia and hypophosphataemia** from intracellular K shift (more pronounced than sulfonylurea); **octreotide is NOT effective** (no role); glucagon less useful (depleted glycogen stores in non-diabetic) **[core]**
- Drug screen, paracetamol, salicylate — assess co-ingestion in deliberate self-harm

b) Outline 5 priorities of management. (5 marks)

- **Airway protection** (GCS 7) — secure airway with RSI if not corrected by dextrose; **50 mL of 50% dextrose IV stat** (25 g) for symptomatic severe hypoglycaemia **[core]**
- **10% dextrose infusion 100-250 mL/hour** via large peripheral or central line, titrated to maintain BSL 5-10 mmol/L; continue for **minimum 24-48 hours for long-acting analogue**; longer if recurrent hypoglycaemia **[core]**
- **Surgical excision / aspiration of subcutaneous depot** — rarely needed but described for massive doses; consider if recurrent and refractory **[core]**
- **Aggressive monitoring:** BSL hourly for 12 hours, 2-hourly for 24-48 hours; replace **potassium and phosphate** (intracellular shift); telemetry; admit to monitored bed **[core]**
- **Psychiatric assessment** for deliberate self-harm — mental health act if required; involve employee occupational health (healthcare worker); social work; risk assessment before discharge **[core]**
- Glucagon 1 mg IM/IV — transient effect only in this scenario (depleted glycogen stores in non-diabetic without recent meal)

c) List 2 disposition considerations specific to this patient. (2 marks)

- **Prolonged monitoring period** (minimum 24 hours, often 48 hours) given long-acting analogue; admit to monitored bed; **do not discharge from ED [core]**
- **Psychiatric admission** after medical stabilisation — formal mental health review, suicide risk assessment, involuntary admission if required; **notification of regulatory body** (AHPRA) may be required for healthcare worker with mental health issues affecting practice **[core]**
- Removal of access to insulin and other medications; carer/family education; outpatient endocrine and psychiatric follow-up

References: Murray's Toxicology 2.5e — Insulin • Australian Toxicology — Insulin Overdose • eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements

Question 16 | Insulin Pump Failure in Type 1 Diabetes (9 marks)

A 17-year-old girl with Type 1 diabetes mellitus on an insulin pump presents to your emergency department with nausea, vomiting and abdominal pain over the past 12 hours. Her pump has displayed a 'no delivery' error for the past 8 hours and she has been unable to contact her diabetes service. On arrival: alert, heart rate 115, blood pressure 110/70, respiratory rate 24, temperature 36.8 °C. Capillary blood glucose 24 millimoles per litre, capillary blood ketones 3.2 millimoles per litre. Venous blood gas: pH 7.28, partial pressure carbon dioxide 32, bicarbonate 16, base excess -9, sodium 134, potassium 3.7, glucose 24, lactate 1.8.

a) Name 3 features that distinguish pump-failure DKA from typical DKA. (3 marks)

- **Rapid onset of ketosis** — pump uses rapid-acting insulin only (no long-acting basal); failure of delivery → ketonaemia within 2-4 hours, severe DKA possible within 4-8 hours **[core]**
- **Often mild biochemical disturbance** (short interval) — moderate metabolic acidosis (pH 7.28; HCO₃ 16) with significant ketonaemia (3.2); glucose often only modestly elevated (24) compared with classical DKA **[core]**
- **Mechanical / device-specific causes:** cannula occlusion, displacement (this patient — 'no delivery'), kinked tubing, depleted reservoir, infusion site infection, battery failure **[core]**
- Pump failure events highly preventable with patient education and a back-up insulin (pen/vial) for emergencies

