

FExNet 2026

Collated SAQs: Vascular Emergencies

ANSWER BOOKLET

Model answers for the 22 vascular SAQs. References cited at the foot of each question.

22 questions • 218 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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Question 1 | Stanford Type A Aortic Dissection (12 marks)

A 58-year-old man presents to your emergency department with sudden-onset severe interscapular pain radiating to the chest and back, associated with diaphoresis and a brief syncopal episode 90 minutes ago. Past history: long-standing untreated hypertension, smoker. On arrival: Glasgow Coma Score 14, heart rate 105 beats per minute, blood pressure 80/45 millimetres of mercury on the right arm and 130/85 on the left arm, respiratory rate 24, peripheral oxygen saturation 94 percent on room air. Examination: diastolic murmur at the right sternal edge, muffled heart sounds, left-sided hemiparesis. Twelve-lead electrocardiogram shows ST-elevation of 2 millimetres in leads II, III and aVF. Bedside echocardiogram demonstrates a pericardial effusion with right ventricular collapse during diastole.

a) Name 4 clinical features that support a diagnosis of Type A aortic dissection. (4 marks)

- **Sudden-onset severe 'tearing' interscapular/chest pain** — maximum at onset (vs gradual onset of acute coronary syndrome); often migratory along the line of dissection **[core]**
- **Blood pressure differential >20 mmHg between arms** (here 80/45 vs 130/85 — 50 mmHg differential) — involvement of subclavian/innominate artery; pulse deficits in 30% **[core]**
- **Aortic regurgitation murmur** (diastolic at right sternal edge) in 40-75% of Type A — proximal dissection disrupts aortic valve cusps; **cardiac tamponade** with pericardial effusion (15-20%) — leading cause of death **[core]**
- **End-organ malperfusion: stroke / focal neurological deficit** (here left hemiparesis — carotid/right innominate involvement), **inferior STEMI** (right coronary artery from aortic root dissection — here II, III, aVF), syncope (15%), mesenteric/renal/spinal cord ischaemia **[core]**
- Risk factors: hypertension (70%), Marfan/Ehlers-Danlos/Loeys-Dietz, bicuspid aortic valve, Turner, vasculitis, pregnancy (3rd trimester), iatrogenic, cocaine, weightlifting

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

- **Resuscitation area, two large-bore IV lines, full monitoring, arterial line in unaffected arm**; group and cross-match 6 units; activate massive transfusion protocol; **cardiothoracic surgery and ICU consultation immediately [core]**
- **Anti-impulse therapy** — reduce dP/dT (rate of pressure rise): first **β-blocker** (esmolol infusion 50-300 micrograms/kg/min OR labetalol 10-20 mg IV boluses) to **target HR <60** and SBP 100-120 mmHg; then add vasodilator (GTN or sodium nitroprusside 0.5-3 micrograms/kg/min) for refractory hypertension. **Never use vasodilator without prior β-blockade** (reflex tachycardia worsens dissection propagation) **[core]**
- **Urgent imaging**: CT angiography chest/abdomen/pelvis is the gold standard if haemodynamically stable; **transoesophageal echocardiography (TOE)** for unstable patients; transthoracic echo (TTE) misses 40% — useful for tamponade only **[core]**
- **Treat tamponade: limited pericardial drainage (50-100 mL)** if haemodynamic collapse — bridge to theatre only (further drainage may cause re-bleed and complete arrest); **permissive hypotension targeting SBP 70-90 mmHg** until theatre **[core]**
- **Emergency cardiothoracic surgery** — aortic root/ascending aorta repair (potentially valve-sparing) is the definitive treatment; mortality without surgery 1-2% per hour for the first 24 hours, 50% at 48 hours, 80% at 14 days; **do NOT delay for retrieval if cardiothoracic surgery is available on-site [core]**
- **Avoid thrombolysis** for the apparent STEMI (coronary ischaemia is from dissection — thrombolysis is contraindicated and fatal); **analgesia** (IV opioids); anti-emetics

c) List 3 differential diagnoses. (3 marks)

- **Acute coronary syndrome (STEMI)** — particularly inferior MI from RCA (may coexist as complication of dissection). **Pulmonary embolism** (massive — chest pain, syncope, haemodynamic collapse) **[core]**

- **Acute pericarditis with tamponade** (any cause); **tension pneumothorax**; **oesophageal rupture (Boerhaave)** — sudden severe chest pain with subcutaneous emphysema; **perforated peptic ulcer**; **acute pancreatitis [core]**
- **Stroke (ischaemic)** — particularly in absence of pain (silent dissection in 5-15%); musculoskeletal pain; anxiety / panic disorder **[core]**
- **Aortic intramural haematoma** (variant — manage similarly to dissection)

References: ESC Aortic Disease Guideline 2014/2024 • AHA/ACC Aortic Disease Guideline 2022 • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 60 • ACEM clinical policies and statements

Question 2 | Stanford Type B Aortic Dissection (11 marks)

A 64-year-old man presents to your emergency department with severe interscapular and lumbar back pain that started suddenly 4 hours ago while gardening. Past history: hypertension, hypercholesterolaemia. On arrival: Glasgow Coma Score 15, heart rate 95 beats per minute, blood pressure 195/110 millimetres of mercury (similar both arms), respiratory rate 18, peripheral oxygen saturation 97 percent on room air, afebrile. Examination: normal heart sounds, no murmur, no neurological deficit, all peripheral pulses palpable and symmetrical. CT angiography chest/abdomen/pelvis confirms an uncomplicated Stanford Type B aortic dissection with the intimal tear originating just distal to the left subclavian artery.

a) Define Stanford Type A and Type B dissection AND state the implication for management. (3 marks)

- **Stanford Type A:** dissection involves the ascending aorta (regardless of distal extent); **requires emergency surgical repair** — mortality 1-2%/hour untreated due to tamponade, valve disruption, coronary involvement, stroke **[core]**
- **Stanford Type B:** dissection distal to the left subclavian artery only (ascending aorta spared); **medical management is first-line** for uncomplicated; **thoracic endovascular aortic repair (TEVAR)** for complicated **[core]**
- **'Complicated' Type B:** end-organ malperfusion (mesenteric, renal, spinal cord, lower limb ischaemia); refractory hypertension/pain despite maximal medical therapy; rapid aortic expansion; rupture or impending rupture; → consider TEVAR within 1-2 weeks **[core]**
- DeBakey classification (alternative): Type I (ascending + arch + descending — surgical), Type II (ascending only — surgical), Type III (descending only — medical)

b) Outline 5 components of medical management for uncomplicated Type B dissection. (5 marks)

- **HDU / monitored bed admission** with arterial line, two IV lines, telemetry, hourly neurovascular observations of all limbs for malperfusion **[core]**
- **β-blocker** — **esmolol IV infusion 50-300 micrograms/kg/min OR labetalol infusion** titrated to **heart rate <60 beats per minute** (reduces dP/dT); transition to oral metoprolol or carvedilol once stable **[core]**
- **Vasodilator (GTN or sodium nitroprusside)** ONLY after β-blockade established, titrated to **SBP target 100-120 mmHg**; alternatives: nicardipine, clevidipine (longer-acting) **[core]**
- **Analgesia** — IV opioids (morphine or fentanyl) — pain control reduces sympathetic drive and BP; anti-emetics; fluid resuscitation only if haemodynamically unstable **[core]**
- **Vascular surgical consultation** for ongoing care, surveillance imaging (CT angiogram at 1, 3, 6, 12 months), and consideration of TEVAR if complications develop; tertiary transfer if not at vascular centre; **long-term BP control** SBP <120, smoking cessation, statin **[core]**
- Tight BP control **halves** the 5-year risk of aortic-related death

c) List 3 indications for TEVAR (thoracic endovascular aortic repair) in Type B dissection. (3 marks)

- **Malperfusion syndrome** — visceral, renal, lower limb, spinal cord ischaemia from extension of dissection **[core]**
- **Rupture or impending rupture** (periaortic haematoma, contrast extravasation); rapid aortic expansion (>5 mm in 6 months); aortic diameter >55 mm **[core]**
- **Refractory hypertension or pain** despite maximal medical therapy; **persistent / recurrent pain** suggesting ongoing extension **[core]**

- INSTEAD trial supports TEVAR for prevention of late aortic-related events in stable Type B

References: ESC Aortic Disease Guideline 2014/2024 • INSTEAD trial • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 60 • Australian and New Zealand Society for Vascular Surgery position statement

Question 3 | Acute Limb Ischaemia (12 marks)

A 74-year-old man presents to your outer-metropolitan emergency department with severe sudden-onset right leg pain 4 hours ago. There is no history of trauma. Past history: atrial fibrillation (anticoagulation refused), hypertension. On arrival: Glasgow Coma Score 15, heart rate 110 irregular, blood pressure 145/80, respiratory rate 18. Examination of the right leg: cool, pale, mottled below the knee, tenderness on calf compression, paraesthesia over the dorsum of the foot, weakness of dorsiflexion (power 3/5), absent dorsalis pedis and posterior tibial pulses by palpation and Doppler. The left leg has normal pulses. Your hospital does not have vascular surgery on site.

a) List the 6 Ps of acute limb ischaemia AND classify this patient per the Rutherford criteria. (4 marks)

- **6 Ps:** **Pain** (sudden, severe), **Pulselessness** (absent), **Pallor**, **Paraesthesia** (neuropathia), **Paralysis** (motor — late, ischaemic muscle), **Poikilothermia** (cold limb) [core]
- **Rutherford classification:** **I (viable)** = no sensory/motor loss, audible Doppler signals; **Ila (marginally threatened)** = minimal toe sensory loss, no motor loss, inaudible arterial Doppler [core]
- **Ilb (immediately threatened)** = sensory loss beyond toes, mild-moderate motor loss, inaudible Doppler — this patient [core]
- **III (irreversible)** = profound sensory and motor loss (paralysis, anaesthesia), absent arterial AND venous Doppler signals, fixed mottling of skin — amputation rather than revascularisation [core]
- Time-critical: **6-hour 'golden window'** before ischaemic muscle injury becomes irreversible (varies by tissue tolerance)

b) Outline 5 immediate management steps while arranging transfer to a vascular centre. (5 marks)

- **Resuscitation, IV access × 2, full bloods:** FBC, U&E, coagulation, CK, lactate, troponin, group and hold, fasting bloods, ECG (likely AF — embolic source) [core]
- **Heparin — unfractionated heparin infusion 5000 units IV bolus, then 1000 units/hour** (titrated to APTT 60-80) — prevents clot extension; **continue until time of revascularisation** [core]
- **Analgesia — IV opioid** (morphine 2.5-5 mg IV titrated); **avoid cooling AND warming the limb; position dependently** (20-30° below heart) to aid arterial perfusion; protect from pressure [core]
- **Urgent vascular surgery referral AND retrieval activation** for transfer to vascular centre; **CT angiogram** of aorta to ankle before transfer if it doesn't delay revascularisation; document time of onset (clock-starts for ischaemia) [core]
- **Pre-empt rhabdomyolysis and hyperkalaemia:** continuous ECG; 0.9% NaCl IV to maintain urine output >1 mL/kg/h; potassium replacement withheld; pre-emptive bicarbonate may be considered; communicate to receiving team [core]
- Patient education: revascularisation may not be possible, may require amputation, mortality 10-20% from co-morbidity

c) Differentiate embolic and thrombotic causes (3 features). (3 marks)

