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100 HIGH-YIELD CARDIOLOGY MCQs

Board-Style Self-Assessment for Medical Students and Interns

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Disclaimer

This book is intended solely for educational and self-assessment purposes for medical students, interns, and healthcare professionals in training. Questions and explanations are aligned with mainstream American Heart Association (AHA), American College of Cardiology (ACC), and European Society of Cardiology (ESC) guidance current as of 2026.

This is a teaching and self-assessment tool, not a substitute for a structured cardiology curriculum, supervised clinical training, or the primary guideline documents. Clinical practice should always be guided by current institutional protocols and individualized patient assessment.

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Preface

Nothing consolidates learning like being tested on it. This collection of 100 questions is designed to do exactly that — to take everything a medical student or intern should know about cardiology and turn it into a rapid, honest self-check.

The questions are written in board-exam style: clinical vignettes and direct-knowledge stems with a single best answer among five options, each followed by a focused explanation of not just what the right answer is, but why the tempting wrong answers are wrong. That second part matters as much as the first — understanding why a distractor fails is often what prevents the same mistake on the next patient or the next exam.

The 100 questions span thirteen areas of cardiology: anatomy and physiology, hypertension, preventive cardiology and lipids, ECG and rhythm disorders, emergency cardiac care, acute coronary syndromes, heart failure, valvular disease, cardiomyopathies,

pericardial disease, congenital heart disease, infective endocarditis, and pharmacology — the same ground covered across this series' companion volumes on Emergency Cardiac Care, Drug Dosing, and the ECG Pocket Guide.

Work through them in order, by section, or at random. Time yourself if you're preparing for an exam. However you use it, my hope is that this question bank sharpens both your recall and your reasoning.

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How to Use This Book

- Each question presents a stem, five options (A-E), the correct answer, and a focused explanation.
- Try to answer each question yourself — ideally covering the answer and explanation — before checking.
- Read the explanation even when you get a question right; it often clarifies why the distractors are wrong, which is where deeper understanding comes from.
- Questions are grouped into 13 sections by topic; a full section map and a compact answer key appear in the appendices for quick self-scoring.
- Use this book alongside the companion volumes in this series — Synopsis of Emergency Cardiac Care, Handy Drugs and Dosing Reference in Cardiology, and the ECG Pocket Guide — for full explanations of concepts referenced only briefly here.

Section 1: Cardiac Anatomy and Physiology

Q1. A 58-year-old man presents to the emergency department with 45 minutes of crushing substernal chest pain radiating to the left arm. An ECG shows 3 mm ST-segment elevation in leads V1 through V4. Which coronary artery is most likely occluded?

A. Left circumflex artery B. Left main coronary artery C. Left anterior descending artery D. Posterior descending artery E. Right coronary artery

Correct Answer: C

Explanation: The LAD supplies the anteroseptal and anterior wall of the left ventricle and the interventricular septum, corresponding to ST elevation in the precordial leads V1-V4. The right coronary artery supplies the inferior wall (leads II, III, aVF), and the left circumflex supplies the lateral wall (leads I, aVL, V5-V6) — neither matches this ECG pattern. Left main occlusion typically produces more diffuse ST changes across multiple territories with hemodynamic compromise, not an isolated anteroseptal pattern.

Q2. In a patient with a right-dominant coronary circulation, present in approximately 85% of individuals, the posterior descending artery most commonly arises from which vessel?

A. First diagonal branch B. Right coronary artery C. Left anterior descending artery D. Left circumflex artery E. Left main coronary artery

Correct Answer: B

Explanation: Coronary dominance is defined by the origin of the posterior descending artery (PDA); in right-dominant circulation (~85% of people), the PDA arises from the RCA and supplies the inferior wall and posterior septum. In left-dominant circulation (the remainder), the PDA arises from the left circumflex instead. The LAD and its diagonal branches supply the anterior wall and septum and do not give rise to the PDA.

Q3. Which coronary vessel supplies the sinoatrial (SA) node in the majority of the population?

A. Left circumflex artery B. Right coronary artery C. Obtuse marginal artery D. Posterior descending artery E. Left anterior descending artery

Correct Answer: B

Explanation: The SA node is supplied by the RCA in approximately 60% of individuals (right-dominant pattern) and by the left circumflex artery in the remainder. This is why proximal RCA disease or occlusion can produce sinus node dysfunction, including sinus bradycardia or sinus arrest. The LAD supplies the anteroseptal LV and septum, not the SA node.

Q4. A 65-year-old man develops an acute inferior STEMI with ST elevation in leads II, III, and aVF. Over the next hour he becomes bradycardic with 2:1 atrioventricular block on telemetry. Ischemia of which structure, supplied by which vessel, best explains this new conduction abnormality?

A. Bundle of His, supplied by LAD septal perforators B. Purkinje network, supplied by the left circumflex artery C. SA node, supplied by the left circumflex artery D. Left bundle branch, supplied by the LAD E. AV node, supplied by the right coronary artery

Correct Answer: E

Explanation: The AV node is supplied by the RCA (via the posterior descending artery) in about 90% of people, which explains why inferior MI from RCA occlusion commonly causes AV block — usually at the level of the AV node, often transient and responsive to atropine. In contrast, anterior MI causing infranodal block (via LAD septal perforator ischemia to the His-Purkinje system) tends to produce wider-complex, less reversible block and carries a worse prognosis. The scenario describes an inferior, not anterior, MI.

Q5. Closure of which cardiac valves produces the first heart sound (S1)?

A. Mitral and tricuspid valves B. Aortic and pulmonic valves C. Mitral valve only D. Pulmonic and tricuspid valves E. Aortic valve only

Correct Answer: A

Explanation: S1 corresponds to closure of the mitral and tricuspid (atrioventricular) valves and marks the onset of ventricular systole. S2, by contrast, corresponds to closure of the aortic and pulmonic (semilunar) valves and marks the onset of diastole. Confusing which valve pair generates which sound is a common exam pitfall.

Q6. Physiologic splitting of the second heart sound (S2), which widens during inspiration, is best explained by which mechanism?

A. Increased venous return delaying pulmonic valve closure B. Increased intrathoracic pressure accelerating pulmonic valve closure C. Decreased pulmonary vascular resistance shortening right ventricular ejection D. Increased left ventricular afterload during inspiration E. Decreased venous return delaying aortic valve closure

Correct Answer: A

Explanation: Inspiration decreases intrathoracic pressure, augmenting venous return to the right heart and increasing right ventricular stroke volume and ejection time, which delays pulmonic valve closure (P2) relative to aortic valve closure (A2). This produces the normal, physiologic widening of the A2-P2 interval heard on inspiration. It is a normal finding and does not indicate underlying cardiac pathology.

Q7. A 72-year-old man with complete (third-degree) AV block is noted on examination to have intermittent, large-amplitude pulsations in the jugular venous pulse that occur irregularly and do not consistently precede the carotid upstroke. What best explains this finding?

A. Absent “x” descent due to cardiac tamponade B. Cannon “a” waves due to atrial contraction against a closed tricuspid valve C. Prominent “c” waves due to tricuspid valve bulging in early systole D. Prominent “y” descent due to constrictive pericarditis E. Large “v” waves due to tricuspid regurgitation

Correct Answer: B

Explanation: In complete heart block, atrial and ventricular contractions are dissociated; when the atrium happens to contract against a closed tricuspid valve (during ventricular systole), the resulting elevated right atrial pressure produces an intermittent, irregular “cannon a wave” in the JVP. Large “v” waves instead reflect regurgitant systolic flow into the right atrium in tricuspid regurgitation and would be regular, occurring with every beat rather than intermittently.

Q8. A patient with early, compensated heart failure is given a modest intravenous fluid bolus, and stroke volume increases as myocardial fibers are stretched by greater venous return. Which statement best describes this principle and its limitation?

A. This is the Frank-Starling mechanism; beyond a certain point on the curve, further increases in preload cause worsening congestion without additional gains in stroke volume B. This is the Frank-Starling mechanism; further increases in preload continue to proportionally increase contractility indefinitely, with no downside C. This is the Bowditch (treppe) effect; further fluid increases heart rate-dependent contractility D. This is a reflex tachycardic response; further fluid administration simply improves diastolic filling time E. This is the Anrep effect; further fluid increases afterload-mediated contractility

Correct Answer: A

Explanation: The Frank-Starling mechanism states that, within physiologic limits, increased preload stretches sarcomeres and increases contractile force and stroke volume. However, in a failing or overloaded ventricle, the curve flattens and then plateaus or descends, so further volume loading produces pulmonary and systemic congestion without further improving cardiac output — the physiologic rationale for diuresis in decompensated heart failure. The Anrep and Bowditch effects describe afterload- and heart-rate-dependent contractility changes, respectively, not the preload-stroke volume relationship described here.

Section 2: Hypertension

Q9. According to current ACC/AHA blood pressure staging, a reading of 128/76 mmHg (confirmed on repeat measurement) is classified as which category?

A. Elevated blood pressure B. Normal blood pressure C. Stage 1 hypertension D. Stage 2 hypertension E. Hypertensive urgency

Correct Answer: A

Explanation: Elevated blood pressure is defined as systolic 120-129 mmHg AND diastolic <80 mmHg, which fits this reading exactly. Normal blood pressure requires systolic <120 AND diastolic <80. Stage 1 hypertension requires systolic 130-139 OR diastolic 80-89, neither of which is met here.

Q10. A 50-year-old man at a routine office visit has a blood pressure of 136/84 mmHg, confirmed on a repeat measurement later in the same visit. He has no other significant findings. How should his blood pressure be classified?

A. Hypertensive emergency B. Stage 2 hypertension C. Elevated blood pressure D. Stage 1 hypertension E. Normal blood pressure

Correct Answer: D

Explanation: Stage 1 hypertension is defined as systolic 130-139 mmHg OR diastolic 80-89 mmHg; this patient's systolic (136) and diastolic (84) values both fall within that range. Elevated blood pressure requires a diastolic <80 mmHg, which is not the case here, and stage 2 hypertension requires systolic ≥ 140 or diastolic ≥ 90 , which he has not reached.

Q11. A patient presents to urgent care with a blood pressure of 210/130 mmHg and a severe headache. She is alert and oriented, has no papilledema on fundoscopic exam, no focal neurologic deficits, normal renal function on basic metabolic panel, and no chest pain or dyspnea. How should this presentation be classified and managed?