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

- **Remove pump and give 'rescue' subcutaneous rapid-acting insulin 0.1 unit/kg** (e.g. for 60 kg = 6 units of aspart) immediately, then every 2-3 hours until ketones <0.6 and pH >7.3 — OR commence IV insulin infusion 0.1 unit/kg/hour per standard DKA protocol **[core]**
- **0.9% NaCl IV 1 L over 1 hour**, then maintenance with electrolyte replacement; add **10% dextrose** when BSL falls to 14 **[core]**
- **Address pump failure:** replace cannula at a new site; replace tubing and reservoir; restart pump only after ketones cleared AND under guidance of diabetes team; consider pen/vial back-up for the next 24-48 hours **[core]**
- **Identify trigger:** cannula displacement (most common in pump users); infusion site infection (examine site for erythema, induration, abscess); pump malfunction (contact manufacturer for replacement) **[core]**
- Diabetes team review before discharge; reinforce sick day rules; provide written pump-failure action plan

c) State 2 sick day rules for patients with type 1 diabetes. (2 marks)

- **Never omit insulin** even when unable to eat — basal insulin requirements continue; in vomiting / anorexia switch to sugary fluids and adjust dose (typically reduce short-acting by 25-50%) **[core]**
- **Test BSL and ketones every 2-4 hours** during illness; if ketones >1.5 OR BSL >15 — give additional rapid-acting insulin 0.1 unit/kg every 2-3 hours and seek medical advice; **present to ED** if ketones >3, persistent vomiting, drowsiness, or unable to tolerate fluids **[core]**
- Maintain hydration (200 mL fluid every hour awake); contact diabetes service for guidance

References: Australian Diabetes Society (ADS) Guidelines • ISPAD Clinical Practice Consensus Guidelines (current) • eTG (Therapeutic Guidelines, current) • ACEM clinical policies and statements

Question 17 | Amiodarone-Induced Thyrotoxicosis (10 marks)

A 64-year-old man on amiodarone 200 milligrams daily for 18 months (prescribed for paroxysmal atrial fibrillation) presents to your emergency department with palpitations, weight loss of 6 kilograms, heat intolerance and worsening dyspnoea over 6 weeks. On arrival: heart rate 140 beats per minute (atrial fibrillation), blood pressure 110/70, respiratory rate 22, peripheral oxygen saturation 93 percent on room air, mild fine tremor, lid lag, no goitre, bibasal crackles. Thyroid function tests: TSH <0.01 milliunits per litre, free T4 65 picomoles per litre, free T3 18 picomoles per litre.

a) Complete the table — name 2 mechanisms by which amiodarone causes thyrotoxicosis and give 1 distinguishing feature for each. (4 marks)

- **Type 1 amiodarone-induced thyrotoxicosis (AIT 1): iodine-induced increased hormone synthesis** in a pre-existing abnormal thyroid (TMNG or latent Graves'); high iodine load (each 200 mg amiodarone tablet contains 75 mg organic iodine) **[core]**
- **Type 2 AIT: destructive thyroiditis** with release of preformed hormone from a normal thyroid; amiodarone-induced direct cytotoxic effect — self-limiting over months **[core]**
- **Type 1 distinguishing features:** goitre / nodules on examination; **increased vascularity on colour-Doppler ultrasound; preserved uptake on 99mTc scan** (if not suppressed by iodine load); IL-6 normal-low **[core]**
- **Type 2 distinguishing features:** **no goitre or thyroid tenderness; reduced vascularity on Doppler; absent radio-iodine uptake;** raised serum IL-6; raised thyroglobulin (release from destruction) **[core]**
- Mixed type and Type 2 most common in Australia/NZ (iodine-replete); Type 1 commoner in iodine-deficient regions

b) Outline 4 treatment priorities including drug choices. (4 marks)