- **Embolic:** **sudden onset, no previous claudication, identifiable source** (atrial fibrillation — this patient, recent MI with LV thrombus, endocarditis, atherosclerotic plaque embolism, paradoxical via PFO); contralateral limb usually normal [core]
- **Thrombotic:** **more gradual onset** (hours-days), **history of claudication and atherosclerotic disease** on the affected side; contralateral limb often also abnormal (reduced ABI, claudication); typically less severe than embolic at presentation due to collateralisation [core]
- **Management implication:** **embolectomy / Fogarty** for embolic (relatively normal vessel proximal and distal to clot); **thrombolysis or bypass / endovascular intervention** for thrombotic (diffuse)

atherosclerotic disease) **[core]**

- Other causes: dissection (extension into iliac or popliteal), trauma (direct vascular injury), popliteal aneurysm thrombosis, vasospasm

References: *ESVS Acute Limb Ischaemia Guideline 2020* • *ANZSVS position statement* • *eTG (Therapeutic Guidelines, current)* • *Tintinalli's Emergency Medicine 9e Ch 64* • *NSW Institute of Trauma and Injury Management*

Question 4 | Lower Limb Deep Vein Thrombosis (10 marks)

A 45-year-old woman presents to your emergency department with a 4-day history of progressive right calf swelling and pain. She returned 5 days ago from an 18-hour flight from Europe. Past history: combined oral contraceptive use for 8 years. On arrival: alert and well, heart rate 88, blood pressure 130/80, afebrile. Examination: right calf 4 centimetres larger in circumference than left at 10 centimetres below the tibial tuberosity, mild pitting oedema, calf tenderness, mildly erythematous, no superficial varicosities. D-dimer 2200 (reference <500). Compression ultrasound confirms thrombus extending from the popliteal vein into the common femoral vein with non-compressibility.

a) List 4 components of the Wells score for DVT AND state how it guides workup. (4 marks)

- **Wells Score for DVT** (each = 1 point unless stated): **active cancer** (within 6 months); **paralysis, paresis, recent plaster immobilisation of lower limb; recently bedridden ≥ 3 days OR major surgery within 12 weeks** (requiring anaesthesia) **[core]**
- **Local tenderness** along the deep venous system; **entire leg swelling; calf swelling >3 cm** compared to asymptomatic leg (measured 10 cm below tibial tuberosity); **pitting oedema** in symptomatic leg only **[core]**
- **Collateral superficial veins** (non-varicose); **previous documented DVT** (1 point); **alternative diagnosis at least as likely as DVT** (–2 points) **[core]**
- **Interpretation:** ≥ 2 = DVT likely (this patient — 2 points from swelling + oedema or 3 with calf circumference) → **proceed to compression USS**; < 2 = DVT unlikely → **D-dimer first**, USS only if positive **[core]**
- D-dimer is sensitive but non-specific — limited utility in cancer, pregnancy, recent surgery, elderly; use age-adjusted cut-off in >50 years (age $\times$ 10 ng/mL)

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

- **Anticoagulation choice:** **direct oral anticoagulant (DOAC) — rivaroxaban 15 mg PO BD for 21 days then 20 mg daily** OR apixaban 10 mg BD for 7 days then 5 mg BD — first-line for uncomplicated proximal DVT; **LMWH if active cancer or DOAC contraindicated** (pregnancy, severe renal impairment, antiphospholipid syndrome) **[core]**
- **Outpatient vs inpatient:** **outpatient management** appropriate for haemodynamically stable, no extensive iliofemoral thrombosis, low bleeding risk, compliant patient, follow-up arranged; **admit** if extensive iliofemoral, phlegmasia, suspicion of PE, high bleeding risk, comorbidity **[core]**
- **Duration of anticoagulation:** **3 months** for provoked DVT (transient risk factor — surgery, OCP, flight); **extended/indefinite** for unprovoked, recurrent, ongoing risk (cancer, antiphospholipid syndrome); weigh against bleeding risk **[core]**
- **Aetiology workup AND patient advice:** **cease OCP** (precipitant + ongoing risk); consider thrombophilia screen (only if results would alter duration of anticoagulation — controversy); compression stockings (class II, 20-30 mmHg) for post-thrombotic syndrome prevention; patient education re symptoms of extension or PE **[core]**
- Iliofemoral DVT or phlegmasia → consider catheter-directed thrombolysis

c) Name 2 considerations for DVT in pregnancy. (2 marks)

- **DOACs and warfarin are contraindicated in pregnancy** (teratogenic; cross placenta); **low-molecular-weight heparin (enoxaparin) is the agent of choice** (does not cross placenta); typically twice-daily weight-adjusted dosing throughout pregnancy AND 6 weeks post-partum **[core]**
- **Diagnostic considerations:** D-dimer commonly elevated in pregnancy (less specific); **left-sided iliofemoral DVT more common** (gravid uterus compresses left iliac vein — May-Thurner like physiology);

proximal extension and PE more likely; obstetric and haematology input **[core]**

- Switch to unfractionated heparin near term (reversible for delivery); regional anaesthesia requires 24-h LMWH-free interval

References: *THANZ — Thrombosis and Haemostasis Society of ANZ position* • *eTG (Therapeutic Guidelines, current)* • *European Society of Cardiology Guideline (current)* • *Tintinalli's Emergency Medicine 9e Ch 56*

Question 5 | Intermediate-Risk (Submassive) Pulmonary Embolism (11 marks)

A 55-year-old woman presents to your emergency department with sudden-onset pleuritic chest pain and dyspnoea following a 12-hour drive home from a family holiday yesterday. Past history: obesity (BMI 38), recent left knee arthroplasty 4 weeks ago. On arrival: Glasgow Coma Score 15, heart rate 115 beats per minute, blood pressure 115/70 millimetres of mercury, respiratory rate 26, peripheral oxygen saturation 91 percent on room air, afebrile. Bedside echocardiography shows a dilated and hypokinetic right ventricle with septal flattening. CT pulmonary angiography confirms bilateral large lobar pulmonary emboli. Troponin 80 nanograms per litre (reference <30), BNP 480, lactate 1.8.

a) Classify this PE per the ESC 2019 categories AND explain why. (3 marks)

- **Intermediate-risk (submassive) PE — specifically intermediate-HIGH risk [core]**
- **BP preserved** (BP 115/70 — not shocked) — excludes high-risk (massive) PE which requires SBP <90 or vasopressor support [core]
- **BOTH RV dysfunction (dilated/hypokinetic on echo) AND elevated cardiac biomarkers** (troponin 80, BNP 480) → intermediate-HIGH risk; if only one or neither → intermediate-low. **sPESI score** would also help risk-stratify [core]
- Mortality at 30 days: massive 25-65%; intermediate-high 5-15%; intermediate-low 3-5%; low <1%

b) Outline 5 management priorities, including indications for thrombolysis or alternatives. (5 marks)

- **Therapeutic anticoagulation:** LMWH (enoxaparin 1 mg/kg SC BD) OR unfractionated heparin infusion if considering thrombolysis or in renal impairment; DOACs (rivaroxaban / apixaban) as primary therapy in stable patients (avoid in haemodynamic compromise) [core]
- **Thrombolysis in intermediate-high risk: not routine** (PEITHO trial — increased bleeding; no clear mortality benefit). **Indications for rescue thrombolysis:** progressive haemodynamic deterioration (SBP fall, RV failure worsening, lactate rising) [core]
- **Catheter-directed thrombolysis or thrombectomy** — emerging alternative for intermediate-high risk PE — ultrasound-assisted (EKOS), aspiration (FlowTrieve) thrombectomy; consider if PERT (PE Response Team) available at tertiary centre [core]
- **Supportive care:** oxygen to SpO₂ >94%; cautious IV fluids (500 mL crystalloid — large volume worsens RV failure); **noradrenaline first-line vasopressor** in deterioration; **avoid intubation if possible** (positive-pressure ventilation reduces preload — peri-intubation arrest common); admit HDU/ICU [core]
- **Monitor for deterioration:** serial echocardiography, lactate, troponin/BNP, RV function; **IVC filter** ONLY if anticoagulation contraindicated; **PERT activation** for shared decision-making in intermediate-high risk [core]
- Indication for thrombolysis (high-risk/massive): alteplase 100 mg IV over 2 hours (or 0.6 mg/kg as 10-min bolus in cardiac arrest)

c) List 3 contraindications to thrombolysis. (3 marks)

- **Absolute:** active major bleeding; intracranial neoplasm, aneurysm or AVM; previous haemorrhagic stroke or ischaemic stroke in last 3 months; recent major head/spine trauma or surgery (within 3 weeks); known bleeding diathesis [core]
- **Relative:** BP >180/110 not responding to therapy; major non-CNS surgery / trauma within 3 weeks (this patient — recent knee replacement); recent GI bleed; pregnancy or 1 week post-partum; non-compressible vascular puncture; age >75 [core]

- Anticoagulation already in therapeutic range (INR >1.7 or DOAC active); traumatic CPR; active peptic ulcer disease **[core]**
- Recent thrombolysis (within 6 months); endocarditis; aortic dissection

References: ESC PE Guideline 2019 • PEITHO trial • ANZICS / THANZ position statements • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 56

Question 6 | Hypertensive Emergency (11 marks)

A 56-year-old man presents to your emergency department with shortness of breath and central chest pain. Past history: hypertension recently diagnosed, on perindopril 5 milligrams and amlodipine 5 milligrams daily, but admits poor adherence. He has measured his blood pressure at home as 210/110 millimetres of mercury. He is convinced he is going to have a stroke. On arrival: Glasgow Coma Score 15, heart rate 105, blood pressure 215/120 right arm and 212/118 left arm, respiratory rate 22, peripheral oxygen saturation 94 percent on room air, bibasal crackles. ECG: sinus rhythm with left ventricular hypertrophy and lateral T-wave inversion. Chest X-ray: mild pulmonary venous congestion with normal mediastinum. Urinalysis: protein 2+, blood 1+.

a) Define hypertensive emergency vs urgency AND list 4 end-organ manifestations demonstrated in this patient or that should be sought. (4 marks)