A. Hypertensive emergency; admit to the ICU for intravenous antihypertensive therapy B. Stage 2 hypertension; start a thiazide diuretic and follow up in one month C. Elevated blood pressure; reassurance and lifestyle counseling only D. Hypertensive urgency; reduce blood pressure gradually with oral agents over 24-48 hours, typically without hospital admission E. White coat hypertension; confirm with ambulatory blood pressure monitoring before treating

Correct Answer: D

Explanation: A blood pressure exceeding 180 systolic and/or 120 diastolic without evidence of acute end-organ damage (no papilledema, normal renal function, no focal neurologic deficits, no chest pain) is classified as hypertensive urgency, which is managed with gradual oral blood pressure reduction rather than IV therapy. Hypertensive emergency requires objective evidence of acute target-organ injury (e.g., encephalopathy, papilledema, acute kidney injury, acute coronary syndrome), which is absent here despite the severe headache and markedly elevated pressure.

Q12. Which of the following antihypertensive drug classes is generally NOT considered first-line therapy for uncomplicated essential hypertension in the absence of a compelling indication?

A. Beta-blockers B. Thiazide/thiazide-like diuretics C. ACE inhibitors D. Calcium channel blockers E. Angiotensin receptor blockers

Correct Answer: A

Explanation: Thiazide/thiazide-like diuretics, calcium channel blockers, and ACE inhibitors/ARBs are all considered first-line agents for uncomplicated hypertension. Beta-blockers are generally reserved for patients with a compelling indication, such as post-myocardial infarction, heart failure with reduced ejection fraction, or certain arrhythmias, rather than being used as routine first-line monotherapy.

Q13. A 45-year-old man has resistant hypertension despite three antihypertensive agents at adequate doses, including a diuretic. Labs reveal a serum potassium of 3.0 mEq/L that is not fully explained by his diuretic dose. What is the most likely underlying diagnosis, and what is the most appropriate next diagnostic step?

A. Primary hyperaldosteronism; plasma aldosterone-to-renin ratio B. Renal artery stenosis; renal artery duplex ultrasound C. Obstructive sleep apnea; polysomnography D. Cushing syndrome; low-dose dexamethasone suppression test E. Pheochromocytoma; plasma free metanephrines

Correct Answer: A

Explanation: Resistant or severe hypertension accompanied by unexplained hypokalemia is a classic clue for primary hyperaldosteronism (Conn syndrome), and the appropriate screening test is a plasma aldosterone-to-renin ratio. Pheochromocytoma typically presents with paroxysmal symptoms such as palpitations, headache, and diaphoresis rather than hypokalemia as the leading clue, and renal artery stenosis is more classically suggested by a renal artery bruit or a rise in creatinine after starting an ACE inhibitor/ARB.

Q14. A 38-year-old obese man with resistant hypertension is noted by his wife to snore loudly and have witnessed pauses in breathing during sleep; he reports significant daytime somnolence. What is the most likely secondary cause of his hypertension?

A. Coarctation of the aorta B. Obstructive sleep apnea C. Pheochromocytoma D. Renal artery stenosis E. Primary hyperaldosteronism

Correct Answer: B

Explanation: Loud snoring, witnessed apneas, obesity, and daytime somnolence are classic features of obstructive sleep apnea, which is the most common secondary cause of hypertension overall and should be confirmed with polysomnography. Pheochromocytoma and primary hyperaldosteronism have different characteristic clues (paroxysmal palpitations/diaphoresis, and hypokalemia, respectively) that are not described in this vignette.

Section 3: Preventive Cardiology and Lipid Management

Q15. For which of the following patients is statin therapy recommended regardless of a calculated 10-year ASCVD risk score?

A. A 60-year-old nonsmoker with a normal lipid panel B. A 50-year-old with mild intermittent claudication and a normal ankle-brachial index C. A 45-year-old with an LDL-C of 195 mg/dL D. A 30-year-old with borderline elevated blood pressure and normal lipids E. A 55-year-old with an LDL-C of 150 mg/dL and no other risk factors

Correct Answer: C

Explanation: An LDL-C ≥ 190 mg/dL (presumed familial hypercholesterolemia) is one of the risk-independent indications for statin therapy, along with established clinical ASCVD, diabetes mellitus age 40-75, and intermediate-risk patients after a risk discussion. An LDL-C of 150 mg/dL alone, without additional risk factors, does not meet an automatic threshold and instead requires risk-score-based decision-making.

Q16. A 62-year-old man is admitted with an ST-elevation myocardial infarction and undergoes successful percutaneous coronary intervention. He is started on a statin prior to discharge. What intensity of statin therapy and LDL-C reduction goal is most appropriate?

A. High-intensity statin, targeting $\geq 50\%$ LDL-C reduction B. Moderate-intensity statin, targeting 30-49% LDL-C reduction C. No statin is needed if his admission LDL-C was already below 100 mg/dL D. Fibrate monotherapy, targeting triglyceride reduction E. Low-intensity statin, targeting less than 30% LDL-C reduction

Correct Answer: A

Explanation: Patients with very-high-risk clinical ASCVD, such as a recent myocardial infarction, should be started on high-intensity statin therapy (e.g., atorvastatin 80 mg or rosuvastatin 20-40 mg), targeting at least a 50% reduction in LDL-C. Moderate-intensity statins, targeting a 30-49% reduction, are generally reserved for lower-risk primary prevention scenarios, and admission LDL-C level does not eliminate the indication for high-intensity statin therapy after ACS.

Q17. The patient from the previous question returns for follow-up six weeks after his STEMI. He has diabetes and a prior remote MI in addition to this recent event, placing him at very-high risk. He is adherent to maximally tolerated high-intensity atorvastatin, but his LDL-C remains 75 mg/dL. What is the most appropriate next step?

A. Continue the current statin alone and reassess in one year B. Add niacin to the statin regimen C. Switch from the statin to a fibrate D. Add ezetimibe, and if the LDL-C goal is still not achieved, add a PCSK9 inhibitor E. Discontinue the statin as a treatment failure and start a bile acid sequestrant

Correct Answer: D

Explanation: For very-high-risk ASCVD patients with additional high-risk features, an LDL-C goal of <55 mg/dL is a widely supported treatment target; when this is not achieved on maximally tolerated statin therapy, ezetimibe is added first, followed by a PCSK9 inhibitor if the goal remains unmet. Discontinuing an effective, well-tolerated statin or substituting fibrate/niacin/bile acid sequestrant would abandon proven LDL-lowering therapy in favor of agents that are not first-line for further LDL reduction in this setting.

Q18. A 22-year-old man is found to have an LDL-C of 310 mg/dL on routine screening. Examination reveals thickened, nodular swelling over the Achilles tendons bilaterally and a grayish ring around the corneal periphery. His father suffered a myocardial infarction at age 42. What is the most likely diagnosis and underlying defect?

A. Metabolic syndrome B. Secondary hypercholesterolemia due to hypothyroidism C. Familial hypercholesterolemia, most commonly due to an LDL receptor mutation D. Type 2 diabetes-related dyslipidemia E. Familial hypertriglyceridemia due to lipoprotein lipase deficiency

Correct Answer: C

Explanation: This presentation is classic for familial hypercholesterolemia: markedly elevated LDL-C (often >190 mg/dL), tendon xanthomas, corneal arcus before age 45, and a family history of premature ASCVD. FH is autosomal dominant and most commonly caused by mutations in the LDL receptor gene. Hypertriglyceridemia from lipoprotein lipase deficiency instead presents with eruptive xanthomas and recurrent pancreatitis, not tendon xanthomas with isolated severe LDL elevation.

Q19. Which of the following is NOT a variable directly incorporated into the pooled cohort equations used to estimate 10-year ASCVD risk?

A. Diabetes and smoking status B. High-sensitivity C-reactive protein level C. Age and sex D. Systolic blood pressure and treatment status E. Total and HDL cholesterol

Correct Answer: B

Explanation: The pooled cohort equations incorporate age, sex, total and HDL cholesterol, systolic blood pressure and treatment status, diabetes status, and smoking status. High-sensitivity CRP is not part of the core pooled cohort equation calculation; it is instead considered a risk-enhancing factor that clinicians may use to help refine treatment decisions in select intermediate-risk patients, similar to a coronary artery calcium score.

Q20. A 45-year-old current smoker with hypertension asks his physician which single lifestyle change would most rapidly and substantially lower his cardiovascular risk. What should the physician recommend?

A. Adding daily aspirin B. Losing 5% of body weight C. Smoking cessation D. Starting a statin E. Starting an ACE inhibitor

Correct Answer: C

Explanation: Smoking cessation produces a rapid and substantial reduction in cardiovascular risk and is considered one of the single most impactful preventive interventions available, with benefit accruing quickly after quitting. While statins and antihypertensive therapy are important and appropriate, and aspirin is no longer routinely recommended for primary prevention in most patients given bleeding risk, none matches the speed and magnitude of risk reduction achieved by stopping smoking. ## Section 4: ECG Basics, Rhythm, and Conduction Disorders — Q21-Q34

Q21. A 45-year-old woman is started on a new antiarrhythmic medication for symptomatic palpitations. A follow-up ECG obtained one week later shows a corrected QT interval (QTc) of 505 ms. Which of the following statements about this finding is correct?

A. This QTc value falls in the high-risk range for torsades de pointes, and the offending drug should be discontinued B. A QTc <480 ms is considered prolonged in women, so this value is not concerning C. QTc prolongation is clinically significant only if the PR interval is also prolonged D. QTc prolongation thresholds apply only to men, not women E. A QTc of 505 ms is a normal finding in the setting of sinus tachycardia

Correct Answer: A

Explanation: In women, a QTc ≥ 480 ms is considered prolonged, and a QTc > 500 ms places the patient in the high-risk category for torsades de pointes regardless of sex; the drug should be stopped and QT-prolonging risk factors corrected. Option B misstates the threshold, since 480 ms itself is already abnormal, not merely “not concerning.” Option D is incorrect because QTc thresholds are sex-specific (≥ 470 ms in men, ≥ 480 ms in women), not male-only. Option E is wrong because QTc is, by definition, already corrected for heart rate.

Q22. A 78-year-old woman with a history of hypertension presents with palpitations. Her pulse is irregularly irregular at 110/min. An ECG shows no discernible discrete P waves, with an irregularly irregular ventricular rhythm. Which rhythm is most consistent with these findings?

A. Sinus arrhythmia B. Atrial fibrillation C. Frequent premature atrial contractions D. Atrial flutter with variable block E. Multifocal atrial tachycardia

Correct Answer: B

Explanation: Atrial fibrillation is characterized by the absence of discrete, organized P waves and an irregularly irregular R-R interval, exactly as described. Multifocal atrial tachycardia is a tempting distractor because it is also irregular, but it is defined by the presence of at least three distinct P wave morphologies (not an absence of P waves), typically seen in patients with severe pulmonary disease. Atrial flutter with variable block

can produce an irregular ventricular response but is distinguished by organized sawtooth flutter waves rather than a complete absence of atrial activity.

Q23. A 58-year-old man presents with palpitations. His heart rate is regular at 150/min. The ECG shows a regular narrow-complex tachycardia with sawtooth-shaped atrial deflections best seen in leads II, III, and aVF, occurring at approximately 300/min. Which of the following best describes this rhythm?