- **Do NOT stop amiodarone** reflexively — discuss with cardiology; amiodarone has a half-life of ~100 days and stopping does not give rapid benefit; may be needed for life-threatening arrhythmia **[core]**
- **Type 1 AIT — carbimazole 60 mg/day OR PTU 400-600 mg/day** (anti-thyroid drugs) AND consider **potassium perchlorate 200 mg 8-hourly** (blocks thyroid iodine uptake — limited availability) **[core]**
- **Type 2 AIT — prednisolone 40-60 milligrams orally daily** for 1-3 months, then taper; anti-inflammatory effect; thyrotoxicosis typically resolves **[core]**
- **Mixed / unclear cases:** combine anti-thyroid drug + corticosteroid; **β-blocker** (e.g. propranolol 40-80 mg PO 4-hourly OR esmolol infusion) for symptomatic control of HR / tremor; **thyroidectomy** for refractory cases or severe cardiac failure where amiodarone cannot be ceased **[core]**
- Treat AF: anticoagulate (CHA₂DS₂-VASc); rate control with β-blocker; cardioversion rarely successful until euthyroid

c) Name 2 amiodarone-related thyroid considerations apart from thyrotoxicosis. (2 marks)

- **Amiodarone-induced hypothyroidism** (commoner than AIT in Australia — iodine-replete region; affects ~5-10% of users) — typically in patients with underlying autoimmune thyroid disease **[core]**
- **Baseline and 6-monthly TSH monitoring** for all patients on amiodarone; **elevated TSH alone** in the first 3 months may reflect transient Wolff-Chaikoff effect — recheck before treating **[core]**
- Other amiodarone toxicities to screen for: pulmonary fibrosis, hepatic dysfunction, corneal deposits, skin (blue-grey discolouration)

References: European Thyroid Association Amiodarone and Thyroid Guideline 2018 • Thyroid Society of Australia and NZ position statement • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 226

Question 18 | Secondary Adrenal Insufficiency from Steroid Withdrawal (9 marks)

A 58-year-old woman with polymyalgia rheumatica, previously on prednisolone 10 milligrams daily for the past 18 months, presents to your emergency department with progressive weakness, nausea and dizziness over 2 weeks. Her rheumatologist stopped prednisolone abruptly 3 weeks ago because she had achieved disease remission. She also has a urinary tract infection diagnosed by her GP yesterday and was commenced on trimethoprim. On arrival: heart rate 105 beats per minute, blood pressure 88/52 millimetres of mercury, temperature 37.8 °C, respiratory rate 18, capillary refill 3 seconds. Initial bloods: sodium 131 millimoles per litre, potassium 4.3 (normal), glucose 3.2 millimoles per litre.

a) Name 3 features that distinguish secondary from primary adrenal insufficiency. (3 marks)

- **Normal potassium** (here 4.3) — aldosterone production preserved (controlled by renin-angiotensin, NOT ACTH) so no hyperkalaemia or salt-wasting; in primary AI K is elevated **[core]**
- **Pallor (not hyperpigmentation)** — low ACTH means no MSH-related hyperpigmentation; whereas primary AI has skin/buccal hyperpigmentation **[core]**
- **Cause: abrupt cessation of exogenous steroid** after >3 weeks use (anyone >5 mg prednisolone for >3 weeks may have HPA axis suppression). May also be from pituitary pathology (tumour, apoplexy, Sheehan's, lymphocytic hypophysitis) — typically with other anterior pituitary deficits **[core]**
- Aldosterone is preserved in secondary; synacthen test still abnormal but more equivocal than in primary

b) Outline 4 management priorities for this patient. (4 marks)

- **Hydrocortisone 100 mg IV stat**, then 50 mg IV 6-hourly or infusion 200 mg/24 hours — same dose as primary; do not wait for confirmatory tests **[core]**
- **0.9% NaCl 1 L IV over 1 hour + dextrose 5-10%** for hypoglycaemia (BSL 3.2); maintenance fluids; monitor BSL and electrolytes **[core]**
- **Treat precipitant:** trimethoprim for UTI (review choice — trimethoprim can cause hyperkalaemia; alternative cephalexin if K rising); blood and urine cultures; CXR; **review all medications** for interactions **[core]**
- **Resume oral steroid — prednisolone 10 mg daily** with slow weaning over months; **sick day rules** (double dose for febrile illness, IM hydrocortisone for vomiting); MedicAlert; outpatient endocrine review; synacthen test once stable **[core]**
- **NEVER stop chronic steroids abruptly** — wean over weeks-months; HPA axis recovery may take 6-12 months

c) List 2 other medications/situations causing iatrogenic HPA suppression. (2 marks)

