Internal Medicine — Categorized Question Bank

Compiled from 6 exam files: IM Exam 2023 (Sherine Asha), Doctor 2020, Doctor 2019, Medicine.2-Final 018, Medicine-Final 017, and 6th Year IM Final 2023 (Tarek & Kittaneh).

How to use this file:

- Questions are grouped by topic in the requested order. A miscellaneous section is at the end.
- Duplicate questions are merged. A tag like **[×3]** after a question means it appeared in 3 exams.
- Within each topic, the most-repeated questions come first.
- The correct answer is written **after** the choices (choices are NOT pre-marked).
- Each item has a short **Explanation** and, where the original answer key looks doubtful, a **Reviewer note** with a correction.

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1. CARDIOLOGY

C1. Septal myectomy in HOCM [×2]

A patient presents with progressive dyspnea and fluid overload (bilateral edema). On exam there is a left lower sternal **systolic murmur (3–6/6 systolic ejection murmur) that accentuates with the Valsalva maneuver.** Best management?

- a) Aortic valve replacement
- b) Surgical septal myectomy / myomectomy
- c) Mitral valve surgery
- d) Observation
- e) ICD

Answer: Surgical septal myectomy.

Explanation: A systolic murmur at the left lower sternal border that **increases with Valsalva** is the hallmark of hypertrophic obstructive cardiomyopathy (HOCM); Valsalva reduces preload and worsens LVOT obstruction. For symptomatic obstructive HCM refractory to medical therapy, septal reduction (surgical myectomy) is indicated.

Reviewer note: Valve replacement is wrong — the problem is dynamic LVOT obstruction from septal hypertrophy, not a diseased valve. Remember most murmurs *decrease* with Valsalva; HOCM and MVP are the classic exceptions that *increase*.

C2. Acute pericarditis — true statement [×3]

A patient with sharp retrosternal chest pain that improves sitting forward and worsens lying flat; pericardial friction rub; ECG with diffuse concave ST elevation and PR depression (preceding URTI). Which is true?

- a) First line is corticosteroids
- b) Colchicine decreases the risk of recurrence
- c) The disease is asymptomatic most of the time
- d) Heparin is first-line therapy
- e) Tamponade is a common complication

Answer: Colchicine decreases the risk of recurrence.

Explanation: First-line treatment of acute pericarditis is NSAIDs + colchicine; colchicine reduces symptoms and recurrence. Corticosteroids are second-line (they actually *increase* recurrence). Anticoagulants are avoided (hemorrhagic effusion risk).

Reviewer note: "Diffuse **concave** ST elevation" is correct for pericarditis; an option saying "convex" is wrong (convex = STEMI). A 2019 ECG variant asked the most consistent finding → **ST depression / PR elevation in aVR**.

C3. Constrictive pericarditis — confirmatory test [×2]

Dyspnea, bilateral leg edema, elevated JVP with prominent/deep X and Y descents; **echocardiogram normal**; multiple URTIs in the past year. Best tool to confirm the diagnosis?

- a) Cardiac MRI
- b) Simultaneous right- and left-heart catheterization
- c) 3D echocardiogram
- d) CT

Answer: Simultaneous right and left heart catheterization.

Explanation: Prominent X and Y descents + right heart failure + a **normal echo** suggest constrictive pericarditis. Simultaneous cath shows ventricular interdependence and equalization of diastolic pressures — the most definitive confirmation (and distinguishes it from restrictive cardiomyopathy).

Reviewer note: A 2019 variant gave a CXR with pericardial calcification → answer "constrictive pericarditis" as the *diagnosis*. When the question asks for the **confirmatory test**, choose catheterization.

C4. Valvular AF — anticoagulant choice [×2]

An 80-year-old female with HTN, DM and severe **rheumatic mitral stenosis** presents with atrial fibrillation. Best anticoagulation?

- a) Warfarin
- b) Dabigatran
- c) Rivaroxaban
- d) Apixaban
- e) Heparin
- f) Aspirin

Answer: Warfarin.

Explanation: AF with significant rheumatic mitral stenosis or a mechanical valve = "valvular AF". DOACs are NOT approved here; warfarin (target INR 2–3) is required.

Reviewer note: Aspirin is inadequate for stroke prevention in AF. DOACs remain not indicated for rheumatic mitral disease, mechanical valves, or LV assist devices.

C5. AF stroke risk factor [×2]

One factor that increases (cardioembolic) stroke risk in atrial fibrillation:

- a) Cigarette smoking
- b) Male gender
- c) Hypertension
- d) Dyslipidemia
- e) Previous DVT/PE

Answer: Hypertension.

Explanation: Of the CHA_2DS_2-VASc components, hypertension is the listed risk factor. Smoking and dyslipidemia are atherosclerotic risk factors, not in the score; the score counts **female** (not male) sex; VTE is not a component.

C6. Infective endocarditis — highest risk [×2]

Which condition carries the highest risk of infective endocarditis?

- a) Mitral valve prolapse
- b) Mitral valve ring repair (annuloplasty ring)
- c) Severe mitral regurgitation
- d) Rheumatic valvular heart disease
- e) Prosthetic valve

Answer: Prosthetic valve (a prosthetic ring counts as prosthetic material).

Explanation: Prosthetic heart valves and prosthetic repair material carry the highest IE risk and warrant antibiotic prophylaxis, along with previous IE, certain congenital heart disease, and cardiac-transplant valvulopathy. Native MVP/MR/rheumatic disease no longer warrant prophylaxis.

Reviewer note: 2017 keyed "prosthetic valve"; 2020 keyed "mitral valve ring repair" — both reflect prosthetic material.

C7. Torsade de pointes — management [×2]

A male presents with palpitations after an infection treated with azithromycin/a macrolide; ECG shows Torsade de pointes. Best next step?

- a) Synchronized cardioversion
- b) Amiodarone
- c) IV magnesium
- d) Procainamide

Answer: IV magnesium.

Explanation: Macrolides prolong QT and can precipitate Torsade. IV magnesium sulfate is first-line (even with normal Mg). Amiodarone is avoided (further QT prolongation); unstable/pulseless patients need defibrillation.

C8. Aortic dissection — first ER step [×2]

A hypertensive patient with severe chest pain (not relieved by nitroglycerin), inter-arm BP differential (e.g., 180/130 left arm vs 110/70 right arm); CXR widened mediastinum. First step?

- a) IV labetalol
- b) IV heparin
- c) Clopidogrel
- d) Coronary angiogram
- e) Aspirin

Answer: IV labetalol.

Explanation: Inter-arm BP differential + tearing pain + widened mediastinum = aortic dissection. Priority is rapid HR and BP control to reduce aortic wall shear stress — IV beta-blocker (labetalol/esmolol) FIRST, before vasodilators. Anticoagulants/antiplatelets are contraindicated.

Reviewer note: A confirmatory-test variant of the same scenario answers **CT angiography of the thorax**; for the first ER step, give IV labetalol.

C9. Coarctation of the aorta — confirmatory test [×2]

Chronic HTN, retrosternal chest pain, BP 180/130, X-ray rib notching (or a young patient with HTN in the arms and weak lower-limb pulses). Confirms the diagnosis?

- a) Thoracic CT angiography
- b) Coronary CT angiography
- c) X-ray
- d) Echocardiogram

Answer: Thoracic CT angiography.

Explanation: Rib notching + upper-extremity hypertension with weak lower-extremity pulses = coarctation of the aorta. Thoracic CT (or MR) angiography defines the coarctation and is confirmatory.

C10. Drug-induced AV block — management

ECG of AV block. A female on atenolol; regular pulse 80, stable vitals. Best management?

- a) Permanent pacemaker
- b) Temporary pacemaker
- c) Decrease the atenolol dose

Answer: Decrease the atenolol dose.

Explanation: If the block is reversible (drug-induced) and the patient is asymptomatic and stable, remove the offending cause (reduce/stop the beta-blocker) rather than implant a device.

Reviewer note: If the strip is a true **complete (3rd-degree) block** with symptoms/instability, a **pacemaker** is indicated. Also note: bradyarrhythmia/heart block needs a **pacemaker**, not an "ICD" — several papers loosely wrote "ICD."

C11. Heart block in the ER

ECG showing 2nd-degree (Mobitz II) or 3rd-degree heart block in the ER. What to do?

- a) Permanent pacemaker
- b) Temporary pacemaker
- c) Atropine
- d) Observation

Answer: Mobitz II → permanent pacemaker. Complete (3rd-degree) block presenting acutely/unstable → temporary pacing as a bridge, then permanent pacemaker.

Explanation: Mobitz II and complete heart block are indications for permanent pacing; an unstable acute complete block is bridged with temporary pacing.

Reviewer note: The student key wrote "3rd degree = temporary because emergency" — more precisely, temporary pacing is only the acute bridge; the definitive treatment of complete block is still a **permanent pacemaker**.

C12. Symptomatic complete heart block

An old woman with long-standing fatigue and dyspnea; ECG shows complete heart block. Best next step?

- a) Observation
- b) Atropine
- c) Permanent pacemaker
- d) Holter monitor

Answer: Permanent pacemaker.

Explanation: Symptomatic complete (3rd-degree) AV block is a class I indication for a permanent pacemaker.

C13. CHA_2DS_2-VASc calculation

A 78-year-old female smoker with HTN, DM, hyperlipidemia and prior TIA. CHA_2DS_2-VASc score?

- a) 1
- b) 3
- c) 5
- d) 7

- e) 9

Answer: 7.

Explanation: CHF 0 + Hypertension 1 + Age ≥75 (2) + Diabetes 1 + Stroke/TIA (2) + Vascular 0 + Sex female 1 = **7**.

Reviewer note: Smoking and hyperlipidemia are NOT part of CHA_2DS_2-VASc — distractors.

C14. Anterior MI on ECG [×2]

A 59-year-old with DM and HTN, 4 hours of retrosternal chest pain with nausea/vomiting; ECG shown. Diagnosis?

- a) Posterior MI
- b) Anterior MI
- c) Lateral MI
- d) Inferoposterior MI
- e) Pericarditis

Answer: Anterior MI (per the exam key).

Explanation: Image-dependent. Anterior MI shows ST elevation in precordial leads V1–V4.

Reviewer note: Diagnosis depends entirely on the ECG provided — verify the lead distribution on the actual strip.

C15. SVT vs VT — management

A patient with palpitations; ECG shown (or a 23-year-old, BP 110/70, HR 160). Best management?

- a) IV adenosine
- b) IV metoprolol
- c) IV diltiazem
- d) Synchronized cardioversion
- e) IV amiodarone

Answer: Stable narrow-complex SVT (AVNRT) → vagal maneuvers, then IV adenosine.

Explanation: A stable patient with a regular narrow-complex tachycardia is treated with vagal maneuvers then IV adenosine. Synchronized cardioversion is for hemodynamic instability.

Reviewer note: Image-dependent. The 2017 paper's key for one strip was "IV amiodarone" — that fits only a stable **wide-complex VT**. For a narrow-complex SVT the stable-patient drug is **adenosine**. Read the QRS width on the strip.

C16. Multifocal atrial tachycardia in COPD

A patient with long-standing COPD presents with palpitations; ECG shown; severe wheezing on exam. Best next step?

- a) Calcium channel blocker (non-dihydropyridine)
- b) Adenosine
- c) Beta blocker
- d) Digoxin

Answer: Non-dihydropyridine calcium channel blocker (verapamil/diltiazem).

Explanation: Likely multifocal atrial tachycardia (classic COPD arrhythmia). Rate control with a non-DHP CCB is preferred; beta-blockers are relatively contraindicated with severe bronchospasm; adenosine is ineffective for MAT.

C17. AF with aortic stenosis — anticoagulation

A patient with AF and **mild aortic stenosis** — long-term anticoagulation?

- a) Warfarin
- b) DOAC
- c) Aspirin
- d) No anticoagulation

Answer: DOAC.

Explanation: Unlike rheumatic *mitral* stenosis or mechanical valves, AF with aortic stenosis (and other non-rheumatic-mitral valve disease) can be treated with a DOAC.

Reviewer note: Contrast directly with C4 — mitral stenosis → warfarin; aortic stenosis → DOAC acceptable.

C18. Takotsubo cardiomyopathy

After a fight with her husband, a woman presents with chest pain and ST elevation; PCI done; echo shows apical dyskinesia with basilar preservation. Most likely cause?

- a) Syndrome X
- b) Stress-induced (Takotsubo) cardiomyopathy
- c) Acute coronary syndrome
- d) Prinzmetal angina

Answer: Stress-induced (Takotsubo) cardiomyopathy.

Explanation: Emotional trigger + ST elevation but non-obstructive coronaries + apical ballooning (apical dyskinesia, preserved base) is classic for Takotsubo.

C19. Long QT with breakthrough Torsade — ICD

A 30-year-old with known long QT syndrome presents with syncope and polymorphic VT; BP 125/80, HR 80. Next step?

- a) Genetic test
- b) ICD
- c) IV amiodarone
- d) IV metoprolol

Answer: ICD.

Explanation: Congenital long QT with syncope and documented polymorphic VT indicates high arrhythmic risk → ICD for secondary prevention.

Reviewer note: If the patient were acutely *in* Torsade and unstable, IV magnesium ± pacing/defibrillation would be the acute treatment — here she is stable post-event, so the definitive step is ICD.

C20. ARVD/ARVC

A female with exertional syncope and family history of sudden cardiac death; ECG shows an epsilon wave; echo shows an enlarged RV with a normal LV. Most likely diagnosis?

- a) HOCM
- b) Arrhythmogenic RV dysplasia/cardiomyopathy
- c) Pulmonary embolism
- d) Athlete's heart

Answer: Arrhythmogenic RV dysplasia (ARVD/ARVC).

Explanation: Exertional syncope + family history of SCD + epsilon wave + dilated/dysfunctional RV with normal LV is characteristic of ARVC.

C21. HFrEF — completing GDMT

A 68-year-old with ischemic cardiomyopathy, LVEF 30%, on a loop diuretic, beta-blocker, ARNI and an MRA. Best next step?

- a) Add SGLT2 inhibitor
- b) Add digoxin
- c) Implant a device
- d) Add an ARB

Answer: Add an SGLT2 inhibitor (complete guideline-directed medical therapy).

Explanation: The four pillars of HFrEF therapy are ARNI/ACEi/ARB, beta-blocker, MRA and an SGLT2 inhibitor. This patient is on three; adding an SGLT2 inhibitor optimizes GDMT before device therapy.

C22. Decompensated HF — what NOT to give

A patient with decompensated heart failure — which drug should NOT be given?

- a) Morphine
- b) Metoprolol
- c) Aspirin
- d) Nitroglycerin

Answer: Metoprolol (do not start/up-titrate a beta-blocker during acute decompensation).

Explanation: Beta-blockers should not be initiated or up-titrated in acute decompensated HF — negative inotropy worsens low-output state. They are started once euvolemic and stable.

Reviewer note: The exam key listed "B or A." Morphine is also now discouraged in acute HF (worse outcomes), but the classically taught contraindication during decompensation is starting a beta-blocker. If one answer is required, choose metoprolol.

C23. HFrEF — drug to reduce mortality

HF patient with EF 35% on captopril, metoprolol and furosemide. Which drug added reduces mortality?

- a) Losartan
- b) Digoxin
- c) Spironolactone

Answer: Spironolactone.

Explanation: A mineralocorticoid receptor antagonist added to an ACE inhibitor + beta-blocker reduces mortality in HFrEF. Digoxin reduces hospitalizations but not mortality; adding an ARB to an ACEi is not recommended.

C24. Aortic stenosis — worst prognostic symptom

In aortic stenosis, which symptom carries the highest 1-year mortality?

- a) Angina
- b) Syncope
- c) Heart failure
- d) Atrial fibrillation

Answer: Heart failure (dyspnea).

Explanation: The triad of symptomatic severe AS is angina, syncope and heart failure, with progressively worse survival: angina ~5 yr, syncope ~3 yr, heart failure ~2 yr (shortest survival / highest 1-year mortality).

C25. Vulnerable plaque

Which indicates an atherosclerotic plaque susceptible to rupture?

- a) Thick fibrous cap
- b) Abundant smooth muscle
- c) Abundant inflammatory cells
- d) Small lipid pool

Answer: Abundant inflammatory cells.

Explanation: A vulnerable plaque has a thin fibrous cap, a large lipid-rich core and abundant inflammatory macrophages with few smooth muscle cells. The other options describe a stable plaque.

C26. Resistant hypertension — required drug class

In the definition of resistant hypertension, three drugs must be used — which class must be included?

- a) Beta blockers
- b) Diuretics
- c) ACE inhibitors
- d) Calcium channel blockers

Answer: A diuretic.

Explanation: Resistant hypertension = BP above goal despite ≥3 drugs of different classes at optimal doses, one of which must be a diuretic (or controlled BP needing ≥4 drugs).

C27. Atrial septal defect [×2]

A 32-year-old female with mild exercise intolerance; BP 100/70, pulse 68 regular; S2 **split throughout the respiratory cycle (fixed split)**; grade 2/6 midsystolic murmur at the 2nd left ICS (± a diastolic rumble at the lower left sternal border); both murmurs increase with inspiration; ECG with right axis deviation. Most likely diagnosis?

- a) Left atrial myxoma
- b) Mitral stenosis
- c) Atrial septal defect
- d) Hypertrophic cardiomyopathy
- e) Pulmonary artery hypertension

Answer: Atrial septal defect.

Explanation: Wide **fixed** splitting of S2, a pulmonary mid-systolic flow murmur (and a tricuspid diastolic flow rumble), right axis deviation, and right-sided murmurs increasing with inspiration are classic for an ASD with left-to-right shunt.

Reviewer note: Exam keys differ (2020 → ASD; 2017 → pulmonary artery hypertension). **Fixed splitting of S2** is the single most specific clue → ASD; PHTN would give a loud P2 and RV pressure-overload signs. Treat ASD as the intended answer.

C28. Edema with vs without raised JVP

A patient with bilateral leg edema and JVP 4 cm above the sternum. All can cause his condition EXCEPT:

- a) Right-sided heart failure
- b) Cirrhosis
- c) Nephrotic syndrome
- d) Pelvic venous obstruction/fibrosis

Answer: Cirrhosis / nephrotic syndrome (whichever does **not** raise the JVP).

Explanation: A raised JVP reflects raised central venous pressure (RHF, or obstruction of venous return). Cirrhosis and nephrotic syndrome cause edema by low oncotic pressure / fluid retention **without** raising JVP.

Reviewer note: The 2020 key marked "right-sided heart failure" as the exception — that is wrong reasoning; RHF is the prototypical cause of a raised JVP. The conditions causing edema *without* a raised JVP are cirrhosis and nephrotic syndrome — re-read the exact stem to pick the true exception.

C29. BNP facts

Which is true about BNP?

- a) BNP decreases with aging
- b) Levels don't affect prognosis
- c) Higher specificity than sensitivity
- d) Falsely low in obese patients

Answer: BNP is falsely low in obese patients.

Explanation: Obesity lowers BNP and can mask heart failure. BNP rises with age, correlates with prognosis, and has high **sensitivity** (good for ruling OUT HF) — higher sensitivity than specificity.

C30. TTE first-line for valvular/right-heart evaluation

Ejection click at the right 2nd ICS — best test? / 58-year-old woman with dyspnea, leg swelling, JVP that increases with inspiration — best next step for diagnosis?

- a) Transthoracic echocardiogram (TTE)
- b) Transesophageal echocardiogram
- c) Right and left heart catheterization

Answer: Transthoracic echocardiogram (TTE).

Explanation: TTE is the first-line, non-invasive test for valvular lesions and right-heart pathology.

Reviewer note: Distinguish from C3 (normal echo + prominent X/Y descents → catheterization). When the echo has not yet been done, start with TTE.

C31. Complete heart block ECG strip

An 80-year-old woman with 3 weeks of dizziness and palpitations, previously well; ECG shown. Diagnosis?

- a) Sinus rhythm with PACs
- b) Sinus bradycardia
- c) Atrial fibrillation
- d) 2nd-degree heart block
- e) 3rd-degree (complete) heart block

Answer: 3rd-degree (complete) AV block (per the exam key).

Explanation: Image-dependent. Complete heart block shows AV dissociation — independent P and QRS rates.

Reviewer note: Confirm AV dissociation on the actual strip before selecting.

C32. Acute limb ischemia — least likely source

A 60-year-old with sudden severe right leg pain; cold leg, no palpable pulses. LEAST likely cause?

- a) Sick sinus syndrome
- b) Infective endocarditis
- c) Paroxysmal atrial fibrillation
- d) Anterior myocardial infarction
- e) Constrictive pericarditis

Answer: Constrictive pericarditis.

Explanation: Acute limb ischemia from arterial embolism arises from cardiac clot sources: AF/sick sinus syndrome (atrial thrombus), infective endocarditis (vegetations), anterior MI (LV mural thrombus). Constrictive pericarditis does not generate systemic arterial emboli.

C33. Severe preeclampsia — acute BP control

A G2P1 woman at 30 weeks with headache and blurry vision; BP 160/90, HR 90, Cr 1.7, platelets 80K. Most appropriate next step?

- a) IV labetalol
- b) Oral methyldopa
- c) Lisinopril
- d) Nifedipine

Answer: IV labetalol.

Explanation: This is severe preeclampsia (severe-range BP with end-organ involvement). IV labetalol is first-line for acute severe hypertension in pregnancy. ACE inhibitors are contraindicated in pregnancy.

Reviewer note: Definitive treatment is delivery + magnesium sulfate for seizure prophylaxis; among the options, IV labetalol is the correct acute BP control.

C34. PPI-induced hypomagnesemia

A 65-year-old with GERD on chronic PPI/antacids; fatigue, cramps, palpitations. Mg 1.0 (low), Ca 7.5 (low), K 3.1 (low). Best next step?

- a) IV magnesium sulfate
- b) Correct hypocalcemia
- c) IV potassium

- d) IV calcium gluconate
- e) Stop PPIs, start H2 blockers

Answer: IV magnesium sulfate.

Explanation: Chronic PPI use causes hypomagnesemia, which in turn causes **refractory** hypokalemia (renal K wasting) and hypocalcemia (impaired PTH). Correct magnesium first — the K and Ca will not correct until Mg is repleted.

C35. HTN in diabetic with CKD — drug combination

Long-standing diabetes, kidney disease and smoking, with elevated BP. Best starting combination?

- a) Lisinopril and amlodipine
- b) Hydralazine and nitrate
- c) Lisinopril and valsartan
- d) Amlodipine and hydrochlorothiazide

Answer: Lisinopril and amlodipine.

Explanation: A diabetic with CKD (and proteinuria) should get a renin-angiotensin blocker (ACE inhibitor) for renal protection, plus a calcium channel blocker.

Reviewer note: Combining an ACEi with an ARB (lisinopril + valsartan) is avoided (hyperkalemia, AKI). This *contradicts* the C36 "slide note" — follow standard guidelines: do not routinely combine ACEi + ARB.

C36. CCB + ARB + diuretic, BP not at goal

HTN and DM, on a CCB, valsartan and a diuretic; BP 135/85. Best next step?

- a) Increase valsartan dose
- b) Add enalapril (ACE inhibitor)
- c) Renal artery ultrasound
- d) Increase diuretic dose

Answer: Per exam key: add enalapril.

Explanation: The 2019 exam, citing course slides, accepted adding an ACE inhibitor to the ARB in a diabetic not at the goal of <130/80.

Reviewer note (CORRECTION): Routine combined ACEi + ARB therapy is **NOT** recommended by current guidelines (ONTARGET trial: more harm — hyperkalemia, renal dysfunction — without CV benefit). The standard correct step is to up-titrate existing agents and confirm adherence/exclude pseudoresistance. This is a case where the exam key reflects the course slide, not current best practice.

2. RESPIRATORY

R1. PFT facts [×3]

Which is true about PFTs? (Combined options across exams.)

- a) FEV1/FVC is not affected by age
- b) Pulmonary hemorrhage has low DLCO
- c) Emphysema has decreased total lung capacity
- d) Obesity doesn't affect the FEV1/FVC ratio
- e) High RV and low TLC in motor neuron disease
- f) DLCO is decreased in asthma

Answer: In motor neuron (neuromuscular) disease: **low TLC with relatively preserved/high RV**; and obesity does not significantly change the **FEV1/FVC ratio**.

Explanation: Neuromuscular disease gives a restrictive pattern (low TLC) but RV is often relatively preserved/high (expiratory muscle weakness). Obesity reduces lung volumes (restrictive) but the FEV1/FVC ratio is preserved. Emphysema **increases** TLC (hyperinflation). Alveolar hemorrhage gives a **high** (not low) DLCO. DLCO is typically **normal** in asthma.

Reviewer note: Different papers use different option sets. Key facts: emphysema → high TLC; alveolar hemorrhage → high DLCO; obesity → preserved FEV1/FVC; neuromuscular disease → low TLC with preserved RV.

R2. Hypersensitivity pneumonitis — diagnosis [×3]

Recurrent dyspnea and fever over months after a parrot/pigeon was brought home; HRCT shows upper-/mid-zone changes; biopsy shows non-caseating granulomas with interstitial fibrosis. Most likely diagnosis?

- a) Cryptogenic organizing pneumonia
- b) Hypersensitivity pneumonitis
- c) Sarcoidosis
- d) Idiopathic pulmonary fibrosis

Answer: Hypersensitivity pneumonitis (bird fancier's lung).

Explanation: Bird (parrot/pigeon) exposure + recurrent dyspnea/fever + upper/mid-zone HRCT changes + non-caseating granulomas with interstitial fibrosis is classic.

Reviewer note: Best initial follow-up investigation is HRCT; bronchoalveolar lavage (lymphocytosis) supports the diagnosis. An IM-2023 variant with "mixed restrictive, non-caseating granulomas, clubbing" → same answer.

R3. OHS — acute hypercapnic failure [×3]

A male with obesity hypoventilation syndrome, non-compliant with home CPAP, presents with dyspnea. ABG: pH 7.16, $PaCO_2$ 72, PaO_2 56 (or pH 7.18 / $PaCO_2$ 75; or pH 7.29 / $PaCO_2$ 64 / PaO_2 58). Best next step?

- a) Mechanical (invasive) ventilation
- b) Use CPAP at a rate of 16
- c) BiPAP
- d) Nasal cannula

Answer: BiPAP (non-invasive bilevel positive airway pressure).

Explanation: Acute-on-chronic hypercapnic respiratory failure in OHS is treated with BiPAP, which provides inspiratory pressure support to augment ventilation and lower $PaCO_2$. CPAP only splints the airway and does not actively ventilate. Invasive ventilation is for BiPAP failure/contraindications.

R4. COPD pharmacotherapy — Group E [×2]

A COPD patient with mMRC 2 and CAT 15, presenting with an exacerbation and ≥1 prior exacerbation/hospitalization this year. Best next step?

- a) LABA
- b) LAMA + LABA
- c) LABA + LAMA + ICS
- d) LAMA

Answer: LAMA + LABA (GOLD Group E).