A. Multifocal atrial tachycardia B. Atrial fibrillation C. Atrial flutter with 2:1 AV conduction D. Sinus tachycardia with first-degree AV block E. AV nodal reentrant tachycardia

Correct Answer: C

Explanation: Atrial flutter typically has an atrial rate of 250-350/min with classic sawtooth flutter waves best seen in the inferior leads, and 2:1 AV conduction produces a characteristic ventricular rate of approximately 150/min, matching this vignette precisely. Atrial fibrillation is excluded because it lacks organized sawtooth flutter waves and instead shows an irregularly irregular rhythm. AVNRT is excluded because it does not produce sawtooth flutter waves; it typically shows P waves hidden in or near the QRS complex.

Q24. A 24-year-old woman with no significant medical history presents to the emergency department after a sudden onset of palpitations and lightheadedness while at work. Heart rate is 190/min. ECG shows a regular, narrow-complex tachycardia with P waves not clearly visible, seemingly buried within the QRS complexes. Which of the following is the most likely diagnosis?

A. Atrial fibrillation B. AV nodal reentrant tachycardia (AVNRT) C. Atrial flutter D. Multifocal atrial tachycardia E. Sinus tachycardia

Correct Answer: B

Explanation: AVNRT is the most common cause of paroxysmal SVT and classically presents with an abrupt onset of a narrow, regular tachycardia in a young, otherwise healthy patient, with P waves often hidden within or immediately adjacent to the QRS complex due to near-simultaneous atrial and ventricular activation. Atrial flutter is excluded by the absence of sawtooth flutter waves and the very rapid rate of 190/min, which is atypical for flutter with fixed conduction. Sinus tachycardia is excluded because of the abrupt, paroxysmal onset and the absence of visible upright P waves preceding each QRS.

Q25. A 30-year-old man with a known history of Wolff-Parkinson-White pattern on prior ECGs presents with palpitations and lightheadedness. He is hemodynamically stable. ECG now shows an irregular wide-complex tachycardia at a rate of 210/min with beat-to-beat variation in QRS morphology. Which of the following medications is contraindicated in this patient because it could accelerate conduction down the accessory pathway and precipitate ventricular fibrillation?

A. IV adenosine B. IV magnesium sulfate C. Procainamide D. Synchronized cardioversion E. Observation with continuous monitoring

Correct Answer: A

Explanation: This ECG pattern is consistent with pre-excited atrial fibrillation conducting over an accessory pathway. AV nodal blocking agents such as adenosine, beta-blockers, calcium channel blockers, and digoxin must be avoided in this setting because blocking the AV node can preferentially shunt conduction down the accessory pathway, potentially accelerating the ventricular rate and precipitating ventricular fibrillation. Procainamide is the preferred pharmacologic agent in a stable patient because it slows conduction through the accessory pathway, and synchronized cardioversion is appropriate if the patient becomes unstable.

Q26. A 68-year-old man with a history of a prior myocardial infarction presents with palpitations and hypotension. ECG shows a regular wide-complex tachycardia at 160/min. Careful review reveals P waves marching through the QRS complexes independently of the ventricular rhythm, along with an occasional narrow “fusion” beat. Which of the following is the most likely diagnosis?

A. Torsades de pointes B. Sinus tachycardia with left bundle branch block C. Atrial fibrillation with pre-excitation D. Supraventricular tachycardia with aberrant conduction E. Monomorphic ventricular tachycardia

Correct Answer: E

Explanation: AV dissociation and fusion beats are classic findings that strongly favor ventricular tachycardia over SVT with aberrancy, especially in a patient with structural heart disease from a prior infarction — a population at high risk for scar-related monomorphic VT. SVT with aberrancy is a common distractor for wide-complex tachycardia, but the presence of AV dissociation and fusion beats argues against it, as SVT with aberrancy typically shows a 1:1 AV relationship. Torsades de pointes is excluded because it is polymorphic (a twisting QRS axis), not a fixed monomorphic wide-complex morphology.

Q27. A 55-year-old woman on multiple QT-prolonging medications develops palpitations and near-syncope. The monitor shows a polymorphic wide-complex tachycardia with a QRS axis that appears to twist around the isoelectric baseline. She has a palpable pulse and a blood pressure of 88/54 mmHg. Which of the following is the most appropriate initial pharmacologic treatment?

A. IV lidocaine B. IV amiodarone C. Oral beta-blocker D. IV magnesium sulfate E. IV calcium gluconate

Correct Answer: D

Explanation: This presentation describes torsades de pointes, a polymorphic VT associated with QT prolongation. IV magnesium sulfate 1-2 g is the treatment of choice and should be given regardless of the patient’s measured serum magnesium level. Amiodarone is a QT-prolonging drug itself and can worsen torsades, making it an inappropriate choice here. Lidocaine is not the first-line agent for torsades, though it is sometimes used for refractory monomorphic VT.

Q28. A telemetry strip shows progressive lengthening of the PR interval over several beats, culminating in a non-conducted P wave with a dropped QRS complex, after which the cycle repeats, producing a pattern of “group beating.” Which of the following best describes this rhythm?

A. Sinus arrhythmia with premature atrial contractions B. Mobitz type I (Wenckebach) second-degree AV block C. Mobitz type II second-degree AV block D. First-degree AV block E. Third-degree (complete) AV block

Correct Answer: B

Explanation: Progressive PR interval lengthening followed by a dropped beat, producing “group beating,” is the hallmark of Mobitz type I (Wenckebach) block, which occurs at the level of the AV node and is usually benign. Mobitz type II is excluded because it is characterized by a constant PR interval with sudden, unpredictable dropped beats rather than progressive lengthening. First-degree AV block is excluded because it involves a uniformly prolonged PR interval (>200 ms) with every P wave conducting, not intermittent dropped beats.

Q29. A 70-year-old man presents with recurrent syncope. His ECG shows a constant PR interval on conducted beats, but intermittently a P wave fails to conduct and no QRS follows, without any preceding PR prolongation. Which of the following is the most appropriate next step in management?

A. Observation alone, as this rhythm is benign B. Beta-blocker initiation C. Permanent pacemaker implantation D. IV atropine as definitive therapy E. Reassurance and outpatient follow-up

Correct Answer: C

Explanation: A constant PR interval with sudden, unheralded dropped QRS complexes describes Mobitz type II block, which reflects infranodal (His-Purkinje) conduction disease and carries a high risk of progression to complete heart block. Permanent pacemaker implantation is generally indicated, especially in a symptomatic patient with syncope. Unlike Mobitz I, Mobitz II is not benign and should not be managed with reassurance alone; atropine acts primarily at the AV node and is unreliable and not considered definitive therapy for infranodal block.

Q30. An ECG obtained in an elderly man with fatigue and dizziness shows P waves occurring regularly at a rate of 90/min and QRS complexes occurring regularly at a rate of 35/min, with no consistent relationship between the P waves and the QRS complexes. Which of the following best describes this finding?

A. Mobitz type II second-degree AV block B. Mobitz type I second-degree AV block C. Accelerated junctional rhythm D. Third-degree (complete) AV block E. AV nodal reentrant tachycardia

Correct Answer: D

Explanation: Complete dissociation between an independent, faster atrial rhythm and a slower, regular ventricular escape rhythm is diagnostic of third-degree (complete) AV block, in which no atrial impulses are conducted to the ventricles. This is distinguished from Mobitz I and II, which are forms of second-degree block in which some P waves do conduct to the ventricles. Complete heart block generally requires pacemaker implantation given the risk of hemodynamic compromise and asystole.

Q31. Which of the following ECG findings is characteristic of right bundle branch block (RBBB)?

A. Progressive PR prolongation followed by a dropped QRS complex B. Sawtooth-shaped deflections in the inferior leads C. A broad, monomorphic R wave in leads I, V5, and V6 with a broad S wave in V1 D. An RSR' ("rabbit ears") pattern in V1 with a wide, slurred S wave in leads I and V6 E. A short PR interval with a delta wave slurring the upstroke of the QRS

Correct Answer: D

Explanation: RBBB classically produces an RSR' pattern ("rabbit ears") in V1 along with a wide, slurred S wave in the lateral leads I and V6, reflecting delayed activation of the right ventricle. Option C describes left bundle branch block instead, which shows a broad monomorphic R wave in the lateral leads and a broad S wave in V1 — essentially the opposite pattern. Option E describes the WPW pattern (short PR, delta wave), not a bundle branch block.

Q32. A 62-year-old man with end-stage renal disease misses two consecutive dialysis sessions and presents with weakness. His potassium level returns at 7.8 mEq/L. Which of the following correctly describes the expected progression of ECG changes as hyperkalemia worsens?

A. P wave flattening → sine wave pattern → peaked T waves → QRS widening B. QRS widening → peaked T waves → PR prolongation → sine wave pattern C. Peaked T waves → PR prolongation/P wave flattening → QRS widening → sine wave pattern D. Sine wave pattern → peaked T waves → QRS widening → PR prolongation E. PR shortening → peaked T waves → ST elevation → QRS narrowing

Correct Answer: C

Explanation: The classic progression of hyperkalemic ECG changes begins with peaked, narrow-based T waves, followed by PR interval prolongation and flattening/loss of the P wave, then progressive QRS widening, and finally a pre-terminal sine wave pattern as the QRS and T wave merge. This sequence reflects the escalating effect of hyperkalemia on cardiac membrane excitability and conduction velocity. The other answer choices scramble this sequence in ways that do not reflect the actual pathophysiologic progression.

Q33. A 74-year-old woman on chronic digoxin therapy for atrial fibrillation presents with nausea, vomiting, and visual halos around lights. The ECG shows a wide-complex tachycardia in which the QRS axis alternates by 180 degrees from beat to beat. Which of the following is the most likely diagnosis?

A. Torsades de pointes B. Accelerated idioventricular rhythm C. Monomorphic ventricular tachycardia D. Digoxin effect E. Digoxin toxicity with bidirectional ventricular tachycardia

Correct Answer: E

Explanation: Bidirectional VT, in which the QRS axis alternates from beat to beat, is a highly specific finding for digoxin toxicity, and the accompanying nausea, vomiting, and visual disturbances (xanthopsia/halos) further support this diagnosis. “Digoxin effect” is a distinct, benign finding on therapeutic digoxin consisting of scooped/sagging ST depression and a shortened QT interval — not a sign of toxicity, and it does not cause a tachyarrhythmia. Torsades de pointes is polymorphic with a continuously twisting axis rather than a fixed alternating bidirectional pattern, and it is not specifically associated with digoxin.