- **Hypertensive emergency:** severely elevated BP (usually >180/120) WITH evidence of **acute target organ damage** — requires IV therapy AND controlled BP reduction over hours; **hypertensive urgency:** same BP WITHOUT acute organ damage — oral therapy over days **[core]**
- **Cardiovascular:** **acute pulmonary oedema** (this patient — crackles, CXR congestion), acute coronary syndrome / unstable angina, **aortic dissection** (must rule out with CTA — BP differential, chest pain) **[core]**
- **CNS:** hypertensive encephalopathy (headache, confusion, seizure, focal deficit — see Q20 PRES); intracranial haemorrhage; ischaemic stroke (in this context — permissive hypertension) **[core]**
- **Renal:** **acute kidney injury** with proteinuria/haematuria (this patient — protein 2+, blood 1+) — malignant nephrosclerosis; **retinal:** papilloedema, haemorrhages, exudates (Keith-Wagener-Barker grades III-IV); **microangiopathic haemolytic anaemia:** schistocytes, thrombocytopenia **[core]**
- Pre-eclampsia/eclampsia in pregnancy; pheochromocytoma; cocaine/methamphetamine

b) Outline 5 management priorities. (5 marks)

- **Resuscitation area, IV access × 2, arterial line;** full bloods including troponin, U&Es, FBC, blood film for schistocytes, TSH, urine for protein/blood; **CT angiography** if dissection suspected (this patient's chest pain + severe HTN warrants exclusion) **[core]**
- **BP target — reduce by 20-25% over 1-2 hours, then to <160/100 over 6 hours; avoid rapid normalisation** (cerebral, coronary, renal hypoperfusion in chronically hypertensive patients) **[core]**
- **Drug selection by end-organ involvement:** **pulmonary oedema** → GTN infusion 5-200 micrograms/min + frusemide 40-80 mg IV; **acute coronary syndrome** → GTN + β-blocker; **aortic dissection** → β-blocker first (esmolol/labetalol) then vasodilator; **pheochromocytoma** → phentolamine first; **encephalopathy/PRES** → labetalol or nicardipine **[core]**
- **This patient (pulmonary oedema + AKI):** **GTN infusion 5 micrograms/min titrated**, frusemide 40 mg IV, CPAP if respiratory distress; consider labetalol 10 mg IV boluses; **avoid sublingual nifedipine** (unpredictable severe hypotension) **[core]**
- **Treat precipitants and admit:** review medication adherence, drug screen (cocaine, methamphetamine), investigation for secondary HTN (renal artery stenosis, pheochromocytoma, Cushing's); **HDU admission;** medication review and outpatient follow-up **[core]**
- Hypertensive urgency: oral therapy in ED — amlodipine 5-10 mg or oral labetalol 200-400 mg; outpatient follow-up

c) Name 2 drugs commonly used and 1 contraindication of each. (2 marks)

- **GTN infusion:** contraindicated in **severe aortic stenosis** (reduced preload — collapse), **right ventricular infarction**, **recent phosphodiesterase-5 inhibitor use** (sildenafil within 24 h, tadalafil 48 h — severe hypotension) **[core]**

- **Labetalol** (β -blocker + α -blocker): contraindicated in **severe asthma, acute heart failure with cardiogenic shock, bradyarrhythmia (heart block)**; **sodium nitroprusside**: cyanide toxicity with prolonged use / hepatic / renal impairment **[core]**
- Nicardipine: avoid in advanced aortic stenosis; hydralazine: avoid in acute coronary syndrome / dissection (reflex tachycardia)

References: *NHF Hypertension Guideline 2016/2024 update* • *ESC Hypertension Guideline 2018* • *eTG (Therapeutic Guidelines, current)* • *Tintinalli's Emergency Medicine 9e Ch 59*

Question 7 | Symptomatic Carotid Artery Stenosis and Transient Ischaemic Attack (10 marks)

A 68-year-old man presents to your emergency department after a transient episode of right-sided weakness and expressive dysphasia lasting 30 minutes, occurring 4 hours ago. He now has no neurological deficit. Past history: hypertension, hypercholesterolaemia, 40 pack-year smoking history. On arrival: Glasgow Coma Score 15, heart rate 78 sinus rhythm, blood pressure 165/95, respiratory rate 16. Examination: a carotid bruit is audible over the left bifurcation. ECG: sinus rhythm. CT brain plain is normal.

a) Calculate the ABCD2 score AND state the implication. (3 marks)

- **ABCD2 score:** Age $\geq 60 = 1$ (this patient 68 — 1), BP $\geq 140/90 = 1$ (165/95 — 1), Clinical features (unilateral weakness = 2; speech impairment without weakness = 1) — has both weakness AND dysphasia → **2 points [core]**
- **Duration** (≥ 60 min = 2; 10-59 = 1; $< 10 = 0$) — here 30 min → 1; **Diabetes** = 1 (not present) → 0. **Total = 1 + 1 + 2 + 1 + 0 = 5 [core]**
- **Interpretation:** ABCD2 ≥ 4 = high risk (7.1% 7-day stroke); **requires urgent inpatient workup** — CTA carotids, echocardiography, telemetry for AF; antiplatelet therapy initiated. **Score has limitations** — current Stroke Foundation recommends **urgent specialist review for all TIA** regardless of score **[core]**
- Stroke risk after TIA: 5% at 7 days, 10% at 30 days WITHOUT treatment; halved with antiplatelet and BP control

b) Outline 5 management priorities. (5 marks)

- **Antiplatelet therapy:** aspirin 300 mg loading, then 100 mg daily AND consider clopidogrel 300 mg loading, then 75 mg daily (combination antiplatelet for 21 days in high-risk TIA / minor stroke — CHANCE/POINT trials) **[core]**
- **Urgent CT angiography of carotids and intracranial vessels** (within 24 hours) — looking for stenosis $> 50\%$ in symptomatic carotid (left internal carotid — the symptomatic side here as right-sided symptoms = left hemisphere); MRI/DWI more sensitive for small infarcts **[core]**
- **Investigate cardio-embolic source:** 24-hour Holter monitor (or longer — extended monitoring for paroxysmal AF — CRYSTAL-AF trial); transthoracic echocardiogram (LV thrombus, valve disease); **fasting lipids, HbA1c**; carotid duplex if CTA unavailable **[core]**
- **Carotid endarterectomy (CEA) referral** within 2 weeks (**ideally within 48 hours** if high risk) for **symptomatic stenosis $> 70\%$** (NASCET, ECST trials); **50-69%** in selected (male, hemispheric symptoms); **carotid stenting** alternative in selected patients (age < 70 , restenosis after CEA, hostile neck anatomy) **[core]**
- **Risk factor modification:** statin (high-intensity — atorvastatin 80 mg or rosuvastatin 20-40 mg); **BP control** (target $< 130/80$ long-term, but no acute lowering < 48 h post-event); smoking cessation; diabetes control; **driving cessation** 4 weeks (private), longer for commercial (Austroads) **[core]**
- All patients need urgent stroke or TIA clinic referral; consider admission for ABCD2 ≥ 4 or crescendo symptoms

c) Name 2 'TIA mimics' to exclude. (2 marks)

- **Migraine with aura** (especially atypical/hemiplegic), **focal seizure with Todd's paresis**, **hypoglycaemia**, **conversion disorder**, **structural brain lesion** (tumour, subdural haematoma) **[core]**
- **Vertebrobasilar TIA** (mimics labyrinthitis — vertigo, ataxia, diplopia, dysarthria); **vestibular cause**; **complicated migraine**; **peripheral neuropathy with sudden deterioration**; **multiple sclerosis flare** **[core]**
- Syncope (not TIA — global cerebral hypoperfusion); transient global amnesia

References: *Stroke Foundation Living Guideline 2024 • NASCET; ECST; CHANCE; POINT; CRYSTAL-AF • Austroads Assessing Fitness to Drive 2022 • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 173*

Question 8 | Chronic Limb-Threatening Ischaemia (10 marks)

A 78-year-old man with type 2 diabetes mellitus, hypertension and 50 pack-year smoking history presents to your emergency department with a non-healing ulcer on the right great toe and rest pain in the foot for the past 3 weeks. The rest pain is worse at night and improved by hanging the leg over the bed. Past history includes intermittent claudication at 50 metres for 2 years. On arrival: alert, heart rate 78, blood pressure 150/85, afebrile. Examination: 1.5 centimetre punched-out ulcer over the right great toe with exposed bone, surrounding erythema, malodorous, mild crepitus; cold right foot, capillary refill 5 seconds, absent dorsalis pedis and posterior tibial pulses by Doppler, ankle-brachial index 0.3.

a) Define chronic limb-threatening ischaemia AND state the Rutherford / Fontaine stage. (3 marks)

- **Chronic limb-threatening ischaemia (CLTI)** (formerly 'critical limb ischaemia') = **chronic ischaemic rest pain ≥ 2 weeks AND/OR non-healing wound / gangrene** in the presence of objective evidence of **peripheral arterial disease** (ABI < 0.4 , ankle pressure < 50 mmHg, toe pressure < 30 mmHg) **[core]**
- **Rutherford:** Category 4 (ischaemic rest pain), 5 (minor tissue loss), 6 (major tissue loss/gangrene) — this patient is **Category 5** (minor tissue loss); **Fontaine III (rest pain) / IV (tissue loss)** **[core]**
- **Distinguish from acute limb ischaemia:** chronic with collateralisation, gradual onset, diabetic + atherosclerotic risk; **amputation rate without revascularisation: 30-40% at 6 months; 1-year mortality 25%** **[core]**
- **WIFI score** (wound, ischaemia, foot infection) for limb threat severity

b) Outline 5 management priorities. (5 marks)

- **Urgent vascular surgical referral** for imaging (CT angiography or duplex/MRA) and revascularisation (endovascular angioplasty/stenting OR open bypass surgery — BASIL-2 trial informs choice in CLTI) **[core]**
- **Foot infection management: swab/tissue culture; broad-spectrum antibiotics** (flucloxacillin + metronidazole; piperacillin-tazobactam 4.5 g IV if severe) — concern for osteomyelitis with exposed bone; **plain X-ray foot**, MRI if osteomyelitis suspected; tetanus prophylaxis; surgical debridement **[core]**
- **Medical management of atherosclerosis: antiplatelet** (aspirin 100 mg + clopidogrel 75 mg combination in CLTI / stenting per recent trials), **statin** (atorvastatin 40-80 mg), **ACE inhibitor**, **HbA1c target $< 7\%$** , **smoking cessation**, **BP control** **[core]**
- **Analgesia and wound care:** opioid for rest pain (escalating); non-adherent dressings; off-loading (total contact cast not appropriate for ischaemic ulcer); avoid compression of foot; **multidisciplinary high-risk foot service** (podiatry, vascular, endocrine, infectious disease) **[core]**
- **Discuss prognosis and goals of care:** amputation may be required if revascularisation not feasible (advanced age, multiple comorbidities); discussion with patient and family; **palliative care referral** if comfort approach chosen; rehabilitation and prosthetic planning if amputation **[core]**
- Rivaroxaban 2.5 mg BD + aspirin (COMPASS, VOYAGER PAD trials) reduces MACE and major adverse limb events in PAD/post-revascularisation

c) List 2 features of diabetic foot infection complicating CLTI. (2 marks)