Explanation: A symptomatic COPD patient with frequent/severe exacerbations is GOLD Group E. Initial therapy is LABA + LAMA. ICS is added (triple therapy) if exacerbations persist on dual bronchodilation or if blood eosinophils are high (≥300).

R5. UIP HRCT — what is NOT seen

What is NOT seen on a high-resolution CT of usual interstitial pneumonia (UIP)?

- a) Peripheral infiltrates
- b) Honeycombing
- c) Subpleural infiltrates
- d) Profuse nodules / mosaic attenuation

Answer: Profuse nodules / mosaic attenuation.

Explanation: UIP shows peripheral, subpleural, basal-predominant reticulation, honeycombing and traction bronchiectasis. Profuse nodules or a mosaic pattern are NOT UIP features and suggest an alternative (hypersensitivity pneumonitis, sarcoidosis).

Reviewer note: An IM-2023 variant asks "not an ILD pattern" with options including "mosaicism with pulmonary nodules" — same concept.

R6. Sarcoidosis — NOT an indication to treat [×2]

Which is NOT an indication for treatment in sarcoidosis?

- a) Declining pulmonary function
- b) Lupus pernio
- c) Erythema nodosum
- d) Hypercalcemia

Answer: Erythema nodosum.

Explanation: Sarcoidosis is treated for significant organ dysfunction: progressive lung disease, cardiac/neurologic involvement, hypercalcemia, ocular disease, or disfiguring skin disease (lupus pernio). Erythema nodosum is self-limited, indicates Löfgren syndrome, and carries a **good** prognosis — not an indication to treat.

R7. CAP disposition — CURB-65 (unit trap) [×2]

A 50-year-old with cough/sputum 3 days; BP 118/70, RR ~20, BUN 10 — most appropriate management? (Variant: 44-year-old with confusion, RR 26, BP 95/65, **urea 8.2 mmol/L**.)

- a) Admit to the ward, IV antibiotics
- b) Discharge with 5 days of oral antibiotics (e.g., levofloxacin)
- c) Admit to the ward on oxygen
- d) Admit to ICU

Answer: Depends on CURB-65: score 0–1 → outpatient oral antibiotics; score 2 → consider ward admission.

Explanation: CURB-65 = Confusion, Urea >7 mmol/L (>19–20 mg/dL), RR ≥30, BP <90/≤60, Age ≥65. Score 0–1 → outpatient; 2 → ward; ≥3 → consider ICU.

Reviewer note (UNIT TRAP): In the 44-year-old case the urea is in **mmol/L** — 8.2 mmol/L ≈ 23 mg/dL, which **is** above threshold. Confusion + high urea → CURB-65 ≥2 → admit. The 50-year-old with normal BP, RR ~20, BUN 10 is low-risk → outpatient oral therapy. Always convert urea units before scoring.

R8. Combined pre- and post-capillary pulmonary HTN [×2]

PHTN found on echo; right heart catheterization shows PCWP/PAWP 25–30 mmHg and increased PVR (~4 WU). Type of pulmonary hypertension?

- a) Pre-capillary (pulmonary arterial) hypertension
- b) Post-capillary hypertension
- c) Combined pre- and post-capillary hypertension
- d) Exercise pulmonary hypertension

Answer: Combined pre- and post-capillary pulmonary hypertension.

Explanation: A high PCWP (>15 mmHg) = post-capillary (left-heart) component; an elevated PVR (>2–3 WU) = added pre-capillary component → combined pre- and post-capillary PHTN.

R9. Chylothorax — best test [×2]

A pleural effusion; pleurocentesis shows milky fluid. Best test to distinguish it?

- a) Cholesterol
- b) Triglycerides (in the pleural fluid)

Answer: Triglyceride level in the pleural fluid.

Explanation: A milky effusion is investigated with pleural fluid triglycerides: >110 mg/dL confirms chylothorax. A pseudochylothorax instead has high cholesterol with low triglycerides.

R10. Asthma controller step-up

A female with dyspnea/cough like childhood asthma; uses a reliever several times with frequent nighttime awakenings. Best next step?

- a) Use the reliever 3 times a week
- b) High-dose ICS + LABA + reliever
- c) Medium-dose ICS + LABA + reliever
- d) LAMA + LABA

Answer: Medium-dose ICS + LABA (with an as-needed reliever).

Explanation: Frequent symptoms and nocturnal awakenings = uncontrolled asthma needing controller step-up (GINA step 3–4: medium-dose ICS–LABA + reliever). SABA-only is inadequate; LAMA+LABA without ICS is a COPD regimen.

Reviewer note: An IM-2023 variant (SABA-only, worsening 3 months) similarly favors stepping up to ICS+LABA; high-dose ICS is for more severe disease.

R11. Asthma "uncontrolled" — first check technique

A 28-year-old with poor asthma control despite SABA + ICS, frequent symptoms. Most appropriate next step?

- a) Add a LABA
- b) Add a leukotriene receptor antagonist
- c) Check inhaler technique and adherence
- d) Increase the ICS dose
- e) Switch to a combination LABA/ICS inhaler

Answer: Check inhaler technique and adherence.

Explanation: Before escalating pharmacotherapy in "uncontrolled" asthma, first confirm correct inhaler technique, adherence, and address triggers/comorbidities — apparent treatment failure is often technique/adherence.

R12. Acute asthma — what is NOT indicated

A 20-year-old with worsening asthma over 3 days, rescue inhaler ineffective; RR 30, O2 sat 93%. Which is NOT indicated?

- a) Montelukast
- b) Glucocorticoids
- c) Albuterol nebulizer
- d) Magnesium
- e) Oxygen

Answer: Montelukast.

Explanation: Acute exacerbation: oxygen, inhaled SABA, systemic glucocorticoids, and IV magnesium for severe attacks. Leukotriene receptor antagonists are controllers with no role in acute exacerbations.

R13. Severe allergic asthma — omalizumab

A 34-year-old with poorly controlled asthma; total IgE 1800 (high), eosinophils 1200 (high). Next best step?

- a) Omalizumab
- b) Benralizumab
- c) Low-dose oral corticosteroids

Answer: Omalizumab.

Explanation: Severe allergic asthma with markedly elevated IgE → omalizumab (anti-IgE). Benralizumab (anti-IL-5R) targets eosinophilic asthma; chronic oral steroids are avoided.

R14. Acute asthma in pregnancy

A 27-week pregnant woman with asthma, SOB and dry cough 10 days; O2 sat 90%, RR 24; reluctant to take medications. Next best step?

- a) O2 + systemic steroids + SABA
- b) O2 + ICS + SABA
- c) O2 + systemic and inhaled steroids
- d) ICS + SABA

Answer: O2 + systemic steroids + SABA.

Explanation: This is an acute exacerbation (hypoxia, tachypnea). Treatment is the same in pregnancy — oxygen, inhaled SABA, systemic corticosteroids. Under-treating asthma is more dangerous to the fetus than the medications.

R15. Confirming the asthma diagnosis — atypical feature

A 25-year-old labelled asthmatic, on PRN SABA. Which feature does NOT support asthma?

- a) Cold and exercise exacerbate symptoms
- b) Symptoms relieved by a beta-2 agonist
- c) Productive cough
- d) Symptoms worse at night
- e) History of childhood asthma

Answer: Productive cough.

Explanation: Asthma causes a **dry** cough with episodic wheeze, nocturnal symptoms, triggers (cold air, exercise), beta-agonist responsiveness, and atopic/childhood history. A productive (purulent) cough is atypical and suggests an alternative diagnosis.

R16. Life-threatening asthma — which feature does NOT qualify

An 18-year-old with an asthma attack: poor air entry bilaterally, almost no wheeze, central cyanosis; ABG pH 7.38, PaO_2 59, $PaCO_2$ 44. Which factor does NOT indicate a life-threatening attack?

- a) No wheezes (silent chest)
- b) Respiratory rate 10
- c) $PaCO_2$ 44 mmHg
- d) Extremely poor air entry bilaterally
- e) Using accessory muscles of respiration

Answer: Using accessory muscles of respiration.

Explanation: Life-threatening features include a silent chest, poor effort/low RR (exhaustion), cyanosis, and a normal or rising $PaCO_2$ (it should be low in an attack). Accessory muscle use indicates a **severe** attack but is not itself a "life-threatening" criterion.

Reviewer note: A "normal" $PaCO_2$ (44) in an acute attack is ominous — it signals tiring; the patient should be hyperventilating with a low $PaCO_2$.

R17. Asthma trigger — least likely

A 23-year-old with recent HTN admitted with an acute asthma attack. LEAST likely trigger?

- a) Aspirin
- b) Beta blockers
- c) ACE inhibitors
- d) Vape
- e) Upper respiratory tract infection

Answer: ACE inhibitors.

Explanation: Aspirin/NSAIDs (aspirin-exacerbated respiratory disease), beta-blockers (bronchoconstriction), vaping and URTIs are recognized triggers. ACE inhibitors cause a dry cough but do not typically trigger bronchospasm/asthma attacks.

R18. Long-term oxygen therapy — indication

Which is an indication for long-term (lifelong) oxygen therapy in COPD?

- a) PaO_2 61 mmHg with right heart failure
- b) PaO_2 56 mmHg with polycythemia
- c) Advanced COPD with O2 sat 89% on room air
- d) O2 sat 93%

Answer: PaO_2 56 mmHg with polycythemia.

Explanation: LTOT is indicated when resting PaO_2 ≤55 mmHg (SaO_2 ≤88%), OR PaO_2 56–59 with cor pulmonale, right heart failure or polycythemia (Hct >55%).

Reviewer note: PaO_2 61 + RHF is above threshold; O2 sat 89% alone is borderline. The cleanest correct answer is the PaO_2 56 + polycythemia option.

R19. Oxygen therapy mechanism in COPD

Which is true about oxygen therapy in COPD patients?

- a) Improves survival in mild COPD with O2 sat 93%
- b) Works by vasoconstriction of pulmonary vasculature
- c) Works by improving hypoxemia and decreasing pulmonary vascular resistance
- d) Improves survival in COPD with a PaO_2 of 57

Answer: It works by improving hypoxemia and decreasing pulmonary vascular resistance.

Explanation: Long-term oxygen relieves hypoxic pulmonary **vasoconstriction**, lowering PVR and pulmonary artery pressure, and improves survival in severe resting hypoxemia. It does not benefit mild COPD.

Reviewer note: A PaO_2 of 57 alone does not meet criteria (need ≤55, or 56–59 with cor pulmonale/polycythemia).

R20. Bronchiectasis — true statement

A male with copious green sputum, suspected bronchiectasis. Which is true?

- a) Ceftriaxone 3×/week decreases exacerbations
- b) Hemoptysis is a rare complication
- c) It can cause a restrictive pattern of disease
- d) Long-term azithromycin reduces exacerbations

Answer: Long-term (macrolide) azithromycin reduces exacerbations; bronchiectasis is primarily an **obstructive** disease.

Explanation: Long-term low-dose macrolide therapy reduces exacerbation frequency (anti-inflammatory + antimicrobial). Hemoptysis is a **common** complication. Bronchiectasis usually causes an obstructive (not restrictive) pattern.

Reviewer note: The IM-2023 paper offers "azithromycin outpatient long term" vs "numerous IV antibiotics in hospital" — choose long-term azithromycin.

R21. PFT — restrictive pattern (IPF)

PFT: FEV1/FVC ~80% (high/normal), FEV1 ~60%, FVC ~60%, DLCO ~50%. Most likely diagnosis?

- a) Emphysema
- b) Idiopathic pulmonary fibrosis (IPF)
- c) Neuromuscular disease

Answer: Idiopathic pulmonary fibrosis (restrictive pattern with low DLCO).

Explanation: A preserved/high FEV1/FVC with proportionally reduced FEV1 and FVC = restrictive pattern. A **low DLCO** points to interstitial disease (IPF). Emphysema shows obstruction; neuromuscular restriction usually preserves DLCO.

R22. Restrictive lung disease — which is restrictive

PFT question — which is true about restrictive lung disease?

- a) FEV1 <60% and FEV1/FVC <70% implies restrictive disease
- b) ILD shows a restrictive pattern
- c) Emphysema is restrictive
- d) Bronchiectasis is restrictive

Answer: ILD shows a restrictive pattern.

Explanation: Restriction = reduced TLC with a preserved/increased FEV1/FVC ratio; ILD is the prototypical restrictive disease. A low FEV1/FVC ratio (<70%) = obstruction (emphysema, bronchiectasis).

R23. ARDS — false statement

ARDS — which statement is FALSE?

- a) Non-cardiogenic pulmonary edema
- b) The PaO_2/FiO_2 ratio signifies severity
- c) Treatment includes mechanical ventilation
- d) The mechanism of hypoxia is hypoventilation

Answer: The mechanism of hypoxia is hypoventilation.

Explanation: ARDS hypoxemia is caused by intrapulmonary **shunting** and V/Q mismatch from alveolar flooding — refractory to supplemental oxygen, not hypoventilation. ARDS is non-cardiogenic pulmonary edema; severity is graded by PaO_2/FiO_2; lung-protective ventilation is the mainstay.

R24. Sarcoidosis — most diagnostic test

A 37-year-old African American woman with chronic dry cough, recurrent episodes, and erythema nodosum (or classic sarcoidosis with hilar lymphadenopathy + erythema nodosum). Most diagnostic test / best next step?

- a) Chest X-ray
- b) Lung biopsy
- c) Lymph node biopsy
- d) Start steroids
- e) Bronchoalveolar lavage

Answer: Tissue biopsy showing non-caseating granulomas (lymph node or lung biopsy).

Explanation: Definitive diagnosis of sarcoidosis requires histology of non-caseating granulomas after excluding other causes.

Reviewer note: Exam keys vary (2018 → lung biopsy; 2019 → lymph node biopsy or start steroids). In classic Löfgren syndrome many clinicians treat without biopsy, but the most **diagnostic** test is tissue biopsy.

R25. Sarcoidosis — good prognostic sign

Dyspnea/dry cough with bilateral hilar enlargement and erythema nodosum. Good prognostic sign?

- a) Erythema nodosum
- b) Shortness of breath
- c) Bilateral hilar enlargement with vascular nodularity
- d) Dry cough

Answer: Erythema nodosum.

Explanation: Erythema nodosum (Löfgren syndrome) carries a good prognosis with a high rate of spontaneous remission. Lupus pernio indicates chronic disease and poor prognosis.

R26. Bird-exposure HP — bronchoalveolar lavage

A 45-year-old with dry cough, low-grade fever, malaise 2 weeks; close contact with a pet parrot; CXR shows bilateral hilar lymphadenopathy and patchy infiltrates. Next best step in evaluation?

- a) Lung biopsy
- b) Start steroids
- c) Tuberculin skin test (PPD)
- d) Bronchoalveolar lavage

Answer: Bronchoalveolar lavage.

Explanation: For suspected hypersensitivity pneumonitis (bird exposure), BAL typically shows marked lymphocytosis and supports the diagnosis non-invasively before lung biopsy.

R27. New clubbing in a smoker with ILD/COPD

A 70-year-old retired smoker with chronic dry cough and exertional dyspnea; clubbing, decreased breath sounds, fine end-inspiratory crackles. Next step?

- a) Spirometry with bronchodilator
- b) Full blood count
- c) Chest CT with contrast
- d) High-resolution chest CT
- e) Antinuclear antibody

Answer: High-resolution chest CT.

Explanation: Clubbing + fine "velcro" end-inspiratory crackles suggest ILD (likely IPF). HRCT is the key next investigation to characterize the interstitial pattern.

R28. New clubbing in COPD — rule out cancer

A 68-year-old with COPD on ipratropium; SaO_2 91%; **new digital clubbing** not present 6 months ago. Most appropriate next step?

- a) Reassurance and follow-up
- b) Home oxygen therapy
- c) DLCO
- d) Chest CT scan with contrast
- e) Start regular bronchodilator

Answer: Chest CT scan with contrast.

Explanation: Clubbing is NOT a feature of COPD. New clubbing in a COPD patient (a smoker) raises concern for lung cancer → chest CT to exclude malignancy.

R29. Definite UIP on HRCT — antifibrotic therapy

A 70-year-old smoker with progressive dyspnea; HRCT shows traction bronchiectasis and honeycombing. Best next step?

- a) Start steroids
- b) Start antifibrotic therapy
- c) Lung biopsy
- d) PFT

Answer: Start antifibrotic therapy.

Explanation: A definite UIP pattern on HRCT (peripheral honeycombing + traction bronchiectasis) in the right context establishes IPF without biopsy. Antifibrotics (pirfenidone, nintedanib) are the treatment; steroids do not benefit IPF.

R30. SVC syndrome — most likely cause

A patient with lung cancer presents with a swollen face and dilated neck/chest veins (SVC syndrome). Most likely cause?

- a) Small cell lung cancer
- b) Squamous cell lung cancer
- c) Adenocarcinoma

Answer: Small cell lung cancer.

Explanation: SVC syndrome is most often caused by a central/mediastinal tumor; small cell lung carcinoma (typically central with bulky mediastinal nodes) is the classic malignant cause.

R31. Lung cancer — true statement

Which is true regarding lung cancer?

- a) Mortality has decreased over the past 20 years
- b) Small cell cancer is chemo-sensitive and has good prognosis
- c) Small cell cancer causes Pancoast tumor by invading the superior sulcus

Answer: Lung cancer mortality has decreased over the past ~20 years.

Explanation: Mortality has declined (reduced smoking, earlier detection, better therapy). Small cell cancer is chemo-sensitive but has a **poor** prognosis. Pancoast tumors are typically **non**-small cell (squamous).

R32. Cavitating lung mass with hypercalcemia — squamous cell

A 60-year-old heavy smoker with fever, productive cough, dyspnea; CXR shows a right-upper-lobe consolidation with a cavitary lesion; serum calcium 11.9. Most likely diagnosis?

- a) Squamous cell carcinoma of the lung
- b) Adenocarcinoma of the lung
- c) Lung abscess
- d) Small cell carcinoma of the lung

Answer: Squamous cell carcinoma of the lung.

Explanation: A central cavitating lung mass with hypercalcemia (paraneoplastic PTHrP) in a heavy smoker is classic for squamous cell carcinoma.

R33. Parapneumonic effusion — mechanism

What is the mechanism of pleural effusion in pneumonia?

- a) Increased oncotic pressure
- b) Decreased oncotic pressure
- c) Increased capillary permeability
- d) Increased hydrostatic pressure
- e) Decreased hydrostatic pressure

Answer: Increased capillary permeability.

Explanation: A parapneumonic effusion is an exudate: pleural inflammation increases capillary permeability, leaking protein-rich fluid. Hydrostatic/oncotic mechanisms produce transudates (HF, cirrhosis).

R34. Low-glucose exudative effusion

Exudative effusion with low glucose — most likely cause? (Variant: fluid protein 3 g/dL — most likely diagnosis.)

- a) Uncomplicated parapneumonic effusion
- b) Rheumatoid effusion
- c) Congestive heart failure
- d) Tuberculosis
- e) Nephrotic syndrome

Answer: Rheumatoid (or tuberculous/empyema) effusion — an exudate with characteristically low glucose.

Explanation: Very low pleural glucose occurs with rheumatoid effusion (often the lowest), empyema/complicated parapneumonic effusion, TB and malignancy. Uncomplicated parapneumonic effusion has near-normal glucose; transudates (CHF, cirrhosis, nephrotic) have normal glucose.

Reviewer note: The 2017 image-based effusion question's key is "Tuberculosis" (exudative lymphocytic effusion). Value/image-dependent — apply Light's criteria first, then use glucose/lymphocyte clues.

R35. Pleural effusion with fever — sample it

A patient with HFrEF and liver cirrhosis; right-sided pleural effusion and fever 39.8°C. Which is correct?

- a) Thoracentesis is indicated
- b) IV furosemide
- c) Pleural fluid LDH differentiates HF vs cirrhosis effusion
- d) Pleural fluid glucose <90 implies empyema

- e) CT is better than US for detecting pleural fluid

Answer: Thoracentesis is indicated.

Explanation: A new pleural effusion with **fever** must be sampled — diagnostic thoracentesis is required to exclude empyema/infection and apply Light's criteria. Ultrasound, not CT, is the preferred bedside tool to detect/guide drainage of pleural fluid.

R36. PFT pattern — IPF vs others

PFT table with FEV1/FVC ~80s, FEV1 ~60s, FVC ~60s, DLCO ~50s. Most likely diagnosis?

- a) Emphysema
- b) IPF
- c) Neuromuscular disease

Answer: IPF (restrictive with reduced DLCO).

Explanation: Preserved/high ratio + reduced volumes = restriction; reduced DLCO points to parenchymal/interstitial disease (IPF).

R37. PE — highest mortality risk

Which carries the highest mortality in PE?

- a) Positive troponin
- b) PESI grade IV
- c) RV dysfunction on echo/CTPA
- d) Bilateral saddle embolism
- e) Vasopressor requirement to maintain SBP above 90 mmHg

Answer: Vasopressor requirement to maintain SBP above 90 mmHg (hemodynamic instability = high-risk/massive PE).

Explanation: Hemodynamic instability — hypotension/shock requiring vasopressors — defines high-risk PE with the highest short-term mortality. Positive troponin and RV dysfunction = intermediate-risk; clot burden alone (saddle embolus) is less prognostically important than hemodynamics.

R38. PE — A–a gradient and mechanism

A 40-year-old with SOB and pleuritic chest pain; PE suspected; ABG pH 7.45, $PaCO_2$ 32, HCO_3 22, PaO_2 67. Expected A–a gradient and mechanism of hypoxia?

- a) Hypoventilation with normal A–a gradient
- b) V/Q mismatch with increased A–a gradient
- c) V/Q mismatch with normal A–a gradient
- d) Shunt with increased A–a gradient
- e) Shunt with normal A–a gradient

Answer: V/Q mismatch with an increased A–a gradient.

Explanation: PE causes hypoxemia by V/Q mismatch, which **widens** the A–a gradient. The patient is also hyperventilating (low $PaCO_2$, respiratory alkalosis). A normal A–a gradient with hypoxemia would instead indicate hypoventilation/low inspired O_2.

R39. Advanced PAH — epoprostenol

A 30-year-old with 1 year of dyspnea and cough, now faint; loud P2, RV heave, leg edema; ECG right axis deviation. Best therapy?

- a) Ambrisentan
- b) Treprostinil
- c) Epoprostenol
- d) Sildenafil

Answer: Epoprostenol (IV prostacyclin) — for advanced/high-risk PAH.

Explanation: Right heart failure and syncope indicate advanced, high-risk PAH; IV prostacyclin (epoprostenol) is the most effective therapy and improves survival; oral agents are for lower-risk disease.

Reviewer note: Exam key marked "C (Not sure)." Epoprostenol is the standard answer for high-risk PAH with right heart failure.

R40. Suspected TB, smear-negative — bronchoscopy

A 60-year-old with cough, hemoptysis, weight loss, night sweats and a RUL cavity; HIV negative; sputum AFB negative ×3. Best next step?

- a) Repeat sputum AFB after one month
- b) Blood culture for mycobacteria
- c) Bronchoscopy
- d) Treat empirically for TB
- e) PPD test

Answer: Bronchoscopy (with bronchoalveolar lavage / biopsy).

Explanation: When suspicion of TB is high but sputum smears are repeatedly negative, bronchoscopy with BAL (and biopsy) obtains a diagnostic sample for AFB smear, culture and NAAT.

R41. Extended anticoagulation — unprovoked VTE

Which indicates anticoagulation BEYOND the standard 3–6 months?

- a) DVT due to a long flight
- b) DVT due to pneumonia and sepsis
- c) DVT due to multiple comorbidities
- d) Unprovoked DVT

Answer: Unprovoked DVT.

Explanation: VTE provoked by a transient major risk factor (surgery, flight, acute illness) is treated for 3 months. An **unprovoked** VTE (or with a persistent risk factor / active cancer) carries high recurrence risk → extended/indefinite anticoagulation if bleeding risk allows.

R42. VTE risk factor stratification — IVF

A pregnant woman by IVF, on bed rest 4 days, leg swelling and SOB; varicose veins. Which is a MODERATE risk factor for PE?

- a) Bed rest for 4 days
- b) Pregnancy
- c) IVF
- d) Varicose veins

Answer: IVF (assisted reproduction).

Explanation: VTE risk factors are strong (major surgery, fracture, prior VTE), moderate (pregnancy/postpartum, IVF/hormonal therapy, prolonged immobility), and weak (varicose veins, short bed rest, obesity). The exam classifies IVF as moderate.

Reviewer note: Pregnancy is also a recognized moderate-risk factor; the exam key is IVF.

R43. Lung cancer — Pancoast tumor management

Management of a Pancoast (superior sulcus) tumor?

- a) Surgery (as part of multimodality treatment)
- b) Chemotherapy alone

Answer: Neoadjuvant chemoradiation followed by surgical resection.

Explanation: Resectable superior sulcus tumors without distant spread are treated with concurrent chemoradiotherapy followed by surgery.

Reviewer note: The IM-2023 paper simply states "Pancoast tumor, do surgery" — surgery is part of multimodality treatment after neoadjuvant chemoradiation.

R44. OHS — false statement

Which is wrong about obesity hypoventilation syndrome?

- a) Most common symptom is exertional dyspnea
- b) HCO_3 levels are usually elevated
- c) Long-standing disease leads to cor pulmonale
- d) Associated with sleep apnea / sleep hypoventilation
- e) Hypercapnia without hypoxia

Answer: Hypercapnia without hypoxia.

Explanation: OHS is defined by daytime hypercapnia AND hypoxemia. Chronic respiratory acidosis is renally compensated with an elevated bicarbonate; longstanding disease causes pulmonary hypertension and cor pulmonale.

R45. COPD ABCD/GOLD classification

A 65-year-old, FEV1 45% predicted, one exacerbation requiring hospitalization, CAT 15, mMRC 2. ABCD assessment classification?

- a) GOLD grade 4, group D
- b) GOLD grade 3, group B
- c) GOLD grade 3, group D
- d) GOLD grade 2, group D
- e) GOLD grade 4, group C

Answer: GOLD grade 3, Group D (per the older 2017 ABCD tool).

Explanation: FEV1 45% = GOLD spirometric grade 3 (30–49%). Under the older ABCD tool, ≥1 hospitalization for exacerbation + high symptoms (CAT ≥10 / mMRC ≥2) = Group D.

Reviewer note: The 2023 GOLD update merged groups C and D into a single **Group E** for high exacerbation risk. By current GOLD this patient is Grade 3, Group E. The 2017 exam answer reflects the older system.

3. ENDOCRINE

E1. Graves disease — diagnosis [×2]

A female with palpitations and pretibial plaques (pretibial myxedema), TSH 0.005, T4 20 (or weight loss, palpitations, dry/bulging eyes, elevated free T4). Most likely diagnosis?

- a) Graves disease
- b) Toxic multinodular goiter
- c) Subacute thyroiditis

Answer: Graves disease.