Q34. A 66-year-old man with a known chronic left bundle branch block presents with acute chest pain. His ECG shows the baseline LBBB pattern, but in a lead with a predominantly positive (upright) QRS complex, there is 2 mm of ST-segment elevation in the same direction as the QRS. Which of the following best describes this finding?

A. This finding indicates digoxin effect superimposed on LBBB B. This is an expected discordant ST change of LBBB and is not diagnostic of ischemia C. This finding is a normal variant seen in most patients with LBBB D. This finding meets modified Sgarbossa criteria (concordant ST elevation) and is highly specific for acute STEMI E. This finding is diagnostic of Wellens syndrome

Correct Answer: D

Explanation: Modified Sgarbossa criteria are used to diagnose STEMI in the presence of LBBB (or a paced rhythm) and include concordant ST elevation ≥ 1 mm in any lead (ST elevation in the same direction as the QRS), which is highly specific for acute infarction. In LBBB, ST segments are normally discordant (opposite in direction) to the QRS as a baseline finding, so concordant elevation is abnormal, ruling out option B. Wellens syndrome refers to biphasic/deeply inverted T waves in V2-V3 in a pain-free interval and is unrelated to LBBB or concordant ST elevation.

Section 5: Emergency Cardiac Care and Resuscitation — Q35-Q40

Q35. During adult CPR performed by a single rescuer without an advanced airway in place, which of the following describes the correct chest compression technique?

A. Rate of 120-140/min, depth of 3 inches, with a compression-to-ventilation ratio of 30:2 B. Rate of 100-120/min, depth of 2-2.4 inches, with continuous compressions and no pauses for ventilation C. Rate of 100-120/min, depth of 1-1.5 inches, with a compression-to-ventilation ratio of 15:2 D. Rate of 80-100/min, depth of 1-1.5 inches, with a compression-to-ventilation ratio of 15:2 E. Rate of 100-120/min, depth of 2-2.4 inches (5-6 cm), with a compression-to-ventilation ratio of 30:2

Correct Answer: E

Explanation: Current adult CPR guidelines recommend a compression rate of 100-120/min, a compression depth of 2-2.4 inches (5-6 cm) with full chest recoil, and a compression-to-ventilation ratio of 30:2 for a single rescuer without an advanced airway. Continuous compressions without pauses for ventilation (option B) apply only once an advanced airway (e.g., endotracheal tube) is in place, at which point ventilations are delivered asynchronously. Depths less than 2 inches (options B and E) are inadequate and associated with worse outcomes.

Q36. A patient collapses and is found unresponsive and pulseless. The cardiac monitor shows ventricular fibrillation. After activating the emergency response system and beginning high-quality CPR, which of the following is the most appropriate next step?

A. Administer IV atropine 1 mg B. Administer synchronized cardioversion at 50 J C. Continue CPR only, as defibrillation is not indicated in VF D. Administer IV amiodarone 300 mg before defibrillation E. Administer an unsynchronized shock at 120-200 J (biphasic)

Correct Answer: E

Explanation: Ventricular fibrillation and pulseless VT are treated with immediate unsynchronized defibrillation, typically at 120-200 J for a biphasic defibrillator, as early defibrillation is the single most important intervention for improving survival in these shockable rhythms. Synchronized cardioversion is reserved for organized rhythms with a discernible QRS (e.g., unstable SVT or VT with a pulse) and is not used for VF, which lacks an organized QRS to synchronize to. Amiodarone is reserved for refractory VF/pVT after defibrillation and epinephrine have already been administered, not as an initial substitute for defibrillation.

Q37. A patient remains in pulseless ventricular tachycardia despite an initial defibrillation attempt and ongoing high-quality CPR. IV access has just been obtained. Which of the following is the most appropriate epinephrine dosing regimen for this cardiac arrest?

A. 1 mg IM every 5 minutes B. 0.1 mg IV/IO every 1-2 minutes C. 10 mg IV/IO every 3-5 minutes D. 1 mg IV/IO every 3-5 minutes E. 1 mg IV/IO as a single one-time dose only

Correct Answer: D

Explanation: During cardiac arrest, epinephrine is dosed at 1 mg IV/IO every 3-5 minutes, repeated throughout the resuscitation as needed. Option B reflects an inappropriately low dose and interval, and option C reflects a tenfold overdose, which is not part of standard adult ACLS dosing. Epinephrine is not a one-time dose (option E); it is repeated at regular intervals throughout the arrest as long as resuscitation continues.

Q38. A patient in ventricular fibrillation has already received three defibrillation attempts, ongoing CPR, and epinephrine, but remains in VF ("refractory VF"). Which of the following represents the correct first-dose antiarrhythmic therapy at this point?

A. Lidocaine 300 mg IV/IO as a single dose B. Amiodarone 300 mg IV/IO every 3-5 minutes as needed C. Amiodarone 300 mg IV/IO, followed by a second dose of 150 mg if needed D.

Amiodarone 150 mg IV/IO, followed by a second dose of 300 mg if needed E. Magnesium sulfate 300 mg IV/IO as first-line therapy

Correct Answer: C

Explanation: For refractory VF/pulseless VT, amiodarone is dosed as an initial 300 mg IV/IO bolus, followed by a second dose of 150 mg IV/IO if the arrhythmia persists — not the reverse order described in option D. Amiodarone is not re-dosed every 3-5 minutes like epinephrine (option B); it is given as these two discrete doses. Magnesium sulfate is used specifically for torsades de pointes or suspected hypomagnesemia, not as first-line therapy for refractory VF in general.

Q39. A 60-year-old man develops sudden hypotension, jugular venous distension, and muffled heart sounds three days after a percutaneous coronary intervention complicated by a coronary perforation. Blood pressure is 78/50 mmHg, and an inspiratory systolic blood pressure drop of 18 mmHg is noted. Which of the following is the most appropriate definitive treatment?

A. Pericardiocentesis B. Immediate needle decompression of the chest C. Emergent synchronized cardioversion D. IV fluid resuscitation alone, with no further intervention E. High-dose IV diuretics

Correct Answer: A

Explanation: This presentation describes cardiac tamponade, evidenced by Beck's triad (hypotension, jugular venous distension, muffled heart sounds) and pulsus paradoxus >10 mmHg, in a patient with a clear precipitating cause (coronary perforation causing pericardial hemorrhage). Pericardiocentesis is the definitive treatment to relieve the pericardial pressure and restore cardiac filling. Needle decompression of the chest is the treatment for tension pneumothorax, a different reversible cause of shock/arrest that presents with absent breath sounds and tracheal deviation, not muffled heart sounds and JVD.

Q40. A 55-year-old man presents to a rural hospital without on-site PCI capability with 45 minutes of ongoing chest pain and ST elevation in leads II, III, and aVF. The nearest PCI-capable center is a 3-hour ground transport away, meaning first-medical-contact-to-device time would far exceed 120 minutes. Which of the following is the most appropriate reperfusion strategy?

A. Observation and serial troponins before deciding on reperfusion B. Immediate transfer for primary PCI regardless of transport time C. Fibrinolytic therapy with a door-to-needle goal of ≤ 30 minutes D. Oral antiplatelet therapy alone, deferring reperfusion until symptoms resolve E. Immediate coronary artery bypass grafting

Correct Answer: C

Explanation: When timely primary PCI cannot be achieved within 120 minutes of first medical contact, fibrinolytic therapy should be administered promptly, with a goal door-to-needle time of ≤ 30 minutes, rather than delaying reperfusion for a prolonged transfer.

Option B is incorrect because forcing a 3-hour transfer would far exceed the acceptable window and delay reperfusion, worsening myocardial salvage. Observation with serial troponins (option A) inappropriately delays reperfusion in a patient who already meets ECG criteria for STEMI and needs immediate treatment. ## Section 6: Acute Coronary Syndromes and Ischemic Heart Disease — Q41-Q52 (12 questions)

Q41. A 45-year-old man presents to the emergency department with acute-onset crushing chest pain. His ECG shows ST-segment elevation isolated to leads V2 and V3. Per standard STEMI ECG diagnostic criteria, what is the minimum ST-segment elevation required in leads V2-V3 to diagnose STEMI in this patient?

A. ≥ 1.5 mm B. ≥ 2 mm C. ≥ 2.5 mm D. ≥ 1 mm E. ≥ 3 mm

Correct Answer: B

Explanation: For men aged ≥ 40 years, STEMI criteria require ≥ 2 mm of ST-segment elevation in leads V2-V3 due to the higher baseline J-point elevation seen in this demographic. The ≥ 2.5 mm threshold applies to men < 40 years (who have higher normal early repolarization voltages), while ≥ 1.5 mm applies to women. The ≥ 1 mm threshold applies to limb leads and other contiguous chest leads (V4-V6), not V2-V3.

Q42. A 62-year-old man presents with substernal chest pain radiating to the jaw. ECG demonstrates ST-segment elevation in leads II, III, and aVF with reciprocal ST depression in I and aVL. Occlusion of which coronary artery is the most likely cause?

A. First diagonal branch B. Left circumflex artery C. Left anterior descending artery D. Right coronary artery E. Left main coronary artery

Correct Answer: D

Explanation: ST elevation localized to the inferior leads (II, III, aVF) classically corresponds to occlusion of the right coronary artery (RCA), which supplies the inferior wall in most individuals (right-dominant circulation). The LAD supplies the anterior/anteroseptal territory (V1-V4), and the LCx supplies the lateral wall (I, aVL, V5-V6). Left main occlusion typically produces diffuse ST changes with ST elevation in aVR, not an isolated inferior pattern.

Q43. A 58-year-old man presents with an acute inferior STEMI. He is given sublingual nitroglycerin for ongoing chest pain and immediately becomes hypotensive to 78/50 mmHg with clear lung fields. Right-sided ECG leads are obtained and reveal ST-segment elevation in V4R. Which of the following is the most appropriate immediate management step?

A. Administer IV metoprolol B. Administer IV morphine C. Administer another dose of sublingual nitroglycerin D. Administer IV furosemide E. Administer an IV normal saline bolus

Correct Answer: E

Explanation: ST elevation in V4R confirms right ventricular (RV) infarction, which is preload-dependent because the infarcted RV cannot generate sufficient output without adequate filling pressure. IV fluid boluses restore preload and improve cardiac output and blood pressure. Nitrates, diuretics, and morphine are all preload-reducing agents that can precipitate profound hypotension in RV infarction and should be avoided; beta-blockers are also relatively contraindicated given the hemodynamic instability.

Q44. Which statement best describes the most significant recent structural change in the major U.S. multisociety guideline for acute coronary syndromes?