- **Polymicrobial infection** (Gram-positive cocci, Gram-negatives, anaerobes — particularly with deep / chronic / ischaemic wounds); **osteomyelitis common** with exposed bone (probe-to-bone positive $\rightarrow 90\%$ predictive); cellulitis often more extensive than apparent **[core]**
- **Reduced inflammatory response** from diabetic peripheral neuropathy and immunopathy — fever and elevated WCC often absent despite severe infection; **gas in tissues / crepitus** suggests **necrotising soft-tissue infection** (rapid deterioration; polymicrobial including Clostridium); urgent surgical

consultation **[core]**

- Charcot foot (neuroarthropathy) may mimic infection; ABI may be falsely elevated in diabetes (calcified vessels) — use toe pressure

References: GVG (Global Vascular Guidelines on CLTI) 2019 • BASIL-2; COMPASS; VOYAGER PAD • eTG (Therapeutic Guidelines, current) • European Society for Vascular Surgery Guideline • Tintinalli's Emergency Medicine 9e Ch 64

Question 9 | Phlegmasia Cerulea Dolens (9 marks)

A 56-year-old man with metastatic pancreatic cancer presents to your emergency department with sudden severe left lower limb swelling and pain over the past 8 hours. On arrival: Glasgow Coma Score 14, heart rate 130 beats per minute, blood pressure 88/55 millimetres of mercury, respiratory rate 26, peripheral oxygen saturation 92 percent on 6 litres oxygen. Examination: the entire left lower limb is massively swollen from groin to foot, deeply cyanotic and purple, marked tenderness, tense compartments, blistering of the skin, paraesthesia of the foot, sensory loss, weakness of dorsiflexion. Bilateral peripheral pulses are present at presentation but the ankle pulses on the left are now becoming weak. Compression ultrasound confirms occlusive thrombus extending from common iliac to popliteal veins.

a) Define phlegmasia cerulea dolens AND name 2 features that distinguish it from uncomplicated proximal DVT. (3 marks)

- **Phlegmasia cerulea dolens (PCD)** = extensive iliofemoral DVT causing **massive venous obstruction** → **compromise of arterial inflow (compartment-syndrome physiology)** → **venous gangrene**; medical emergency **[core]**
- **Distinguishing features:** **massive limb swelling with cyanosis** (blue limb — vs pale limb in arterial ischaemia); **compartment-syndrome physiology** with tense compartments, neurological deficit; **haemodynamic instability** (sequestration of large volumes in the limb) **[core]**
- **Mortality 25-40%; venous gangrene and amputation 20-50%**; almost always underlying malignancy (this patient — pancreatic Ca), May-Thurner syndrome, post-surgical, antiphospholipid, thrombophilia **[core]**
- Phlegmasia alba dolens — less severe variant (pale + swollen, without cyanosis) — earlier form

b) Outline 4 management priorities. (4 marks)

- **Resuscitation:** IV crystalloid (20 mL/kg) for hypotension; **aggressive correction of hypovolaemia** (large fluid sequestration in affected limb); **leg elevation 30-45°** to reduce venous pressure; analgesia **[core]**
- **Therapeutic anticoagulation immediately:** **unfractionated heparin infusion 80 units/kg bolus then 18 units/kg/hour** (better titration than LMWH in this haemodynamically unstable patient); target APTT 60-80 **[core]**
- **Urgent vascular / interventional radiology referral** for **catheter-directed thrombolysis** (intrathrombus alteplase, often with pharmacomechanical thrombectomy — AngioJet, Trellis); **surgical thrombectomy** as alternative; **IVC filter** may be considered to prevent PE during thrombolysis **[core]**
- **Compartment syndrome:** monitor compartment pressures if available; **fasciotomy** if compartment syndrome confirmed; **amputation** if venous gangrene irreversible; monitor for reperfusion injury (hyperkalaemia, rhabdomyolysis, metabolic acidosis); admit ICU **[core]**
- Treat underlying malignancy; long-term LMWH (CLOT trial — cancer-associated thrombosis)

c) List 2 additional considerations specific to cancer-associated thrombosis. (2 marks)

- **Anticoagulation choice:** **LMWH** remains preferred in cancer-associated VTE (CLOT, CATCH trials — superior to warfarin); **DOACs (rivaroxaban, apixaban, edoxaban)** are non-inferior alternatives (SELECT-D, Hokusai VTE Cancer, CARAVAGGIO) but increased bleeding risk in GI/genitourinary cancer **[core]**
- **Duration:** **indefinite** while active cancer present and ongoing treatment; **shared decision-making** with palliative care if life expectancy <6 months; **monitor for recurrent thrombosis and bleeding** (Khorana score for primary VTE prevention in outpatients) **[core]**
- Treat underlying malignancy where appropriate; consider goals of care discussion

References: ASCO Venous Thromboembolism in Cancer Guideline • CLOT; CATCH; SELECT-D; Hokusai VTE Cancer; CARAVAGGIO • THANZ position statement • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 56

Question 10 | Upper Limb Deep Vein Thrombosis (Paget-Schroetter and Catheter-Related) (9 marks)

A 24-year-old elite swimmer presents to your emergency department with painful swelling of the right (dominant) arm over the past 24 hours, with prominent venous distention over the chest wall. There is no recent trauma. On arrival: Glasgow Coma Score 15, heart rate 78, blood pressure 125/70, afebrile. Examination: marked swelling of the entire right arm with cyanotic discoloration, prominent collateral chest wall veins, mild tenderness. Compression ultrasound shows non-compressible thrombus extending from the right subclavian and axillary veins.

a) State the likely diagnosis AND name 2 predisposing factors. (3 marks)

- **Paget-Schroetter syndrome (effort-induced upper limb DVT)** — primary thrombosis of the subclavian or axillary vein, typically in young athletes with repetitive upper-limb use (swimmers, baseball pitchers, weightlifters) — variant of **venous thoracic outlet syndrome** [core]
- **Mechanical compression of the subclavian vein** between the first rib, clavicle and costoclavicular ligament during overhead arm movements → endothelial trauma and thrombosis (especially with congenital anatomical variants — anomalous muscular insertions, cervical rib) [core]
- **Other causes of upper limb DVT: catheter-related** (CVC, PICC, pacemaker leads, port — >50% of all upper limb DVT); **malignancy** (particularly head and neck or lung); **thrombophilia** (factor V Leiden, antiphospholipid); pacemaker / implantable defibrillator [core]
- Hereditary or acquired hypercoagulability should be screened in young patients

b) Outline 4 management priorities. (4 marks)

- **Therapeutic anticoagulation** (LMWH or DOAC) for minimum 3 months; **commence immediately** while arranging definitive management [core]
- **Catheter-directed thrombolysis** via interventional radiology — preferred for primary effort-induced thrombosis in young patients to preserve venous patency and reduce post-thrombotic syndrome; **followed by definitive decompression surgery (first rib resection / scalenectomy)** within weeks-months to address the underlying anatomical compression [core]
- **Remove the offending catheter** (if catheter-related DVT) — **do NOT remove if still needed** and patient on anticoagulation; consider replacement with smaller-bore or alternative site after anticoagulation initiated [core]
- **Investigation for thrombophilia** (young patient — factor V Leiden, prothrombin gene, protein C/S, antithrombin, antiphospholipid); **cancer screening** if no clear trigger (age-appropriate); **imaging** with MR/CT venography to assess thoracic outlet anatomy [core]
- Avoid repetitive overhead activities until decompression; rehabilitation under specialist vascular and physiotherapy guidance

c) Name 2 differentials for unilateral upper limb swelling. (2 marks)

- **Cellulitis** (warm, erythematous, fever, no venous distention); **lymphoedema** (chronic, often post-axillary node dissection — breast cancer, lymphoma); **superior vena cava obstruction** (both upper limbs ± facial swelling, dyspnoea — malignant or pacemaker-related) [core]
- **Lymphangitis** (red streaks tracking from peripheral wound); **compartment syndrome** (post-trauma); **arterial thrombosis** (pale, cold, pulseless — different presentation); **haematoma**; **septic arthritis** with effusion [core]
- Superficial thrombophlebitis (along course of vein, less swelling); reflex sympathetic dystrophy

References: ACR/ASRA position • THANZ position statement • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 56 • European Society for Vascular Surgery Guideline

Question 11 | Bilateral Renal Artery Stenosis and Flash Pulmonary Oedema (10 marks)

A 78-year-old woman is brought to your emergency department with sudden-onset severe dyspnoea, now her third presentation in 6 months with similar episodes that resolved with diuresis. Past history: hypertension, smoking, peripheral vascular disease. Medications: perindopril 8 milligrams, amlodipine 10 milligrams, metoprolol 50 milligrams, frusemide 40 milligrams. On arrival: Glasgow Coma Score 14, heart rate 110, blood pressure 220/110, respiratory rate 32, peripheral oxygen saturation 78 percent on room air. Examination: diffuse bilateral crepitations, abdominal bruit. CXR: bilateral 'bat-wing' alveolar infiltrates. Bloods: creatinine 280 micromoles per litre (baseline 180), potassium 5.5, BNP 1800. Bedside echo: preserved LVEF 60%, no regional wall motion abnormality.

a) Name 4 features supporting renovascular hypertension with 'flash' pulmonary oedema. (4 marks)

- **'Flash' pulmonary oedema** = recurrent acute pulmonary oedema with **preserved LV systolic function** (LVEF 60% here) on a background of severe hypertension — the Pickering syndrome **[core]**
- **Severe hypertension refractory to ≥ 3 medications** including a diuretic (this patient on 4 agents); **worsening renal function with ACE inhibitor** (creatinine 280 from baseline 180 — bilateral RAS physiology); accelerated renal impairment **[core]**
- **Older age, diffuse atherosclerotic disease**, smoker, peripheral vascular disease, coronary disease; **abdominal bruit** heard on examination (renal artery stenosis specificity); asymmetric kidney sizes (if known) **[core]**
- **BNP elevated** (1800), absent ECG ischaemia, preserved LVEF — hypertension-driven diastolic failure (HFpEF); episodes resolve rapidly with diuresis and BP control **[core]**
- **Fibromuscular dysplasia** — alternative cause of renovascular hypertension in younger women (string of beads on angiogram)