Explanation: Hyperthyroidism with **extrathyroidal** manifestations — pretibial myxedema and ophthalmopathy (proptosis) — is specific for Graves disease (TSH-receptor antibody mediated). Toxic multinodular goiter and thyroiditis do not cause eye disease or pretibial myxedema.

E2. HbA1c → average glucose [×2]

If HbA1c is 7%, the estimated average glucose over the past 3 months?

- a) 150 mg/dL
- b) 210 mg/dL
- c) 240 mg/dL

Answer: ~150 mg/dL.

Explanation: Estimated average glucose ≈ 28.7 × A1c − 46.7. A1c 7% ≈ 154 mg/dL. Anchors: A1c 6% ≈ 126, 7% ≈ 154, 8% ≈ 183, 9% ≈ 212.

E3. Primary hyperparathyroidism — surgical criteria [×2]

An asymptomatic patient with calcium 13, high PTH and a T-score of −3.5 (or asymptomatic, corrected Ca 10.6, high PTH, high urinary calcium). Best next step?

- a) Sestamibi scan
- b) Surgical referral for parathyroidectomy
- c) Bisphosphonate
- d) Observation
- e) Cinacalcet

Answer: Surgical referral for parathyroidectomy (for the Ca 13 / T-score −3.5 case).

Explanation: Primary hyperparathyroidism is treated surgically when criteria are met: calcium >1 mg/dL above the upper limit, osteoporosis (T-score ≤ −2.5) or fragility fracture, age <50, or significant renal involvement. Ca 13 + T-score −3.5 clearly meet criteria.

Reviewer note: The two cases differ. Ca 13 + T-score −3.5 → operate. The borderline case (corrected Ca 10.6, mildly high) may meet **observation** criteria — the 2019 key for that milder case is "probably observation." Imaging (sestamibi) is for surgical localization AFTER the decision to operate, not the decision-maker.

E4. Thyroid nodule — FNA vs uptake scan [×2]

A 2.5 cm complex thyroid nodule, TSH 4.3 (or a euthyroid 1 cm solid nodule). Best next step?

- a) FNA

- b) Radioactive iodine uptake test
- c) Observe
- d) Levothyroxine
- e) Reevaluate in 4 weeks

Answer: FNA (fine-needle aspiration).

Explanation: A nodule ≥1 cm with a non-suppressed (normal/high) TSH is evaluated with ultrasound-guided FNA cytology. A radionuclide uptake scan is done instead when TSH is **low** (to find a hyperfunctioning "hot" nodule).

Reviewer note: Decision pivot: low TSH → uptake scan first; normal/high TSH with a nodule ≥1 cm → FNA.

E5. Pheochromocytoma — alpha before beta [×2]

Pheochromocytoma on hydralazine, BP 150/80, phenoxybenzamine unavailable (or on an ACE inhibitor + CCB, BP 185/100). Best management?

- a) Propranolol then doxazosin
- b) Doxazosin then propranolol
- c) Add metoprolol
- d) Add hydralazine
- e) Add phenoxybenzamine

Answer: Alpha-blockade FIRST (doxazosin / phenoxybenzamine), THEN beta-blockade.

Explanation: In pheochromocytoma, alpha-blockade must precede beta-blockade. A beta-blocker first leaves alpha-mediated vasoconstriction unopposed → hypertensive crisis. So doxazosin (or phenoxybenzamine) first, then add propranolol; surgery follows adequate blockade.

Reviewer note: ALWAYS alpha before beta. Adrenalectomy is definitive but only after adequate medical preparation.

E6. Adrenal incidentaloma — screen for hyperaldosteronism [×2]

An adrenal mass found incidentally on CT; cosyntropin and low-dose dexamethasone suppression tests negative (or a small homogeneous adenoma, BP 170/100, normal metanephrines and dexamethasone suppression). Best next step?

- a) CT abdomen
- b) Prolactin level
- c) Plasma aldosterone and renin (aldosterone-to-renin ratio)

Answer: Measure plasma aldosterone and renin (aldosterone-to-renin ratio).

Explanation: An adrenal incidentaloma is screened for three functional possibilities: pheochromocytoma (metanephrines), cortisol excess (dexamethasone suppression), and — if hypertensive — primary aldosteronism (aldosterone-to-renin ratio). With metanephrines and cortisol testing already negative, the hypertensive patient is screened for hyperaldosteronism.

E7. Acromegaly — screening test [×2]

What is the screening test for acromegaly?

- a) Oral glucose tolerance test (OGTT)
- b) Insulin-like growth factor-1 (IGF-1)
- c) IGF binding protein-3

Answer: IGF-1.

Explanation: IGF-1 is the **screening** test (stable, integrates GH secretion). The **confirmatory** test is the oral glucose tolerance test showing failure of GH to suppress.

E8. Acromegaly — confirmatory test

Signs/symptoms of acromegaly — definitive (confirmatory) test?

- a) IGF-BP3
- b) Glucose (OGTT) suppression test
- c) MRI
- d) Insulin stimulation test

Answer: Glucose (OGTT) suppression test.

Explanation: Acromegaly is confirmed by an OGTT: normally glucose suppresses GH; in acromegaly GH fails to suppress. MRI then localizes the pituitary adenoma after biochemical confirmation.

E9. Prediabetes vs diabetes — IFG [×2]

A female obese patient: fasting glucose 111, then a week later 124 (or two readings, 106 then 124). What do you tell the patient?

- a) She has prediabetes and should start lifestyle changes
- b) She has diabetes and should start metformin
- c) Her labs are normal — do nothing

Answer: She has prediabetes — start lifestyle modification.

Explanation: Fasting glucose 100–125 = impaired fasting glucose (prediabetes). Diabetes requires fasting glucose ≥126 (confirmed), A1c ≥6.5%, or random glucose ≥200 with symptoms. Both readings here are in the prediabetes range.

E10. Switching PTU to methimazole in pregnancy [×2]

A pregnant patient with Graves disease on PTU, now 16 weeks, stable but still with a thyroid bruit and exophthalmos. Best next step?

- a) Decrease the PTU dose
- b) Stop PTU and start methimazole
- c) Refer to surgery

Answer: Switch from PTU to methimazole after the first trimester.

Explanation: PTU is preferred in the **first** trimester (methimazole risks teratogenicity — aplasia cutis, embryopathy). After the first trimester, switch to methimazole because PTU carries higher hepatotoxicity risk. At 16 weeks the switch is appropriate.

E11. Diagnosing diabetes in a symptomatic patient [×2]

A patient with random blood sugar 220, polyuria and polydipsia (or polyuria, fasting glucose 240, HbA1c 7.5%). Best advice / next step?

- a) Do fasting blood sugar
- b) Diagnose type 2 DM and start treatment

- c) Prediabetes — give metformin
- d) Do HbA1c
- e) Lifestyle modification + metformin
- f) Repeat the tests

Answer: Diagnose diabetes (symptomatic hyperglycemia) and start treatment — lifestyle modification + metformin (insulin if markedly hyperglycemic/symptomatic).

Explanation: Classic hyperglycemic symptoms with a random glucose ≥200, a fasting glucose ≥126, or A1c ≥6.5% are diagnostic — one test suffices when symptoms are present. First-line therapy is lifestyle modification + metformin.

Reviewer note: Exam keys were inconsistent (2020 → "B or C"; 2019 → "B or C"). The defensible answer: this IS diabetes (no need to repeat or just do A1c), and standard initial treatment is metformin + lifestyle. Insulin is reserved for very high glucose (>300), ketosis, or marked symptoms.

E12. Discordant screening tests — repeat the abnormal one

A 58-year-old asymptomatic man; fasting glucose 129 (diabetes range) and HbA1c 5.6% (normal). Best next diagnostic test?

- a) HbA1c
- b) Repeat fasting blood glucose
- c) Oral glucose tolerance test
- d) Random blood glucose
- e) Fructosamine test

Answer: Repeat fasting blood glucose.

Explanation: When screening tests are discordant, the abnormal test should be repeated for confirmation. An asymptomatic patient needs two abnormal results.

Reviewer note: Contrast with E11, where a symptomatic patient needs only one diagnostic test.

E13. New type 2 DM — screen for complications

A 56-year-old obese female, new type 2 DM, asymptomatic, HbA1c 7.1%. In addition to lifestyle changes and metformin, best next step?

- a) Check urine albumin and refer for a dilated eye exam
- b) Refer to neurology for peripheral neuropathy
- c) Check serum C-peptide and islet cell antibodies
- d) Refer for exercise stress test
- e) CT pancreas

Answer: Check urine albumin and refer for a dilated eye exam.

Explanation: At the diagnosis of type 2 diabetes, screen immediately for microvascular complications — urine albumin-to-creatinine ratio (nephropathy) and a dilated retinal exam (retinopathy) — because type 2 DM may have been present, undiagnosed, for years.

E14. C-peptide distinguishes DM2 from DM1

A 30-year-old diagnosed with diabetes — which finding supports type 2 over type 1?

- a) Waist circumference 70 cm
- b) Other autoimmune diseases

- c) Anti-GAD antibody
- d) Elevated C-peptide

Answer: Elevated C-peptide.

Explanation: C-peptide reflects endogenous insulin secretion: elevated/preserved in type 2 DM (insulin resistance with retained beta-cell function), low/absent in type 1 DM. Anti-GAD antibodies and associated autoimmune disease point to type 1.

E15. Obese diabetic wanting weight loss — GLP-1 agonist

A diabetic with BMI 35, HbA1c 8.5% on metformin, wants to lose weight, actively exercising. Drug of choice?

- a) GLP-1 receptor agonist
- b) SGLT2 inhibitor
- c) Sulfonylurea
- d) Insulin

Answer: GLP-1 receptor agonist.

Explanation: For obesity needing extra glycemic control and weight loss, a GLP-1 receptor agonist is preferred (significant weight loss). Sulfonylureas and insulin cause weight gain; SGLT2 inhibitors cause modest weight loss but less than GLP-1 agonists.

E16. HFrEF + CKD diabetic — SGLT2 inhibitor

A 65-year-old with HFrEF and stage 3 CKD; worsening fatigue, weight gain, edema; eGFR 45. Which diabetes drug gives the greatest cardiorenal benefit?

- a) Insulin
- b) Glipizide
- c) Sitagliptin
- d) Liraglutide
- e) Dapagliflozin

Answer: Dapagliflozin (SGLT2 inhibitor).

Explanation: SGLT2 inhibitors reduce heart failure hospitalizations and slow CKD progression (cardiorenal protection), benefiting HFrEF and CKD — even without diabetes.

E17. SGLT2 inhibitor — mechanism

Mechanism of SGLT2 inhibitors in lowering blood glucose?

- a) Decreases renal (proximal tubular) glucose reabsorption
- b) Increases insulin secretion
- c) Decreases hepatic gluconeogenesis

Answer: Decreases renal proximal-tubular glucose reabsorption, causing glucosuria.

Explanation: SGLT2 inhibitors block the sodium-glucose cotransporter-2 in the proximal tubule, reducing glucose reabsorption and increasing urinary glucose excretion — lowering blood glucose independently of insulin.

E18. Hypogonadism — primary vs secondary [×2]

A 19-year-old with delayed puberty and small testes; high LH, low testosterone (or delayed puberty + **anosmia**, no facial hair, small testes; LH/FSH low-normal, testosterone low). Best diagnostic test / diagnosis?

- a) Testicular biopsy
- b) Give testosterone
- c) Karyotyping
- d) Kallmann syndrome
- e) Klinefelter syndrome
- f) Constitutional delay of puberty

Answer: High LH + low testosterone → primary (hypergonadotropic) hypogonadism → karyotype (Klinefelter 47,XXY). Low LH/FSH + anosmia → Kallmann syndrome.

Explanation: **High** LH/FSH with low testosterone = primary testicular failure; in a young male with small firm testes this is most often Klinefelter — confirm with a karyotype. **Low/inappropriately-normal** LH/FSH with low testosterone = secondary hypogonadism; with anosmia this is Kallmann syndrome.

Reviewer note: The cases hinge on the gonadotropins: HIGH LH → karyotype for Klinefelter; LOW LH with anosmia → Kallmann. Read the LH/FSH carefully.

E19. Mildly elevated prolactin + bitemporal hemianopia

Bitemporal hemianopia and prolactin 32 ng/mL (normal <20). LEAST likely cause?

- a) Exercise
- b) Hypothyroidism
- c) Non-functioning pituitary adenoma
- d) A 2 cm functional prolactinoma
- e) Antipsychotic drugs

Answer: A 2 cm functional prolactinoma.

Explanation: A 2 cm prolactinoma (macroadenoma) would cause a prolactin in the hundreds–thousands, not a mild 32. A mildly elevated prolactin with bitemporal hemianopia is better explained by "stalk effect" from a non-functioning macroadenoma compressing the stalk, or by hypothyroidism/medications/physiologic causes.

E20. Central hyperthyroidism

Hyperthyroid symptoms with T4 30 and TSH 3.5 (non-suppressed TSH). Diagnosis?

- a) Primary hyperthyroidism
- b) Subclinical hyperthyroidism
- c) Central (secondary) hyperthyroidism

Answer: Central (secondary) hyperthyroidism.

Explanation: In primary hyperthyroidism the elevated hormones suppress TSH (TSH low). Here T4 is high but TSH is NOT suppressed — a central cause: a TSH-secreting pituitary adenoma or thyroid hormone resistance.

E21. Hypercalcemia with low PTH — least likely cause

A case of hypercalcemia with low PTH. LEAST likely cause?

- a) Malignancy
- b) Familial hypocalciuric hypercalcemia (FHH)

- c) Chronic kidney disease

Answer: Familial hypocalciuric hypercalcemia.

Explanation: PTH-**independent** hypercalcemia (suppressed PTH) is caused by malignancy, vitamin D excess, granulomatous disease, etc. FHH is PTH-**dependent** — PTH is normal or mildly high with low urinary calcium — so it would not present with a low PTH.

Reviewer note: CKD usually causes secondary hyperparathyroidism with hypocalcemia; a hypercalcemic CKD patient typically has tertiary hyperparathyroidism (high PTH), so CKD also fits poorly with "low PTH" — but FHH is the intended exception (PTH characteristically non-suppressed).

E22. Cushing syndrome — test that is NOT useful

A Cushing syndrome case — which test is NOT useful?

- a) Late-night salivary cortisol
- b) 24-hour urine free cortisol
- c) Low-dose dexamethasone suppression test
- d) Midnight serum cortisol
- e) 8 a.m. serum cortisol

Answer: 8 a.m. (morning) serum cortisol.

Explanation: Screening for Cushing relies on loss of cortisol rhythm/feedback: late-night salivary/serum cortisol, 24-hour urinary free cortisol, and the low-dose dexamethasone suppression test. A single morning cortisol is unhelpful (overlaps widely between normal and Cushing patients).

E23. Confirmed Cushing — next step is plasma ACTH

A patient with facial plethora, central obesity, wide pink striae; non-suppressible cortisol after 1 mg dexamethasone and urine free cortisol >3× normal (confirmed). Most appropriate next step?

- a) Late-night salivary cortisol
- b) MRI of the pituitary
- c) Plasma ACTH level
- d) CT of the adrenal glands
- e) Inferior petrosal sinus sampling

Answer: Plasma ACTH level.

Explanation: Once Cushing **syndrome** is confirmed, determine the cause by measuring plasma ACTH: suppressed ACTH → adrenal source (adrenal CT); normal/high ACTH → ACTH-dependent cause (pituitary MRI ± inferior petrosal sinus sampling).

E24. 21-hydroxylase deficiency — test

A patient with 21-hydroxylase deficiency — most appropriate test?

- a) 17-hydroxyprogesterone
- b) 17-hydroxypregnenolone
- c) Cortisol and aldosterone
- d) DHEA

Answer: 17-hydroxyprogesterone.

Explanation: 21-hydroxylase deficiency (the commonest congenital adrenal hyperplasia) causes accumulation of the substrate 17-hydroxyprogesterone, which is markedly elevated and diagnostic.

E25. CK elevation — not hyperparathyroidism

Which is NOT associated with an increase in creatine kinase (CK)?

- a) Myocardial infarction
- b) Hypothyroidism
- c) Hyperparathyroidism
- d) Muscular dystrophy

Answer: Hyperparathyroidism.

Explanation: CK rises with MI (CK-MB), hypothyroidism (myopathy), and muscular dystrophy. Hyperparathyroidism is not a recognized cause of CK elevation (its proximal myopathy is typically neurogenic with normal CK).

E26. Hypothyroidism — least associated

Which is LEAST likely associated with hypothyroidism?

- a) Hypertension
- b) Hyponatremia
- c) Hypokalemia
- d) Macrocytosis

Answer: Hypokalemia.

Explanation: Hypothyroidism is associated with diastolic hypertension, hyponatremia (impaired free-water excretion) and macrocytosis. It does not characteristically cause hypokalemia.

E27. Factitious hypoglycemia

A 32-year-old pharmacist diaphoretic and restless at work; glucose 41, responded to IV glucose; prolonged fast shows HIGH insulin and UNDETECTABLE C-peptide. Most likely cause?

- a) Insulinoma
- b) Adrenal insufficiency
- c) Acromegaly
- d) Factitious (exogenous insulin)
- e) Renal failure

Answer: Factitious hypoglycemia (surreptitious exogenous insulin).

Explanation: High insulin with **low/undetectable** C-peptide = exogenous insulin (injected insulin contains no C-peptide). An insulinoma shows high insulin **with** high C-peptide. A healthcare worker is the classic factitious scenario.

E28. Insulin resistance — least associated

Which diabetic condition is LEAST likely associated with insulin resistance?

- a) Type 2 diabetes
- b) Gestational diabetes
- c) Cortisol-associated (steroid-induced) diabetes

- d) Pancreatitis-associated diabetes

Answer: Pancreatitis-associated diabetes.

Explanation: Type 2 DM, gestational diabetes and cortisol/Cushing-related diabetes are driven by insulin **resistance**. Diabetes from chronic pancreatitis (type 3c) results from loss of islet cells and insulin **deficiency**.

E29. Early-pregnancy hyperthyroidism

A 27-year-old pregnant woman (8 weeks) with sweating, palpitations, sweaty palms and an enlarged tender thyroid; TSH 0.02, T4 15.03. Next step?

- a) Symptomatic treatment as hCG-related, schedule follow-up
- b) Start carbimazole
- c) Abdominal ultrasound to rule out molar pregnancy
- d) ESR and CRP

Answer: Per exam key: rule out molar pregnancy with ultrasound (key marked "C, not sure").

Explanation: Early-pregnancy hyperthyroidism is often transient gestational thyrotoxicosis (hCG cross-stimulates the TSH receptor); marked thyrotoxicosis can also signal a molar pregnancy (very high hCG).

Reviewer note: Exam key was marked "C (Not sure)." A **tender** thyroid is atypical for both gestational thyrotoxicosis and molar pregnancy and could suggest thyroiditis. The most defensible general approach in early pregnancy with mild hCG-mediated hyperthyroidism is supportive care with follow-up; consider obstetric ultrasound if hCG is disproportionately high or molar features are present. Treat this key as uncertain.

E30. Inpatient glycemic control — basal-bolus insulin

A patient with controlled diabetes on metformin is admitted to hospital. What for glycemic control?

- a) Basal-bolus (scheduled) insulin
- b) Continue metformin
- c) Sliding-scale insulin alone

Answer: Switch to scheduled (basal-bolus) insulin.

Explanation: Inpatient, oral agents (especially metformin) are usually held and glycemic control is managed with insulin. A scheduled basal-bolus regimen is preferred over sliding-scale insulin alone, which reactively chases hyperglycemia.

Reviewer note: Metformin is held inpatient due to lactic acidosis risk with acute illness, contrast, or renal impairment.

E31. Metformin — eGFR cut-off

A diabetic on metformin with CKD — when is metformin contraindicated / stopped?

- a) Creatinine >1.5
- b) Creatinine >1.5 (men) / >1.4 (women)
- c) eGFR <30
- d) eGFR <15
- e) When starting hemodialysis

Answer: eGFR <30 mL/min/1.73m².

Explanation: Current guidance uses eGFR: metformin is contraindicated at eGFR <30 (lactic acidosis risk) and should not be started below 45.

Reviewer note: Older creatinine-based thresholds (1.5/1.4) are outdated; the eGFR <30 cut-off is the modern standard.

4. NEPHROLOGY

N1. NAGMA — diarrhea vs distal RTA [×2]

A female with fatigue. ABG: HCO_3 16, PCO_2 30s, pH 7.18, Na 136, K 2.5, Cl 110. Urine pH 5.8, urine chloride 40. Most likely cause?

- a) DKA
- b) Sepsis
- c) Vomiting
- d) Diarrhea
- e) Distal (type 1) RTA

Answer: Diarrhea vs distal RTA — distinguished by the urine pH and urine anion gap.

Explanation: This is a normal-anion-gap (hyperchloremic) metabolic acidosis with hypokalemia. The two main causes are GI bicarbonate loss (diarrhea) and renal tubular acidosis. The urine pH discriminates: diarrhea → appropriately **acidic** urine (<5.5); distal (type 1) RTA → inappropriately **alkaline** urine (>5.5, because the kidney cannot acidify).

Reviewer note: With a urine pH of 5.8 the kidney **is** acidifying, which argues FOR diarrhea and somewhat AGAINST distal RTA — yet the 2023 paper's key says "distal RTA." A positive urine anion gap supports RTA, a negative one supports diarrhea. The exam key here is debatable; check the exact urine pH/urine anion gap given.

N2. RBC casts + low C3 → PSGN [×2]

A 16-year-old with increasing weight, bilateral edema, abdominal distention; BP 90/60; Cr 8, U/A +1 protein and +1 blood, **low C3** (or RBC casts, previously healthy, low C3). Most likely diagnosis?

- a) ATN
- b) IgA nephropathy
- c) Post-streptococcal glomerulonephritis (PSGN)
- d) Interstitial nephritis

Answer: Post-streptococcal glomerulonephritis.

Explanation: A nephritic picture (edema, fluid overload, hematuria with RBC casts, mild proteinuria) with a **low C3** in a young patient after a streptococcal infection is classic for PSGN. IgA nephropathy has a **normal** complement.

Reviewer note: Complement is the key discriminator: low C3 → PSGN (also lupus nephritis, MPGN, endocarditis); normal C3 → IgA nephropathy, anti-GBM disease, ANCA vasculitis.

N3. Resistant HTN — spironolactone / except [×2]

A patient on a CCB, beta-blocker, HCTZ and an ARB — most appropriate next step? (Variant: a woman on 4 antihypertensives still hypertensive — do all EXCEPT.)

- a) Ambulatory 24-hour blood pressure reading
- b) Add spironolactone
- c) Do TSH
- d) Stop amlodipine

Answer: Add spironolactone (the preferred 4th-line agent). The EXCEPTION (wrong step) is stopping amlodipine.

Explanation: In resistant hypertension, after confirming adherence and excluding white-coat effect (ambulatory BP) and secondary causes, the most effective add-on is a mineralocorticoid receptor antagonist (spironolactone). Stopping an effective agent (amlodipine) is not part of management.

Reviewer note: Two phrasings, two answers. "Most appropriate next step" on 4-drug failure → add spironolactone. "Do all EXCEPT" → the exception is stopping amlodipine. Ambulatory BP and TSH are both legitimate in a resistant-HTN work-up.

N4. Acute pyelonephritis — culture + IV antibiotics [×2]

Flank pain and fever; WBC 15,000; U/A with 20 WBCs and **white (leukocyte) casts** (or loin pain, fever, renal angle tenderness, WBC casts). Best next step?

- a) Start IV steroids
- b) Send urine culture and start IV antibiotics

Answer: Send urine culture and start IV antibiotics.

Explanation: Fever, costovertebral angle tenderness, pyuria and **white casts** indicate acute pyelonephritis. Management is a urine culture plus empiric IV antibiotics.

N5. Hypertension before 20 weeks → chronic HTN [×2]

A pregnant patient at 12–14 weeks of gestation has hypertension. What type?

- a) Chronic hypertension
- b) Gestational hypertension
- c) Preeclampsia
- d) Masked hypertension

Answer: Chronic hypertension.

Explanation: Hypertension diagnosed before 20 weeks of gestation (or pre-pregnancy) is **chronic** hypertension. Gestational hypertension and preeclampsia are defined by new hypertension **after** 20 weeks.

N6. ACE inhibitor mechanism [×2]

True about ACE inhibitors / mechanism of ACE inhibitors:

- a) Decreases single-nephron (intraglomerular) filtration pressure
- b) Increases glomerular filtration rate
- c) Causes bilateral edema
- d) Causes hypokalemia
- e) Increases cardiac remodeling

Answer: ACE inhibitors decrease single-nephron (intraglomerular) filtration pressure.

Explanation: ACE inhibitors dilate the **efferent** arteriole, lowering intraglomerular pressure — the basis of their renoprotective, anti-proteinuric effect. They cause a small drop in GFR, cause **hyperkalemia** (not hypokalemia), and reduce cardiac remodeling.

N7. Thiazides do NOT cause hypocalcemia [×2]

Thiazide diuretics do NOT cause which of the following?

- a) Hypomagnesemia
- b) Hypocalcemia

- c) Hyperuricemia
- d) Hyperlipidemia
- e) Hypokalemia

Answer: Hypocalcemia (thiazides cause **hyper**calcemia).

Explanation: Thiazides cause hypokalemia, hypomagnesemia, hyperuricemia, hyperglycemia, hyperlipidemia and hyponatremia. They **reduce** urinary calcium excretion → hypercalcemia.

Reviewer note: Mnemonic — "hyperGLUC": hyperGlycemia, hyperLipidemia, hyperUricemia, hyperCalcemia (plus hypokalemia, hypomagnesemia, hyponatremia).

N8. Myeloma kidney — mechanism that does NOT apply [×2]

A patient with multiple myeloma — which is NOT a mechanism of nephropathy?

- a) Hyperuricemia due to tumor lysis syndrome
- b) Hypercalcemia causing tubular damage
- c) Recurrent infections from decreased immunity
- d) Bence-Jones (light chain) protein causing tubular damage
- e) Renal infarcts / direct calcium-crystal stones

Answer: Renal infarcts (and direct calcium-crystal kidney stones are not the recognized mechanism).

Explanation: Myeloma damages the kidney via light-chain (cast) nephropathy, hypercalcemia (tubular injury, nephrocalcinosis), hyperuricemia/tumor lysis, AL amyloidosis, and recurrent infection. Renal arterial **infarction** is not a mechanism of myeloma kidney.

Reviewer note: Two papers phrase this differently. The 2023 paper makes "recurrent infections" look like a distractor, but recurrent infection IS a contributor; the clearest non-mechanism is renal infarction (IM-2023 option).

N9. Stable CKD — what to add [×2]

A stable CKD patient with phosphate 3.5, LDL 150, Hb 9 (or CKD-4, phosphorus 3.5, bicarb 22, Hb 9, asymptomatic). What to add?

- a) EPO (erythropoietin) analogues
- b) Phosphate binder
- c) Statin / lipid-lowering agent

Answer: Statin / lipid-lowering agent (per the exam keys).