A. The 2025 ACC/AHA/ACEP/NAEMSP/SCAI guideline merged STEMI and NSTEMI/unstable angina into a single unified Acute Coronary Syndromes guideline B. Fibrinolytic therapy has been removed from all guideline recommendations C. STEMI and NSTEMI/unstable angina now have entirely separate, unrelated guideline documents for the first time D. Guidelines now recommend medical therapy alone for all ACS presentations, eliminating routine PCI E. The guideline eliminated troponin testing in favor of ECG criteria alone

Correct Answer: A

Explanation: In 2025, the ACC/AHA, along with ACEP, NAEMSP, and SCAI, consolidated the previously separate STEMI and NSTEMI/unstable angina guidelines into one unified “Acute Coronary Syndromes” guideline document, reflecting the shared pathophysiology and overlapping management pathways across the ACS spectrum. Fibrinolytic therapy remains a guideline-endorsed option when timely primary PCI is unavailable. Troponin testing and risk stratification remain central to ACS diagnosis and management, not eliminated.

Q45. A patient develops STEMI symptoms at a rural hospital without on-site PCI capability. The nearest PCI-capable center is far enough away that the anticipated first-medical-contact-to-device time will exceed 120 minutes. What is the most appropriate next step?

A. Transfer for primary PCI regardless of the delay, since PCI is always superior B. Give anticoagulation alone and defer reperfusion therapy entirely C. Discharge home with outpatient cardiology follow-up D. Administer fibrinolytic therapy with a door-to-needle time goal of ≤ 30 minutes, then arrange transfer E. Observe with serial ECGs and troponins before deciding on reperfusion strategy

Correct Answer: D

Explanation: When primary PCI cannot be performed within 120 minutes of first medical contact, fibrinolytic therapy should be administered promptly, with a door-to-needle target of ≤ 30 minutes, followed by transfer to a PCI-capable center. Primary PCI is preferred when it can be achieved within 90 minutes (or 120 minutes with transfer), but excessive delay negates its mortality benefit compared to timely fibrinolysis. Withholding reperfusion therapy while awaiting further workup would result in unacceptable delay and increased myocardial loss.

Q46. A 38-year-old woman presents with recurrent episodes of chest pain that occur at rest, often waking her at night. During an episode captured on telemetry, transient ST-

segment elevation is noted that resolves completely with resolution of pain. Coronary angiography reveals no significant fixed stenosis. Which of the following is the most appropriate long-term pharmacologic treatment, and which class of drug should be avoided?

A. ACE inhibitor; avoid nitrates B. Calcium channel blocker; avoid nonselective beta-blockers C. Loop diuretic; avoid calcium channel blockers D. Nonselective beta-blocker; avoid calcium channel blockers E. High-intensity statin alone; avoid nitrates

Correct Answer: B

Explanation: This presentation is classic for Prinzmetal (variant) angina caused by coronary vasospasm, typically occurring at rest or nocturnally with transient ST elevation and near-normal coronaries on angiography. Calcium channel blockers, often combined with nitrates, are first-line therapy because they relax vascular smooth muscle and prevent spasm. Nonselective beta-blockers should be avoided because unopposed alpha-mediated vasoconstriction can worsen coronary vasospasm.

Q47. A 68-year-old woman presents with NSTEMI confirmed by a rise in troponin and dynamic ST depressions. Despite aspirin, a P2Y12 inhibitor, anticoagulation, and nitrates, she continues to have refractory chest pain and develops hypotension with cool, mottled extremities. What is the recommended timing for invasive coronary angiography in this very-high-risk NSTEMI-ACS presentation?

A. Elective outpatient angiography in 2-4 weeks B. Immediate, within 2 hours C. Delayed, 25-72 hours, after serial troponins D. Angiography is not indicated; continue medical management only E. Early, within 24 hours

Correct Answer: B

Explanation: Patients with NSTEMI-ACS who have very-high-risk features such as hemodynamic instability/cardiogenic shock, refractory angina despite optimal medical therapy, or life-threatening arrhythmias warrant an immediate invasive strategy within 2 hours. High-risk but hemodynamically stable patients (e.g., GRACE score indicating elevated risk, dynamic troponin changes) generally undergo early angiography within 24 hours, while intermediate/low-risk patients with negative serial troponins may have delayed or selectively invasive management. Continuing medical therapy alone would be inappropriate given her refractory symptoms and shock physiology.

Q48. A 50-year-old man presents with acute chest pain. ECG shows ST-segment elevation in leads V1 and V2. This pattern most strongly suggests occlusion of which coronary artery segment?

A. Proximal left anterior descending artery B. Obtuse marginal branch C. Right coronary artery D. Posterior descending artery E. Left circumflex artery

Correct Answer: A

Explanation: ST elevation localized to V1-V2 (anteroseptal leads) reflects ischemia of the interventricular septum, which is supplied by the proximal left anterior descending (LAD)

artery and its septal perforator branches. Occlusion further down the LAD typically produces more extensive anterior changes involving V3-V4. The RCA and LCx supply the inferior and lateral walls, respectively, and would not produce this anteroseptal pattern.

Q49. A 55-year-old man presents with chest pain. ECG shows ST-segment depression in leads V1-V3 with tall, broad R waves in the same leads, without significant ST elevation elsewhere. What is the most appropriate next diagnostic step?

A. Discharge with outpatient stress testing B. Assume this represents right bundle branch block and defer further workup C. Obtain posterior leads V7-V9 to assess for posterior wall STEMI D. Order a D-dimer to evaluate for pulmonary embolism E. Repeat troponin in 6 hours without further ECG evaluation

Correct Answer: C

Explanation: ST depression in V1-V3 with tall R waves is a mirror-image pattern suggestive of an isolated posterior wall STEMI, which is often missed on the standard 12-lead ECG. Obtaining posterior leads V7-V9 can reveal true ST elevation confirming posterior infarction, which warrants the same urgent reperfusion strategy as any other STEMI. Deferring to delayed troponin retesting or unrelated workups (D-dimer, presumed conduction abnormality) would dangerously delay time-sensitive reperfusion therapy.

Q50. A 64-year-old man is recovering on hospital day 4 after an inferior STEMI treated with primary PCI. He acutely develops respiratory distress, a new harsh holosystolic murmur best heard at the apex radiating to the axilla, and bilateral crackles consistent with flash pulmonary edema. Which mechanical complication is most likely?

A. Free wall rupture B. Left ventricular aneurysm C. Ventricular septal rupture D. Dressler syndrome E. Papillary muscle rupture

Correct Answer: E

Explanation: Acute severe mitral regurgitation from papillary muscle rupture presents with a new holosystolic murmur and abrupt-onset flash pulmonary edema, typically occurring within the first week post-MI. Inferior MI classically causes rupture of the posteromedial papillary muscle because it has single-vessel (RCA-derived) blood supply, unlike the dual-supplied anterolateral papillary muscle. Free wall rupture presents with sudden PEA/tamponade rather than a murmur, and ventricular septal rupture produces an oxygen saturation step-up between the right atrium and right ventricle rather than acute MR physiology.

Q51. A 60-year-old woman is 5 days post-anterior STEMI when she develops a new, loud holosystolic murmur at the left lower sternal border along with hypotension and biventricular failure signs. Right heart catheterization reveals a significant oxygen saturation step-up between the right atrium and right ventricle. Which mechanical complication is present?

A. Free wall rupture B. Left ventricular pseudoaneurysm C. Pericardial effusion from Dressler syndrome D. Papillary muscle rupture E. Ventricular septal rupture

Correct Answer: E

Explanation: A new holosystolic murmur combined with an oxygen step-up from the right atrium to the right ventricle on catheterization is diagnostic of a ventricular septal rupture (VSR), which creates a left-to-right shunt through the defect. Papillary muscle rupture causes acute mitral regurgitation without an intracardiac shunt or oxygen step-up. Free wall rupture typically presents catastrophically with sudden pulseless electrical activity and tamponade rather than a new murmur with hemodynamic step-up.

Q52. A 70-year-old man develops cardiogenic shock 12 hours after a large anterior STEMI, with SBP 78/50 mmHg, cool extremities, and oliguria despite adequate revascularization. Which vasopressor is generally considered first-line in this setting?

A. Dopamine B. Norepinephrine C. Vasopressin D. Epinephrine E. Phenylephrine

Correct Answer: B

Explanation: Norepinephrine is generally the first-line vasopressor in cardiogenic shock because it provides potent alpha-mediated vasoconstriction with modest beta-1 inotropic support, improving perfusion pressure with a more favorable arrhythmia profile than older agents. Dopamine has fallen out of favor as first-line therapy due to associations with increased arrhythmias and mortality compared to norepinephrine in shock trials. Refractory cases may require escalation to mechanical circulatory support such as an IABP, Impella, or VA-ECMO.

Section 7: Heart Failure — Q53-Q60 (8 questions)

Q53. A 66-year-old woman with dyspnea on exertion undergoes echocardiography, which reveals a left ventricular ejection fraction of 45%. How should her heart failure be classified?

A. Heart failure with reduced ejection fraction (HFrEF) B. Isolated diastolic dysfunction, not heart failure C. Heart failure with mildly reduced ejection fraction (HFmrEF) D. Heart failure with preserved ejection fraction (HFpEF) E. Heart failure with improved ejection fraction

Correct Answer: C

Explanation: HFmrEF is defined by an LVEF of 41-49%, which fits this patient's echocardiographic finding of 45%. HFrEF requires LVEF $\leq 40\%$, and HFpEF requires LVEF $\geq 50\%$; her value of 45% falls squarely in the mildly reduced category. "Heart failure with improved ejection fraction" is a distinct category reserved for patients whose EF has recovered from a previously reduced baseline, which is not described here.

Q54. A 55-year-old man is newly diagnosed with HFrEF (LVEF 30%) after presenting with exertional dyspnea. He is not currently taking any heart failure medications. According to current guideline-directed medical therapy strategy, what is the preferred approach to initiating pharmacotherapy?

A. Start an ACE inhibitor alone and titrate it to the maximum dose before adding a beta-blocker B. Rapidly initiate low doses of all four pillar drug classes (ARNI or ACEI/ARB, evidence-based beta-blocker, MRA, and SGLT2 inhibitor) within 1-2 weeks, then uptitrate C. Defer all pharmacotherapy until after cardiac resynchronization therapy is placed D. Start a beta-blocker alone and wait three months before adding any other agent E. Start only an SGLT2 inhibitor, since it has the greatest mortality benefit of the four classes

Correct Answer: B

Explanation: Current guidance favors rapid, simultaneous initiation of low doses of all four pillar drug classes — ARNI (or ACEI/ARB if ARNI is unavailable), an evidence-based beta-blocker, a mineralocorticoid receptor antagonist, and an SGLT2 inhibitor — within the first 1-2 weeks of diagnosis, followed by systematic uptitration, rather than the older sequential approach of maximizing one drug before adding the next. This strategy achieves earlier morbidity and mortality benefit, since each class acts through complementary mechanisms. Waiting to maximize a single agent before adding others, or deferring pharmacotherapy for device therapy, delays proven mortality benefit.