- **Inhaled corticosteroids** (high-dose fluticasone, especially in children); **topical / intra-articular corticosteroids**; **chronic opioid use**; **megestrol acetate** **[core]**
- **Chronic high-dose oral steroids** for any indication (asthma, IBD, rheumatological); **etomidate** (transient adrenal suppression — particularly in septic shock — single induction dose generally OK in haemodynamically stable patients); **ketoconazole, fluconazole, itraconazole, rifampicin** (induce hepatic metabolism of cortisol — increase steroid requirement) **[core]**
- Post-bilateral adrenalectomy; post-pituitary surgery / radiotherapy; Sheehan's syndrome

References: Endocrine Society Adrenal Insufficiency Guideline 2016 • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 225

Question 19 | Hyperthyroidism in Pregnancy (10 marks)

A 28-year-old woman at 16 weeks gestation in her first pregnancy presents to your emergency department with palpitations, tremor and a 4 kilogram weight loss despite normal appetite. She has a previous history of Graves' disease in remission for 4 years off treatment. Her GP has performed thyroid function tests today: TSH <0.01 milliunits per litre, free T4 50 picomoles per litre (elevated), free T3 18 picomoles per litre (elevated), TSH-receptor antibody titre 8.0 international units per litre (elevated). On arrival: heart rate 110, blood pressure 130/80, respiratory rate 18, afebrile, mild lid lag, no goitre.

a) Name 4 differential diagnoses for new hyperthyroidism in pregnancy AND state which is most likely for this patient. (4 marks)

- **Recurrence of Graves' disease** (this patient — previous Graves, raised TRAb titre) — typically presents in first or second trimester **[core]**
- **Gestational transient thyrotoxicosis (GTT) / hyperemesis gravidarum-related** — high β -hCG cross-stimulates TSH receptor; typically self-limiting by 20 weeks; TRAb **negative** **[core]**
- **Gestational trophoblastic disease** (molar pregnancy / choriocarcinoma) — very high β -hCG; uterine size large for dates; ultrasound shows 'snowstorm' appearance **[core]**
- **Other:** toxic multinodular goitre, toxic adenoma, subacute thyroiditis, amiodarone-induced (uncommon in pregnancy), factitious; **most likely diagnosis** in this patient: **recurrence of Graves' disease** given history and raised TRAb **[core]**
- Struma ovarii — rare; β -hCG-secreting tumours

b) Outline 4 specific management considerations in pregnancy. (4 marks)

- **Drug choice by trimester: propylthiouracil (PTU)** 100-150 mg 8-hourly is preferred in the **first trimester** (carbimazole/methimazole associated with aplasia cutis and choanal/oesophageal atresia 'methimazole embryopathy'). **Switch to carbimazole** (15-30 mg/day) in the second trimester to reduce PTU hepatotoxicity **[core]**
- **Target free T4 at the upper end of normal** for trimester (not aggressive control — risk of fetal hypothyroidism from transplacental drug transfer); monthly TFTs and dose titration **[core]**
- **Avoid:** radio-iodine (absolutely contraindicated — fetal thyroid ablation); **β -blocker only short-term** (propranolol can cause IUGR, fetal bradycardia, neonatal hypoglycaemia — limit to weeks); **thyroidectomy** reserved for refractory cases ideally in second trimester **[core]**
- **Monitor fetal thyroid function** and check **maternal TRAb titre at 18-22 weeks** — TRAb crosses placenta and can cause fetal/neonatal Graves' (fetal goitre, tachycardia, growth restriction); high TRAb requires obstetric referral; deliver in tertiary centre **[core]**
- Multidisciplinary review: endocrinology, obstetrics, neonatology

c) Name 2 risks of untreated hyperthyroidism in pregnancy. (2 marks)