Explanation: Phosphate of 3.5 is normal — a phosphate binder is not needed. The exam keys treat the elevated LDL with a statin (cardiovascular disease is the leading cause of death in CKD).

Reviewer note: Exam keys favor "statin." However, an Hb of 9 in CKD also merits anemia evaluation (iron studies, then EPO once iron-replete). If forced to one answer, follow the key (statin), but recognize anemia work-up is clinically appropriate too.

N10. Mixed metabolic acidosis + alkalosis from vomiting [×2]

A 67-year-old with massive vomiting 3 days; pH 7.24, PCO_2 24; Na 140, K 3.2, Cl 79, HCO_3 10 (or confusion + persistent vomiting; Na 140, Cl 60, HCO_3 11, K 3.4, pH 7.28). Acid-base disorder?

- a) High anion gap metabolic acidosis
- b) Metabolic alkalosis
- c) High anion gap metabolic acidosis WITH metabolic alkalosis

- d) HAGMA with respiratory compensation
- e) Non-anion gap metabolic acidosis

Answer: High anion gap metabolic acidosis WITH a concurrent metabolic alkalosis.

Explanation: Compute the anion gap (Na − Cl − HCO_3): e.g., 140 − 60 − 11 = 69 — markedly **high**. Then check the delta ratio / corrected bicarbonate: prolonged vomiting also produces a metabolic **alkalosis**, so the bicarbonate is "less low than expected" for the gap size → mixed high-anion-gap acidosis plus metabolic alkalosis.

Reviewer note: Vomiting alone causes alkalosis; severe vomiting with hypovolemia/starvation also generates a high-gap acidosis (ketosis, lactic acidosis). Always compute the gap and delta ratio.

N11. CKD-3 with volume overload — NOT hemodialysis

A CKD-3 patient, doing well; signs of pleural effusion and +2 edema; Hb 9, HCO_3 12. Which is NOT part of current management?

- a) Hemodialysis
- b) IV furosemide
- c) Salt restriction
- d) Sodium bicarbonate

Answer: Hemodialysis.

Explanation: A stable CKD-3 patient with volume overload and acidosis is managed with diuretics (IV furosemide), salt restriction and oral sodium bicarbonate. Hemodialysis is not indicated in stable CKD-3 — it is reserved for advanced CKD/ESRD with refractory complications.

N12. Symptomatic uremia — start dialysis

A stage 5 CKD patient (GFR 7) with colon cancer; nausea, vomiting, metallic taste; normal K, no acidosis. Next step?

- a) Start hemodialysis
- b) Refer to urgent hemodialysis
- c) Refer for preemptive kidney transplant
- d) Observe with monthly visits

Answer: Start dialysis — uremic symptoms are an indication to initiate dialysis.

Explanation: Uremic symptoms (nausea, vomiting, metallic taste, encephalopathy/anorexia) are a clinical indication to initiate dialysis regardless of a specific GFR number.

Reviewer note: Exam key marked "A or B." Whether "urgent" vs a planned outpatient start depends on severity, but the principle is clear: symptomatic uremia → start dialysis. A preemptive transplant is precluded by active malignancy.

N13. CKD stage 5 — least likely reason to start dialysis

A 70-year-old with stage 5 CKD. LEAST likely reason to start dialysis?

- a) Encephalopathy
- b) eGFR less than 15
- c) Hyperkalemia
- d) Itching
- e) Pericarditis

Answer: eGFR less than 15.

Explanation: Dialysis is started for **clinical** indications: uremic encephalopathy, uremic pericarditis, refractory hyperkalemia/acidosis, refractory fluid overload, intractable uremic symptoms (incl. severe pruritus). An eGFR <15 defines CKD stage 5 but, alone in an asymptomatic patient, is not an indication to initiate dialysis.

N14. CKD-4 lost to follow-up — least appropriate step

A CKD-4 patient lost to follow-up for 2 years; creatinine rose; bicarbonate 16; severe itching relieved by antihistamines. LEAST appropriate step?

- a) Check PTH and phosphorus
- b) Hemodialysis
- c) Give bicarbonate

Answer: Hemodialysis.

Explanation: This patient has correctable problems: metabolic acidosis (treat with bicarbonate) and CKD-mineral bone disease/pruritus (check PTH and phosphorus). The pruritus is relieved by antihistamines, so it is not severe uremic itching. There is no urgent dialysis indication, so hemodialysis is the least appropriate immediate step.

N15. Rhabdomyolysis — AKI

A 76-year-old diabetic on a statin with diffuse muscle tenderness and muddy-brown casts (high uric acid, high potassium). Most likely cause of AKI?

- a) Rhabdomyolysis
- b) Acute tubular necrosis
- c) Acute interstitial nephritis

Answer: Rhabdomyolysis (causing pigment-induced ATN).

Explanation: Diffuse muscle tenderness in a statin user with AKI, hyperuricemia and hyperkalemia (released from muscle) indicates rhabdomyolysis; myoglobin causes pigment nephropathy / ATN with muddy-brown granular casts and a markedly elevated CK.

N16. Ischemic ATN after shock

A 51-year-old hospitalized for acute MI complicated by severe hypotension on multiple pressors; urine output drops; urea 59, creatinine 2.9; U/A shows tubular epithelial cell casts. Most likely pathologic cause of AKI?

- a) Mesangial immune complex deposition
- b) Acute tubular necrosis
- c) Hyperplastic arteriolosclerosis
- d) Podocyte foot process effacement
- e) Glomerular crescent formation

Answer: Acute tubular necrosis.

Explanation: Prolonged hypotension/shock causes ischemic acute tubular necrosis; the hallmark urinary finding is renal tubular epithelial cell casts and "muddy-brown" granular casts.

N17. Thiazide abuse — hypocalciuria

A psychotic patient with hyponatremia, hypokalemia, normal urinary potassium and **hypocalciuria**. Most likely cause?

- a) Thiazide abuse
- b) Bartter syndrome
- c) Vomiting

Answer: Thiazide abuse (surreptitious diuretic use).

Explanation: Hyponatremia + hypokalemia with **hypocalciuria** is characteristic of thiazide effect (thiazides reduce urinary calcium). Bartter syndrome causes **hypercalciuria**. Surreptitious diuretic use is common in psychiatric patients.

Reviewer note: Calcium handling is the discriminator: low urine calcium → thiazide; high urine calcium → Bartter (loop-type).

N18. Diabetes insipidus

A schizophrenic patient with hypernatremia, low urine sodium and **low urine osmolality**. Diagnosis?

- a) Diabetes insipidus
- b) SIADH
- c) Primary polydipsia

Answer: Diabetes insipidus.

Explanation: Hypernatremia with an inappropriately **dilute** urine (low urine osmolality) indicates inability to concentrate urine — diabetes insipidus. SIADH causes hyponatremia with concentrated urine.

Reviewer note: Psychiatric patients on lithium are prone to nephrogenic diabetes insipidus — a clue if lithium is mentioned.

N19. NSAID-induced nephrotic syndrome

A young lady took ibuprofen for shoulder pain for 1 month; nephrotic-range proteinuria (~10 g/day). Most likely diagnosis?

- a) Focal segmental glomerulosclerosis (FSGS)
- b) Minimal change disease
- c) Membranous nephropathy

Answer: Minimal change disease (NSAID-associated) — though FSGS is also possible.

Explanation: NSAIDs can cause nephrotic syndrome, classically minimal change disease (often with acute interstitial nephritis); they are also associated with membranous nephropathy.

Reviewer note: Exam key marked "FSGS mostly." Standard teaching links NSAID-induced nephrotic syndrome most strongly with minimal change disease (± interstitial nephritis). Consider the exact histology described; the key's "FSGS" is questionable.

N20. Membranous nephropathy with solid tumor

A patient with a history of lung cancer and findings of nephrotic syndrome. Diagnosis?

- a) Membranous nephropathy
- b) Minimal change disease
- c) FSGS

Answer: Membranous nephropathy.

Explanation: Membranous nephropathy is the classic **solid-tumor**-associated nephrotic syndrome (lung, GI cancers). Minimal change disease is more associated with Hodgkin lymphoma.

N21. Light-chain nephropathy — normal albumin clue

A 67-year-old with fatigue and frothy urine; Hb 9.0, calcium 10.8, creatinine 2.3; **large proteinuria with normal serum albumin**, no significant hematuria. Most likely diagnosis?

- a) Membranous nephropathy
- b) Lupus nephritis
- c) Light chain (cast) nephropathy
- d) Minimal change disease
- e) Chronic pyelonephritis

Answer: Light chain nephropathy (multiple myeloma).

Explanation: Heavy proteinuria with a **normal** serum albumin argues against true nephrotic syndrome and toward overflow proteinuria of light chains. With anemia, hypercalcemia and renal failure in an older patient → multiple myeloma / light-chain nephropathy.

Reviewer note: A normal albumin despite "large proteinuria" is the trick — dipstick mainly detects albumin and may be negative; the protein is monoclonal light chains.

N22. Myeloma — bone pain + light-chain proteinuria

A patient with bone pain; U/A +1 protein but urine albumin/creatinine ratio shows ~4.5 g protein. Diagnosis?

- a) Multiple myeloma
- b) Diabetic nephropathy
- c) FSGS

Answer: Multiple myeloma.

Explanation: A large discrepancy — minimal dipstick protein but very high total urine protein — indicates non-albumin proteinuria (monoclonal light chains), since the dipstick detects mainly albumin. With bone pain → multiple myeloma.

N23. Dipstick misses light chains

A lymphoma patient excreting 1.5 g of urinary protein/day has a NEGATIVE dipstick for protein. The reason?

- a) Only heavy-chain sequences are recognized by the strip
- b) The urine is not concentrated enough
- c) Tamm-Horsfall protein blocks the reaction
- d) The protein is too small to be detected
- e) Dipsticks preferentially detect albumin compared with immunoglobulin

Answer: Dipsticks preferentially detect albumin and poorly detect immunoglobulin/light chains.

Explanation: Urine dipsticks are designed to detect **albumin** and are relatively insensitive to immunoglobulin light chains (Bence-Jones protein). A patient excreting monoclonal light chains can have a falsely negative dipstick despite heavy proteinuria — quantified by a 24-hour collection or protein electrophoresis.

N24. Metabolic acidosis — apply Winter's formula

ABG: pH 7.3, PCO_2 38, HCO_3 13. Interpretation?

- a) Metabolic acidosis with a relative respiratory acidosis (inadequate compensation)
- b) Pure metabolic acidosis with appropriate compensation
- c) Metabolic acidosis with respiratory alkalosis

Answer: Metabolic acidosis with an inadequate respiratory response (relative respiratory acidosis).

Explanation: Low HCO_3 + low pH = metabolic acidosis. Winter's formula: expected $PCO_2 = 1.5 \times HCO_3 + 8 \pm 2 = 1.5 \times 13 + 8 \approx 27.5$. The measured PCO_2 of 38 is much **higher** than expected — inadequate compensation → a superimposed respiratory acidosis.

Reviewer note: Always apply Winter's formula: actual PCO_2 above the expected value = concurrent respiratory acidosis; below = concurrent respiratory alkalosis.

N25. AG metabolic acidosis with respiratory alkalosis

A 67-year-old with 1 day of dyspnea; HR 110, BP 98/54, RR 26; Na 140, K 5.8, Cl 104, creatinine 6.1 (was 1.2). ABG pH 7.26, PCO_2 14, HCO_3 10. Best characterization?

- a) Anion gap metabolic acidosis with respiratory compensation
- b) Anion gap metabolic acidosis with a metabolic alkalosis
- c) Anion gap metabolic acidosis with respiratory alkalosis
- d) Non-anion gap and anion gap metabolic acidosis
- e) Non-anion gap metabolic acidosis

Answer: Anion gap metabolic acidosis with respiratory ALKALOSIS (a mixed disorder).

Explanation: Anion gap = 140 − 104 − 10 = 26 (high → AG metabolic acidosis from uremia/AKI). Expected PCO_2 by Winter's = 1.5×10 + 8 = 23 ± 2. Measured PCO_2 of 14 is **lower** than expected — over-breathing beyond compensation → superimposed respiratory alkalosis.

Reviewer note: The trap is calling this "metabolic acidosis with respiratory compensation." Apply Winter's: PCO_2 below the expected range = concurrent respiratory alkalosis.

N26. Type IV RTA in a diabetic

A diabetic with CAD on amlodipine and metoprolol; acidosis and hyperkalemia; urine pH 5.7. Diagnosis?

- a) Type IV (hyperkalemic) RTA
- b) Distal (type 1) RTA
- c) Proximal (type 2) RTA

Answer: Type IV renal tubular acidosis.

Explanation: Type IV RTA (hyporeninemic hypoaldosteronism, common in diabetic nephropathy) is the only RTA that causes **hyper**kalemia, with a normal-anion-gap metabolic acidosis and an acidic urine pH (the kidney can still acidify). Types 1 and 2 RTA cause **hypo**kalemia.

N27. CKD anemia

True about CKD:

- a) 90% of patients with late-stage CKD have anemia
- b) Anemia causes significant morbidity and mortality in CKD
- c) CKD rarely causes iron deficiency
- d) Iron deficiency criteria in CKD are the same as in the general population

- e) Erythropoietin secretion is not controlled by hypoxia-inducible factor

Answer: Anemia causes significant morbidity and mortality in CKD patients.

Explanation: Anemia is common and worsens outcomes in CKD — multifactorial (erythropoietin deficiency, iron deficiency with criteria that **differ** from the general population, shortened RBC survival). EPO production **is** regulated by hypoxia-inducible factor.

Reviewer note: The 2020 exam marked "A" (with a question mark). Statement B is unambiguously correct; the prevalence figure in A is an overstatement and the key's choice is debatable.

N28. CKD hypocalcemia — vitamin D

A CKD patient with a hip fracture — likely cause of hypocalcemia?

- a) Decreased renal 1-alpha-hydroxylation of vitamin D
- b) Decreased calcium intake
- c) Hypoparathyroidism

Answer: Decreased renal 1-alpha-hydroxylation of vitamin D.

Explanation: The diseased kidney cannot convert 25-OH vitamin D to active 1,25-$(OH)_2$ vitamin D (calcitriol), reducing intestinal calcium absorption → hypocalcemia — central to CKD-mineral and bone disorder, which drives secondary hyperparathyroidism and renal osteodystrophy (fracture risk).

N29. ADPKD — leading cause of death

Most common cause of mortality in ADPKD?

- a) Infections
- b) Ruptured cerebral aneurysm
- c) Transformation to malignancy
- d) Renal failure
- e) Cardiovascular complications

Answer: Cardiovascular complications.

Explanation: Although ADPKD is famous for berry aneurysms, the leading cause of death is **cardiovascular** disease (hypertension is nearly universal and accelerates CV events), as in CKD generally.

Reviewer note: Exam key marked "E." Ruptured cerebral aneurysm is a feared but less common cause of death.

N30. Calcineurin inhibitors — not a side effect

Regarding calcineurin inhibitors — which is NOT a kidney-related side effect?

- a) Hyperkalemia
- b) Hyperphosphatemia
- c) Hypertension
- d) Hypomagnesemia

Answer: Hyperphosphatemia.

Explanation: Calcineurin inhibitors (cyclosporine, tacrolimus) cause nephrotoxicity, hypertension, hyperkalemia, hypomagnesemia and **hypo**phosphatemia (renal phosphate wasting). Hyperphosphatemia is not a typical effect.

N31. Diabetic nephropathy — true statement

A 40-year-old with diabetic nephropathy — which is true?

- a) Absence of retinopathy excludes it
- b) Most patients develop nephropathy 10–20 years after diagnosis

Answer: Most patients develop nephropathy 10–20 years after the diagnosis of diabetes.

Explanation: Diabetic nephropathy typically appears 10–20 years after diabetes onset. Retinopathy usually accompanies type 1 diabetic nephropathy, but its absence does not exclude nephropathy (especially in type 2 DM).

N32. Kimmelstiel-Wilson nodules

A 48-year-old with malaise, peripheral neuropathy; creatinine 3.5, glucose 190, HbA1c 9.9%; U/A 1+ glucose, 3+ protein. Most likely renal pathology?

- a) Acute pyelonephritis
- b) Acute tubular necrosis
- c) Membranous nephropathy
- d) Chronic glomerulonephritis
- e) Kimmelstiel-Wilson nodules

Answer: Kimmelstiel-Wilson nodules (nodular glomerulosclerosis of diabetic nephropathy).

Explanation: Long-standing poorly controlled diabetes (A1c 9.9%) with peripheral neuropathy and heavy proteinuria → diabetic nephropathy; its pathognomonic lesion is nodular glomerulosclerosis (Kimmelstiel-Wilson nodules).

N33. Hypokalemic metabolic alkalosis — IV fluids + K

A patient with hypokalemia and metabolic alkalosis — management?

- a) IV fluids (normal saline) plus IV potassium
- b) Potassium alone
- c) Bicarbonate

Answer: IV fluids (normal saline) plus IV potassium.

Explanation: Chloride-responsive metabolic alkalosis (e.g., from vomiting) with hypokalemia is corrected by restoring volume and chloride with isotonic saline and repleting potassium.

N34. Asymptomatic bacteriuria — do not treat

An elderly diabetic woman with a positive urine culture (E. coli, sensitive to ciprofloxacin) but NO urinary symptoms, fever or systemic signs. Best next step?

- a) Start antibiotics based on sensitivity
- b) Repeat the urine culture
- c) Observe

Answer: Observe — do not treat asymptomatic bacteriuria.

Explanation: Asymptomatic bacteriuria should NOT be treated in most adults, including diabetics and the elderly — treatment does not improve outcomes and promotes resistance. Exceptions where treatment IS indicated: pregnancy and before urologic procedures with expected mucosal trauma.

5. GASTROENTEROLOGY (GI)

G1. SAAG interpretation of ascites [×3]

New-onset ascites; serum albumin 3.5, ascitic albumin 2.7, total protein 3.5 (or SAAG case ~3.7 minus ~2.5 with ascitic protein 4). Most likely diagnosis?

- a) Cirrhosis
- b) Heart failure
- c) Budd-Chiari syndrome
- d) Ovarian carcinomatosis / malignancy

Answer: Use the SAAG (serum albumin − ascitic albumin). **Low SAAG (<1.1)** → peritoneal disease (malignancy/carcinomatosis, TB). **High SAAG (≥1.1)** → portal hypertension; then high SAAG + **high** protein (>2.5) → cardiac/Budd-Chiari, high SAAG + **low** protein → cirrhosis.

Explanation: SAAG <1.1 g/dL indicates absence of portal hypertension (malignancy, peritoneal carcinomatosis, TB peritonitis). SAAG ≥1.1 indicates portal hypertension; the ascitic total protein subdivides it — high protein suggests cardiac/Budd-Chiari (post-hepatic), low protein suggests cirrhosis.

Reviewer note: Worked example — serum 3.5 − ascites 2.7 = SAAG 0.8 (**low**) → not portal hypertension → malignancy/ovarian carcinomatosis. The IM-2023 "SAAG (3.7−2.5)=1.2 with protein 4" gives a **high** SAAG with **high** protein → that points to cardiac/Budd-Chiari, yet the IM-2023 key says "ovarian cancer" — recheck the numbers; the key conflicts with a high SAAG. Always compute SAAG first, then use the ascitic protein.

G2. Achalasia — atypical feature / false statement [×3]

Which does NOT favor a diagnosis of achalasia / wrong about achalasia?

- a) Dysphagia to solids AND liquids
- b) Dilation of the esophagus
- c) Aperistalsis of the esophagus
- d) Significant weight loss
- e) Failure of LES relaxation

Answer: Significant weight loss (the false statement is anything denying the typical features).

Explanation: Achalasia: dysphagia to **both** solids and liquids from the start, manometry showing aperistalsis and failure of LES relaxation, and a dilated ("bird-beak") esophagus. Significant/rapid weight loss is NOT typical — marked weight loss should raise concern for malignancy (pseudoachalasia).

Reviewer note: The 2017 paper phrases it as "FALSE: it usually presents with dysphagia to **solids** with significant weight loss" — false because achalasia causes solid-AND-liquid dysphagia and is not typically associated with major weight loss.

G3. IBD extraintestinal manifestations — activity correlation [×3]

In IBD, which extraintestinal manifestation does NOT parallel disease activity? (And: which is associated with increasing severity?)

- a) Uveitis
- b) Episcleritis
- c) Peripheral / pauci-immune arthritis
- d) Aphthous stomatitis
- e) Erythema nodosum
- f) Primary sclerosing cholangitis

- g) Pyoderma gangrenosum
- h) Sacroiliitis / ankylosing spondylitis

Answer: Those that do NOT parallel activity: ankylosing spondylitis/sacroiliitis, primary sclerosing cholangitis, and uveitis. Those that DO parallel activity: peripheral arthritis, episcleritis, erythema nodosum, aphthous stomatitis.

Explanation: IBD extraintestinal manifestations split into activity-tracking (peripheral arthritis, episcleritis, erythema nodosum, aphthous ulcers) and independent-course (axial arthritis/ankylosing spondylitis, sacroiliitis, PSC, uveitis).

Reviewer note: Exam keys differ. The 2020 paper's "associated with increasing severity" answer was "erythema nodosum" (an activity-paralleling lesion). The 2023/IM-2023 "does NOT parallel activity" answer is uveitis (or PSC/ankylosing spondylitis). Sort by the two lists above.

G4. Hepatitis B serology — past infection [×2]

A patient with a previous (resolved) HBV infection — which serology? / Evidence of previous HBV infection.

- a) HBsAg
- b) Anti-HBc IgG (anti-core IgG)
- c) Anti-HBc IgM (anti-core IgM)
- d) Anti-HBs
- e) Anti-HBe

Answer: Anti-HBc IgG (with anti-HBs), HBsAg negative.

Explanation: Recovery from HBV: HBsAg negative, anti-HBs positive (immunity), anti-HBc IgG positive (past natural infection). Anti-HBc IgM indicates **acute/recent** infection. Vaccination gives anti-HBs WITHOUT anti-HBc — so anti-HBc IgG distinguishes past infection from vaccination.

Reviewer note: Anti-HBc IgG is the key marker of prior natural infection (present in both resolved infection and chronic carriers); isolated anti-HBs without anti-HBc = vaccine immunity.

G5. Eosinophilic esophagitis [×2]

A patient with asthma presents with dysphagia; endoscopy shows a mid-esophageal food impaction with rings and furrows (or asthma + eczema, trouble swallowing solids; edematous esophagus with rings and furrows). Most likely diagnosis / management?

- a) Eosinophilic esophagitis
- b) GERD
- c) Achalasia
- d) Esophageal spasm
- e) Give oral / swallowed topical steroids

Answer: Eosinophilic esophagitis — managed with PPIs, swallowed topical (or short-course oral) corticosteroids and dietary elimination.

Explanation: An atopic patient with solid-food dysphagia / food impaction and endoscopic rings ("feline esophagus") and linear furrows has eosinophilic esophagitis (esophageal eosinophilia on biopsy).

Reviewer note: The 2018 case mentions verapamil for HTN — a CCB can worsen GERD/LES tone but is not the cause; the correct management addresses the eosinophilic esophagitis (steroids), not the antihypertensive.

G6. Crohn's disease — false statement [×2]

Crohn's disease — which statement is FALSE? / Wrong about Crohn's.

- a) Smoking is associated with disease activity / reduces treatment efficacy
- b) It can involve the esophageal mucosa
- c) Patients younger than 35 have a better prognosis
- d) It most commonly involves the distal ileum

Answer: "Patients younger than 35 have a better prognosis" is FALSE — young age at onset is a **poor** prognostic factor.

Explanation: In Crohn's, a **younger** age at diagnosis is associated with a more aggressive course and **worse** prognosis. Smoking worsens Crohn's and reduces treatment efficacy. Crohn's can affect any part of the GI tract, including the esophagus, but most commonly involves the terminal/distal ileum.

Reviewer note: The IM-2023 paper explicitly lists "age of onset above 35 is a poor prognostic factor" — that is FALSE; it is YOUNG onset that is poor.

G7. Pancreatitis — correct statement [×2]

Pancreatitis — which is correct?

- a) An amylase level of 1000 correlates with severe disease
- b) Lipase is more specific than amylase
- c) IV antibiotics should be started immediately

Answer: Lipase is more specific than amylase.

Explanation: Serum lipase is more specific (and stays elevated longer) than amylase. The **degree** of enzyme elevation does NOT correlate with severity. Prophylactic antibiotics are NOT routine — reserved for documented infected necrosis.

G8. IBD — induction vs maintenance [×2]

A patient with ulcerative colitis (left-sided colitis) / not used in INDUCTION therapy. Which is NOT used to induce remission?

- a) Mesalamine
- b) Prednisone
- c) Azathioprine
- d) Budesonide
- e) Infliximab

Answer: Azathioprine.

Explanation: Azathioprine (a thiopurine) has a slow onset (weeks–months) and is used for **maintenance**, not induction. Induction agents include 5-ASA (mesalamine), corticosteroids (prednisone, budesonide) and biologics (infliximab).

G9. Variceal upper GI bleeding — least appropriate step [×2]

A cirrhotic (HBV) with melena, Hb 8–9; vitals stable. Which is the LEAST appropriate / "do all EXCEPT" next step?

- a) IV octreotide
- b) IV PPI
- c) IV ceftriaxone

- d) PRBC transfusion
- e) Endoscopy within 12 hours

Answer: The least appropriate is liberal/unnecessary PRBC transfusion when Hb is already ~8 (a restrictive strategy targets Hb ~7–8).

Explanation: Standard variceal-bleed management: IV octreotide (splanchnic vasoconstriction), IV PPI (empiric until source confirmed), IV ceftriaxone (prophylaxis reduces mortality/rebleeding), restrictive PRBC transfusion (target Hb ~7–8), endoscopy within 12 hours. Over-transfusion can raise portal pressure and worsen rebleeding.

Reviewer note: Exam keys are inconsistent. The other four are all guideline-recommended; the defensible "least appropriate" answer is unnecessary/liberal PRBC transfusion. Verify the exact stem wording.

G10. Celiac serology — anti-tTG IgA [×2]

A young female with iron deficiency anemia and diarrhea; normal IgG and IgA (or a type 1 DM patient with diarrhea/malabsorption). Best initial test?

- a) Anti-tissue transglutaminase IgA
- b) Anti-tissue transglutaminase IgG
- c) Anti-gliadin IgA
- d) Anti-gliadin IgG

Answer: Anti-tissue transglutaminase IgA.

Explanation: Anti-tTG IgA is the first-line screening test for celiac disease and is reliable when total IgA is **normal**. (If IgA-deficient, use an IgG-based test — anti-tTG IgG or deamidated gliadin peptide IgG.)

Reviewer note: Confirm total IgA is normal before relying on the IgA-based test. Type 1 diabetes is strongly associated with celiac disease.

G11. Cirrhotic ascites — true statement

A 44-year-old with HCV cirrhosis develops new ascites. Which is true regarding ascites?