Q55. A patient with HFrEF is being started on an SGLT2 inhibitor as part of guideline-directed medical therapy. What is the standard once-daily dose of dapagliflozin used for this indication?

A. 100 mg B. 10 mg C. 5 mg D. 25 mg E. 50 mg

Correct Answer: B

Explanation: Dapagliflozin, like empagliflozin, is dosed at 10 mg once daily for heart failure regardless of diabetes status, and this dose has demonstrated reductions in cardiovascular death and heart failure hospitalization across the LVEF spectrum. Lower doses (such as 5 mg) are not the standard heart failure dose, and higher doses (25-100 mg) exceed the studied and approved dosing for this indication.

Q56. Which of the following statements about BNP/NT-proBNP interpretation is accurate?

A. BNP is unaffected by any comorbid conditions and is always a pure reflection of ventricular wall stress B. Levels are falsely low in patients with atrial fibrillation C. Levels are falsely low in patients with renal impairment D. Levels may be falsely low in patients with obesity, which can mask underlying heart failure E. Levels are typically falsely elevated in obesity, potentially masking underlying heart failure

Correct Answer: D

Explanation: BNP/NT-proBNP levels can be falsely low in obese patients, likely due to increased clearance and altered production by adipose tissue, which can mask or underestimate underlying heart failure severity. Conversely, levels tend to be falsely elevated in renal impairment (due to reduced clearance) and in atrial fibrillation (due to atrial stretch), which can lead to overestimation of heart failure in those settings. Clinicians must interpret natriuretic peptide levels in the context of these confounding comorbidities.

Q57. A 70-year-old man with a history of hypertension presents in acute respiratory distress with bilateral crackles, elevated JVP, and a blood pressure of 178/96 mmHg, consistent with acute decompensated heart failure and pulmonary edema. Which combination represents the most appropriate initial management?

A. Immediate initiation of a high-dose oral beta-blocker B. IV normal saline bolus to improve forward flow C. IV morphine as a first-line agent to reduce anxiety and afterload D. IV loop diuretic, IV nitrates, and noninvasive positive pressure ventilation E. Oral loop diuretic and outpatient follow-up in one week

Correct Answer: D

Explanation: For hypertensive acute pulmonary edema, first-line management includes IV loop diuretics to relieve volume overload, IV nitrates to reduce preload and afterload given the elevated blood pressure, and noninvasive positive pressure ventilation to improve oxygenation and reduce work of breathing. Morphine is now discouraged or used only with caution, as it has been associated with worse outcomes in acute heart failure. IV fluid boluses and oral diuretics would be inappropriate and potentially harmful in a volume-overloaded, hypertensive patient in respiratory distress.

Q58. A patient hospitalized for acute decompensated heart failure is started on IV furosemide. Over the next 48 hours, his congestive symptoms and JVP improve, but his serum creatinine rises from 1.0 to 1.6 mg/dL. Which of the following best describes this clinical scenario?

A. Nephrotic syndrome B. Contrast-induced nephropathy C. Rhabdomyolysis-induced acute kidney injury D. Cardiorenal syndrome E. Acute interstitial nephritis

Correct Answer: D

Explanation: Cardiorenal syndrome refers to worsening renal function occurring in the setting of heart failure treatment, such as diuresis, and represents a common clinical challenge requiring careful balancing of decongestion against renal perfusion. This patient's rising creatinine temporally linked to diuretic therapy for heart failure, without exposure to contrast, muscle injury, or signs of an allergic interstitial process, fits this pattern. Management requires individualized judgment about continuing diuresis for decongestion versus adjusting therapy to protect renal function.

Q59. A patient presents with elevated jugular venous pressure, hepatomegaly, bilateral lower extremity edema, and ascites, consistent with right heart failure. What is the most common underlying cause of right heart failure?

A. Idiopathic pulmonary arterial hypertension B. Tricuspid valve endocarditis C. Left heart failure D. Primary pulmonic valve stenosis E. Right ventricular infarction

Correct Answer: C

Explanation: Left heart failure is by far the most common cause of right heart failure, as elevated left-sided filling pressures are transmitted backward into the pulmonary circulation, increasing RV afterload over time and leading to RV dysfunction. Pulmonary

hypertension, RV infarction, and primary pulmonic or tricuspid valve disease can each cause right heart failure independently, but these are far less common etiologies than secondary right heart failure from underlying left heart disease. Recognizing left heart failure as the leading cause helps direct the initial diagnostic workup.

Q60. A 62-year-old man with HFrEF (LVEF 28%) remains symptomatic despite being on the maximally tolerated dose of carvedilol along with an ARNI, an MRA, and an SGLT2 inhibitor. He is in normal sinus rhythm with a resting heart rate of 82 beats per minute. Which additional agent, which inhibits the If (funny) current in the sinoatrial node, should be considered?

A. Amiodarone B. Diltiazem C. Ivabradine D. Verapamil E. Digoxin

Correct Answer: C

Explanation: Ivabradine is indicated for HFrEF patients in sinus rhythm with a resting heart rate ≥ 70 beats per minute despite maximally tolerated beta-blocker therapy, working by selectively inhibiting the If (funny) current in the sinoatrial node to reduce heart rate without affecting contractility or blood pressure. Non-dihydropyridine calcium channel blockers such as diltiazem and verapamil have negative inotropic effects and are generally avoided in HFrEF. Digoxin can be considered for symptom control in HFrEF but does not act on the SA node's If current and is not the agent described here.

Section 8: Valvular Heart Disease — Q61-Q70 (10 questions)

Q61. A 74-year-old man presents with exertional dyspnea and near-syncope. On auscultation, there is a harsh crescendo-decrescendo systolic murmur best heard at the right upper sternal border, radiating to the carotid arteries. Echocardiography reveals an aortic valve area of 0.7 cm^2 , a mean transvalvular gradient of 48 mmHg, peak jet velocity of 4.3 m/s, and an LV ejection fraction of 55%. Which of the following is the most appropriate next step in definitive management?

A. Perform balloon aortic valvuloplasty as definitive therapy B. Continue clinical observation with serial echocardiography C. Initiate diuretics and an ACE inhibitor D. Start high-intensity statin therapy to slow valve calcification E. Refer for aortic valve replacement (surgical or transcatheter)

Correct Answer: E

Explanation: This patient has severe, symptomatic aortic stenosis (valve area $< 1.0 \text{ cm}^2$, mean gradient $> 40 \text{ mmHg}$, peak velocity $> 4 \text{ m/s}$) with exertional dyspnea and near-syncope — a class I indication for valve replacement (SAVR or TAVR depending on surgical risk and anatomy). Balloon valvuloplasty is not definitive therapy in adults because restenosis occurs rapidly; it is used only as a palliative bridge in select cases. Observation is inappropriate once severe AS becomes symptomatic, as untreated symptomatic severe AS carries a poor prognosis, and statins have not been shown to slow hemodynamic progression of calcific AS.

Q62. Which symptom, when it develops in a patient with severe aortic stenosis, is associated with the worst prognosis if the valve is not replaced?

A. Heart failure symptoms (dyspnea) B. Palpitations C. Exertional dizziness without loss of consciousness D. Exertional syncope E. Angina

Correct Answer: A

Explanation: The classic triad of severe symptomatic AS — angina, syncope, and heart failure — carries progressively worsening average survival without valve replacement, with heart failure symptoms conferring the shortest survival of the three. Angina and syncope also indicate a need for urgent valve replacement, but development of overt heart failure signifies the most advanced hemodynamic decompensation and the poorest untreated prognosis.

Q63. A 58-year-old woman develops sudden dyspnea, hypotension, and pulmonary edema three days after an acute myocardial infarction. On examination, a new murmur at the apex is faint and difficult to appreciate. Which of the following is the most likely diagnosis?

A. Ventricular septal rupture B. Papillary muscle rupture causing acute severe mitral regurgitation C. Recurrent myocardial infarction D. Left ventricular free wall rupture with tamponade E. Acute infective endocarditis

Correct Answer: B

Explanation: Papillary muscle rupture is a mechanical complication of MI (classically inferior MI affecting the posteromedial papillary muscle) that causes acute severe MR, presenting with flash pulmonary edema and cardiogenic shock. A key exam point is that the murmur may be soft or even absent because LA and LV pressures equalize rapidly in acute severe MR, unlike chronic MR's loud holosystolic murmur. Ventricular septal rupture instead produces a harsh holosystolic murmur with a palpable thrill and an oxygen step-up on right heart catheterization, while free wall rupture typically causes tamponade and pulseless electrical activity rather than a new murmur.

Q64. A 38-year-old woman with a childhood history of recurrent untreated sore throats presents with progressive dyspnea and new-onset atrial fibrillation. Auscultation reveals a low-pitched diastolic rumble at the apex preceded by an opening snap occurring unusually soon after S2. What is the most likely underlying etiology, and what does the short S2-to-opening-snap interval indicate?

A. Mitral annular calcification; the short interval indicates milder mitral stenosis B. Infective endocarditis; the short interval indicates more severe mitral stenosis C. Rheumatic heart disease; the short interval indicates more severe mitral stenosis D. Rheumatic heart disease; the short interval indicates milder mitral stenosis E. Congenital mitral stenosis; the interval is unrelated to disease severity

Correct Answer: C

Explanation: Rheumatic heart disease remains the most common cause of mitral stenosis worldwide, and this patient's history of recurrent (likely untreated streptococcal)

pharyngitis supports this etiology. A shorter interval between S2 and the opening snap reflects a higher left atrial pressure forcing the stenotic mitral valve open earlier and more forcefully, which correlates with more severe stenosis. Atrial fibrillation is a common complication of mitral stenosis due to left atrial enlargement and pressure overload.

Q65. A 30-year-old man with a known bicuspid aortic valve presents with a wide pulse pressure (150/40 mmHg), a bounding “water-hammer” pulse, and an early decrescendo diastolic murmur at the left sternal border. A separate rumbling mid-diastolic murmur is also heard at the apex. What is the mechanism of this apical murmur?

A. Concurrent rheumatic mitral stenosis is present B. Papillary muscle dysfunction from ischemia C. The regurgitant aortic jet interferes with anterior mitral leaflet opening and mitral inflow D. Fixed subvalvular left ventricular outflow tract obstruction E. A left-to-right shunt across an atrial septal defect

Correct Answer: C

Explanation: This patient has severe chronic aortic regurgitation, evidenced by the wide pulse pressure, water-hammer pulse, and decrescendo diastolic murmur. The apical rumble is an Austin Flint murmur, produced when the regurgitant aortic jet strikes the anterior mitral leaflet and interferes with normal mitral inflow, mimicking mitral stenosis without any actual mitral valve pathology. It is distinguished from true mitral stenosis by the absence of an opening snap and the presence of the peripheral signs of AR.