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

- **Non-invasive ventilation (CPAP 10-15 cm H₂O)** for respiratory failure and pulmonary oedema; **oxygen** to SpO₂ >94%; **intubation** if deteriorating or fatigue **[core]**
- **Aggressive diuresis**: frusemide 40-80 mg IV bolus then infusion 5-10 mg/hour; **aggressive BP control**: GTN infusion 5-200 micrograms/min (preload AND afterload reduction); target SBP <160/100 within 1-2 hours **[core]**
- **WITHDRAW the ACE inhibitor** (perindopril) — worsens renal function in bilateral RAS by inhibiting efferent arteriolar constriction; **monitor electrolytes** (hyperkalaemia, K 5.5) **[core]**
- **Inpatient workup**: **CT or MR angiography of renal arteries** (gold standard); duplex ultrasound of renal arteries; vascular surgery / interventional cardiology referral for **renal artery angioplasty + stenting** (controversy — CORAL trial showed no benefit in routine RAS, but selected patients with flash pulmonary oedema or rapidly deteriorating renal function may benefit) **[core]**
- Optimisation of medical therapy is first-line; intervention reserved for refractory cases

c) List 2 ED contraindications to ACE inhibitor / ARB in hypertension management. (2 marks)

- **Bilateral renal artery stenosis OR single functioning kidney with RAS** — precipitates acute renal failure; **hyperkalaemia >5.5**; **pregnancy** (teratogenic — class D); **severe aortic stenosis** (reduces preload — collapse); **angioedema history** **[core]**
- **Concurrent sacubitril/valsartan (ARNI)** — must allow 36 h washout; **severe hyponatraemia**; **volume-depleted state** (initial dose-related hypotension); **acute kidney injury** with ongoing rise in creatinine **[core]**

- Caution with NSAIDs (triple whammy — ACE inhibitor + NSAID + diuretic) — AKI

References: CORAL trial • Pickering syndrome • ESH/ESC HTN Guideline • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 59

Question 12 | Acute Mesenteric Venous Thrombosis (10 marks)

A 42-year-old man presents to your emergency department with 4 days of progressive diffuse colicky abdominal pain, nausea and vomiting. Past history: factor V Leiden heterozygous, previous lower limb DVT 2 years ago (anticoagulation ceased at 6 months by patient choice), 30 pack-year smoking history. On arrival: Glasgow Coma Score 14, heart rate 115, blood pressure 110/70, respiratory rate 22, temperature 37.6 °C. Examination: diffuse abdominal tenderness with mild peritonism, absent bowel sounds. Bloods: white cell count $18 \times 10^9/L$, lactate 4.2 millimoles per litre, creatinine 110, D-dimer markedly elevated. CT with portal venous phase contrast confirms extensive superior mesenteric vein thrombosis extending into the portal vein with bowel wall thickening and pneumatosis of a short small bowel segment.

a) Name 3 features distinguishing mesenteric venous from arterial ischaemia. (3 marks)

- **Subacute onset (days)** vs the abrupt 'pain out of proportion to examination' of acute mesenteric arterial occlusion; younger patient (40-50 years) vs typical 70+ years for arterial **[core]**
- **Predisposition to hypercoagulability:** factor V Leiden (this patient), JAK2-positive myeloproliferative disease, antiphospholipid, oral contraceptive, malignancy, cirrhosis with portal hypertension, recent abdominal surgery (particularly splenectomy); **30-50% have inherited thrombophilia [core]**
- **CT findings:** filling defect in the SMV/portal vein on portal venous phase (must request); congested thickened bowel wall (target sign); **NOT** the abrupt cut-off of SMA occlusion. Lactate often less markedly elevated than arterial **[core]**
- Bowel infarction more variable / patchy than arterial; **mortality 20-50%** (less than 60-90% for arterial)

b) Outline 5 management priorities. (5 marks)

- **Resuscitation:** aggressive IV crystalloid (third-spacing significant); two large-bore IV lines; nasogastric tube; nil by mouth; analgesia (IV opioid) **[core]**
- **Therapeutic anticoagulation immediately: unfractionated heparin infusion 80 units/kg bolus then 18 units/kg/hour** (target APTT 60-80) — prevents propagation, allows bowel salvage; **do not delay for imaging confirmation** if high clinical suspicion **[core]**
- **Empiric broad-spectrum antibiotics** (piperacillin-tazobactam 4.5 g IV) — translocation across compromised bowel; tetanus prophylaxis if any perforation **[core]**
- **Surgical consultation:** most patients can be managed non-operatively with anticoagulation alone; **laparotomy** if peritonism, bowel perforation, transmural ischaemia (here pneumatosis suggests bowel wall compromise); resection of necrotic bowel with re-look laparotomy at 24 hours **[core]**
- **Investigate underlying thrombophilia and predisposition:** JAK2 mutation (myeloproliferative disease), antiphospholipid, paroxysmal nocturnal haemoglobinuria, cancer screening, liver disease evaluation; **long-term anticoagulation** typically indefinite given thrombophilia and recurrence risk; haematology referral **[core]**
- Catheter-directed thrombolysis via transjugular intrahepatic portosystemic approach for refractory cases

c) Name 2 causes of acute portal vein thrombosis. (2 marks)

- **Cirrhosis / portal hypertension** (most common cause in Australia — up to 25% of cirrhotic patients develop PVT); **myeloproliferative neoplasms** (JAK2-positive — polycythaemia vera, essential thrombocythaemia) **[core]**
- **Local inflammation/infection:** pancreatitis, diverticulitis, appendicitis, cholecystitis (pylephlebitis); abdominal surgery (particularly splenectomy); **thrombophilia** (factor V Leiden, prothrombin, protein C/S, antiphospholipid); **oral contraceptive / pregnancy [core]**
- Hepatocellular carcinoma with tumour invasion of portal vein

References: WSES Mesenteric Venous Thrombosis 2017 • eTG (Therapeutic Guidelines, current) • European Society for Vascular Surgery Guideline • Tintinalli's Emergency Medicine 9e Ch 81

Question 13 | Femoral Pseudoaneurysm Post-Catheterisation (9 marks)

A 68-year-old man returns to your emergency department 3 days after percutaneous coronary intervention via the right common femoral artery, with progressive right groin pain and a pulsatile swelling at the puncture site. On arrival: Glasgow Coma Score 15, heart rate 88, blood pressure 130/80, afebrile. Examination: tender pulsatile mass 4 centimetres diameter in the right groin with audible 'to-and-fro' bruit on auscultation. The right lower limb pulses are present and the leg is well-perfused. He remains on dual antiplatelet therapy (aspirin and ticagrelor).

a) Define femoral pseudoaneurysm AND name 2 key risk factors. (3 marks)

- **Femoral pseudoaneurysm** = contained extravascular collection of blood in continuity with the arterial lumen through a defect in the arterial wall, contained by surrounding tissue (NOT a true aneurysm — no intact vessel wall); characterised by 'to-and-fro' Doppler flow pattern through the neck **[core]**
- **Risk factors:** large-bore arterial puncture (cardiac catheterisation, intra-aortic balloon pump, ECMO), antiplatelet/anticoagulation (this patient — DAPT), punctuated below the inguinal ligament (femoral artery vs SFA), inadequate manual compression post-procedure **[core]**
- **Other risk factors:** obesity, hypertension, advanced age, female sex, calcified vessels, repeated procedures, vascular closure device failure **[core]**
- Complications: rupture (rare but life-threatening); compression of femoral nerve/vein; distal embolisation; skin necrosis; infection

b) Outline 4 management options. (4 marks)

- **Conservative observation** with compression bandage and serial ultrasound — appropriate for small (<2-3 cm) asymptomatic pseudoaneurysms; 90% thrombose spontaneously within 4 weeks; cease anticoagulation/antiplatelet (in conjunction with cardiology for the indication — balance with stent thrombosis risk) **[core]**
- **Ultrasound-guided manual compression** (10-30 minutes) of the neck of the pseudoaneurysm — occludes flow long enough to thrombose; success rate 60-90%; less effective if on anticoagulation **[core]**
- **Ultrasound-guided thrombin injection** (bovine or human thrombin into the sac under real-time ultrasound) — **first-line** for moderate pseudoaneurysm (3-7 cm); success >95%; complications include arterial thrombosis, anaphylaxis to thrombin **[core]**
- **Surgical or endovascular repair:** for large (>7 cm), rapidly expanding, infected, associated with limb ischaemia / nerve compression, failed less invasive measures; **covered stent** or open repair with primary closure or patch angioplasty **[core]**
- Indications for emergency intervention: rupture, large rapidly expanding, skin necrosis, neurovascular compromise, uncontrolled infection

c) Name 2 other vascular complications of femoral arterial access. (2 marks)

- **Retroperitoneal haematoma** — from puncture above the inguinal ligament (external iliac artery) with bleeding into the retroperitoneum; presents with hip/back pain, hypotension, falling haemoglobin; often delayed; diagnose with CT; transfusion and occasionally embolisation **[core]**
- **Arteriovenous fistula** (femoral artery to vein — bruit, thrill, leg swelling); **arterial thrombosis with distal ischaemia; arterial dissection; infection** (rare in modern practice with sterile technique); cholesterol embolisation **[core]**
- Nerve injury (femoral nerve compression by haematoma)

References: European Society for Vascular Surgery Guideline • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 65

Question 14 | Aortic Endograft Complications and Endoleak (9 marks)

A 72-year-old man presents to your emergency department with a 1-week history of vague abdominal discomfort and a 24-hour history of severe back pain. Past history: endovascular aortic aneurysm repair (EVAR) 6 years ago for a 6.2 centimetre infrarenal abdominal aortic aneurysm; he has missed his routine surveillance CT scans for 2 years. On arrival: Glasgow Coma Score 14, heart rate 105, blood pressure 95/55, respiratory rate 22, afebrile. Examination: tender pulsatile abdominal mass 12 centimetres in diameter, no peritonism. Bedside echocardiography: no pericardial effusion. Urgent CT angiography shows a growing abdominal aortic aneurysm sac of 8 centimetres with a Type 1A endoleak at the proximal anchoring site and a small retroperitoneal haematoma.

a) Define the 5 types of endoleak (Types I-V). (4 marks)

- **Type I — proximal (1a) or distal (1b) attachment site leak** — failure of seal between graft and native artery; **highest risk of rupture**; this patient (Type 1a); requires urgent intervention **[core]**
- **Type II — retrograde flow from lumbar / inferior mesenteric branches** into the sac; most common type (40-60% of all endoleaks); often resolves; intervention if sac expansion **[core]**
- **Type III — graft component junction or fabric defect**; uncommon but high-pressure leak — high rupture risk; requires repair **[core]**
- **Type IV — graft porosity** (historical, with old graft materials); not seen with modern grafts; **Type V (endotension)** — sac expansion without demonstrable leak; controversial; may need re-intervention **[core]**
- All EVAR patients require lifelong annual surveillance (CT or duplex) — failure of surveillance is a major issue with EVAR (this patient — 2 years missed)

b) Outline 3 management priorities for this patient. (3 marks)