- a) Fluid restriction is a standard part of management
- b) Diagnostic paracentesis is not indicated
- c) Increased angiotensin II secretion is part of the pathophysiology
- d) Cirrhotic ascites forms from increased hepatic sinusoid fenestrations
- e) Ascites occurs at a rate of 50% per year in cirrhotics

Answer: Increased angiotensin II secretion is part of the pathophysiology.

Explanation: Splanchnic vasodilation reduces effective arterial blood volume, activating RAAS (increased angiotensin II and aldosterone) → sodium and water retention. Management is **sodium** restriction + diuretics (fluid restriction only for significant hyponatremia); diagnostic paracentesis IS indicated for new ascites.

G12. SAAG with cardiac risk factors — cirrhosis

A 64-year-old with CAD, hyperlipidemia and diabetes; serum protein 7, serum albumin 3.4; ascitic protein 2.2, ascitic albumin 1.6. Cause of ascites?

- a) Cirrhosis
- b) Peritoneal carcinomatosis
- c) Nephrotic syndrome
- d) Congestive heart failure

Answer: Cirrhosis (SAAG = 3.4 − 1.6 = 1.8, high SAAG, with a **low** ascitic protein of 2.2).

Explanation: SAAG ≥1.1 → portal hypertension. The ascitic total protein is low (<2.5), favoring cirrhosis over a cardiac cause.

Reviewer note: The cardiac risk factors are a distractor — cardiac ascites would have a high SAAG but a **high** ascitic protein (>2.5). Here the protein is low → cirrhosis.

G13. Non-variceal upper GI bleed — endoscopy within 24 h

A patient with hematemesis from peptic ulcer disease, stabilized with IV fluids and PPIs. Next best step?

- a) Nasogastric tube
- b) Observe
- c) Endoscopy within 24 hours
- d) IV octreotide
- e) IV ceftriaxone

Answer: Endoscopy within 24 hours.

Explanation: After hemodynamic stabilization, upper GI endoscopy within 24 hours is indicated for non-variceal upper GI bleeding (PUD) — diagnostic and therapeutic. Octreotide and ceftriaxone are specific to **variceal** bleeding.

G14. Zenker's diverticulum

A 71-year-old with 2 years of intermittent swallowing problems, halitosis and nighttime coughing; good appetite, stable weight. Most likely etiology?

- a) Achalasia
- b) Zenker's diverticulum
- c) Esophageal candidiasis
- d) Esophageal peptic stricture
- e) Esophageal adenocarcinoma

Answer: Zenker's diverticulum.

Explanation: Oropharyngeal dysphagia with **halitosis** (food retention), regurgitation of undigested food and nocturnal cough/aspiration, with preserved appetite and stable weight, is classic for a Zenker's diverticulum.

G15. Fulminant Wilson disease vs autoimmune hepatitis [×2]

A hypothyroid female on levothyroxine; ALT 1500s, AST 1000s, ALP ~200, GGT ~100, bilirubin 2 (or a patient with asterixis/choreiform movements; ALT 1500, AST 2000, ALP 25 low, Hb 10). Most likely diagnosis?

- a) Wilson disease
- b) Hemochromatosis
- c) NAFLD
- d) Alcoholic liver disease
- e) Autoimmune hepatitis

Answer: For very high transaminases + LOW alkaline phosphatase + hemolytic anemia + neuropsychiatric signs → fulminant Wilson disease. For the hypothyroid case with ALT 1500s → autoimmune hepatitis (autoimmune association).

Explanation: Fulminant Wilson disease shows markedly elevated transaminases with a characteristically **low** alkaline phosphatase, Coombs-negative hemolytic anemia, and neuropsychiatric features. Autoimmune hepatitis presents with high transaminases and clusters with other autoimmune diseases (e.g., autoimmune thyroid disease).

Reviewer note: Two distinct cases. Pattern-recognition: very high ALT/AST + LOW ALP + hemolysis + neuro signs → Wilson. High ALT/AST in a patient with another autoimmune disease (hypothyroidism) → autoimmune hepatitis. Alcoholic hepatitis usually has AST:ALT >2:1 with both typically <300–400 — NOT in the thousands.

G16. PSC — false statement [×2]

Which does NOT favor a diagnosis of primary sclerosing cholangitis? / Wrong about PSC.

- a) Onion-skin fibrosis on histology
- b) P-ANCA antibodies
- c) AST and ALT in the thousands
- d) Patient has ulcerative colitis
- e) Abnormal cholangiogram (beaded appearance)

Answer: AST and ALT in the thousands.

Explanation: PSC is a **cholestatic** disease: elevated ALP and GGT, an abnormal cholangiogram with multifocal strictures and "beading," periductal "onion-skin" fibrosis, p-ANCA positivity, strong association with ulcerative colitis. Transaminases in the thousands indicate hepatocellular injury (acute viral/ischemic hepatitis), not PSC.

G17. Primary biliary cholangitis — ursodeoxycholic acid [×2]

An elderly female with fatigue and itching; high ALP and GGT, ALT 50, AST 60, AMA positive, ASMA negative (or PBC with pruritus). Drug of choice / treatment?

- a) Ursodeoxycholic acid
- b) Prednisone
- c) Azathioprine
- d) Obeticholic acid
- e) Antihistamines

Answer: Ursodeoxycholic acid.

Explanation: Cholestatic enzymes with a **positive AMA** are diagnostic of primary biliary cholangitis. Ursodeoxycholic acid is first-line and slows progression. Obeticholic acid is second-line; cholestyramine is used specifically for the pruritus.

Reviewer note: AMA positive → PBC → ursodeoxycholic acid. For PBC-related pruritus specifically, the symptomatic drug is cholestyramine; UDCA is the disease-modifying treatment.

G18. Acute mesenteric ischemia

Sudden abdominal pain 12 hours (HR 110, BP 110/65, RR 22), cool and pale, maroon stool ×3 in 24 h, blood pH 7.15. Diagnosis?

- a) Volvulus
- b) Mesenteric ischemia

Answer: Mesenteric ischemia.

Explanation: Acute severe abdominal pain (classically out of proportion to exam), GI bleeding, hemodynamic compromise and a metabolic (lactic) acidosis indicate acute mesenteric ischemia with bowel infarction.

G19. Ischemic colitis

An elderly hypertensive diabetic with lower abdominal pain and lower GI bleeding; endoscopy shows friable colonic mucosa with ulceration at the splenic flexure. Cause?

- a) IBD
- b) Ischemic colitis
- c) Infectious colitis
- d) Microscopic colitis

Answer: Ischemic colitis.

Explanation: Ischemic colitis affects elderly patients with vascular risk factors and characteristically involves "watershed" areas — the splenic flexure and rectosigmoid junction — with crampy pain and hematochezia.

G20. Obscure GI bleeding — capsule endoscopy

An elderly patient with longstanding melena; Hb 10, positive FOBT; upper endoscopy and colonoscopy both normal twice. Next best step?

- a) Small-intestine (video) capsule endoscopy
- b) Repeat endoscopy and colonoscopy in 2 weeks
- c) Hemicolectomy
- d) CT without contrast

Answer: Small-intestine (video) capsule endoscopy.

Explanation: Obscure GI bleeding (bleeding with normal upper and lower endoscopy) points to a small-bowel source (angiodysplasia, small-bowel tumor). Video capsule endoscopy evaluates the small intestine.

G21. Rectal bleeding in an older adult — colonoscopy

A 55-year-old with rectal bleeding and a long history of hemorrhoids, requesting anti-hemorrhoidal medication. Best advice?

- a) Stool for hemoccult testing
- b) Colonoscopy to check for pathology
- c) Use anti-hemorrhoidals for three weeks
- d) Upper GI endoscopy
- e) Surgical consultation

Answer: Colonoscopy to evaluate for pathology.

Explanation: Rectal bleeding in a patient ≥45–50 years must NOT be attributed to hemorrhoids without excluding colorectal cancer. A colonoscopy is required before treating presumed hemorrhoidal bleeding.

G22. Ascending cholangitis — ERCP

A 65-year-old with gallstones; fever, chills, jaundice, RUQ pain; scleral icterus; elevated WBC and liver enzymes; ultrasound shows a dilated CBD. Next appropriate step?

- a) MRCP to evaluate for obstruction
- b) ERCP after initial resuscitation

- c) Admit and start IV antibiotics
- d) Proceed directly to cholecystectomy
- e) Oral antibiotics and discharge

Answer: Per the exam key: ERCP after initial resuscitation.

Explanation: Charcot's triad (fever, jaundice, RUQ pain) with a dilated CBD = acute (ascending) cholangitis from choledocholithiasis. Management is resuscitation + IV antibiotics AND biliary decompression — ERCP both confirms and relieves the obstruction.

Reviewer note: Both "admit and start IV antibiotics" and "ERCP after resuscitation" are correct components — antibiotics and resuscitation come FIRST, then ERCP. The single "next step" the key chose is ERCP after resuscitation; in practice antibiotics are started concurrently.

G23. Crohn's — skip lesions and cobblestoning

A 27-year-old with bloody diarrhea; colonoscopy shows erythema/ulceration in the ascending, descending and sigmoid colon, sparing the transverse colon and rectum, with a cobblestone appearance. Diagnosis?

- a) Ulcerative colitis
- b) Crohn's disease

Answer: Crohn's disease.

Explanation: **Skip lesions** (sparing of segments such as the transverse colon and rectum) and a **cobblestone** mucosa are characteristic of Crohn's disease. Ulcerative colitis causes continuous inflammation that always involves the rectum.

G24. Crohn's — feature NOT typical

One feature NOT typical of Crohn's disease:

- a) Stricturing and fistula formation
- b) Transmural involvement
- c) Skip areas on colonoscopy
- d) Rectal involvement
- e) Terminal ileum involvement

Answer: Rectal involvement.

Explanation: Crohn's characteristically **spares** the rectum, with skip lesions, transmural inflammation (strictures and fistulas) and terminal-ileal predominance. Continuous rectal involvement is the hallmark of ulcerative colitis.

G25. Mild distal UC — mesalamine first

A 28-year-old with 3 months of bloody diarrhea 2–3×/day, no fever/weight loss/nocturnal symptoms; colonoscopy shows erythema/friability limited to the rectum and sigmoid; biopsy confirms mild ulcerative colitis. Best next step?

- a) Mesalamine (5-ASA)
- b) Infliximab
- c) Azathioprine
- d) Total colectomy
- e) IV methylprednisolone

Answer: Mesalamine (5-ASA).

Explanation: Mild-to-moderate distal (proctosigmoid) ulcerative colitis is treated first with 5-ASA (mesalamine), topical and/or oral. Steroids, biologics and immunomodulators are escalation for moderate-severe or refractory disease.

G26. Crohn's maintenance — not prednisolone

A 23-year-old with Crohn's, doing well after discharge, on prednisolone 40 mg, azathioprine and infliximab. Which should NOT be used for long-term maintenance?

- a) Adalimumab
- b) Methotrexate
- c) Azathioprine
- d) Prednisolone
- e) Infliximab

Answer: Prednisolone.

Explanation: Systemic corticosteroids induce remission but must NOT be used for long-term maintenance (cumulative toxicity — osteoporosis, diabetes, infection, adrenal suppression). Maintenance uses thiopurines, methotrexate, or biologics.

G27. Stool occult blood screening — average risk

In which case do you advise colon cancer screening with a stool occult blood test?

- a) Average-risk individual (≈45–75 years)
- b) High-risk patient with a strong family history
- c) Patient with prior colorectal cancer

Answer: Average-risk adults (≈45–75 years) — a fecal occult blood/FIT test is an acceptable screening modality.

Explanation: For **average**-risk individuals, stool-based testing (annual FIT or guaiac FOBT) is one acceptable colorectal cancer screening option (colonoscopy every 10 years is another). High-risk patients (strong family history, prior CRC/adenomas, IBD, hereditary syndromes) require **colonoscopy**.

Reviewer note: The original stem was truncated in the source; the principle is that stool occult-blood screening is for average-risk patients only.

G28. Child-Pugh score — not a component

Which is NOT part of the Child-Pugh score?

- a) Albumin
- b) Esophageal varices
- c) Ascites
- d) Encephalopathy
- e) Prothrombin time

Answer: Esophageal varices.

Explanation: Child-Pugh uses five parameters: bilirubin, albumin, INR/prothrombin time, ascites and hepatic encephalopathy. Esophageal varices are NOT a component.

G29. Fat malabsorption — not Plummer-Vinson

Which is NOT associated with fat malabsorption?

- a) Chronic pancreatitis
- b) Cystic fibrosis
- c) Plummer-Vinson syndrome
- d) Pancreatic cancer
- e) Zollinger-Ellison syndrome

Answer: Plummer-Vinson syndrome.

Explanation: Fat malabsorption results from pancreatic exocrine insufficiency (chronic pancreatitis, cystic fibrosis, pancreatic cancer) or impaired fat digestion (Zollinger-Ellison — acid inactivates lipase). Plummer-Vinson syndrome (iron deficiency, esophageal webs, dysphagia) does NOT cause fat malabsorption.

G30. Malabsorption — not diverticulosis

Which is NOT associated with malabsorption?

- a) Intestinal telangiectasia
- b) Crohn's disease
- c) Celiac disease
- d) Intestinal diverticulosis
- e) Intestinal lymphangiectasia

Answer: Intestinal diverticulosis.

Explanation: Crohn's, celiac and lymphangiectasia all cause malabsorption (mucosal disease / lymphatic obstruction). Colonic diverticulosis does NOT cause malabsorption.

Reviewer note: Small-bowel diverticulosis can cause bacterial overgrowth and malabsorption, but the typical exam answer treats diverticulosis as the non-malabsorptive option.

G31. GERD — not a contributing factor

Which is NOT a contributing factor to GERD?

- a) Decreased salivation
- b) Increased resting tone of the lower esophageal sphincter
- c) Transient LES relaxations
- d) Impaired esophageal peristalsis
- e) Delayed gastric emptying

Answer: Increased resting tone of the lower esophageal sphincter.

Explanation: GERD is promoted by a **low**/incompetent LES tone, transient LES relaxations, impaired esophageal clearance (decreased peristalsis and salivation) and delayed gastric emptying. Increased LES tone would protect against reflux.

G32. Stool osmotic gap — osmotic diarrhea

A patient with non-bloody diarrhea; stool osmotic gap 150. Most likely diagnosis?

- a) Lactose intolerance
- b) Carcinoma of the colon
- c) Bacterial infection
- d) Celiac disease

Answer: Lactose intolerance (osmotic diarrhea).

Explanation: A **high** stool osmotic gap (>100 mOsm/kg) indicates osmotic diarrhea — unabsorbed osmotically active solute drawing water into the lumen. Secretory diarrhea has a **low** osmotic gap and persists with fasting.

G33. Inflammatory diarrhea

Diarrhea with numerous RBCs and WBCs in the stool. Type of diarrhea?

- a) Secretory
- b) Osmotic
- c) Inflammatory
- d) Fatty

Answer: Inflammatory.

Explanation: Blood and leukocytes in the stool indicate an inflammatory (exudative) diarrhea — mucosal inflammation/ulceration (invasive bacterial infection, IBD).

G34. HBV → HCC without cirrhosis

A patient with an RUQ mass and chronic hepatitis infection for years, no signs of cirrhosis; AFP 13,500. Which virus most likely caused the HCC?

- a) Hepatitis A
- b) Hepatitis B
- c) Hepatitis C
- d) Hepatitis D
- e) Hepatitis E

Answer: Hepatitis B.

Explanation: HBV can cause hepatocellular carcinoma **without** pre-existing cirrhosis (directly oncogenic — viral DNA integration). HCV-related HCC almost always occurs on established cirrhosis. A markedly elevated AFP supports HCC.

Reviewer note: The "no cirrhosis" detail is the key clue pointing to HBV over HCV.

G35. Budd-Chiari syndrome

Abdominal distention and ascites; AST 710, ALT 520, ALP 190, bilirubin 3, Hb 18.5. Diagnosis?

- a) Budd-Chiari syndrome
- b) Cirrhosis
- c) Acute viral hepatitis

Answer: Budd-Chiari syndrome.

Explanation: Hepatic venous outflow obstruction (Budd-Chiari) presents with ascites, abdominal pain, hepatomegaly and elevated transaminases. The **high hemoglobin (18.5)** hints at an underlying myeloproliferative disorder (e.g., polycythemia vera) — a common prothrombotic cause.

G36. Hepatic encephalopathy precipitants

One of the following is NOT associated with precipitation of hepatic encephalopathy:

- a) Hyperkalemia
- b) Spontaneous bacterial peritonitis
- c) Diuretic abuse
- d) GI bleeding
- e) Constipation

Answer: Hyperkalemia.

Explanation: Hepatic encephalopathy is precipitated by GI bleeding, infection (SBP), constipation, and **hypo**kalemia and alkalosis (which increase renal ammonia production and ammonia transfer across the blood-brain barrier). Diuretic abuse precipitates it via hypokalemic alkalosis. Hyperkalemia is not a precipitant.

G37. Recurrent hepatic encephalopathy — add rifaximin

A 64-year-old with HCV cirrhosis, recurrent hepatic encephalopathy despite lactulose (2–3 soft stools/day); alert but with asterixis. In addition to working up reversible causes, next best step?

- a) Salt restriction
- b) IV ceftriaxone
- c) Increase the lactulose dose
- d) Addition of rifaximin
- e) High-protein diet

Answer: Addition of rifaximin.

Explanation: When hepatic encephalopathy recurs despite adequately titrated lactulose (the goal of 2–3 soft stools/day is met), adding the non-absorbable antibiotic rifaximin reduces recurrence. Protein restriction is no longer recommended.

G38. Cirrhotic GI bleed — ceftriaxone prophylaxis

A 50-year-old alcoholic with vomiting fresh red blood; vitals stable; increased abdominal girth and telangiectasias. Preferred agent?

- a) Rifaximin
- b) Metronidazole
- c) Ceftriaxone
- d) Cefazolin

Answer: Ceftriaxone.

Explanation: Cirrhosis with upper GI (variceal) bleeding warrants prophylactic IV antibiotics — ceftriaxone — which reduce infections (including SBP), rebleeding and mortality.

G39. NAFLD/NASH — true statement

Which is true about metabolic (fatty) liver disease?

- a) ALT/AST ratio is low in advanced fibrosis
- b) Elevated ferritin in ~50% of NASH
- c) ALT and AST are 10× the upper limit of normal
- d) ALP is 5× the upper limit of normal

Answer: Per the exam key: elevated ferritin in ~50% of NASH (and the AST/ALT ratio RISES with advancing fibrosis).

Explanation: In NAFLD/NASH, transaminases are usually only mildly elevated (typically <2–3× ULN, not 10×); ALT is initially higher than AST, but as fibrosis advances the AST/ALT ratio rises (≥1). Hyperferritinemia is common in NASH (~half of patients).

Reviewer note: The option "ALT/AST ratio is low in advanced fibrosis" is FALSE — the AST/ALT ratio increases (AST becomes higher than ALT) as fibrosis progresses.

G40. NAFLD — most common cause of death

Most common cause of death in NAFLD?

- a) Cholangiocarcinoma
- b) Cardiovascular events
- c) Renal failure
- d) Liver cirrhosis
- e) Hepatocellular carcinoma

Answer: Cardiovascular events.

Explanation: NAFLD is closely tied to the metabolic syndrome; the leading cause of death is **cardiovascular** disease, ahead of liver-related death.

6. HEMATOLOGY / ONCOLOGY

H1. IPSS / WHO MDS classification — not a variable [×3]

Not part of the IPSS / WHO classification (of myelodysplastic syndrome):

- a) Spleen size / transfusion requirement
- b) WHO category
- c) Karyotype / cytogenetics
- d) Blast percentage
- e) Cytopenias (anemia, thrombocytopenia)
- f) Creatinine

Answer: Spleen size (and creatinine) — not part of the IPSS.

Explanation: The IPSS / IPSS-R for myelodysplastic syndrome uses bone marrow **blast percentage**, **cytogenetics/karyotype**, and the number/degree of **cytopenias** (anemia, thrombocytopenia, neutropenia). Spleen size, creatinine and transfusion requirement (the latter is in some revised models but not the original IPSS) are NOT classic IPSS variables.

Reviewer note: Different papers list different distractors. The 2020 paper's answer was "creatinine"; the 2023/IM-2023 papers' answer was "spleen size." Both are correct as non-IPSS variables. Core IPSS variables: blasts, cytogenetics, cytopenias.

H2. Autoimmune hemolytic anemia [×2]

A patient with a history of ITP presenting with elevated LDH, reticulocytes 8%, low Hb, positive DAT (direct antiglobulin test) — or spherocytes with LDH 1200, retic 8%, positive DAT. Most likely diagnosis?

- a) Autoimmune lymphoproliferative disorder
- b) Autoimmune hemolytic anemia

Answer: Autoimmune hemolytic anemia.

Explanation: Hemolysis (high LDH, reticulocytosis, low Hb) with a **positive direct antiglobulin (Coombs) test** indicates autoimmune hemolytic anemia. A patient with both ITP and autoimmune hemolytic anemia has Evans syndrome.

Reviewer note: A positive DAT plus spherocytes points to **warm** autoimmune hemolytic anemia.

H3. Hemophilia A vs B — mixing study [×3]

A patient with hemarthrosis; PT 13/14 (normal), PTT 80/15 (prolonged); on mixing study PTT corrects to 40/15; Factor VIII assay 90%, Factor IX assay <1%. Most likely diagnosis?

- a) Severe Hemophilia A
- b) Severe Hemophilia B
- c) Von Willebrand disease type 1
- d) Von Willebrand disease type 2
- e) Glanzmann thrombasthenia

Answer: Severe Hemophilia B.

Explanation: An isolated prolonged PTT that **corrects** on a mixing study indicates a **factor deficiency** (not an inhibitor). Factor IX <1% with a normal Factor VIII = Hemophilia B (Christmas disease). Hemophilia A would have a low Factor VIII.

Reviewer note: Mixing study logic: corrects → factor deficiency; does NOT correct → an inhibitor (e.g., acquired hemophilia, antiphospholipid antibody). See H10 for the contrasting "does not correct" case.

H4. TTP — least likely finding [×2]

A patient with CNS symptoms and microangiopathic hemolytic anemia (TTP). LEAST likely to be present? / Not found in a TTP case.

- a) Low platelets
- b) Low ADAMTS13 activity
- c) Prolonged INR / elevated INR

Answer: Prolonged (elevated) INR.

Explanation: TTP is a thrombotic microangiopathy with thrombocytopenia, microangiopathic hemolytic anemia, neurologic signs, and **low ADAMTS13** activity — but the coagulation studies (PT/INR, PTT) are characteristically **normal** (unlike DIC, where they are deranged).

Reviewer note: Normal PT/INR/PTT distinguishes TTP from DIC — a high-yield discriminator.

H5. Hairy cell leukemia — CD markers [×2]

A patient with massive splenomegaly (8–10 cm below the costal margin) and flow cytometry showing CD19, CD20, CD11c/CD25/CD103 (B-cell markers) with very few blasts in the marrow. Most likely diagnosis?

- a) Mantle cell lymphoma
- b) CML
- c) Hairy cell leukemia
- d) Peripheral T-cell lymphoma
- e) Waldenström macroglobulinemia

Answer: Hairy cell leukemia.

Explanation: Massive splenomegaly with a B-cell immunophenotype and characteristic surface markers (CD11c, CD25, CD103), pancytopenia and a "dry tap" / hypocellular aspirate suggests hairy cell leukemia. T-cell lymphoma would express T-cell markers; CML would show the Philadelphia chromosome.

Reviewer note: The exam wrote "CD100" — likely a typo for one of the hairy-cell markers (CD103/CD11c/CD25). The B-cell phenotype + massive splenomegaly + scant marrow blasts is the picture of hairy cell leukemia.

H6. CML diagnosis — Philadelphia chromosome [×2]

A 61-year-old with CML chronic-phase symptoms; normal labs (no pancytopenia). Best diagnostic step?

- a) Detect the Philadelphia t(9;22) translocation
- b) Abdominal CT
- c) Bone marrow biopsy

Answer: Detect the Philadelphia chromosome t(9;22) translocation (BCR-ABL).

Explanation: CML is defined by the Philadelphia chromosome — the t(9;22) BCR-ABL fusion. Detecting it (cytogenetics / FISH / PCR) is the key diagnostic step and identifies the target for tyrosine kinase inhibitors.

Reviewer note: The IM-2023 paper notes the peripheral blood shows basophilia — a classic CML clue. A bone marrow biopsy is part of the work-up but the *defining* test is the t(9;22).

H7. Sickle cell — transfusion indication [×2]

A young patient with dyspnea who had an RBC transfusion. Which is NOT an indication for transfusion in sickle cell disease?

- a) Acute chest syndrome
- b) Vaso-occlusive crisis
- c) Pyelonephritis
- d) Stroke
- e) Symptomatic anemia / aplastic crisis

Answer: Vaso-occlusive crisis (uncomplicated) — and pyelonephritis is also not a transfusion indication.

Explanation: Transfusion (often exchange transfusion) in sickle cell disease is indicated for acute chest syndrome, stroke, severe symptomatic anemia, aplastic/splenic sequestration crises, and before major surgery. An **uncomplicated vaso-occlusive (pain) crisis** is managed with hydration and analgesia, NOT transfusion.

Reviewer note: The 2023 paper's intended answer is "pyelonephritis"; the IM-2023 paper agrees ("not an indication: pyelonephritis"). An uncomplicated pain crisis is also not a transfusion indication — pick whichever the option set offers as the clearly non-indication.

H8. PE with malignancy — anticoagulation on discharge [×2]

Multiple pulmonary emboli on CT with widespread metastases; started on heparin/LMWH for 5 days. What to give on discharge?

- a) Hold heparin and start warfarin (INR goal 2–3)
- b) Continue heparin / LMWH
- c) Hold heparin and start a DOAC
- d) Stop anticoagulation

Answer: Continue LMWH (or a DOAC) — cancer-associated thrombosis.

Explanation: For cancer-associated VTE, LMWH has long been preferred; DOACs (e.g., apixaban, edoxaban, rivaroxaban) are now an accepted alternative, especially without a high GI/GU bleeding risk. Warfarin is less effective than LMWH/DOACs in active cancer.

Reviewer note: The IM-2023 key marks "continue LMWH." Current guidelines accept either LMWH or a DOAC for cancer-associated VTE — warfarin is the inferior option here.

H9. Auer rods — acute leukemia

A long case with an image of Auer rods. Diagnosis?

- a) Acute (myeloid) leukemia
- b) Chronic lymphocytic leukemia
- c) Lymphoma

Answer: Acute leukemia (acute myeloid leukemia).

Explanation: Auer rods are azurophilic, needle-like cytoplasmic inclusions seen in the myeloblasts of acute myeloid leukemia (AML) — essentially pathognomonic.

H10. Acquired hemophilia with inhibitor — mixing study does NOT correct [×2]

A patient with knee hemarthrosis/joint pain; markedly prolonged PTT, normal PT, normal platelets and WBC, low Hb. On mixing with normal plasma the PTT does NOT correct. Diagnosis / next step?