- **Maternal:** thyroid storm (often precipitated by delivery, infection, pre-eclampsia, surgery); cardiac failure; atrial fibrillation; pre-eclampsia **[core]**
- **Fetal / neonatal:** miscarriage; preterm birth; intrauterine growth restriction; fetal goitre; neonatal Graves' disease (TRAb-mediated); fetal death; congenital malformations **[core]**
- Postpartum exacerbation common — frequent TFT monitoring in puerperium

References: American Thyroid Association Pregnancy Guideline 2017 • RANZCOG Statement on Thyroid in Pregnancy • Thyroid Society of Australia and NZ position statement • eTG (Therapeutic Guidelines, current)

Question 20 | Cushing's Syndrome — Emergency Department Presentation (9 marks)

A 42-year-old man presents to your emergency department with progressive weight gain over 12 months, easy bruising, lower back pain, and new diagnosis of diabetes mellitus and hypertension in the past 6 months. On arrival: heart rate 88, blood pressure 180/105 millimetres of mercury, temperature 36.8 °C, body mass index 38. Examination: facial plethora and rounded ('moon') facies, supraclavicular and dorsocervical fat pads, central obesity with thin extremities, wide purple striae over abdomen and thighs, proximal myopathy, multiple ecchymoses, fungal skin infection in groin. Random capillary blood glucose 18 millimoles per litre, potassium 2.9 millimoles per litre.

a) List 3 clinical features that have high discriminatory value for Cushing's syndrome. (3 marks)

- **Wide (>1 cm) purple striae** (thin atrophic skin permits visualisation of dermal vasculature); facial plethora; dorsocervical and supraclavicular fat pads **[core]**
- **Proximal muscle weakness** (catabolic effect — difficulty rising from chair, climbing stairs); easy bruising / ecchymoses; thin skin **[core]**
- **Unexplained osteoporotic fractures** (vertebral, ribs) in young adult; new diabetes + hypertension + central obesity in same patient (especially if rapid onset) **[core]**
- Hypokalaemia (mineralocorticoid effect of high cortisol); psychiatric (depression, mania, psychosis); recurrent infections; menstrual disturbance; impotence

b) Outline 4 initial investigations to confirm diagnosis AND determine cause. (4 marks)

- **Confirm hypercortisolism — 3 first-line tests, any 2 of which must be abnormal** : (1) **24-hour urinary free cortisol** ($\geq 3\times$ upper limit), (2) **late-night salivary cortisol** (loss of diurnal rhythm), (3) **1 mg overnight dexamethasone suppression test** (failure to suppress <50 nmol/L) **[core]**
- **ACTH measurement** to determine ACTH-dependent vs ACTH-independent: **elevated/normal ACTH** → ACTH-dependent (pituitary adenoma Cushing's disease 70%, ectopic ACTH 10-15%); **suppressed ACTH** → ACTH-independent adrenal cause (adrenal adenoma/carcinoma) **[core]**
- **High-dose dexamethasone suppression (8 mg) and CRH stimulation test** — differentiate pituitary (Cushing's disease — suppresses) from ectopic ACTH (does not suppress); inferior petrosal sinus sampling for equivocal cases **[core]**
- **Imaging**: pituitary MRI for Cushing's disease (microadenoma in 60-80%); CT abdomen for adrenal mass; CT thorax for small cell lung carcinoma (ectopic ACTH); chest X-ray; smoking history **[core]**
- Always exclude exogenous steroid use **FIRST** (commonest cause — including topical, inhaled, intra-articular)

c) List 2 immediate ED treatment priorities. (2 marks)

- **Manage hypertension and hyperglycaemia — acute**: short-acting anti-hypertensives (avoid spironolactone monotherapy as potassium losses already high); insulin sliding scale or long-acting; potassium replacement for hypokalaemia **[core]**
- **Endocrine referral** for definitive evaluation; **treat infection** (common — immunosuppressed); **thromboprophylaxis** (hypercoagulable state — high VTE risk); **bone protection** (vitamin D, calcium, bisphosphonate); psychiatric review if depressed/psychotic **[core]**
- Steroidogenesis inhibitors (metyrapone, ketoconazole) for severe acute Cushing's awaiting definitive treatment