- **Resuscitation with permissive hypotension** — target SBP 80-90 mmHg; large-bore IV access × 2; type and cross-match 6 units; **massive transfusion protocol primed**; **do NOT aggressively resuscitate to normotension** (rupture more likely with normalisation) **[core]**
- **Urgent vascular surgery and interventional radiology consultation** for definitive repair — **endovascular re-intervention** (proximal extension cuff, fenestrated/branched grafts, embolisation), **open conversion** for failed endovascular options or rupture; **theatre / interventional suite ready** **[core]**
- **Continuous monitoring**: arterial line, central access, urinary catheter, ICU/HDU; serial physical examination for expansion of mass / haemodynamic deterioration; communication with patient and family re prognosis **[core]**
- Type 1a endoleak with sac growth: 5-year rupture risk >30% without re-intervention

c) Name 2 other late complications of EVAR. (2 marks)

- **Aortoenteric fistula** — communication between aortic graft and adjacent bowel (duodenum 3rd/4th part most commonly), presenting with 'sentinel' GI bleed, followed by catastrophic haemorrhage; high mortality; CT and endoscopy; open surgical repair **[core]**
- **Graft infection** (typically 6-24 months post-implant; staphylococci, streptococci, enterics); presents with fever, pain, leukocytosis, perigraft fluid on CT; **graft thrombosis / limb occlusion** with acute limb ischaemia; **migration** of graft components; **kinking/stenosis** **[core]**
- Renal complications from contrast/embolisation/branch coverage

References: ESVS AAA Guideline 2024 • Australian and New Zealand Society for Vascular Surgery position statement • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 64

Question 15 | Reperfusion Injury and Lower Limb Compartment Syndrome Post-Revascularisation (10 marks)

A 65-year-old man underwent emergency femoro-popliteal thromboembolectomy 4 hours ago for Rutherford IIb acute limb ischaemia. He is now in your ICU/HDU. The post-operative course has been complicated by progressive calf swelling, increasing pain on passive ankle movement and oliguria. On reassessment: Glasgow Coma Score 14, heart rate 120, blood pressure 110/70, respiratory rate 24, peripheral oxygen saturation 95 percent on 6 litres oxygen, temperature 36.4 °C. Right calf is tense and swollen with severe pain on passive dorsiflexion of toes. Bloods: potassium 6.1, creatinine 280, creatine kinase 42 000 U/L, myoglobin in urine, pH 7.21, lactate 4.5, bicarbonate 14. ECG shows tall peaked T waves.

a) Name 4 features of reperfusion syndrome. (4 marks)

- **Hyperkalaemia** (K 6.1 with peaked T waves) — release of intracellular K from injured muscle; high risk of ventricular arrhythmia; this is the **most rapidly fatal** component **[core]**
- **Rhabdomyolysis** (CK 42 000 — severe; normal <200; rhabdomyolysis >1000, severe >5000) → myoglobinaemia and myoglobinuria → **pigment-induced acute kidney injury** (Cr 280) **[core]**
- **Metabolic acidosis** (pH 7.21, HCO_3^- 14, lactate 4.5) — lactic acidosis from tissue hypoxia and impaired clearance; potentiates hyperkalaemia **[core]**
- **Compartment syndrome** (swollen tense calf, pain on passive stretch, paraesthesia developing — the 'P's of compartment syndrome): tissue oedema after reperfusion exceeds compartment pressure → muscle and nerve ischaemia **[core]**
- Other features: ARDS, cardiac arrhythmias, DIC, multi-organ failure

b) Outline 4 management priorities. (4 marks)

- **Treat hyperkalaemia immediately** (see Renal+Resp): **calcium gluconate 10% 10 mL IV** (membrane stabilisation), **insulin 10 units + 50 mL 50% dextrose IV**, **sodium bicarbonate 50 mmol IV**, **salbutamol nebulised 20 mg**, **resonium / cation exchange resin**; **urgent haemodialysis** **[core]**
- **Aggressive IV crystalloid 0.9% NaCl 200-500 mL/hour** to maintain urine output >1-2 mL/kg/hour (typically 4-12 L in first 24 hours); avoid potassium-containing fluids; **urinary alkalinisation** (sodium bicarbonate infusion to target urine pH >6.5) — controversial but widely used to reduce myoglobin precipitation **[core]**
- **Urgent surgical fasciotomy** for compartment syndrome — **4-compartment lower leg fasciotomy** via medial and lateral incisions; do not measure pressures if clinical syndrome evident; better to perform prophylactic fasciotomy at time of revascularisation in high-risk cases **[core]**
- **Renal replacement therapy** (haemodialysis or CRRT) — for refractory hyperkalaemia, severe acidosis, oliguric AKI with fluid overload, uraemia; **ICU admission** for organ support; monitor for ARDS, DIC, ongoing arrhythmia **[core]**
- Mannitol — historically used for rhabdomyolysis-AKI but evidence weak; avoid nephrotoxic drugs

c) Name 2 indications for fasciotomy. (2 marks)

- **Clinical compartment syndrome**: pain out of proportion to injury, pain on passive stretch, tense compartment, paraesthesia, pallor, pulselessness (late); **compartment pressure >30 mmHg OR within 30 mmHg of diastolic BP** (delta pressure <30) **[core]**
- **Prophylactic fasciotomy at time of revascularisation** for: ischaemia duration >4-6 hours, combined arterial-venous injury, massive limb swelling on table, marked rhabdomyolysis, phlegmasia cerulea dolens **[core]**
- Crush syndrome / electrical injury / severe burn / supracondylar fracture (paediatric)

References: *European Society for Vascular Surgery Guideline* • *NSW Institute of Trauma and Injury Management* • *eTG (Therapeutic Guidelines, current)* • *Tintinalli's Emergency Medicine 9e Ch 64*

Question 16 | Buerger's Disease and Raynaud's Phenomenon (9 marks)

A 32-year-old man presents to your emergency department with painful blackened tips of his right second and third toes. He smokes 30 cigarettes per day and has done so since age 16. There is no history of diabetes, hypercholesterolaemia or family history of vascular disease. On arrival: alert, haemodynamically stable, afebrile. Examination: **distal digital gangrene** of right 2nd and 3rd toes, palpable femoral and popliteal pulses, weak dorsalis pedis, absent posterior tibial pulse, with reduced capillary refill in toes. Ankle-brachial index 0.7. Bloods: inflammatory markers normal, autoimmune screen negative, fasting lipids and HbA1c normal.

a) State the likely diagnosis AND list 3 Shionoya diagnostic criteria. (3 marks)

- **Buerger's disease (thromboangiitis obliterans)** — non-atherosclerotic segmental inflammatory occlusive disease of small-to-medium arteries and veins of distal extremities; **strongly associated with smoking** (absolute requirement); typically young men <45 years **[core]**
- **Shionoya criteria** (any 3 of 5): (1) **smoking history**; (2) **onset before age 50**; (3) **infrapopliteal arterial occlusive disease**; (4) **upper limb involvement OR migratory phlebitis**; (5) **absence of atherosclerotic risk factors other than smoking** **[core]**
- **Distinguished from atherosclerotic PAD**: young age, normal lipid/glucose, predominantly distal (infrapopliteal vs aortoiliac/femoropopliteal), **thrombophlebitis migrans** in 40%, **'corkscrew' collaterals** on angiography **[core]**
- Distinguished from vasculitis: normal inflammatory markers, normal autoimmune screen; pathology shows acute thrombus with preserved internal elastic lamina

b) Outline 4 management priorities. (4 marks)

- **Absolute smoking cessation** — **only proven disease-modifying intervention**; even single cigarettes cause exacerbation; smoking cessation support, varenicline / NRT / counselling **[core]**
- **Wound care and analgesia**: non-adherent dressings; opioid for rest pain; vasodilators (intravenous prostacyclin / iloprost infusion for severe cases — modest evidence; cilostazol 100 mg BD; calcium channel blockers); **antiplatelet** (aspirin 100 mg) **[core]**
- **Avoid revascularisation** if possible — distal disease usually unsuitable for bypass; **amputation** only for uncontrollable pain or gangrene (toe/ray amputation preserves ambulation); **spinal cord stimulation** experimental for severe rest pain **[core]**
- **Vascular surgical and rheumatological consultation**; treatment of secondary infection; psychological support (chronic disease, young patient, smoking addiction); follow-up in chronic vascular clinic **[core]**
- Tetanus prophylaxis for digital wounds

c) Differentiate primary and secondary Raynaud's phenomenon (2 features each). (2 marks)

- **Primary Raynaud's**: female predominant, onset 15-30 years, symmetric digital involvement, **no underlying disease, nailfold capillaroscopy normal, no ulceration** or ischaemic changes; benign course **[core]**
- **Secondary Raynaud's**: any age (often older), **associated connective tissue disease** (systemic sclerosis, SLE, rheumatoid arthritis, dermatomyositis), asymmetric, **positive autoantibodies, abnormal nailfold capillaroscopy, digital ulceration / pitting** **[core]**
- Triggered by cold and emotion; treat with calcium channel blocker (nifedipine 30-60 mg/day), PDE-5 inhibitor; severe → IV iloprost / endothelin antagonist

References: Shionoya criteria 1998 • EULAR Raynaud's recommendations • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 64

Question 17 | Takayasu Arteritis (9 marks)

A 25-year-old Asian woman presents to your emergency department with progressive dizziness, syncope on exertion, weakness, and weight loss of 5 kilograms over 3 months. She describes left arm pain on use. On arrival: alert, heart rate 65 sinus rhythm, **blood pressure 145/85 right arm and unrecordable on the left arm**, temperature 37.8 °C. Examination: **weak left radial pulse, audible bruits over carotid and subclavian arteries bilaterally**, abdominal bruit, normal heart sounds. Bloods: ESR 95, CRP 80 (elevated), Hb 102 (normocytic), platelets 480, normal autoimmune screen. CT angiography: long-segment narrowing of the left subclavian, common carotid arteries, and thoracic aorta wall thickening.

a) Name 4 features that confirm Takayasu arteritis. (4 marks)

- **'Pulseless disease'** — diminished or absent pulses (left radial here); **blood pressure differential >10 mmHg between arms** (unrecordable vs 145/85); **arterial bruits** over carotid, subclavian, abdominal vessels **[core]**
- **Demographics:** **young woman <40 years**, Asian (Japanese, Korean, Chinese, South Asian) ethnicity — 'oriental disease'; F:M ratio 9:1; onset 15-35 years typically **[core]**
- **Imaging:** **concentric thickening of large vessel walls** (aorta, primary branches), **long-segment stenoses, occlusions, aneurysms**; CT/MR angiography (less invasive than conventional); PET-CT for active inflammation **[core]**
- **ACR/EULAR classification (2022):** age ≤60, imaging evidence of vasculitis, plus clinical/laboratory criteria — pulse deficit, claudication, BP differential, bruit, elevated inflammatory markers; **distinguished from giant cell arteritis** (older patients; temporal/cranial involvement) **[core]**
- Phases: **pre-pulseless / systemic** (fever, malaise, weight loss); **vascular / occlusive** (claudication, stroke, MI, renovascular HTN); often overlapping

b) Outline 3 management priorities. (3 marks)