- a) Hemophilia A
- b) Acquired von Willebrand disease
- c) Fibrinogen dysfunction
- d) Acquired hemophilia with inhibitor development
- e) Platelet dysfunction
- f) Mixing study

Answer: Acquired hemophilia with an inhibitor (a factor VIII inhibitor); the diagnostic next step in the workup is a **mixing study**.

Explanation: An isolated prolonged PTT that **fails to correct** on a 1:1 mix with normal plasma indicates a circulating **inhibitor** (most often an acquired factor VIII inhibitor), not a simple factor deficiency. A patient with no lifelong bleeding history who develops new bleeding and an uncorrecting PTT has acquired hemophilia.

Reviewer note: Contrast directly with H3 — corrects → factor deficiency (e.g., hemophilia B); does NOT correct → inhibitor (acquired hemophilia, or antiphospholipid antibody). Some exams list "mixing study" as the answer because that is the test that establishes the inhibitor.

H11. ITP — first-line treatment / not a feature [×2]

A 32-year-old with easy bruising, petechiae; platelets 12,000, Hb and WBC normal, PT/PTT normal, large platelets on smear (or a 21-year-old with purpura after a recent URI). Best first-line treatment? / Which is NOT associated with ITP?

- a) Plasmapheresis
- b) Corticosteroids
- c) Platelet transfusion
- d) Rituximab
- e) Severe hypocellular bone marrow
- f) Normal or high megakaryocytes in the marrow

Answer: First-line treatment is corticosteroids. The feature NOT associated with ITP is a severe **hypocellular** bone marrow.

Explanation: Immune thrombocytopenia is isolated thrombocytopenia (normal Hb/WBC, normal PT/PTT) with large platelets and **normal or increased** marrow megakaryocytes. First-line treatment is corticosteroids (± IVIG). A hypocellular marrow indicates aplastic anemia, not ITP. Platelet transfusion is reserved for life-threatening bleeding.

Reviewer note: ITP marrow has normal/high megakaryocytes — a hypocellular marrow points away from ITP toward aplastic anemia.

H12. ITP — not used in treatment

Which is NOT used in the treatment of ITP?

- a) Corticosteroids
- b) IVIG
- c) Rituximab
- d) Platelet transfusion (routine)

Answer: Routine platelet transfusion.

Explanation: Transfused platelets are rapidly destroyed by the same autoantibodies, so platelet transfusion is reserved only for severe/life-threatening bleeding. Standard ITP therapy is corticosteroids, IVIG, and second-line agents (rituximab, TPO agonists, splenectomy).

H13. Iron deficiency anemia — wrong statement

Wrong about iron deficiency anemia:

- a) High reticulocyte count
- b) High TIBC
- c) Low ferritin
- d) Low serum iron
- e) Low MCV

Answer: High reticulocyte count.

Explanation: Iron deficiency anemia is a **hypoproliferative** anemia — the reticulocyte count is **low/inappropriately normal**. Characteristic labs: low MCV, low serum iron, low ferritin, high TIBC, low transferrin saturation.

H14. Beta thalassemia trait

A pregnant lady with fatigue, SOB; MCV 60, low MCH, HbF <1% (normal), HbA2 4.5% (normal 1.5–3.5%). Diagnosis?

- a) Iron deficiency anemia
- b) Beta thalassemia trait
- c) Sickle cell anemia

Answer: Beta thalassemia trait.

Explanation: Microcytic anemia with an **elevated HbA2** is diagnostic of beta thalassemia trait. Iron deficiency would have a low/normal HbA2; the MCV is often disproportionately low relative to the degree of anemia in thalassemia.

H15. Hodgkin lymphoma — Ann Arbor staging

A patient with fever and drenching sweats, Reed-Sternberg cells, enlarged lymph nodes on both sides of the diaphragm, and a hypodense liver lesion. Ann Arbor stage?

- a) II
- b) III A
- c) III B
- d) IV A
- e) IV B

Answer: IV B.

Explanation: Liver involvement is **extranodal** organ involvement → stage IV. The presence of B symptoms (fever, drenching night sweats, weight loss) adds the suffix "B" → stage IV B.

H16. TTP — peripheral smear and diagnosis

A 30-year-old disoriented woman; decreased Hb, normal WBC, decreased platelets, Cr 4, LDH 859, low haptoglobin, bilirubin 3, schistocytes on smear. Diagnosis?

- a) TTP
- b) DIC
- c) ITP

Answer: TTP (thrombotic thrombocytopenic purpura).

Explanation: Microangiopathic hemolytic anemia (schistocytes, high LDH, low haptoglobin, indirect hyperbilirubinemia) + thrombocytopenia + neurologic signs + renal involvement is the pentad of TTP. Coagulation tests are normal (vs DIC).

H17. Polycythemia — aquagenic pruritus

A 55-year-old with itching after hot showers. All can cause this presentation EXCEPT:

- a) Polycythemia rubra vera
- b) Hemochromatosis
- c) Renal cell carcinoma
- d) Dehydration

Answer: Hemochromatosis.

Explanation: Aquagenic pruritus (itching after a hot shower/bath) is classically associated with polycythemia vera; it can also occur with secondary polycythemia (renal cell carcinoma) and with dehydration/dry skin. Hemochromatosis is not a recognized cause of aquagenic pruritus.

H18. Vitamin B12 deficiency — not a finding

A 48-year-old with weakness, forgetfulness, symmetric paresthesia, abnormal gait; Hb 8, MCV 115. Which is NOT an expected finding?

- a) Hypersegmented neutrophils
- b) Reduced reticulocyte count
- c) Loss of position sensation in the toes
- d) Megaloblastic bone marrow changes
- e) Hypocellular bone marrow

Answer: Hypocellular bone marrow.

Explanation: B12 deficiency causes a megaloblastic anemia: macrocytosis, hypersegmented neutrophils, a **hypercellular** megaloblastic marrow (with ineffective erythropoiesis), a low reticulocyte count, and subacute combined degeneration (loss of position/vibration sense). A **hypocellular** marrow is not a feature.

H19. Macrocytic anemia — not chronic blood loss

Macrocytic anemia is NOT seen in which of the following?

- a) Myelodysplastic syndrome
- b) Chronic hemolytic anemia
- c) Vitamin B12 deficiency
- d) Chronic GI blood loss
- e) Folate deficiency

Answer: Chronic GI blood loss.

Explanation: Chronic blood loss causes iron deficiency → a **microcytic** anemia. Macrocytosis occurs with B12/folate deficiency, MDS, and chronic hemolysis (reticulocytosis raises MCV).

H20. Warfarin over-anticoagulation with bleeding

A patient on warfarin develops melena; INR 7.9, Hb 9.3. In addition to omitting warfarin, next step?

- a) Give vitamin K
- b) Observe
- c) Increase warfarin

Answer: Hold warfarin and give vitamin K (and, for active significant bleeding, prothrombin complex concentrate / fresh frozen plasma).

Explanation: A markedly elevated INR with active bleeding requires reversal: stop warfarin, give vitamin K, and for serious bleeding give 4-factor prothrombin complex concentrate (or FFP) for immediate correction.

H21. CLL — clinical picture and diagnosis

A 72-year-old with bilateral painless lymphadenopathy, lymphocyte count 30×10^9/L, weight loss; non-tender mobile nodes and splenomegaly; smear shows mature lymphocytes with smudge cells. Diagnosis?

- a) Diffuse large B-cell lymphoma
- b) Chronic lymphoid leukemia (CLL)
- c) Acute myeloid leukemia
- d) Chronic myeloid leukemia
- e) Burkitt's lymphoma

Answer: Chronic lymphoid (lymphocytic) leukemia.

Explanation: An older patient with marked lymphocytosis, painless lymphadenopathy, splenomegaly and **smudge cells** on the smear is classic for CLL.

H22. CLL — indication to treat / not expected finding

All are indications to start treatment for CLL EXCEPT — or, not expected in a CLL patient.

- a) Progressive splenomegaly
- b) Anemia refractory to treatment
- c) Weight loss / night sweats (B symptoms)
- d) Lymphocyte count 65×10^9 (an absolute count alone)
- e) t(9;22) — Philadelphia chromosome

Answer: A lymphocyte count alone is NOT an indication to treat; and t(9;22) is NOT expected in CLL.

Explanation: CLL is treated for **symptomatic/progressive** disease: B symptoms, progressive marrow failure (anemia, thrombocytopenia), bulky/progressive lymphadenopathy or splenomegaly, rapid lymphocyte doubling. An elevated absolute lymphocyte count by itself is not an indication. The **t(9;22)** Philadelphia chromosome defines CML, not CLL.

Reviewer note: Two related questions. "Indication to treat EXCEPT" → a static high lymphocyte count. "Not expected in CLL" → t(9;22).

H23. Multiple myeloma — diagnostic test

A 70-year-old with back pain, fatigue, dyspnea; Hb 8, creatinine 2.4, calcium 12, total protein 9.8, rouleaux on smear, ESR 105, multiple lytic skull/spine lesions. Most appropriate test?

- a) CT chest/abdomen
- b) Temporal artery biopsy
- c) Cytogenetics for the Philadelphia chromosome

- d) Serum protein electrophoresis
- e) Peripheral blood flow cytometry

Answer: Serum protein electrophoresis.

Explanation: Anemia, renal failure, hypercalcemia, lytic bone lesions, a high total protein with rouleaux and a very high ESR indicate multiple myeloma. Serum (and urine) protein electrophoresis detects the monoclonal (M) protein.

H24. Multiple myeloma — not a feature

Which is NOT seen in multiple myeloma?

- a) Anemia
- b) Osteoblastic (sclerotic) lesions
- c) Renal failure
- d) Amyloidosis

Answer: Osteoblastic (sclerotic) lesions.

Explanation: Multiple myeloma causes **osteolytic** (lytic, "punched-out") bone lesions, not osteoblastic ones. Its features include anemia, renal failure, hypercalcemia and AL amyloidosis.

H25. Aplastic anemia

A patient with fatigue and ecchymosis; Hb 9, platelets 70K, WBC 2000; bone marrow biopsy shows hypocellularity with no fibrosis. Diagnosis?

- a) Aplastic anemia
- b) Primary myelofibrosis
- c) ITP

Answer: Aplastic anemia.

Explanation: Pancytopenia with a **hypocellular** bone marrow and **no fibrosis** is aplastic anemia. Primary myelofibrosis would show marrow fibrosis; ITP has isolated thrombocytopenia with a normal/hypercellular marrow.

H26. Anemia of chronic disease — wrong statement

Wrong about anemia of chronic disease:

- a) Hepcidin decreases iron absorption
- b) Treat the underlying disease and EPO
- c) High TIBC and transferrin saturation

Answer: "High TIBC and transferrin saturation" is wrong.

Explanation: Anemia of chronic disease shows a **low** TIBC and **low/normal** transferrin saturation, with **normal or high** ferritin (an acute-phase reactant). Inflammation raises hepcidin, which reduces iron absorption and traps iron in macrophages. (Contrast with iron deficiency: high TIBC, low ferritin.)

H27. Blood transfusion reaction — wrong action

In a suspected blood transfusion reaction, which is WRONG?

- a) Check the patient ID and blood compatibility

- b) Secure IV cannula
- c) Monitor vitals, BP and urine output
- d) Discard the transfused units

Answer: Discard the transfused units.

Explanation: When a transfusion reaction is suspected, STOP the transfusion but **return the blood unit (and a fresh patient sample) to the blood bank** for investigation (re-typing, cross-match, cultures) — do not discard it. Maintain IV access, monitor vitals and urine output, and verify patient/unit identification.

H28. IDA — not a risk factor

Which is NOT a risk factor for iron deficiency anemia?

- a) Vegan diet
- b) Old age
- c) Multiparity
- d) Menorrhagia

Answer: Per the exam key: old age.

Explanation: Iron deficiency results from inadequate intake (vegan diet), increased demand/loss (multiparity, menorrhagia), or blood loss. The exam treats "old age" as the non–risk-factor option.

Reviewer note (CORRECTION): In practice, iron deficiency anemia in an **older adult must be treated as occult GI blood loss** (e.g., colorectal cancer) until proven otherwise — so old age is clinically a major red flag, not a benign finding. The exam key's logic is that age alone is not a *cause*; however, IDA in the elderly always warrants a GI work-up.

H29. IV iron after bariatric surgery

A patient with previous bariatric surgery; MCV 69, ferritin 4, Hb 7. Management?

- a) IV iron
- b) Oral iron
- c) Blood transfusion only

Answer: IV iron.

Explanation: Severe iron deficiency anemia after bariatric surgery results from impaired iron absorption (bypassed duodenum / reduced gastric acid). Oral iron is poorly absorbed in this setting, so **IV iron** is preferred for effective repletion.

7. RHEUMATOLOGY

Rh1. Limited scleroderma — not a feature [×2]

A patient with blue fingers diagnosed with limited scleroderma. Which is NOT a feature?

- a) Face involvement
- b) Loud P2 (pulmonary hypertension)
- c) Anterior trunk involvement
- d) Negative autoantibodies

Answer: Anterior trunk involvement (and negative autoantibodies) — these are NOT features of limited scleroderma.

Explanation: Limited cutaneous systemic sclerosis (CREST) involves skin **distal** to the elbows/knees plus the **face** — it characteristically **spares the trunk**. It is associated with pulmonary hypertension (loud P2) and anti-centromere antibodies. Truncal/proximal skin involvement defines **diffuse** scleroderma.

Reviewer note: The IM-2023 paper lists "spared anterior trunk, spared face, negative autoantibodies" — limited scleroderma **spares the trunk** but **involves the face** and is **antibody-positive** (anti-centromere). So "spared face" and "negative autoantibodies" are the false statements; trunk sparing is TRUE of limited disease.

Rh2. Dermatomyositis — antibody [×2]

A patient with hand lesions and a rash on the neck, chest and upper back, with proximal myopathy (or a female with difficulty combing hair / rising from a chair and a heliotrope rash). Which antibody?

- a) Anti-Jo-1
- b) Anti-Mi-2
- c) Anti-SRP
- d) Anti-dsDNA
- e) Anti-RNP

Answer: Anti-Mi-2 (classic for dermatomyositis with skin disease) — or anti-Jo-1 if antisynthetase features (ILD) predominate.

Explanation: Dermatomyositis with prominent cutaneous disease (heliotrope rash, shawl sign, Gottron papules) is associated with **anti-Mi-2** antibodies. **Anti-Jo-1** marks the antisynthetase syndrome (myositis + interstitial lung disease + mechanic's hands).

Reviewer note: The IM-2023 paper notes "probably Mi-2 because muscle weakness/skin disease." If the case emphasizes interstitial lung disease and mechanic's hands, the answer shifts to anti-Jo-1 — read the stem for ILD vs pure skin/muscle disease.

Rh3. ANCA vasculitis (MPA) — rituximab [×2]

A patient with fluffy/pulmonary infiltrates on CXR and foot drop (MPA features); ANA negative, ANCA positive (MPO+). Best step in management?

- a) Methotrexate
- b) Rituximab
- c) Plasmapheresis

Answer: Rituximab.

Explanation: ANCA-associated vasculitis (microscopic polyangiitis, MPO/p-ANCA positive) with organ-threatening disease is treated with induction immunosuppression — **rituximab** (or cyclophosphamide) plus glucocorticoids. Plasmapheresis is added in selected severe cases (e.g., severe renal failure or diffuse alveolar hemorrhage). Methotrexate is for non-organ-threatening disease only.

Reviewer note: The IM-2023 paper keys "use rituximab." Plasma exchange is adjunctive in severe presentations, not the primary induction agent.

Rh4. Behçet disease — true statement [×2]

A patient with oral ulcers (or painful ulcers with violaceous borders, oral ulcers, positive pathergy test). What is true about this disease?

- a) Pulmonary aneurysm is the common cause of death
- b) Neurological manifestations are common
- c) The disease is more common in females
- d) Most associated with IBD

Answer: Pulmonary artery aneurysm is a recognized cause of death in Behçet disease.

Explanation: Behçet disease causes recurrent oral and genital ulcers, uveitis, skin lesions and a positive pathergy test. Pulmonary artery aneurysms are a feared complication and an important cause of mortality. Neurologic involvement occurs but is not the most common feature; the disease has a male predominance in classic high-prevalence regions.

Reviewer note: The 2019 paper pairs the pathergy-positive oral/genital ulcer picture with an "IBD" answer option — that question was asking which condition the *findings* resemble; oral ulcers + pathergy are most specific for **Behçet**, though aphthous ulcers also occur in IBD. For "true about the disease," the pulmonary aneurysm statement is correct.

Rh5. Polyarteritis nodosa — true statement [×2]

PAN — which is true about this condition?

- a) Most patients have HBV
- b) Renal involvement causes renal infarction (microaneurysms/thrombosis)
- c) The disease causes glomerulonephritis

Answer: Renal involvement in PAN causes renal **infarction** (from medium-vessel arteritis with microaneurysms), NOT glomerulonephritis.

Explanation: Polyarteritis nodosa is a **medium-vessel** vasculitis: it causes microaneurysms and infarcts (renal, mesenteric) and characteristically **spares the glomeruli and lungs** — it does NOT cause glomerulonephritis. It is associated with hepatitis B, but only a minority of cases are HBV-related now (so "most patients have HBV" is overstated/false).

Reviewer note: Key PAN facts: medium-vessel, renal **infarcts** (not GN), no pulmonary involvement, ANCA-negative, HBV-associated in a minority.

Rh6. RA — earliest radiographic finding [×2]

What is the first / earliest plain radiographic sign of rheumatoid arthritis?

- a) Joint space narrowing
- b) Juxta-articular osteopenia
- c) Soft-tissue swelling
- d) Bone erosions
- e) Symmetric joint space loss

Answer: Soft-tissue swelling (the earliest) — followed by juxta-articular osteopenia.

Explanation: The radiographic sequence in RA is: soft-tissue swelling (earliest) → juxta-articular (periarticular) osteopenia → joint space narrowing → marginal erosions. Erosions are a later finding.

Reviewer note: Exam keys differ — 2018 marked "juxta-articular osteopenia" as the earliest, 2017 effectively as well. The very **first** sign is soft-tissue swelling; the earliest *bony* change is juxta-articular osteopenia. If the option set includes soft-tissue swelling, that is the earliest overall.

Rh7. RA — most common eye finding (scleritis) [×2]

A patient with RA on methotrexate develops a painful red eye with **limbal sparing**, photosensitivity, and pain on pressing the globe. Most common eye finding in active RA?

- a) Conjunctivitis
- b) Scleritis
- c) Uveitis
- d) Optic neuritis
- e) Cataract

Answer: Scleritis (per the described painful red eye with limbal sparing and globe tenderness).

Explanation: The painful red eye with limbal sparing and tenderness on globe pressure described here is **scleritis** — a serious, painful ocular complication of RA. (The most common ocular manifestation of RA overall is keratoconjunctivitis sicca / dry eye from secondary Sjögren's.)

Reviewer note: Exam keys conflict — 2017 keyed "conjunctivitis," 2020 keyed "scleritis." The clinical description (severe pain, limbal sparing, globe tenderness) is textbook **scleritis** — that is the defensible answer. The "most common" ocular finding in RA is actually dry eye, which is not listed; among the listed options for *this clinical picture*, scleritis fits best.

Rh8. RA — poor vs good prognostic factors [×2]

Which is associated with a poor RA prognosis? / In a smoker with RA, what indicates a good prognosis?

- a) Male gender
- b) Shared epitope (HLA-DRB1)
- c) Positive RF and anti-CCP
- d) Normal hand X-ray and MRI
- e) Late start of disease-modifying drugs

Answer: Poor prognosis: shared epitope, positive RF/anti-CCP, erosions on imaging, delayed DMARD start. Good prognosis: a **normal hand X-ray and MRI** (no erosive disease).

Explanation: Poor prognostic markers in RA include the HLA-DRB1 "shared epitope," seropositivity (RF and anti-CCP), early erosive disease, extra-articular manifestations, and delayed initiation of DMARDs. The absence of structural damage (normal X-ray/MRI) is a good prognostic sign.

Reviewer note: Two related questions. "Poor prognosis" → shared epitope / seropositivity. "Good prognosis" → no erosive damage on imaging.

Rh9. Spondyloarthropathy — MRI for sacroiliitis [×2]

A young HLA-B27-positive patient with inflammatory back pain, bilateral sacroiliac tenderness, morning stiffness >1 hour relieved by movement; X-ray normal (or inflammatory back pain responding to NSAIDs). Best next step / best imaging?

- a) No further test needed
- b) Sacroiliac joint MRI
- c) CT
- d) X-ray
- e) Ultrasound

Answer: Sacroiliac joint MRI.

Explanation: In early axial spondyloarthritis, plain X-rays are often normal. MRI of the sacroiliac joints is the most sensitive test — it detects **bone marrow edema** (active sacroiliitis) before structural changes appear on radiographs.

Rh10. Osteoarthritis — clinical features [×2]

A 68-year-old with bilateral knee pain along the joint line, worse with weight-bearing and better at rest; bony enlargement and crepitus; no systemic features; normal labs. Common clinical finding associated with the diagnosis?

- a) Prevalence increases with age
- b) Synovial fluid WBC count of 15,000
- c) Urate crystals in the pre-patellar bursa
- d) Presence of rheumatoid factor
- e) Association with Raynaud's phenomenon

Answer: Prevalence increases with age.

Explanation: This is osteoarthritis (mechanical pain, bony enlargement, crepitus, normal inflammatory markers). OA prevalence rises with age. A synovial WBC of 15,000 indicates an inflammatory arthritis (OA fluid is non-inflammatory, <2,000); rheumatoid factor and Raynaud's are not OA features.

Rh11. Anti-Ro/SSA and congenital heart block [×2]

A mother whose neonate has complete (congenital) heart block requiring a pacemaker. Which maternal antibody is most likely?

- a) Anti-endomysial antibodies
- b) Anti-SCL-70 antibodies
- c) Anti-Ro/SSA antibodies
- d) Rheumatoid factor
- e) Anti-dsDNA antibodies

Answer: Anti-Ro/SSA antibodies.

Explanation: Maternal anti-Ro/SSA (and anti-La/SSB) antibodies cross the placenta and damage the fetal cardiac conduction system, causing neonatal lupus and congenital complete heart block. These antibodies are seen in Sjögren's syndrome and SLE.

Rh12. Gout — true statement / acute attack [×2]

Which is true about gout? (And: a gout case in an acute attack — which is true?)

- a) 20–30% of cases affect the MTP joint
- b) The presentation is usually monoarticular
- c) Acute attacks are treated with allopurinol
- d) Serum uric acid is usually high during acute attacks

- e) Attacks subside over days to weeks

Answer: The presentation is usually monoarticular (and attacks self-resolve over days to weeks).

Explanation: Acute gout is typically **monoarticular** (most often the first MTP joint — podagra). Attacks resolve spontaneously over days to weeks. Urate-lowering drugs (allopurinol) are NOT started for an acute attack (they can prolong it) — they are for chronic management. Serum uric acid can be **normal** during an acute attack, so a normal level does not exclude gout.

Rh13. Anti-synthetase syndrome

A 36-year-old with progressive proximal muscle weakness, joint pain, dyspnea; cracked hyperkeratotic skin on the sides of the fingers; elevated creatine kinase; anti-Jo-1 positive; chest CT shows interstitial lung disease. Most likely diagnosis?

- a) Systemic sclerosis
- b) Polymyositis
- c) Dermatomyositis
- d) Anti-synthetase syndrome
- e) Mixed connective tissue disease

Answer: Anti-synthetase syndrome.

Explanation: The triad of myositis (high CK, proximal weakness), interstitial lung disease, and "mechanic's hands" (cracked hyperkeratotic skin) with **anti-Jo-1** (an anti-aminoacyl-tRNA synthetase antibody) defines the antisynthetase syndrome.

Rh14. CREST syndrome — anti-centromere antibody

A case of CREST syndrome — which antibody?

- a) Anti-centromere antibody
- b) Anti-Jo-1
- c) Anti-SCL-70 (anti-topoisomerase)

Answer: Anti-centromere antibody.

Explanation: Limited cutaneous systemic sclerosis (CREST — Calcinosis, Raynaud's, Esophageal dysmotility, Sclerodactyly, Telangiectasia) is associated with **anti-centromere** antibodies. Anti-SCL-70 (anti-topoisomerase I) is associated with **diffuse** systemic sclerosis.

Rh15. Primary Raynaud's — supporting feature

Which supports a diagnosis of primary Raynaud's?

- a) Age <25 (young age at onset)
- b) Male gender
- c) Presence of telangiectasia
- d) Pitting nails / nailfold capillary changes

Answer: Young age at onset (<25), and a benign picture.

Explanation: Primary (idiopathic) Raynaud's typically begins at a young age, is symmetric, has a normal nailfold capillaroscopy, negative autoantibodies, and no tissue damage. Telangiectasias, abnormal nailfold capillaries, digital ulcers and positive serology suggest **secondary** Raynaud's (e.g., systemic sclerosis).

Rh16. GCA — most specific symptom

A patient with headache, temporal tenderness, jaw claudication and other GCA symptoms. Most specific symptom for the diagnosis?

- a) Headache
- b) Jaw claudication
- c) Temporal tenderness

Answer: Jaw claudication.

Explanation: Among the features of giant cell (temporal) arteritis, **jaw claudication** is the most specific symptom (it has the highest positive likelihood ratio). Headache and scalp tenderness are common but less specific.

Rh17. RA — likely RF-negative condition

Which is likely to be RHEUMATOID FACTOR NEGATIVE?

- a) Adult Still's disease
- b) Subacute bacterial endocarditis
- c) Cryoglobulinemia
- d) Sarcoidosis
- e) Sjögren's syndrome

Answer: Adult Still's disease.

Explanation: Adult-onset Still's disease is characteristically **seronegative** — RF and ANA are typically negative. RF can be positive in subacute bacterial endocarditis, cryoglobulinemia, sarcoidosis and Sjögren's syndrome (RF is not specific to RA).

Rh18. SLE — scarring alopecia

Which causes scarring alopecia in SLE patients?

- a) Discoid rash
- b) Acute cutaneous rash
- c) Subacute cutaneous rash
- d) Lupus panniculitis
- e) Lupus pernio

Answer: Discoid rash.

Explanation: Discoid lupus erythematosus causes a chronic, scarring skin lesion — including **scarring alopecia** — due to follicular destruction. Acute and subacute cutaneous lupus are non-scarring.

Rh19. Sjögren's syndrome — not a finding

Which is NOT found in Sjögren's syndrome?

- a) Anti-Ro and anti-La
- b) Anti-Smith
- c) Rheumatoid factor
- d) ANA

Answer: Anti-Smith.

Explanation: Sjögren's syndrome is associated with anti-Ro/SSA and anti-La/SSB antibodies, a positive ANA, and rheumatoid factor. **Anti-Smith** antibody is highly specific for **SLE**, not Sjögren's.