Q66. A patient with long-standing severe pulmonary hypertension due to COPD develops peripheral edema, ascites, and an elevated jugular venous pressure. A holosystolic murmur is heard at the left lower sternal border that increases in intensity with inspiration. What is the most likely mechanism of this murmur?

A. Ebstein anomaly B. Tricuspid stenosis C. Primary tricuspid regurgitation from rheumatic disease D. Primary tricuspid regurgitation from infective endocarditis E. Secondary (functional) tricuspid regurgitation from right ventricular dilation

Correct Answer: E

Explanation: The murmur described — holosystolic at the left lower sternal border, increasing with inspiration (Carvallo’s sign) — is characteristic of tricuspid regurgitation. In this patient, chronic pulmonary hypertension has led to right ventricular dilation, which is the most common mechanism of TR (functional/secondary rather than primary valve pathology). Primary TR from endocarditis is classically seen in intravenous drug users and would not typically follow a course of progressive pulmonary hypertension from COPD.

Q67. A 22-year-old thin woman presents with palpitations. Auscultation reveals a mid-systolic click followed by a late systolic murmur at the apex. When she stands up abruptly from a seated position, the click moves earlier, closer to S1, and the murmur becomes longer. What is the diagnosis, and why does standing produce this change?

A. Mitral regurgitation from a flail leaflet; standing has no effect on timing B. Hypertrophic obstructive cardiomyopathy; standing decreases LVOT obstruction C. Mitral valve

prolapse; standing increases LV volume, delaying prolapse D. Aortic stenosis; standing increases murmur intensity E. Mitral valve prolapse; standing decreases LV volume, causing earlier prolapse of the mitral leaflets

Correct Answer: E

Explanation: This presentation is classic for mitral valve prolapse. Maneuvers that decrease LV volume (standing, Valsalva strain phase) cause the prolapsing leaflet to billow into the left atrium earlier in systole, moving the click and murmur closer to S1. Conversely, maneuvers that increase LV volume (squatting, passive leg raise) delay the click and murmur. This is in contrast to HOCM, where standing worsens rather than improves outflow obstruction.

Q68. A murmur increases in intensity during the strain phase of the Valsalva maneuver (decreased preload) and decreases in intensity with squatting (increased preload). This pattern is most characteristic of which condition, and how does it differ from aortic stenosis?

A. Both AS and HOCM show this same increase with decreased preload B. Aortic stenosis; this is the opposite pattern of HOCM C. Mitral regurgitation; it increases with decreased preload similarly to HOCM D. Hypertrophic obstructive cardiomyopathy; this is the opposite pattern of AS, whose murmur generally decreases with reduced preload E. Aortic regurgitation; its intensity is unaffected by preload changes

Correct Answer: D

Explanation: In HOCM, reducing preload (Valsalva strain, standing) decreases LV chamber size, which worsens dynamic LVOT obstruction from systolic anterior motion of the mitral valve and increases murmur intensity; increasing preload (squatting) enlarges the LV cavity and lessens obstruction, decreasing the murmur. This is the opposite of most other systolic murmurs, including aortic stenosis, whose intensity generally decreases with reduced preload/reduced flow across the valve — a classic bedside maneuver used to distinguish the two conditions.

Q69. Which of the following echocardiographic parameters is consistent with severe aortic stenosis?

A. Peak aortic jet velocity >4 m/s B. Mean transvalvular gradient of 20–30 mmHg C. Aortic valve area <1.5 cm² D. Mean transvalvular gradient <20 mmHg E. Aortic valve area >1.0 cm²

Correct Answer: A

Explanation: Severe aortic stenosis is defined by a valve area <1.0 cm², a mean gradient >40 mmHg, or a peak jet velocity >4 m/s. A peak velocity >4 m/s meets this threshold, whereas a valve area <1.5 cm² and a gradient of 20–30 mmHg both fall in the moderate range rather than severe.

Q70. A 66-year-old man with ischemic cardiomyopathy (LVEF 30%) and left ventricular dilation has a holosystolic murmur at the apex radiating to the axilla. Echocardiography

shows no leaflet flail or prolapse and structurally normal, intact chordae tendineae. What is the mechanism of this patient's mitral regurgitation?

A. Secondary (functional) MR from LV dilation causing papillary muscle displacement and leaflet tethering B. Infective endocarditis with leaflet destruction C. Rheumatic mitral valve disease D. Ruptured chordae tendineae E. Primary degenerative disease from myxomatous valve tissue

Correct Answer: A

Explanation: This patient's MR is secondary (functional), resulting from LV dilation in the setting of ischemic cardiomyopathy that displaces the papillary muscles apart and tethers the mitral leaflets, preventing proper coaptation despite structurally normal valve tissue. This contrasts with primary (degenerative) MR, which arises from intrinsic valve pathology such as myxomatous disease, prolapse, or chordal rupture — all of which are excluded here by the normal leaflets and chordae on echocardiography.

Section 9: Cardiomyopathies — Q71-Q76 (6 questions)

Q71. A 17-year-old competitive basketball player collapses suddenly during practice and dies. Autopsy reveals asymmetric septal hypertrophy. Which of the following is the most likely underlying etiology?

A. An autosomal dominant mutation in a sarcomere protein gene B. Recent viral myocarditis C. Chronic poorly controlled hypertension D. Anthracycline chemotherapy toxicity E. Cardiac amyloid deposition

Correct Answer: A

Explanation: Hypertrophic cardiomyopathy, most commonly caused by autosomal dominant mutations in sarcomere protein genes, is the leading cause of sudden cardiac death in young athletes, and asymmetric septal hypertrophy is its classic morphologic finding. Hypertensive heart disease typically produces concentric, symmetric hypertrophy rather than asymmetric septal hypertrophy and would be unusual in a healthy teenager. Amyloidosis and anthracycline toxicity are causes of infiltrative or dilated cardiomyopathy, not the asymmetric septal hypertrophy described here.

Q72. A 28-year-old woman is newly diagnosed with hypertrophic obstructive cardiomyopathy. She has mild exertional dyspnea, and echocardiography shows a resting LVOT gradient of 45 mmHg. Which of the following is the most appropriate initial pharmacologic therapy?

A. A beta-blocker B. Digoxin C. An ACE inhibitor D. A dihydropyridine calcium channel blocker (e.g., amlodipine) E. Furosemide

Correct Answer: A

Explanation: Beta-blockers (or non-dihydropyridine calcium channel blockers such as verapamil if beta-blockers are not tolerated) are first-line therapy for symptomatic HOCM, as they reduce heart rate and contractility, allowing more time for LV filling and reducing

dynamic outflow obstruction. ACE inhibitors, diuretics, dihydropyridine CCBs, and digoxin are generally avoided in obstructive HCM because they reduce preload/afterload or increase contractility, all of which can worsen LVOT obstruction. Septal reduction therapy is reserved for severe, drug-refractory obstructive symptoms.

Q73. A 26-year-old woman presents three weeks postpartum with progressive dyspnea, orthopnea, and bilateral crackles. She has no prior cardiac history, and echocardiography reveals an LV ejection fraction of 30% with global LV dilation. Which of the following is the most likely diagnosis?

A. Preeclampsia-related pulmonary edema B. Amniotic fluid embolism C. Peripartum cardiomyopathy D. Acute pulmonary embolism E. Takotsubo cardiomyopathy

Correct Answer: C

Explanation: Peripartum cardiomyopathy is a form of dilated cardiomyopathy that presents with new systolic heart failure occurring from the last month of pregnancy through five months postpartum, consistent with this patient's timeline. Takotsubo cardiomyopathy is typically triggered by acute physical or emotional stress and shows apical ballooning rather than global dilation. Amniotic fluid embolism and pulmonary embolism present acutely with respiratory failure and hemodynamic collapse rather than a subacute course with reduced EF and LV dilation.

Q74. A 68-year-old postmenopausal woman develops sudden chest pain shortly after learning of her husband's unexpected death. ECG shows anterior ST elevations, and troponin is mildly elevated. Echocardiography demonstrates apical ballooning with hyperdynamic basal contraction. Coronary angiography shows no significant obstructive disease. Which of the following is the most likely diagnosis?

A. Spontaneous coronary artery dissection requiring stent placement B. Acute pericarditis C. Anterior STEMI due to acute LAD occlusion D. Takotsubo (stress) cardiomyopathy E. Coronary vasospasm requiring emergent stenting

Correct Answer: D

Explanation: Takotsubo cardiomyopathy is triggered by acute emotional or physical stress, mimics an anterior STEMI clinically and on ECG, but is distinguished by angiographically normal or non-obstructive coronary arteries and characteristic apical ballooning with basal hyperkinesis. It predominantly affects postmenopausal women and is usually reversible with supportive care, unlike a true STEMI, which requires urgent revascularization.

Q75. Which of the following findings is most consistent with restrictive cardiomyopathy rather than dilated cardiomyopathy?

A. Reduced ejection fraction with a dilated LV chamber B. Severely dilated LV with eccentric hypertrophy C. Dynamic LVOT obstruction with systolic anterior motion of the mitral valve D. Normal or near-normal ejection fraction with impaired diastolic filling and

normal or near-normal wall thickness or chamber size E. Apical ballooning with normal coronary arteries

Correct Answer: D

Explanation: Restrictive cardiomyopathy is characterized by impaired ventricular filling (diastolic dysfunction) with a normal or near-normal ejection fraction and normal or near-normal wall thickness or chamber size, caused by conditions such as cardiac amyloidosis, sarcoidosis, and hemochromatosis. This differs from dilated cardiomyopathy, which features a dilated, poorly contracting LV with reduced EF, and from HOCM, which shows dynamic LVOT obstruction from SAM of the mitral valve.

Q76. What is the most common overall cause of dilated cardiomyopathy?

A. Peripartum state B. Viral myocarditis C. Idiopathic genetic mutation D. Chronic alcohol use E. Ischemic heart disease

Correct Answer: E

Explanation: Ischemic heart disease is the most common overall cause of dilated cardiomyopathy, resulting from chronic myocardial injury and remodeling following coronary artery disease and prior infarction. Idiopathic, viral myocarditis, chronic alcohol use, peripartum, and genetic/familial forms are all recognized causes but are individually less common than ischemic etiology as a group cause.

Section 10: Pericardial Disease — Q77-Q80 (4 questions)

Q77. A 34-year-old man presents with sharp chest pain that worsens when lying flat and improves when sitting forward, five days after a viral upper respiratory infection. On examination, a pericardial friction rub is heard. ECG shows diffuse concave ST elevation with PR depression, and echocardiography shows no pericardial effusion. What is the diagnosis and most appropriate first-line treatment?