- **High-dose corticosteroid:** **prednisolone 1 mg/kg/day (up to 60 mg) PO** with slow taper over months; **methylprednisolone 1 g IV daily × 3 days** for severe organ-threatening disease; rheumatology consultation **[core]**
- **Steroid-sparing agent:** **methotrexate, azathioprine, mycophenolate, leflunomide** (conventional DMARDs); **biologic — tocilizumab (IL-6 inhibitor)** emerging as an effective steroid-sparing agent **[core]**
- **BP control and revascularisation:** **BP measured in lower limbs OR non-occluded limb** (left arm BP unreliable); **endovascular angioplasty/stenting OR open bypass** for symptomatic stenosis (renal artery for renovascular HTN; carotid for stroke risk); **defer revascularisation** until active inflammation controlled **[core]**
- Antiplatelet (aspirin), statin, cardiovascular risk modification; complications: aortic regurgitation, stroke, renovascular HTN, cardiomyopathy, aortic aneurysm

c) Differentiate Takayasu from giant cell arteritis. (2 marks)

- **Takayasu:** age <40-50 years (typically <40); Asian female; **large vessel — aorta and primary branches** (subclavian, carotid, renal); claudication, BP differential, bruits, renovascular HTN **[core]**
- **Giant cell arteritis (GCA / temporal arteritis):** age >50 years (mean 70); Caucasian predominant; **temporal and extracranial branches of carotid** (jaw claudication, scalp tenderness, headache); **visual loss** (anterior ischaemic optic neuropathy); association with polymyalgia rheumatica; ESR markedly elevated; **temporal artery biopsy** for diagnosis **[core]**
- Overlap exists — large-vessel GCA may involve aorta; both respond to corticosteroids

References: ACR/EULAR Takayasu Classification 2022 • EULAR Large-Vessel Vasculitis Guidelines 2018 • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 281

Question 18 | Ruptured Thoracic Aortic Aneurysm (10 marks)

A 75-year-old man with known 7.5 centimetre descending thoracic aortic aneurysm on surveillance presents to your emergency department with sudden-onset severe interscapular back pain and dyspnoea. Past history: hypertension, COPD, ex-smoker, previous AAA repair 8 years ago. On arrival: Glasgow Coma Score 13, heart rate 130, blood pressure 70/45, respiratory rate 32, peripheral oxygen saturation 90 percent on 10 litres oxygen, peripheral pulses weak. CXR: widened mediastinum with left-sided pleural effusion and tracheal deviation. CT angiography confirms an 8.5 centimetre saccular ruptured descending thoracic aneurysm with active contrast extravasation into the left haemothorax.

a) Name 3 features supporting the diagnosis. (3 marks)

- **Known thoracic aortic aneurysm** (7.5 cm — risk of rupture rises exponentially >6 cm); **sudden-onset severe interscapular back pain** — classic for thoracic aortic rupture or dissection **[core]**
- **Haemodynamic shock** (HR 130, BP 70/45) with peripheral pulses weak — Class III/IV haemorrhagic shock; **widened mediastinum and left haemothorax** on CXR **[core]**
- **CT angiography with contrast extravasation** — definitive sign of active rupture; **periaortic haematoma, haemothorax, contained vs free rupture** determines emergent intervention **[core]**
- Marfan, bicuspid aortic valve, hypertension, previous AAA (this patient — same atherosclerotic substrate)

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

- **Permissive hypotension** — target SBP 70-90 mmHg with **cautious crystalloid + early blood products** (avoid normalising BP — rupture worsens); **activate massive transfusion protocol**; tranexamic acid 1 g IV **[core]**
- **Anti-impulse therapy** with **β-blocker (esmolol or labetalol)** to target HR <80; SBP titrated up only for evidence of malperfusion; **analgesia** with IV opioid (titrated to avoid hypotension) **[core]**
- **Activate cardiothoracic / vascular surgical team; thoracic endovascular aortic repair (TEVAR)** is first-line for ruptured descending TAA (lower mortality than open repair); **open repair** for ascending / arch / unsuitable anatomy **[core]**
- **Treat haemothorax: large-bore chest drain (32-36 Fr)** ipsilateral to haemothorax — **BEWARE** rapid decompression of a contained rupture may precipitate fatal bleeding (be ready with surgical team); intubate if respiratory failure / GCS deteriorating **[core]**
- **Inform family:** high mortality (50% in-hospital even with treatment; >90% in field); discuss goals of care if appropriate; involve clinical psychologist / pastoral care **[core]**
- Pre-empt complications: spinal cord ischaemia (CSF drainage in elective TEVAR), stroke, renal failure

c) List 2 indications for elective TAA repair. (2 marks)

- **Diameter ≥5.5 cm for descending** (≥5.0 cm for ascending or in Marfan/bicuspid AV; ≥4.5 cm in Loeys-Dietz / Marfan with family history of dissection); **rapid expansion ≥5 mm in 6 months OR ≥10 mm/year** **[core]**
- **Symptomatic aneurysm** (pain — even without rupture); **aortic valve regurgitation** from ascending dilatation; **concomitant cardiac surgery** (consider repair at lower thresholds if undergoing CABG/valve) **[core]**
- Saccular morphology (higher rupture risk than fusiform)

References: ESC Aortic Disease Guideline 2014/2024 • AHA/ACC 2022 • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 60 • Australian and New Zealand Society for Vascular Surgery position statement

Question 19 | Catastrophic Limb Haemorrhage and Tourniquet Application (9 marks)

A 28-year-old man is brought to your emergency department by ambulance 30 minutes after a high-speed motorcycle crash. He sustained open injuries to the right lower limb with partial amputation at mid-thigh and uncontrolled arterial bleeding at scene. Paramedics applied a Combat Application Tourniquet (CAT) to the right thigh 25 minutes ago which controlled the bleeding. On arrival: Glasgow Coma Score 13, heart rate 130, blood pressure 75/40, respiratory rate 28, peripheral oxygen saturation 96 percent on 10 litres oxygen, cool peripheries, capillary refill 4 seconds, ashen and confused, no obvious head injury. Tourniquet is in place and effective.

a) Outline 3 priorities of catastrophic haemorrhage management. (3 marks)

- **<C>ABC approach** — catastrophic external haemorrhage takes precedence over airway in penetrating / explosive trauma per current military and civilian (FACEM, ITIM, Pre-Hospital Trauma Life Support) guidelines; **tourniquet OR direct pressure** first [core]
- **Tourniquet remains in situ until definitive surgical control** — **do NOT remove in resuscitation area** if effective and limb is non-salvageable or surgery imminent; **record time of application** (here 25 min); communicate to surgical team [core]
- **Massive transfusion protocol activation** with balanced ratio (1:1:1 PRBC:FFP:platelets); **tranexamic acid 1 g IV within 3 hours** (CRASH-2); **haemostatic dressings** (QuikClot, Combat Gauze, chitosan) for any other bleeding sites; **permissive hypotension** (SBP 80-90 mmHg) until controlled [core]
- Pelvic binder if pelvic instability suspected; junctional tourniquet for proximal limb / groin haemorrhage

b) Outline 4 considerations for tourniquet use. (4 marks)

- **Indications:** **life-threatening external haemorrhage** from extremity not controlled by direct pressure; **partial / complete amputation**; multiple casualties with limb haemorrhage; tactical environments; entrapment with ongoing bleeding [core]
- **Application technique:** place **5-8 cm proximal to wound** (not over a joint); **commercially manufactured tourniquet (CAT, SOFTT, EMT)** preferred over improvised; **tighten until pulses distal absent AND bleeding stops**; **NOT** just until bleeding stops — partial occlusion worsens venous bleeding [core]
- **Duration tolerance:** **up to 2 hours generally safe**; >6 hours associated with muscle necrosis, nerve injury, rhabdomyolysis, limb loss; **document time of application**; consider downgrading to direct pressure or proximal control once in controlled environment [core]
- **Complications:** rhabdomyolysis and reperfusion syndrome on release (see Q15 — hyperkalaemia, acidosis, AKI); **nerve palsy** (transient or permanent); compartment syndrome after release; **pain** from tourniquet itself; **do NOT remove in non-surgical setting** for >2 hours unless surgery imminent [core]
- Document tourniquet time + location prominently (forehead with marker, or label on tourniquet) — must accompany patient

c) List 2 non-tourniquet haemostatic strategies. (2 marks)

- **Direct pressure** with sterile gauze, packing of cavity wounds (haemostatic gauze — kaolin-based QuikClot Combat Gauze or chitosan-based ChitoGauze) — first-line for junctional bleeding (groin, axilla); **pressure bandaging** [core]
- **Pelvic binder** (commercial — T-POD, SAM Pelvic Sling) for pelvic ring disruption; **resuscitative endovascular balloon occlusion of the aorta (REBOA)** in highly selected patients with refractory pelvic / abdominal haemorrhage; **damage-control surgery / interventional radiology embolisation** [core]
- Wound packing with haemostatic agents; manual proximal arterial compression (e.g. external compression of femoral artery at groin)

References: *NSW Institute of Trauma and Injury Management • Pre-Hospital Trauma Life Support (PHTLS) current • Stop the Bleed Australia • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 14*

Question 20 | Posterior Reversible Encephalopathy Syndrome (PRES) (9 marks)

A 36-year-old woman with end-stage renal disease on haemodialysis presents to your emergency department with severe headache, blurred vision, confusion, and one witnessed tonic-clonic seizure 30 minutes ago. She has missed her last 2 dialysis sessions. Past history: hypertension, lupus nephritis, on tacrolimus and prednisolone for renal transplant 1 year ago. On arrival: Glasgow Coma Score 12, heart rate 95, blood pressure 230/130 millimetres of mercury, respiratory rate 18, temperature 37.0 °C, peripheral oxygen saturation 97 percent on room air. Examination: cortical blindness, normal pupils, no focal weakness, no neck stiffness, papilloedema. CT brain plain shows symmetrical bilateral occipital and posterior parietal hypodensities without haemorrhage.

a) Name 3 features that confirm PRES. (3 marks)

- **Clinical tetrad: headache, altered mental status, seizure, visual disturbance** (cortical blindness, hemianopsia, blurring) — typically in setting of severe hypertension or other trigger **[core]**
- **Triggers: severe hypertension** (most common — exceeds cerebral autoregulation, this patient BP 230/130); **eclampsia / pre-eclampsia**; **immunosuppressants** (tacrolimus, ciclosporin — this patient post-transplant), **cytotoxics** (cisplatin, bevacizumab); **autoimmune disease** (lupus, vasculitis); **renal failure** with fluid overload **[core]**
- **MRI/CT findings: symmetrical, bilateral vasogenic oedema** predominantly involving **parieto-occipital regions** (posterior watershed — less sympathetic innervation, more susceptible to autoregulatory breakdown); T2/FLAIR hyperintensity; usually no haemorrhage or restriction on DWI; **reversible** on imaging with treatment **[core]**
- Distinguished from PCA stroke (asymmetric, restricted diffusion, wedge-shaped); from venous sinus thrombosis (CT venogram)

b) Outline 4 management priorities. (4 marks)