Rh20. Sjögren's syndrome — clinical diagnosis

A patient with dry eyes, dry mouth, bilateral parotid gland enlargement and joint pain; anti-SSA/SSB positive, ANA positive, RF positive. Diagnosis?

- a) RA
- b) Sjögren's syndrome
- c) SLE
- d) Scleroderma

Answer: Sjögren's syndrome.

Explanation: Sicca symptoms (dry eyes/mouth) with bilateral parotid enlargement and anti-SSA/SSB positivity is primary Sjögren's syndrome. ANA and RF are commonly positive but non-specific.

Rh21. SLE — autoantibody for monitoring disease activity [×2]

Which autoantibody has a role in monitoring disease activity?

- a) Anti-Ro (SSA) in Sjögren's
- b) Rheumatoid factor in RA
- c) Anti-dsDNA in SLE
- d) Anti-Sm in SLE
- e) ANA in SLE

Answer: Anti-dsDNA in SLE.

Explanation: Anti-dsDNA titers **fluctuate with SLE disease activity** (rising titers, often with falling complement, correlate with flares, especially lupus nephritis). Anti-Sm is specific for SLE but does not track activity; ANA is a screening test that does not correlate with activity.

Rh22. Lupus nephritis — evidence of active disease

A patient with SLE and lupus nephritis. Evidence of active disease?

- a) Low C3/C4 and high anti-dsDNA
- b) Negative anti-dsDNA
- c) High ESR and CRP
- d) High hemoglobin and high anti-dsDNA

Answer: Low C3/C4 with high anti-dsDNA.

Explanation: Active lupus (especially nephritis) is marked by **consumption of complement** (low C3 and C4) and **rising anti-dsDNA** titers. CRP is often only mildly elevated in lupus flares (a markedly high CRP suggests infection).

Rh23. Lupus nephritis — induction therapy

A 25-year-old with SLE and new-onset renal disease (proteinuria, hematuria, RBC casts, rising creatinine), started on high-dose corticosteroids. Most appropriate additional therapy?

- a) Hydroxychloroquine
- b) Azathioprine

- c) Mycophenolate mofetil
- d) Methotrexate
- e) Cyclophosphamide (oral)

Answer: Mycophenolate mofetil.

Explanation: Induction therapy for proliferative lupus nephritis is high-dose corticosteroids plus an immunosuppressant — **mycophenolate mofetil** or (IV) cyclophosphamide. Mycophenolate is widely used as first-line induction (and maintenance). All SLE patients should also be on hydroxychloroquine, but it is not the induction agent for nephritis.

Rh24. Granulomatosis with polyangiitis (GPA)

A 42-year-old with persistent cough and blood-streaked sputum 2 weeks, dark urine and joint pain; Hb 9.5, creatinine 2.1 (baseline 0.9); urinalysis with hematuria, RBC casts, proteinuria; c-ANCA (PR3) positive. Most likely diagnosis?

- a) Goodpasture syndrome
- b) Microscopic polyangiitis
- c) Granulomatosis with polyangiitis (GPA)
- d) Sarcoidosis

Answer: Granulomatosis with polyangiitis (GPA).

Explanation: A pulmonary-renal syndrome (alveolar hemorrhage + glomerulonephritis) with **c-ANCA / PR3** positivity is characteristic of granulomatosis with polyangiitis. Microscopic polyangiitis is usually p-ANCA/MPO positive and lacks granulomatous upper-airway disease.

Rh25. CKD + acute gout — colchicine

A patient with CKD stage 3b has sudden severe first-MTP joint pain; aspiration shows negative birefringent crystals. First-line treatment?

- a) NSAIDs
- b) Intra-articular steroids
- c) Colchicine
- d) Allopurinol
- e) Febuxostat

Answer: Colchicine (or intra-articular/systemic corticosteroids).

Explanation: Negatively birefringent needle-shaped crystals = acute gout. In a patient with CKD, NSAIDs are relatively contraindicated; colchicine (dose-reduced for renal function) or corticosteroids are preferred. Urate-lowering drugs (allopurinol, febuxostat) are NOT started during an acute attack.

Rh26. SLE — malar rash, photosensitivity, avascular necrosis

A patient with malar rash and photosensitivity develops osteonecrosis of the femoral head (avascular necrosis / slipped capital femoral epiphysis–like hip pain).

- a) The skin findings indicate SLE; avascular necrosis is a recognized complication (disease and corticosteroid therapy).

Answer: SLE — avascular necrosis of the femoral head is a recognized complication.

Explanation: Malar rash and photosensitivity are SLE features. Osteonecrosis (avascular necrosis), especially of the femoral head, occurs in SLE both from the disease itself and from corticosteroid therapy.

Reviewer note: The IM-2023 source listed this as a brief item; the teaching point is the association of SLE (and steroid use) with avascular necrosis of the hip.

Rh27. Methotrexate — stop before pregnancy

A patient on methotrexate planning pregnancy.

- a) Methotrexate is teratogenic and must be stopped ~3 months before conception.

Answer: Stop methotrexate at least ~3 months before pregnancy (and use effective contraception until then).

Explanation: Methotrexate is a teratogen (and an abortifacient); it must be discontinued well before conception — generally ~3 months beforehand — and is contraindicated during pregnancy.

Rh28. Psoriatic arthritis — unique feature

A unique/characteristic feature of psoriatic arthritis (vs other spondyloarthropathies)?

- a) Enthesopathy
- b) Dactylitis
- c) Arthritis mutilans
- d) Asymmetric arthritis
- e) Spondylitis

Answer: Arthritis mutilans.

Explanation: Enthesitis, dactylitis, asymmetric arthritis and spondylitis are shared across the spondyloarthropathies. **Arthritis mutilans** — a severe, deforming, resorptive ("telescoping/pencil-in-cup") arthritis — is the most characteristic/unique form of psoriatic arthritis.

8. INFECTIOUS DISEASE (ID)

ID1. Most common cause of infective endocarditis [×2]

Most common cause of (acute) infective endocarditis?

- a) Staphylococcus aureus
- b) Streptococcus pyogenes / viridans
- c) Enterococcus
- d) Neisseria gonorrhoeae

Answer: Staphylococcus aureus.

Explanation: Staphylococcus aureus is now the most common overall cause of infective endocarditis, including acute IE and IE in injection drug users and healthcare-associated cases. Viridans streptococci classically cause subacute IE on previously abnormal valves.

Reviewer note: The IM-2023 paper notes the options did not include viridans strep — so the answer is staph aureus. If "viridans streptococci" appears as an option for a subacute, native-valve, dental-source case, that may be the intended answer; for the *overall* most common cause, it is S. aureus.

ID2. Most sensitive sample for COVID/SARS PCR [×2]

Most sensitive site/sample for SARS-CoV-2 (COVID) detection by PCR?

- a) Bronchial lavage
- b) Nasopharyngeal swab
- c) Feces / anal swab
- d) Sputum

Answer: Nasopharyngeal swab.

Explanation: The nasopharyngeal swab is the standard, most sensitive readily-obtained sample for SARS-CoV-2 PCR. Lower respiratory samples (bronchoalveolar lavage) can have higher yield in intubated patients but are invasive and not the routine test.

ID3. Antibiotic causing arthropathy / tendon injury [×2]

A patient used an antibiotic and then had shoulder/joint pain or could not raise his limb. Which antibiotic most likely caused this?

- a) Levofloxacin (a fluoroquinolone)
- b) Tetracycline

Answer: Levofloxacin (fluoroquinolone).

Explanation: Fluoroquinolones (levofloxacin, ciprofloxacin) cause tendinopathy and tendon rupture (classically the Achilles, but also shoulder), and arthropathy — a recognized class adverse effect.

ID4. HIV treatment indication [×2]

HIV treatment indication?

- a) All patients positive for HIV
- b) Only patients with a low CD4 count
- c) Only symptomatic patients

Answer: All patients positive for HIV.

Explanation: Current guidelines recommend antiretroviral therapy for **all** HIV-positive individuals regardless of CD4 count or symptoms — early treatment improves outcomes and reduces transmission.

ID5. Sacroiliitis after fever — Brucella [×2]

A patient with sacroiliitis after fever (or a typical brucellosis case). Most likely pathogen?

- a) Brucella
- b) Staphylococcus aureus
- c) Salmonella

Answer: Brucella.

Explanation: Brucellosis classically causes sacroiliitis and other osteoarticular involvement (spondylitis), along with undulant fever, sweats and hepatosplenomegaly — often after contact with livestock or unpasteurized dairy.

ID6. Hand hygiene — true statement [×2]

True about hand hygiene:

- a) Alcohol should be rubbed on the hands for a maximum of 10 seconds
- b) Long nails and nail polish are not infective if scrubbed with alcohol
- c) Alcohol scrub eradicates HBV / kills hepatitis B
- d) Hand gloves are an acceptable substitute for hand hygiene

Answer: Alcohol-based hand rub is effective against hepatitis B virus (and most pathogens).

Explanation: Alcohol-based hand rub is effective against enveloped viruses including HBV, HCV and HIV. Hand rub should be applied for ~20–30 seconds (not "max 10 s"); long/artificial nails and nail polish harbor pathogens; gloves do NOT replace hand hygiene.

Reviewer note: The IM-2023 paper keys "hand hygiene kills hep B: correct." Alcohol is less reliable against non-enveloped viruses (e.g., norovirus) and C. difficile spores — for those, soap-and-water washing is needed.

ID7. Post-imipenem diarrhea — C. difficile [×2]

A patient had pneumonia, took imipenem, then developed diarrhea (or a typical pseudomembranous colitis case). Most likely causative pathogen?

- a) E. coli
- b) Clostridioides difficile
- c) Clostridium perfringens

Answer: Clostridioides difficile.

Explanation: Broad-spectrum antibiotics (e.g., carbapenems, clindamycin, fluoroquinolones, cephalosporins) disrupt the gut flora and predispose to C. difficile colitis — the most common cause of antibiotic-associated and hospital-acquired infectious diarrhea.

ID8. Most nephrotoxic antibiotic combination [×2]

The combination with the worst nephrotoxicity:

- a) Vancomycin with amikacin / gentamicin
- b) Ceftriaxone with azithromycin
- c) Penicillin with metronidazole

Answer: Vancomycin with an aminoglycoside (amikacin / gentamicin).

Explanation: Both vancomycin and aminoglycosides are independently nephrotoxic; combining them substantially increases the risk of acute kidney injury.

ID9. C. difficile — false statement

All are true about Clostridioides difficile disease EXCEPT:

- a) It is diagnosed by detection of serum antibodies to toxin A and B
- b) It is caused by a Gram-positive bacillus
- c) The recurrence rate can reach 20%
- d) It is the most common cause of hospital-acquired diarrhea
- e) It is treated with metronidazole

Answer: "It is diagnosed by detection of serum antibodies to toxin A and B" is FALSE.

Explanation: C. difficile is diagnosed by **stool** testing — toxin enzyme immunoassay and/or NAAT (PCR) for toxin genes — not serum antibodies. It is a Gram-positive anaerobic bacillus, has a recurrence rate up to ~20–25%, and is treated with oral vancomycin or fidaxomicin (metronidazole is now second-line).

ID10. Anti-pseudomonal antibiotic

Which is NOT an anti-pseudomonal antibiotic? (And: which drug CAN treat Pseudomonas?)

- a) Ceftriaxone
- b) Cefepime
- c) Ciprofloxacin
- d) Ceftazidime
- e) Gentamicin
- f) Cefazolin

Answer: Ceftriaxone (and cefazolin) are NOT anti-pseudomonal. Anti-pseudomonal agents include cefepime, ceftazidime, ciprofloxacin, gentamicin (and piperacillin-tazobactam, carbapenems).

Explanation: Among cephalosporins, **ceftazidime** and **cefepime** have anti-pseudomonal activity; **ceftriaxone** and **cefazolin** do not. Ciprofloxacin and aminoglycosides (gentamicin/amikacin) are also anti-pseudomonal.

ID11. Brucella — true statement

Which is true about Brucella?

- a) Brucella abortus is the most virulent species
- b) It is commonly transmitted human-to-human
- c) It is a Gram-positive bacillus
- d) It should be treated with antibiotics for several weeks
- e) People who work with animals should receive a brucella vaccine

Answer: Brucellosis should be treated with antibiotics for several weeks.

Explanation: Brucellosis requires prolonged combination antibiotic therapy (e.g., doxycycline plus rifampin or an aminoglycoside) for several weeks to prevent relapse. Brucella is a Gram-**negative** coccobacillus; human-

to-human transmission is rare; B. melitensis (not abortus) is the most virulent species; there is no human vaccine.

ID12. Brucellosis — diagnostic test

Most appropriate test (highest yield) to diagnose brucellosis?

- a) Serum agglutination test
- b) PCR
- c) Blood culture
- d) Western blot
- e) RPR

Answer: Serum agglutination test (and blood culture).

Explanation: The standard serologic test for brucellosis is the **serum agglutination test**, which detects anti-Brucella antibodies and has high diagnostic yield. Blood culture is definitive but slower and less sensitive (the organism is fastidious/slow-growing).

Reviewer note: Exam key marked "A, C?" — both serology and culture are used. Serology (agglutination titers) is the most practical high-yield test; culture is the gold standard when positive.

ID13. Brucellosis — most common cause of death

Most common cause of death in brucellosis?

- a) Meningitis
- b) Pneumonia
- c) Liver abscess
- d) Leukopenia
- e) Endocarditis

Answer: Endocarditis.

Explanation: Although uncommon overall, **endocarditis** is the leading cause of death in brucellosis.

ID14. HIV-defining illness

Which is NOT an HIV/AIDS-defining illness?

- a) Kaposi sarcoma
- b) Tuberculosis
- c) CMV retinitis
- d) Herpes zoster
- e) Mononucleosis syndrome
- f) Invasive candidiasis

Answer: Herpes zoster (and a mononucleosis-like syndrome) — these are NOT AIDS-defining illnesses.

Explanation: AIDS-defining illnesses include Kaposi sarcoma, esophageal/tracheal/bronchial candidiasis, CMV retinitis, Pneumocystis pneumonia, CNS toxoplasmosis, and others. Herpes zoster (shingles) is common in HIV but is NOT itself AIDS-defining; an acute mononucleosis-like illness is the seroconversion syndrome, not an AIDS-defining condition.

Reviewer note: Different papers list different distractors (2020 → herpes zoster; 2018 → mononucleosis syndrome). Both are correct as non–AIDS-defining.

ID15. Norovirus — outbreak of mild diarrhea

A patient with abdominal discomfort, 4 loose non-bloody stools, no fever; a household contact had similar self-limited symptoms. Most likely organism?

- a) Rotavirus
- b) Norovirus
- c) Staphylococcus aureus

Answer: Norovirus.

Explanation: Norovirus causes brief, self-limited, non-bloody watery diarrhea/vomiting that spreads readily among close contacts (households, institutions, cruise ships). Management of mild cases is supportive (oral rehydration).

ID16. Animal reservoir mismatch

Which is a mismatch (organism–animal reservoir)?

- a) Chlamydia (psittaci) — bats
- b) Salmonella enteritidis — chicken
- c) Pasteurella — cats
- d) Cryptococcus neoformans — pigeons
- e) Brucella canis — dogs

Answer: Chlamydia — bats (mismatch).

Explanation: Chlamydophila psittaci (psittacosis) is associated with **birds** (parrots, parakeets, poultry), not bats. The other pairs are correct: Salmonella–poultry, Pasteurella–cat/dog bites, Cryptococcus–pigeon droppings, Brucella canis–dogs.

ID17. Risk of HCV after needle-stick

The risk of HCV infection after a needle-stick injury:

- a) 0.3%
- b) 3%
- c) 30%

Answer: ~3%.

Explanation: Approximate transmission risks after a percutaneous needle-stick from an infected source: HIV ~0.3%, HCV ~3%, HBV up to ~30% (highest).

ID18. Vancomycin — not a side effect

Which is NOT a side effect of vancomycin?

- a) Red man syndrome
- b) Neutropenia
- c) Phlebitis
- d) Seizures
- e) Nephrotoxicity

Answer: Seizures.

Explanation: Vancomycin can cause red man (vancomycin infusion) syndrome, neutropenia, phlebitis at the infusion site, nephrotoxicity and ototoxicity. Seizures are not a typical vancomycin adverse effect (they are associated with, e.g., imipenem).

ID19. TB — true statement

Which is true regarding tuberculosis?

- a) Gram-positive bacilli
- b) Intracellular organism
- c) Minimum treatment duration is 9 months

Answer: TB is an intracellular organism.

Explanation: Mycobacterium tuberculosis is an **intracellular** acid-fast bacillus (it survives within macrophages). It is **not** classified as Gram-positive (it is acid-fast). Standard treatment of drug-susceptible TB is 6 months.

ID20. TB treatment regimen

Which is true about TB treatment?

- a) INH + Rifampin 2 months, then add Ethambutol + Streptomycin 4 months
- b) INH + Rifampin + Ethambutol + (Streptomycin/Pyrazinamide) for the first 2 months, then INH + Rifampin for 4 months
- c) INH + Rifampin + Ethambutol for 2 months, then INH + Rifampin for 4 months
- d) Four drugs for the first 4 months, then 2 drugs for 2 months

Answer: A 4-drug intensive phase for the first 2 months, then a 2-drug (INH + Rifampin) continuation phase for 4 months.

Explanation: Standard drug-susceptible TB treatment is a 6-month regimen: an intensive phase of isoniazid, rifampin, pyrazinamide and ethambutol for 2 months, followed by a continuation phase of isoniazid and rifampin for 4 months.

ID21. Vaccine contraindicated in immunodeficiency [×2]

Which vaccine is contraindicated in persons with underlying immunodeficiency?

- a) Influenza (injectable) vaccine
- b) Conjugated pneumococcal vaccine
- c) Measles-mumps-rubella (MMR) vaccine
- d) Hepatitis B vaccine
- e) Tetanus vaccine

Answer: Measles-mumps-rubella (MMR) vaccine.

Explanation: MMR is a **live attenuated** vaccine and is contraindicated in significant immunodeficiency. Inactivated/subunit vaccines (injectable influenza, conjugated pneumococcal, hepatitis B, tetanus) are safe in immunocompromised patients.

ID22. Hydatid cyst disease — false statement

All about hydatid cyst disease are true EXCEPT:

- a) The etiologic agent is Echinococcus granulosus

- b) The disease occurs after ingestion of parasite eggs
- c) Humans are the only reservoir
- d) Person-to-person infection does not occur
- e) The majority of lesions occur in the liver

Answer: "Humans are the only reservoir" is FALSE.

Explanation: Echinococcus granulosus has a dog (definitive host)–sheep (intermediate host) life cycle; humans are **accidental, dead-end** intermediate hosts, not the reservoir. Humans acquire it by ingesting eggs; the liver is the most common site; person-to-person spread does not occur.

ID23. Influenza vaccine — accurate statement

Which is most accurate about the injectable influenza vaccine?

- a) Indicated in pregnancy
- b) Should be given in December in temperate regions
- c) Covers only 2 strains of influenza

Answer: It is indicated in pregnancy.

Explanation: The inactivated injectable influenza vaccine is recommended (and safe) in pregnancy at any trimester. It is best given **before** the influenza season (autumn, e.g., October), and modern vaccines are trivalent or quadrivalent (3–4 strains).

ID24. HIV — TMP-SMX prophylaxis

In an HIV patient, prophylactic TMP-SMX protects against?

- a) Pneumocystis pneumonia
- b) CMV retinitis
- c) TB
- d) Oral candidiasis

Answer: Pneumocystis pneumonia.

Explanation: TMP-SMX prophylaxis in HIV (started when CD4 <200) prevents Pneumocystis jirovecii pneumonia (and also toxoplasmosis).

ID25. Needle-stick from HBV source, non-immune

A dentist sustains a needle-stick from a chronic-HBV patient; HBsAg negative, HBsAb negative; vaccinated 25 years ago, no boosters/titers since. Next best step?

- a) Hepatitis B vaccine + HBV immunoglobulin
- b) Hepatitis B vaccine alone
- c) Reevaluate after a week
- d) Do nothing because he was vaccinated

Answer: Hepatitis B vaccine + HBV immunoglobulin.

Explanation: After exposure to an HBV-positive source, a person who is a **non-responder / has no protective anti-HBs** (HBsAb negative) should receive both HBV immunoglobulin (passive immunity) and the HBV vaccine series (active immunity).

ID26. Hepatitis — most accurate statement

Which is the most accurate statement about hepatitis?

- a) To diagnose HAV hepatitis, do HAV RNA PCR
- b) 90% of HBV patients clear the infection
- c) HEV causes chronic hepatitis in 75% of cases
- d) HAV infection usually leads to cirrhosis

Answer: ~90% of (adult) HBV-infected patients clear the infection.

Explanation: About 90–95% of immunocompetent adults with acute hepatitis B clear the virus and recover; only ~5–10% become chronic carriers (chronicity is much higher in neonatal infection). HAV is diagnosed serologically (anti-HAV IgM) and does not cause chronic disease/cirrhosis; HEV is usually self-limited (chronic HEV occurs mainly in immunosuppressed patients).

ID27. Diabetic with ear discharge — malignant otitis externa

A diabetic with ear pus, a painful pinna and an edematous external ear canal. Most likely cause of the discharge?

- a) Staphylococcus aureus
- b) Fungal
- c) Pseudomonas aeruginosa
- d) Streptococcus pneumoniae

Answer: Pseudomonas aeruginosa.

Explanation: Malignant (necrotizing) otitis externa, classically in elderly diabetics, is caused by **Pseudomonas aeruginosa** — a potentially invasive infection of the external canal and skull base.

ID28. Neutropenic patient — isolation type

A patient on chemotherapy with WBC 1000 and 20% neutrophils. Best isolation method?

- a) Protective (reverse) isolation
- b) Droplet isolation
- c) Contact isolation
- d) Negative-pressure isolation

Answer: Protective (reverse) isolation.

Explanation: A severely neutropenic patient (absolute neutrophil count ~200) needs **protective/reverse isolation** to shield the patient from environmental pathogens, since the goal is to protect the immunocompromised patient rather than contain a contagious illness.

ID29. Acute infective diarrhea — supportive care

A patient with diarrhea, Na 136, K 3.5, a contact history and stable vitals. Management?

- a) IV fluids (rehydration)
- b) Antibiotics
- c) Anti-motility agent

Answer: IV fluids / rehydration (supportive care).

Explanation: Most acute infectious (viral) diarrhea is self-limited and managed with rehydration. Empiric antibiotics are not routinely indicated, and anti-motility agents are avoided when an inflammatory/invasive infection is suspected.

9. PSYCHIATRY

P1. Sign of alcohol dependence [×2]

A sign of alcohol addiction / dependence:

- a) Morning drinking
- b) Drinking only on weekends
- c) Occasional social drinking

Answer: Morning drinking.

Explanation: Early-morning drinking (an "eye-opener" to relieve withdrawal symptoms) is a classic marker of alcohol dependence — it reflects physiological dependence and is one of the CAGE questionnaire items.

P2. Depression — true statement [×2]

A depression question — which is true?

- a) Depression commonly presents with physical (somatic) symptoms
- b) It is the 5th cause of disability
- c) Older women commit suicide more than older men

Answer: Depression commonly presents with physical (somatic) symptoms.

Explanation: Major depression frequently presents with somatic complaints (fatigue, pain, sleep and appetite disturbance), and many patients present to non-psychiatric clinicians with physical symptoms. Depression is a leading cause of disability worldwide (among the top causes, not specifically "5th"); completed suicide rates are generally higher in older **men** than older women.

Reviewer note: This is a sensitive topic. If you or someone you know is struggling with depression or suicidal thoughts, reaching out to a professional or trusted person for support is important.

P3. Drug-induced parkinsonism — treatment [×2]

A drug used in the treatment of drug-induced Parkinsonism:

- a) Levodopa
- b) Fluoxetine
- c) Anticholinergic (e.g., procyclidine / benztropine)

Answer: An anticholinergic (procyclidine / benztropine) — and the offending drug should be stopped/reduced.

Explanation: Drug-induced parkinsonism (from dopamine-blocking agents — antipsychotics, metoclopramide) is managed first by **withdrawing or reducing the offending drug**; anticholinergics (procyclidine, benztropine) help control the extrapyramidal symptoms. Levodopa is generally ineffective because the dopamine receptors are being blocked.

Reviewer note: Exam keys vary — one paper listed "levodopa," another "procyclidine." The mechanistically correct answer is to stop the causative dopamine antagonist and use an **anticholinergic**; levodopa is not the treatment for drug-induced parkinsonism.

P4. Serotonin syndrome vs neuroleptic malignant syndrome

A 32-year-old with severe depression, recently stopped phenelzine (an MAOI) 1 month ago; given sertraline, then olanzapine; now confused, diaphoretic, temp 40.1, BP 167/97, pulse 112, with limb rigidity. What happened?

- a) Catatonia
- b) Serotonin syndrome
- c) Malignant hyperthermia
- d) Neuroleptic malignant syndrome

Answer: Per the exam key: neuroleptic malignant syndrome.

Explanation: Hyperthermia, autonomic instability, altered mental status and **rigidity** after an antipsychotic (olanzapine) is the picture of neuroleptic malignant syndrome.

Reviewer note: This case is genuinely ambiguous. The combination of a recently-stopped MAOI (phenelzine) plus an SSRI (sertraline) is a classic setup for **serotonin syndrome** — which also causes hyperthermia, autonomic instability and altered mentation, but more typically with **neuromuscular hyperactivity (clonus, hyperreflexia)** rather than lead-pipe rigidity. The "rigidity after olanzapine" detail favors NMS; the "MAOI + SSRI" detail favors serotonin syndrome. Distinguish by neuromuscular findings: clonus/hyperreflexia → serotonin syndrome; lead-pipe rigidity + bradyreflexia → NMS. The exam key (NMS) is defensible but not certain.

P5. Clozapine — agranulocytosis

Which drug is most likely to cause agranulocytosis?

- a) Olanzapine
- b) Haloperidol
- c) Clozapine
- d) Sertraline

Answer: Clozapine.

Explanation: Clozapine carries a well-known risk of **agranulocytosis**, which is why patients on clozapine require regular monitoring of the absolute neutrophil count.

P6. Schizophrenia — neuroanatomical changes

Which change is related to schizophrenia?

- a) Hyperactivity of the cingulate gyrus
- b) Enlarged lateral and third ventricles
- c) Hyperactivity of the temporal-parietal area

Answer: Enlarged lateral and third ventricles.

Explanation: Schizophrenia is associated with structural brain changes — notably **enlarged lateral and third ventricles** and reduced cortical/hippocampal volume.

Reviewer note: The 2019 key marked "B & C." Ventricular enlargement (B) is the best-established structural finding.

P7. Borderline personality disorder

Which personality disorder is associated with impulsivity, an unstable self-image and affect, and unstable interpersonal relationships?

- a) Borderline
- b) Histrionic
- c) Avoidant
- d) Schizoid

Answer: Borderline.

Explanation: Borderline personality disorder is characterized by instability of affect, self-image and relationships, marked impulsivity, and fear of abandonment.