References: Endocrine Society Cushing's Syndrome Guideline 2015 • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 225

Question 21 | Post-Bariatric Hypoglycaemia (9 marks)

A 38-year-old woman is brought to your emergency department after a witnessed loss of consciousness while shopping. She had a Roux-en-Y gastric bypass 3 years ago with 50 kilograms weight loss. The episode occurred approximately 90 minutes after a meal of muffin and orange juice. Paramedics documented a capillary blood glucose of 1.8 millimoles per litre; she regained consciousness after intravenous 10% dextrose. On arrival: alert, blood sugar level 5.4 millimoles per litre, previously similar episodes treated as 'low blood sugar' with snacks.

a) State the likely diagnosis AND give 2 features supporting it. (3 marks)

- **Post-bariatric hypoglycaemia (post-prandial / late-dumping hypoglycaemia)** — typically occurs 1-3 years post-Roux-en-Y bypass or gastrectomy; pathophysiology: rapid delivery of glucose to small bowel → exaggerated GLP-1 / insulin response → rebound hypoglycaemia 1-3 hours post-prandial **[core]**
- **Timing** — 1-3 hours after a high-glycaemic-index meal (this patient muffin + juice 90 min before); **resolution with carbohydrate; NO fasting hypoglycaemia** (which would suggest insulinoma) **[core]**
- **Whipple's triad** documented: (1) symptoms consistent with hypoglycaemia, (2) low plasma glucose (1.8) measured at time of symptoms, (3) symptoms resolve with correction of hypoglycaemia **[core]**
- Distinguish from **early dumping** (within 15-30 min — hyperosmolar fluid in small bowel; vasomotor symptoms WITHOUT hypoglycaemia)

b) Outline 4 management strategies (acute and long-term). (4 marks)

- **Acute:** 10% dextrose infusion (this ED visit) for severe hypoglycaemia (LOC); monitor BSL hourly until stable then every 2 hours for 8 hours; observe in ED for at least 4-6 hours post-meal to ensure no recurrence **[core]**
- **Dietary modification (first-line):** low-glycaemic-index meals; small frequent meals (6+ per day); separate solid food and liquid by 30 minutes; high protein + complex carbohydrates; avoid simple sugars and rapidly absorbed carbohydrates (juice, white bread, sweets); fibre supplementation **[core]**
- **Pharmacological (second-line):** **acarbose 25-100 mg with each meal** (α -glucosidase inhibitor — slows carbohydrate absorption); **diazoxide** (reduces insulin release); **octreotide 50 micrograms SC three times daily before meals** for refractory cases; **GLP-1 receptor antagonists** (avexitide) emerging **[core]**
- **Surveillance and follow-up:** continuous glucose monitoring; bariatric surgery / endocrine team; consider gastric pouch reduction or reversal of bypass if refractory; education on driving and occupational safety (loss-of-awareness episodes → driving restriction per Austroads) **[core]**
- Differential: insulinoma (work-up with 72-hour fast — plasma glucose, insulin, C-peptide, proinsulin, β -OHb)

c) List 2 other ED considerations for non-diabetic hypoglycaemia. (2 marks)

- **Workup of fasting hypoglycaemia** (unlike this patient — post-prandial): insulinoma (insulin / C-peptide / proinsulin all elevated at time of hypoglycaemia); factitious insulin (insulin high, C-peptide LOW); sulfonylurea (insulin and C-peptide both elevated, drug screen positive); IGF-2 producing tumour **[core]**
- **Driving advice** (per Austroads Assessing Fitness to Drive 2022): hypoglycaemia with impaired awareness or loss of consciousness — non-private driver must not drive; private driver must not drive until reviewed and risk addressed **[core]**
- Recurrent unexplained hypoglycaemia warrants endocrine investigation