- **Controlled BP reduction: target reduction of MAP by 20-25% over 1-2 hours; labetalol IV boluses 10-20 mg or infusion 0.5-2 mg/min, OR nicardipine infusion**; avoid rapid normalisation (cerebral hypoperfusion); arterial line **[core]**
- **Treat seizures: benzodiazepine** (midazolam 10 mg IM, diazepam 10 mg IV) for termination; **levetiracetam 1500-3000 mg IV** as second-line; **EEG monitoring** for non-convulsive status; neurology consultation **[core]**
- **Address precipitants: urgent dialysis** for fluid overload and uraemia (this patient — missed sessions); **review and reduce / cease tacrolimus**; treat infection; consider seizure trigger investigation **[core]**
- **Monitor for complications: haemorrhagic transformation** (in 10-15% — increased mortality), **cerebral infarction, cerebellar / brainstem extension** with herniation; follow-up MRI in 2-4 weeks for resolution; admit HDU/ICU **[core]**
- Reversibility is the rule with prompt treatment; 70-90% have full neurological recovery

c) List 2 differentials. (2 marks)

- **Cerebral venous sinus thrombosis (CVST)** — headache, seizures, focal deficit; CT venogram diagnostic; anticoagulation. **Posterior circulation ischaemic stroke** (PCA territory — typically unilateral, restricted diffusion on MRI) **[core]**
- **Subarachnoid haemorrhage** (blood on CT, severe sudden headache); **encephalitis** (fever, CSF abnormalities); **eclampsia / pre-eclampsia** (if pregnant); **hypertensive encephalopathy without PRES** (more diffuse cerebral involvement); **reversible cerebral vasoconstriction syndrome (RCVS)** **[core]**
- Hypoglycaemia, hyponatraemia, drug intoxication (sympathomimetic)

References: *European Stroke Organisation PRES guidelines* • *eTG (Therapeutic Guidelines, current)* • *Tintinalli's Emergency Medicine 9e Ch 173* • *National Heart Foundation of Australia*

Question 21 | Acute Severe Aortic Regurgitation (10 marks)

A 45-year-old man presents to your emergency department with sudden-onset severe dyspnoea and chest pain. Past history: known bicuspid aortic valve and aortic root dilatation (4.8 centimetres) under surveillance, Marfan-like habitus. On arrival: Glasgow Coma Score 15 (anxious), heart rate 130 sinus tachycardia, blood pressure 100/40 millimetres of mercury, respiratory rate 32, peripheral oxygen saturation 89 percent on 10 litres oxygen, temperature 36.8 °C. Examination: bibasal crepitations, soft early diastolic murmur at left sternal edge, S3 gallop, collapsing pulse, no peripheral pulse differential between arms. Bedside transthoracic echocardiography: severe aortic regurgitation with rapid LV diastolic pressure equilibration, hyperdynamic LV with normal size, no obvious flail leaflet but possible aortic root dissection on parasternal views.

a) Name 3 features that distinguish acute severe AR from chronic AR. (3 marks)

- **Acute** (this patient): **normal LV size** (no time for compensatory dilatation), **marked tachycardia** (to maintain stroke volume), **narrowed pulse pressure** (SBP not elevated; here 100/40 — diastolic markedly reduced), **rapid clinical deterioration** with cardiogenic shock **[core]**
- **Chronic**: **marked LV dilatation**, wide pulse pressure (Corrigan, Quincke, de Musset, Müller signs), **compensated**; symptoms occur late **[core]**
- **Causes of acute severe AR**: **aortic dissection** (this patient — Marfan-like, bicuspid AV, root dilatation); **infective endocarditis** with valve destruction; **chest trauma**; **spontaneous chordal/leaflet rupture** **[core]**
- Acute severe AR is poorly tolerated — rapid haemodynamic collapse; chronic AR may be asymptomatic for years

b) Outline 5 management priorities. (5 marks)

- **Activate cardiothoracic surgical team immediately** — **definitive treatment is emergency aortic valve replacement** (with concomitant aortic root replacement if dissection or aneurysm); medical therapy is bridging only **[core]**
- **Afterload reduction**: **sodium nitroprusside infusion 0.5-3 micrograms/kg/min** OR GTN — reduces regurgitant volume; **inotropy**: dobutamine 5-15 micrograms/kg/min for decompensated heart failure; **avoid β -blockers** in pure acute AR (loss of tachycardia compensation worsens regurgitation) — EXCEPT if dissection coexists (then β -blocker required for dissection management) **[core]**
- **Avoid intra-aortic balloon pump** in pure aortic regurgitation (worsens regurgitation by inflating in diastole); **NIV / CPAP** for pulmonary oedema; **intubation** with caution — positive pressure may reduce preload further; etomidate / ketamine for haemodynamics **[core]**
- **Investigate cause**: **CT angiography aorta URGENT** to exclude dissection (this patient's substrate — bicuspid AV, aortic root dilatation, Marfan habitus); **transoesophageal echocardiography** for leaflet integrity, endocarditis (vegetations), aortic root pathology; **blood cultures $\times 3$** for endocarditis; empiric antibiotics (flucloxacillin + gentamicin) if endocarditis likely **[core]**
- **Search for underlying syndrome**: Marfan, Loeys-Dietz, vascular Ehlers-Danlos (genetic testing later); family screening; communication with family re prognosis; consider transfer to cardiothoracic centre if not on-site **[core]**
- Mortality without surgery 50% at 1 week; surgery reduces to 10-20%

c) Name 2 connective tissue diseases associated with aortic root pathology. (2 marks)

- **Marfan syndrome** (FBN1 mutation, autosomal dominant) — **tall stature, arachnodactyly, aortic root dilatation and dissection** (most common cause of death), **aortic regurgitation**, mitral prolapse, pectus deformity, lens dislocation, scoliosis **[core]**

- **Loeys-Dietz syndrome** (TGFB1/2, SMAD3, TGFB2/3 mutations) — more aggressive aortic disease than Marfan; arterial tortuosity, cleft palate, hypertelorism; **vascular Ehlers-Danlos** (COL3A1) — fragile vessels, thin skin, easy bruising, arterial / GI rupture; **bicuspid aortic valve** (this patient — 1-2% of population, often with root dilatation) **[core]**
- Turner syndrome (XO) — bicuspid valve + coarctation + aortic dilatation

References: AHA/ACC Valvular Heart Disease Guideline 2020 • ESC Aortic Disease Guideline 2024 • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 56

Question 22 | Young Adult with Spontaneous Aortic Dissection — Connective Tissue Disease (9 marks)

A 28-year-old previously well man, 6 foot 6 (198 cm) tall and slim build, is brought to your emergency department with sudden-onset severe chest and back pain that occurred while playing basketball. He has no medical history. On arrival: Glasgow Coma Score 15, heart rate 88 sinus rhythm, blood pressure 150/90 (similar both arms), respiratory rate 22, peripheral oxygen saturation 96 percent on room air, temperature 36.8 °C. Examination: **arachnodactyly, pectus excavatum, scoliosis, high arched palate, positive thumb and wrist (Steinberg, Walker-Murdoch) signs**, soft early diastolic murmur. CT angiography shows Stanford Type A aortic dissection extending from a dilated aortic root (5.0 centimetres). Bedside echocardiography confirms severe aortic regurgitation.

a) Name 4 features supporting Marfan syndrome (Ghent criteria). (4 marks)

- **Aortic root dilatation** (Z-score ≥ 2 — major criterion); **ectopia lentis** (lens dislocation — major criterion); **family history of Marfan or FBN1 mutation**; together any 2 of the above make the diagnosis **[core]**
- **Systemic score ≥ 7 of 20** (skeletal/systemic features): **wrist AND thumb signs** (Steinberg, Walker-Murdoch — 3 points), pectus excavatum / carinatum, hindfoot deformity, pes planus, pneumothorax history, dural ectasia **[core]**
- **Skeletal: tall stature** (this patient 198 cm), **arachnodactyly, arm span > height** (ratio >1.05), reduced upper-to-lower-segment ratio <0.85 , **scoliosis, pectus deformity, high arched palate** **[core]**
- **Ocular:** ectopia lentis (50-80%), myopia, retinal detachment; **cardiovascular:** mitral valve prolapse, **aortic root dilatation** and dissection — this patient; **pulmonary:** spontaneous pneumothorax **[core]**
- FBN1 gene (fibrillin-1) mutation; autosomal dominant; family screening essential

b) Outline 3 specific management priorities for young Marfan patient with acute Type A dissection. (3 marks)

- **Emergency cardiothoracic surgery** — aortic root and ascending aorta replacement; **valve-sparing root replacement (David procedure)** preferred in young patients to preserve native aortic valve (avoids lifelong anticoagulation); **mechanical (Bentall) alternative** **[core]**
- **Anti-impulse therapy** (β -blocker first — esmolol or labetalol) targeting HR <60 and SBP 100-120; **ARB (losartan) long-term** in Marfan — blocks TGF- β signalling, may slow aortic dilatation (PEARL, COMPARE trials) **[core]**
- **Family screening and genetic counselling:** FBN1 mutation (autosomal dominant — 50% risk per child); echocardiography for first-degree relatives; **multi-disciplinary follow-up:** cardiology, cardiothoracic surgery, ophthalmology, orthopaedics; lifetime aortic surveillance; avoid contact sports and isometric exercise lifelong **[core]**
- Driving and occupational counselling; MedicAlert

c) List 2 other causes of aortic dissection in young patients. (2 marks)

- **Loeys-Dietz syndrome, vascular Ehlers-Danlos** (different fibrillopathies — see Q21); **Turner syndrome** (45,X — bicuspid AV, coarctation, aortic dilatation); **bicuspid aortic valve** (1-2% of population) **[core]**
- **Pregnancy** (3rd trimester or post-partum — relaxin-mediated weakening of aortic wall; especially in Marfan / Turner / bicuspid AV); **cocaine / methamphetamine** (sympathomimetic surge); **iatrogenic** (cardiac catheterisation, cardiac surgery); **chest trauma; aortitis** (Takayasu, syphilis, giant cell) **[core]**
- Idiopathic (no identifiable cause in 5-10%)

References: *Revised Ghent Criteria 2010 • ESC Aortic Disease Guideline 2024 • AHA/ACC Aortic Disease Guideline 2022 • eTG (Therapeutic Guidelines, current) • Tintinalli's Emergency Medicine 9e Ch 60*