P8. Psychiatric disorder associated with autism

Which psychiatric disorder is most associated with autism spectrum disorder?

- a) Major depressive disorder
- b) Obsessive-compulsive disorder
- c) Generalized anxiety disorder
- d) ADHD

Answer: Per the exam key: obsessive-compulsive disorder.

Explanation: The 2019 exam keyed OCD as the disorder most associated with ASD.

Reviewer note: This is debatable. **ADHD** is, in fact, one of the most commonly co-occurring conditions with autism spectrum disorder (and DSM-5 now permits a concurrent diagnosis). Anxiety disorders and OCD are also frequently comorbid. The exam key (OCD) is defensible because of overlapping repetitive/ritualistic behaviors, but ADHD is at least an equally strong answer — treat the key as uncertain.

P9. Bipolar II disorder

A female with multiple episodes of major depression and hypomania but never a full manic episode. Diagnosis?

- a) Bipolar I
- b) Bipolar II
- c) MDD
- d) Cyclothymia

Answer: Bipolar II.

Explanation: Bipolar II disorder is defined by at least one major depressive episode plus at least one **hypomanic** episode, **without** a full manic episode (a full manic episode would make it bipolar I).

P10. Catatonia — treatment

Most appropriate treatment for catatonia?

- a) Haloperidol
- b) Sertraline
- c) Lorazepam
- d) Clozapine
- e) Risperidone

Answer: Lorazepam.

Explanation: A benzodiazepine (**lorazepam**) is first-line for catatonia; electroconvulsive therapy is used for refractory cases. Antipsychotics can worsen catatonia and risk neuroleptic malignant syndrome.

P11. PTSD — evidence-based treatment

Most evidence-based treatment for moderate-to-severe PTSD?

- a) Benzodiazepines
- b) Exposure (trauma-focused) therapy
- c) Trazodone
- d) Bupropion

Answer: Exposure (trauma-focused) psychotherapy.

Explanation: Trauma-focused psychotherapy — including prolonged exposure and cognitive processing therapy — has the strongest evidence for PTSD. Benzodiazepines are not recommended (no benefit and possible harm); SSRIs/SNRIs are first-line pharmacotherapy.

P12. OCD — neuroanatomy

A patient with OCD — which brain region is most implicated?

- a) Amygdala
- b) Orbitofrontal cortex
- c) Dorsolateral prefrontal cortex
- d) Nucleus accumbens
- e) Broca's area

Answer: Orbitofrontal cortex.

Explanation: OCD is associated with hyperactivity in the **cortico-striato-thalamo-cortical** circuit, particularly the **orbitofrontal cortex** (and caudate/anterior cingulate).

P13. Major depressive disorder — neurobiology

A patient with major depressive disorder — most commonly associated neurobiological finding?

- a) Decreased monoamine (serotonin/norepinephrine) neurotransmission
- b) Increased serotonin receptor activity

Answer: Altered monoamine neurotransmission (the classic monoamine hypothesis — reduced serotonergic/noradrenergic activity).

Explanation: The traditional neurobiological model of depression involves dysregulation of monoamine (serotonin, norepinephrine, dopamine) neurotransmission, supported by the efficacy of agents that increase monoamine availability.

Reviewer note: The exam key was marked "C maybe (can't remember the options)." The original options were not fully recorded; the well-established teaching point is the monoamine hypothesis of depression.

P14. Delirium — features

A delirium question — which is true?

- a) Fluctuating course
- b) Usually leads to dementia
- c) Stable level of consciousness

Answer: Delirium has an acute onset with a **fluctuating** course.

Explanation: Delirium is an acute, fluctuating disturbance of attention and awareness, often with an identifiable medical cause. It is typically reversible when the cause is treated; it does not itself "lead to" dementia, although dementia is a major risk factor for developing delirium.

P15. Opioid withdrawal / toxicity

A patient with opioid withdrawal vs toxicity — naloxone or methadone?

- a) Naloxone for opioid toxicity/overdose
- b) Methadone for withdrawal/maintenance

Answer: Naloxone reverses opioid **toxicity/overdose**; methadone is used for **withdrawal** management and maintenance.

Explanation: Naloxone is a competitive opioid antagonist that rapidly reverses opioid overdose (respiratory depression). Methadone (a long-acting opioid agonist) is used to treat opioid withdrawal and for maintenance therapy in opioid use disorder.

10. DERMATOLOGY

D1. Verruca vulgaris — false statement [×2]

Verruca vulgaris — which is FALSE?

- a) Caused by human papillomavirus
- b) Can be contagious by contact
- c) Contiguous autoinoculation from hand to genitalia can occur
- d) Can resolve if left without treatment
- e) Less common in children than in adults

Answer: "Less common in children than in adults" is FALSE.

Explanation: Common warts (verruca vulgaris) are caused by HPV, spread by contact and autoinoculation, and frequently resolve spontaneously. They are **more** common in **children** and young adults, not less — so that statement is false.

D2. Isotretinoin + headache — pseudotumor cerebri [×2]

A patient on isotretinoin with a constant headache unresponsive to analgesia (a 21-year-old also recently took doxycycline and developed a severe headache). Best next step?

- a) Decrease the isotretinoin dose
- b) Follow up after some time
- c) Increase the analgesia dose
- d) Stop isotretinoin (and the tetracycline) immediately and refer to ophthalmology for a dilated exam
- e) Check LFTs/KFTs and stop the drugs

Answer: Stop the offending drug(s) and refer for an ophthalmologic (dilated fundus) exam — to evaluate for idiopathic intracranial hypertension (pseudotumor cerebri).

Explanation: Isotretinoin and tetracyclines (doxycycline) can cause **idiopathic intracranial hypertension** (pseudotumor cerebri) — a persistent headache unresponsive to analgesia, sometimes with visual symptoms and papilledema. The drug must be stopped and the patient evaluated (fundoscopy for papilledema) to prevent vision loss.

Reviewer note: The combination of isotretinoin **plus** a tetracycline markedly increases the risk of intracranial hypertension — these two should not be used together. The headache is a red flag, not a dosing nuisance.

D3. Acne — correct statement

Which statement is correct (about acne)?

- a) Infantile acne has no association with adult acne
- b) A woman can safely become pregnant 1 month after stopping oral isotretinoin
- c) Topical steroids are used for severe acne
- d) Staphylococcus epidermidis is strongly involved in acne pathogenesis

Answer: Per the exam key: a woman can become pregnant after a defined interval off isotretinoin (the key marked "A").

Explanation: Isotretinoin is a potent teratogen. Pregnancy must be avoided during treatment and for a period afterward.

Reviewer note (CORRECTION): The exam keyed "A" (infantile acne has no association with adult acne) — but option B as written ("safe to conceive 1 month after stopping isotretinoin") is the more clearly testable point and the standard teaching is that pregnancy should be avoided for **at least 1 month** after discontinuing **oral** isotretinoin — so a 1-month interval is, in fact, generally considered acceptable. Topical steroids are NOT used for acne (they can cause steroid acne); the main acne organism is *Cutibacterium (Propionibacterium) acnes*, not S. epidermidis. Several options here are poorly worded — verify against the course material.

D4. Pemphigus vulgaris — autoantibody

An old patient with flaccid bullae on the trunk, a positive Nikolsky sign and oral mucosal involvement. Which autoantibody is implicated?

- a) Anti-collagen 7
- b) Anti-desmoglein 3
- c) Anti-BP180
- d) Anti-collagen 4

Answer: Anti-desmoglein 3.

Explanation: Pemphigus vulgaris (flaccid bullae, positive Nikolsky sign, prominent mucosal involvement) is caused by autoantibodies to **desmoglein 3** (and desmoglein 1) — desmosomal adhesion proteins. Anti-BP180/BP230 cause bullous pemphigoid (tense bullae); anti-collagen 7 causes epidermolysis bullosa acquisita.

D5. Skin cancer in transplant patients — squamous cell carcinoma

A kidney transplant patient develops a cancerous ulcer on the shin. Most likely skin cancer?

- a) Melanoma
- b) Basal cell carcinoma
- c) Squamous cell carcinoma
- d) Kaposi sarcoma
- e) Lymphoma

Answer: Squamous cell carcinoma.

Explanation: Chronic immunosuppression (as after organ transplantation) markedly increases the risk of cutaneous **squamous cell carcinoma**, which also tends to be more aggressive in transplant recipients. Sun exposure and chronic wounds/ulcers are additional risk factors.

D6. Erythema nodosum — most common cause

Most common cause of erythema nodosum?

- a) Sarcoidosis
- b) Tuberculosis
- c) IBD
- d) Streptococcal infection

Answer: Streptococcal infection.

Explanation: Erythema nodosum (tender, red nodules on the shins) is most commonly triggered by a preceding **streptococcal infection**. Sarcoidosis, TB, IBD, drugs and pregnancy are other recognized causes.

D7. Actinic keratosis — precancerous

Which is considered precancerous?

- a) Actinic keratosis
- b) Syringoma
- c) Skin tags
- d) Seborrheic keratosis

Answer: Actinic keratosis.

Explanation: Actinic (solar) keratosis is a sun-induced **precancerous** lesion that can progress to cutaneous squamous cell carcinoma. Syringomas, skin tags and seborrheic keratoses are benign.

D8. Psoriasis — systemic steroids / infantile presentation

Psoriasis — points: systemic steroids, can appear in infants.

- a) Systemic corticosteroids should be avoided in psoriasis (risk of rebound/pustular flare on withdrawal); psoriasis can occur in infants.

Answer: Systemic corticosteroids are generally avoided in psoriasis — abrupt withdrawal can trigger a severe rebound or pustular flare; psoriasis can present in infancy (e.g., napkin/diaper psoriasis).

Explanation: Although potent, systemic steroids are not standard psoriasis therapy because tapering off can precipitate a rebound flare, including generalized pustular psoriasis. Psoriasis can begin at any age, including infancy.

Reviewer note: The IM-2023 source listed this only as a brief item ("Psoriasis systemic steroids, appear in infants"); the original full question/options were not recorded — the teaching points are summarized above.

11. NEUROLOGY

N-1. Subarachnoid hemorrhage — first imaging [×2]

A patient with the worst headache of their life / suspected subarachnoid hemorrhage. Best next step / what to order?

- a) MRA
- b) CT with contrast
- c) CT without contrast
- d) MRI

Answer: CT head WITHOUT contrast.

Explanation: A non-contrast CT of the head is the first-line investigation for suspected subarachnoid hemorrhage — it rapidly detects acute blood. If the CT is negative but suspicion remains, a lumbar puncture (for xanthochromia) is the next step. Contrast is not needed to see acute blood.

N-2. Vascular (multi-infarct) dementia [×2]

An old male whose son reports a **stepwise** decline in cognitive function (or multiple dementia-causing infarcts). Most likely cause of dementia?

- a) Alzheimer disease
- b) Creutzfeldt-Jakob disease
- c) Vascular (multi-infarct) dementia

Answer: Vascular (multi-infarct) dementia.

Explanation: A **stepwise**, stuttering decline in cognition (as opposed to the gradual, continuous decline of Alzheimer disease) is characteristic of vascular dementia, caused by accumulating cerebrovascular infarcts.

N-3. Drug-induced Parkinsonism — treatment [×2]

(See Psychiatry P3 — this question also appears under Neurology.) A drug used in drug-induced Parkinsonism: levodopa vs an anticholinergic (procyclidine).

Answer: Withdraw the offending dopamine antagonist; use an anticholinergic (procyclidine/benztropine) for symptom control. Levodopa is generally ineffective.

Reviewer note: See P3 for the full discussion — exam keys conflicted between "levodopa" and "procyclidine."

N-4. Reducing thromboembolic stroke risk — exception [×2]

All of the following decrease thromboembolic stroke risk EXCEPT:

- a) Stopping smoking
- b) Hypertension
- c) Clopidogrel
- d) Warfarin
- e) Aspirin

Answer: Hypertension.

Explanation: Smoking cessation, antiplatelets (aspirin, clopidogrel) and anticoagulation (warfarin) all **reduce** stroke risk. **Hypertension** is a major **risk factor** that *increases* stroke risk — so it is the exception (it does not decrease risk).

N-5. Levodopa for drug-induced Parkinson — see P3

(Cross-reference: drug-induced Parkinsonism treatment is covered in Psychiatry P3 / Neurology N-3.)

N-6. Spinal cord lesion localization

A patient with loss of motor and sensory function in the upper and lower limbs, **face spared**, bilateral Babinski signs, and loss of bladder control. Which area is affected?

- a) Cervical spinal cord lesion
- b) Brain stem
- c) Cerebrum
- d) Lumbar lesion
- e) Peripheral lesion (NMJ)

Answer: Cervical spinal cord lesion.

Explanation: Involvement of **all four limbs** with **face sparing**, upper motor neuron signs (bilateral Babinski) and bladder dysfunction localizes to the **cervical spinal cord** (above the level supplying the arms, below the brainstem — so the face is spared).

N-7. Lacunar infarct — pure motor stroke

A hypertensive patient with weakness of the right lower face, hand and leg, with NO sensory findings. Most probable stroke?

- a) Right middle cerebral artery
- b) Left middle cerebral artery
- c) Lacunar infarct of the posterior limb of the internal capsule
- d) Subarachnoid hemorrhage

Answer: Lacunar infarct of the posterior limb of the internal capsule.

Explanation: A **pure motor** hemiparesis (face, arm and leg equally affected) with **no sensory, visual or cortical** signs is the classic lacunar syndrome — typically a small-vessel infarct in the posterior limb of the internal capsule, strongly associated with hypertension.

N-8. Wernicke encephalopathy — thiamine

An alcoholic patient with confusion, ataxia and nystagmus. Best next step?

- a) Thiamine
- b) Benzodiazepines
- c) Vitamin B12
- d) Vitamin B6 (pyridoxine)

Answer: Thiamine (vitamin B1).

Explanation: The triad of confusion, ataxia and ophthalmoplegia/nystagmus in an alcoholic is **Wernicke encephalopathy** — a thiamine deficiency emergency. Give IV thiamine **before** glucose (glucose administration before thiamine can precipitate or worsen Wernicke encephalopathy).

N-9. Cluster headache — wrong statement

Which is wrong about cluster headache?

- a) Associated with lacrimation and rhinorrhea
- b) Lasts 20–120 minutes
- c) More common in males
- d) Steroids can be used in management
- e) Sleep improves the symptoms

Answer: "Sleep improves the symptoms" is WRONG.

Explanation: Cluster headaches are severe unilateral periorbital headaches with ipsilateral autonomic features (lacrimation, rhinorrhea, ptosis), lasting roughly 15–180 minutes, more common in men, and often **occur during sleep** (waking the patient). Patients are typically restless/agitated. Sleep does not relieve them.

N-10. Bulbar disease — feature that does NOT belong

One of the following is NOT associated with bulbar disease:

- a) Absent gag reflex
- b) Dysphagia
- c) Emotional lability
- d) Tongue fasciculations

Answer: Emotional lability.

Explanation: **Bulbar** palsy is a **lower** motor neuron syndrome — flaccid weakness with an absent gag reflex, dysphagia and tongue fasciculations/wasting. **Emotional lability** (pseudobulbar affect) is a feature of **pseudobulbar** palsy, an *upper* motor neuron syndrome.

N-11. Juvenile myoclonic epilepsy — drug of choice

A patient with juvenile myoclonic seizures. Drug of choice?

- a) Valproic acid
- b) Carbamazepine
- c) Diazepam
- d) Topiramate

Answer: Valproic acid.

Explanation: Valproic acid is the most effective drug for juvenile myoclonic epilepsy (a generalized epilepsy). Note that carbamazepine can **worsen** myoclonic and absence seizures.

Reviewer note: Valproate is highly teratogenic — in women of childbearing potential, alternatives (e.g., levetiracetam, lamotrigine) are often preferred.

N-12. Multiple sclerosis — true statement

Which is true about MS?

- a) Relapsing-remitting is the most common presentation
- b) More common in tropical areas
- c) Affects the grey matter of the brain and spinal cord

Answer: Relapsing-remitting is the most common presentation.

Explanation: Relapsing-remitting MS is the most common form at presentation (~85%). MS is more common in **temperate** (higher-latitude) regions, not tropical ones, and primarily affects **white matter** (myelin), although grey matter involvement is increasingly recognized.

N-13. Drug NOT used in Parkinson disease

A drug that is NOT used in Parkinson disease treatment:

- a) Levodopa
- b) Entacapone
- c) Dopamine receptor **antagonist**
- d) COMT inhibitors
- e) Anticholinergics

Answer: Dopamine receptor antagonist.

Explanation: Parkinson disease treatment uses dopamine **agonists**, levodopa, COMT inhibitors (entacapone), MAO-B inhibitors and anticholinergics — all of which **increase** dopaminergic activity. A dopamine receptor **antagonist** would block dopamine and *worsen* Parkinsonism (indeed, antagonists cause drug-induced parkinsonism).

N-14. Essential tremor — true statement

What is true about essential tremor?

- a) Jaw tremor is common
- b) Treated with GABA
- c) Inheritance is autosomal recessive
- d) Beta blockers help decrease the tremor

Answer: Beta-blockers (e.g., propranolol) help decrease the tremor.

Explanation: Essential tremor is an action/postural tremor, typically of the hands, that responds to **propranolol** (and primidone). It is usually **autosomal dominant**, often improves transiently with alcohol, and jaw/voice tremor can occur but hand tremor predominates.

Reviewer note: The exam option "treated with GABA" is loosely worded — primidone (a GABAergic-acting drug) is used, but the cleanest correct answer is the beta-blocker response.

N-15. Gabapentin — mechanism of action

Mechanism of action of gabapentin?

- a) Norepinephrine
- b) Serotonin
- c) Prevents breakdown of neurotransmitters
- d) Acts on the GABA / calcium-channel system

Answer: It acts on the GABAergic / voltage-gated calcium-channel system.

Explanation: Despite its name, gabapentin does not bind GABA receptors directly — it binds the α2δ subunit of voltage-gated **calcium channels**, reducing excitatory neurotransmitter release. The exam groups its action with the GABA system.

N-16. Carotid artery dissection / Horner-type presentation

A 45-year-old with a sudden severe left-sided headache, nausea, somnolence; right-sided ptosis and a right pupil larger than the left. Most likely diagnosis?

- a) Carotid artery dissection
- b) Cavernous sinus thrombosis
- c) Cluster headache
- d) Posterior communicating artery aneurysm

Answer: Per the exam key: posterior communicating artery aneurysm.

Explanation: A painful **third nerve (oculomotor) palsy** — ptosis with a **dilated** ("blown") pupil — classically indicates a posterior communicating artery aneurysm compressing CN III (a surgical emergency, as it may herald rupture).

Reviewer note: The pupil findings determine the answer. A **dilated** pupil with ptosis = oculomotor (CN III) palsy → posterior communicating artery aneurysm (exam key). A **constricted** (miotic) pupil with ptosis = Horner syndrome → suggests **carotid artery dissection**. Re-read the stem carefully: "pupil bigger" points to a CN III palsy / PCom aneurysm.

N-17. Trigeminal neuralgia — treatment

Trigeminal neuralgia — treatment?

- a) Carbamazepine
- b) Ibuprofen
- c) Corticosteroid

Answer: Carbamazepine.

Explanation: Carbamazepine is the first-line treatment for trigeminal neuralgia (paroxysmal, severe, lancinating unilateral facial pain).

N-18. Lumbar puncture — contraindications

Lumbar puncture should be preceded (by neuroimaging) in all of the following EXCEPT:

- a) Meningismus / Kernig sign
- b) Focal neurological deficit
- c) Papilledema
- d) Known brain mass
- e) Decreased level of consciousness

Answer: Kernig sign (meningismus) — this does NOT require imaging before LP.

Explanation: Before a lumbar puncture, neuroimaging (CT) is needed when there is concern for raised intracranial pressure or a mass lesion: focal neurological deficits, papilledema, a known intracranial mass, or a significantly depressed level of consciousness. **Meningismus / a positive Kernig sign** is a sign of meningeal irritation and is itself an *indication* for LP — it does not mandate prior imaging.

N-19. Myasthenia gravis — workup

A patient with suspected myasthenia gravis — do all of the following EXCEPT:

- a) Calcium-channel antibodies
- b) Anti-acetylcholine receptor (AChR) antibodies
- c) Edrophonium (Tensilon) test

Answer: Calcium-channel antibodies.

Explanation: Myasthenia gravis is investigated with **anti-AChR antibodies** (and anti-MuSK), the edrophonium (Tensilon) test, and repetitive nerve stimulation/single-fiber EMG. **Voltage-gated calcium-channel antibodies** are the antibodies of **Lambert-Eaton myasthenic syndrome**, not myasthenia gravis.

N-20. Common peroneal nerve injury — foot drop

Loss of dorsiflexion and loss of sensation over the dorsum of the foot, with loss of big-toe dorsiflexion. Most commonly affected nerve?

- a) Common peroneal (fibular) nerve
- b) Tibial nerve
- c) Femoral nerve
- d) Sciatic nerve

Answer: Common peroneal (fibular) nerve.

Explanation: The common peroneal nerve, vulnerable as it wraps around the fibular head, supplies the dorsiflexors and evertors of the foot. Injury causes **foot drop** — loss of ankle/toe dorsiflexion and eversion — with sensory loss over the dorsum of the foot and lateral leg.

12. MISCELLANEOUS

This section collects questions that do not fit neatly into the 11 topic categories above — including a surgery question, immunology items, and general/internal-medicine questions.

M1. Breast cancer screening — who does NOT need a mammogram (Surgery)

Which of the following does NOT need mammogram screening?

- a) A 38-year-old asymptomatic woman at average risk
- b) A 42-year-old woman with a family history of breast cancer
- c) A 50-year-old woman with a family history of breast cancer
- d) A 60-year-old woman at average risk

Answer: A 38-year-old asymptomatic woman at average risk.

Explanation: Routine screening mammography for **average-risk** women begins at age 40 (some guidelines 45–50) and continues through about age 74. A 38-year-old at average risk with no symptoms is below the screening start age. Women with a significant family history start earlier, and a 60-year-old is within the routine screening window.

Reviewer note: This was a surgery question included in the 2020 paper. The key point: average-risk screening starts at ≥40; a strong family history prompts earlier screening (often 10 years before the youngest affected relative's age at diagnosis).

M2. Most common primary immunodeficiency

What is the most common (primary) immune deficiency?

- a) Common variable immunodeficiency (CVID)
- b) Severe combined immunodeficiency (SCID)
- c) Selective IgA deficiency
- d) Hyper-IgM syndrome

Answer: Selective IgA deficiency.

Explanation: Selective IgA deficiency is the most common primary immunodeficiency. Most affected individuals are asymptomatic; some have recurrent sinopulmonary or GI infections, and there is a risk of anaphylactic transfusion reactions to blood products containing IgA.

M3. Risk factor for duodenal adenocarcinoma

Which is a risk factor for duodenal (small-bowel) adenocarcinoma?

- a) Younger age
- b) Female sex
- c) Celiac disease
- d) GERD

Answer: Celiac disease.

Explanation: Celiac disease is a recognized risk factor for small-bowel adenocarcinoma (including duodenal) and for enteropathy-associated T-cell lymphoma. Other risk factors include Crohn's disease and hereditary polyposis syndromes (FAP, Peutz-Jeghers). Small-bowel adenocarcinoma is a disease of **older** adults, and GERD is unrelated.

Reviewer note: This item could also sit under Gastroenterology; it is placed here as a less commonly tested, "fringe" GI-oncology fact.

M4. Acute upper-extremity DVT in a young athlete

A young weightlifter develops sudden swelling, heaviness and bluish discoloration of the dominant arm after intense training. Most likely diagnosis?

- a) Paget-Schroetter syndrome (effort thrombosis of the axillary-subclavian vein)
- b) Cellulitis
- c) Lymphedema
- d) Superficial thrombophlebitis

Answer: Paget-Schroetter syndrome (effort-related axillary-subclavian vein thrombosis).

Explanation: Effort thrombosis (Paget-Schroetter syndrome) is a primary upper-extremity DVT occurring in young, active individuals after repetitive strenuous arm activity, usually related to thoracic outlet compression of the subclavian vein. It presents with acute arm swelling, heaviness and cyanotic discoloration.

Reviewer note: Included here as a general internal-medicine/vascular item; if a paper groups it with VTE, it can be read alongside the Respiratory PE/DVT questions.

M5. Health-screening / preventive-care principle

General preventive-care principle reflected across several exam items: screening tests are applied to **asymptomatic, average-risk** populations within defined age ranges; a **symptomatic** patient or one at **high risk** is investigated diagnostically rather than "screened."

Explanation: This recurring theme underlies several questions in this bank — colorectal cancer screening (G27), the mammography question (M1), and diabetes screening vs diagnosis (E11–E12). When a patient has symptoms or red-flag features, move directly to definitive diagnostic testing rather than a screening test.

END OF QUESTION BANK

Summary of contents

- 11 topic sections plus a Miscellaneous section, compiled from 6 exam papers.
- Duplicate questions across papers were merged, with a **[×N]** tag indicating how many papers a question appeared in.
- Within each topic, the most frequently repeated questions appear first.
- Where the original exam answer key appeared doubtful, a **Reviewer note** explains the issue and gives a corrected/▶ best-evidence answer.

Key reviewer corrections to be aware of (study these carefully):

1. **C36 / C35** — Routine combined ACE inhibitor + ARB therapy is NOT recommended (ONTARGET trial); some exam keys reflect an outdated course slide.
2. **R7** — CURB-65 "urea" is in mmol/L (>7 mmol/L ≈ >19–20 mg/dL); convert units before scoring.
3. **N19 (Nephrology)** — NSAID-induced nephrotic syndrome is classically minimal change disease (± interstitial nephritis); a key marking "FSGS" is questionable.
4. **N1 (Nephrology)** — Distal RTA vs diarrhea is decided by urine pH / urine anion gap; a urine pH of 5.8 actually argues toward diarrhea.
5. **P4** — Serotonin syndrome vs neuroleptic malignant syndrome: distinguish by neuromuscular findings (clonus/hyperreflexia vs lead-pipe rigidity); the MAOI+SSRI history is a strong serotonin-syndrome clue.
6. **P8** — ADHD is at least as strongly associated with autism spectrum disorder as OCD; the exam key (OCD) is uncertain.
7. **N16 (Neurology)** — A dilated pupil + ptosis = CN III palsy (posterior communicating artery aneurysm); a constricted pupil + ptosis = Horner syndrome (carotid dissection).
8. **H28** — Iron deficiency anemia in an older adult must be worked up as occult GI blood loss (e.g., colorectal cancer).
9. **R45 / R4** — GOLD has merged groups C and D into a single Group E; older papers still use the ABCD system.
10. **C28 (Nephrology edema)** — A raised JVP reflects raised central venous pressure; cirrhosis and nephrotic syndrome cause edema *without* a raised JVP — re-read the stem to identify the true exception.

Always cross-check answers against your current course material and standard textbooks — exam answer keys can contain errors, which is why reviewer notes are included throughout.