References: Endocrine Society Hypoglycaemia in Diabetes and Non-Diabetic Guideline • Australian Diabetes Society (ADS) Guidelines • eTG (Therapeutic Guidelines, current) • Austroads Fitness to Drive 2022

Question 22 | Fasting Hypoglycaemia Workup (9 marks)

A 28-year-old office worker presents to your emergency department by ambulance following a witnessed loss of consciousness at work in the morning before breakfast. Paramedics documented a capillary blood glucose of 1.2 millimoles per litre with rapid recovery after 50 millilitres of 50% dextrose intravenously. She has had two similar fasting episodes in the past 6 months. She is not diabetic, takes no medications, and denies recent dieting, alcohol, or self-administered insulin. On arrival: alert, blood sugar level now 6.8 millimoles per litre, examination unremarkable. She is 65 kilograms with a body mass index of 24.

a) List 4 critical investigations to send at the time of hypoglycaemia (paired specimens). (4 marks)

- **Plasma glucose** (laboratory confirmed — capillary may be inaccurate at low values) AT THE TIME of symptoms (Whipple's triad) **[core]**
- **Plasma insulin AND C-peptide AND proinsulin** — distinguish endogenous insulin secretion (all three elevated — insulinoma or sulfonylurea) from exogenous insulin (insulin high, C-peptide LOW — factitious / Munchausen) **[core]**
- **β-hydroxybutyrate** — suppressed by insulin (low <2.7 mmol/L in insulin-mediated hypoglycaemia such as insulinoma, sulfonylurea, insulin administration); raised in non-insulin-mediated (alcohol, starvation, IGF-2 tumour, severe liver disease) **[core]**
- **Sulfonylurea / meglitinide screen** in urine and plasma — clandestine ingestion in healthcare workers, mental health diagnoses, or by accident; **insulin antibodies** for autoimmune hypoglycaemia **[core]**
- Cortisol and ACTH — adrenal insufficiency; growth hormone; IGF-1 and IGF-2 if low; lactate and ammonia for inborn errors

b) Construct a diagnostic algorithm — based on the results, name 3 possible diagnoses. (3 marks)

- **Insulinoma**: insulin high, C-peptide high, proinsulin high, β-OHB low, sulfonylurea negative — typical of pancreatic islet cell tumour; investigate with 72-hour fast, endoscopic ultrasound, MRI pancreas **[core]**
- **Sulfonylurea ingestion** (deliberate or accidental): insulin high, C-peptide high, proinsulin high, β-OHB low, **sulfonylurea screen POSITIVE** — may be Munchausen, in healthcare worker, or accidental in confused elderly **[core]**
- **Exogenous insulin (factitious / Munchausen)**: insulin high, **C-peptide LOW**, proinsulin low, β-OHB low; analogue-specific immunoassays available **[core]**
- **Non-insulin-mediated** (β-OHB high): alcohol-induced (no synthesis substrate); starvation; cortisol/GH deficiency; non-islet-cell tumour producing IGF-2; severe liver disease; sepsis

c) Outline 2 disposition / safety considerations. (2 marks)

- **Admit for monitoring and observation** given recurrent episodes with loss of consciousness; **fasting study under supervision** (up to 72-hour) for diagnostic confirmation **[core]**
- **Driving restriction (Austroads)**: hypoglycaemia with impaired awareness or loss of consciousness — must not drive until reviewed and cause addressed; **occupational review** for safety-critical work; **mandatory psychiatric review** if factitious / deliberate suspected **[core]**
- Endocrine referral for definitive workup; education on emergency management for family / carers (oral glucose, glucagon kit)

References: Endocrine Society Adult Hypoglycaemic Disorders Guideline 2009 • Australian Diabetes Society (ADS) Guidelines • eTG (Therapeutic Guidelines, current) • Austroads Fitness to Drive 2022 • Tintinalli's Emergency Medicine 9e Ch 224