A. Acute pericarditis; NSAIDs plus colchicine B. Acute pericarditis; high-dose corticosteroids as first-line therapy C. Aortic dissection; CT angiography and emergent surgery D. Pulmonary embolism; therapeutic anticoagulation E. STEMI; emergent percutaneous coronary intervention

Correct Answer: A

Explanation: This patient meets at least 2 of the 4 diagnostic criteria for acute pericarditis (positional/pleuritic chest pain, a friction rub, and typical diffuse concave ST elevation with PR depression), and an effusion is not required for diagnosis. First-line treatment is NSAIDs combined with colchicine, which also reduces the risk of recurrence. Corticosteroids are not first-line therapy because they are associated with a higher rate of recurrent pericarditis and are reserved for cases refractory to or with contraindications to NSAIDs/colchicine.

Q78. A 55-year-old man with metastatic malignancy presents with hypotension (82/58 mmHg), jugular venous distension, and muffled heart sounds. Pulsus paradoxus is

measured at 18 mmHg. Echocardiography shows a large pericardial effusion with right atrial and right ventricular diastolic collapse. What is the most appropriate next step in management?

A. Emergent coronary artery bypass grafting B. Urgent pericardiocentesis C. Observation with a repeat echocardiogram in one week D. IV diuretics to reduce preload E. High-dose NSAIDs and colchicine

Correct Answer: B

Explanation: This patient has cardiac tamponade, evidenced by Beck's triad (hypotension, JVD, muffled heart sounds), pulsus paradoxus, and echocardiographic RA/RV diastolic collapse, requiring urgent pericardiocentesis to relieve the hemodynamically significant fluid accumulation. Diuretics are inappropriate and potentially harmful in tamponade because they further reduce preload in a patient who is critically dependent on it to maintain cardiac output. NSAIDs/colchicine treat pericarditis but do not address the hemodynamic emergency of tamponade.

Q79. A 60-year-old man with a history of pulmonary tuberculosis treated 20 years ago presents with progressive peripheral edema, ascites, and elevated jugular venous pressure that rises with inspiration. An early diastolic sound is heard after S2. Cardiac catheterization shows equalization of diastolic pressures across all four cardiac chambers. What is the diagnosis and definitive treatment?

A. Restrictive cardiomyopathy; heart transplantation B. Tricuspid stenosis; balloon valvuloplasty C. Cardiac tamponade; pericardiocentesis D. Right heart failure from COPD; diuretics alone E. Constrictive pericarditis; surgical pericardiectomy

Correct Answer: E

Explanation: This presentation — prior tuberculosis, Kussmaul's sign (JVP rising with inspiration), a pericardial knock, and equalization of diastolic pressures across all four chambers on catheterization — is classic for constrictive pericarditis, for which prior TB is a well-recognized cause. Definitive treatment for symptomatic constrictive pericarditis is surgical pericardiectomy. Restrictive cardiomyopathy can mimic this presentation but is distinguished by invasive hemodynamics, imaging, or biopsy rather than by equalized diastolic pressures with a pericardial knock and known TB exposure.

Q80. In a patient with a first episode of acute pericarditis being treated with NSAIDs, which additional medication has been shown to significantly reduce the risk of recurrence?

A. High-dose prednisone B. Furosemide C. Colchicine D. Warfarin E. Amiodarone

Correct Answer: C

Explanation: Colchicine, added to NSAID therapy, is recommended as part of first-line treatment for acute pericarditis because it significantly reduces the risk of recurrent pericarditis compared to NSAIDs alone. Corticosteroids are generally avoided as initial therapy because they have been associated with an increased, not decreased, risk of

recurrence. Amiodarone, furosemide, and warfarin have no established role in reducing pericarditis recurrence. ## Section 11: Congenital Heart Disease — Q81-Q86

Q81. A 32-year-old woman undergoes a pre-employment physical examination and is found to have a soft systolic ejection murmur at the left upper sternal border. On auscultation, S2 is split and the splitting does not change with inspiration or expiration. She has no symptoms and has never been told of a heart murmur before. Echocardiography reveals a defect in the interatrial septum with left-to-right shunting. Which of the following is the most likely diagnosis?

A. Ventricular septal defect B. Tetralogy of Fallot C. Patent ductus arteriosus D. Coarctation of the aorta E. Atrial septal defect

Correct Answer: E

Explanation: A fixed, widely split S2 that does not vary with respiration is the classic auscultatory finding of an atrial septal defect (ASD), caused by chronic right heart volume overload that equalizes right and left heart filling regardless of the respiratory cycle. ASDs, most commonly the ostium secundum type, are frequently asymptomatic and diagnosed incidentally in adulthood, with a systolic ejection murmur at the left upper sternal border from increased flow across the pulmonic valve. A VSD produces a holosystolic murmur at the left lower sternal border, not a fixed split S2. PDA produces a continuous machine-like murmur, which is not described here.

Q82. Which of the following is the most common congenital heart defect in children overall, classically producing a harsh holosystolic murmur best heard at the left lower sternal border?

A. Atrial septal defect B. Coarctation of the aorta C. Patent ductus arteriosus D. Tetralogy of Fallot E. Ventricular septal defect

Correct Answer: E

Explanation: Ventricular septal defect (VSD) is the most common congenital heart defect in children overall. Small VSDs are frequently asymptomatic and many close spontaneously, while large, uncorrected defects can lead to heart failure and, over time, pulmonary hypertension with shunt reversal (Eisenmenger syndrome). The harsh holosystolic murmur at the left lower sternal border reflects turbulent flow across the septal defect during systole. ASD produces a fixed split S2 rather than a holosystolic murmur, and PDA produces a continuous murmur, not a holosystolic one.

Q83. A premature infant born at 29 weeks' gestation is noted to have a continuous "machine-like" murmur best heard at the left infraclavicular area. The mother's prenatal history is notable for a rubella infection early in pregnancy. Which of the following pharmacologic agents can be used to promote closure of this infant's cardiac defect?

A. Furosemide B. Digoxin C. Indomethacin D. Alprostadil (prostaglandin E1) E. Propranolol

Correct Answer: C

Explanation: This infant has a patent ductus arteriosus (PDA), suggested by the continuous, machine-like murmur at the left infraclavicular area and risk factors of prematurity and maternal rubella infection during pregnancy. Indomethacin (or another NSAID) inhibits prostaglandin synthesis and can be used to pharmacologically close a PDA in premature infants. Prostaglandin E1 (alprostadil) does the opposite — it is used to keep a ductus arteriosus open in ductal-dependent congenital lesions, making it incorrect here. Furosemide, digoxin, and propranolol have no role in ductal closure.

Q84. A 4-year-old boy with known tetralogy of Fallot suddenly becomes more cyanotic, tachypneic, and irritable while crying during a clinic visit. Which of the following is the most appropriate immediate management?

A. Perform emergent pericardiocentesis B. Administer IV adenosine C. Administer sublingual nitroglycerin D. Place the child in the knee-to-chest position and give supplemental oxygen E. Administer IV furosemide

Correct Answer: D

Explanation: This child is having a hypercyanotic (“tet”) spell, a complication of tetralogy of Fallot triggered by increased right-to-left shunting across the VSD due to dynamic worsening of right ventricular outflow tract obstruction. The knee-to-chest position increases systemic vascular resistance, which reduces the right-to-left shunt and improves pulmonary blood flow, and supplemental oxygen further helps as a pulmonary vasodilator; both are first-line acute measures. Furosemide and nitroglycerin do not address the underlying shunt physiology, adenosine is used for supraventricular tachycardia, and pericardiocentesis is irrelevant to this presentation.

Q85. A 15-year-old girl with Turner syndrome is found to have hypertension on a routine visit. Examination reveals diminished and delayed femoral pulses compared to the radial pulses, and blood pressure in the arms is significantly higher than in the legs. A chest radiograph is most likely to show which of the following findings?

A. Egg-on-a-string appearance B. Kerley B lines C. Rib notching D. Water-bottle-shaped heart silhouette E. Boot-shaped heart

Correct Answer: C

Explanation: This presentation — hypertension, radio-femoral pulse delay, and an arm-leg blood pressure gradient in a patient with Turner syndrome — is classic for coarctation of the aorta, which is also associated with bicuspid aortic valve. Chronic collateral flow through enlarged intercostal arteries produces rib notching on chest radiograph, typically seen in older children and adults. A boot-shaped heart is characteristic of tetralogy of Fallot, egg-on-a-string suggests transposition of the great arteries, and a water-bottle silhouette suggests a large pericardial effusion — none of which fit this clinical picture.

Q86. Tetralogy of Fallot classically consists of all of the following components EXCEPT:

A. Right ventricular hypertrophy B. Right ventricular outflow tract obstruction C. Overriding aorta D. Ventricular septal defect E. Patent ductus arteriosus

Correct Answer: E

Explanation: The four components of tetralogy of Fallot are a ventricular septal defect, right ventricular outflow tract (pulmonic) obstruction, an overriding aorta, and right ventricular hypertrophy. A patent ductus arteriosus is a separate congenital lesion and is not one of the four defining components of tetralogy of Fallot, although a PDA may coexist and can be important for maintaining pulmonary blood flow in severe cases before surgical repair. Confusing tetralogy of Fallot's components with other congenital lesions is a common test-taking pitfall.

Section 12: Infective Endocarditis and Cardiac Infections — Q87-Q91

Q87. A 28-year-old man with a history of intravenous heroin use presents with fever, malaise, and pleuritic chest pain. Blood cultures grow gram-positive cocci in clusters, and transthoracic echocardiography reveals a vegetation on the tricuspid valve. Which of the following organisms is the most likely cause of this patient's infective endocarditis?

A. *Streptococcus gallolyticus* B. *Candida albicans* C. HACEK organisms D. Viridans group streptococci E. *Staphylococcus aureus*

Correct Answer: E

Explanation: *Staphylococcus aureus* is the most common overall cause of infective endocarditis and is classically implicated in acute presentations and in intravenous drug use-associated endocarditis, which characteristically affects the right-sided (tricuspid) valve, as in this patient. Viridans group streptococci more typically cause subacute native valve endocarditis associated with dental disease, not the acute right-sided presentation seen here. HACEK organisms are a classic but less common cause of culture-negative endocarditis, which does not fit this case since cultures were positive.

Q88. According to the Modified Duke criteria for the diagnosis of infective endocarditis, which of the following is classified as a MAJOR criterion?

A. A new valvular regurgitant murmur with a vegetation seen on echocardiography B. Known intravenous drug use C. Osler nodes on the finger pads D. Fever of 38.2°C E. Roth spots on fundoscopic examination

Correct Answer: A

Explanation: Evidence of endocardial involvement — such as a new valvular regurgitant murmur or a vegetation demonstrated on echocardiography — together with persistently positive blood cultures for a typical infective endocarditis organism, constitutes the two major criteria in the Modified Duke criteria. Fever $\geq 38^{\circ}\text{C}$, vascular phenomena, immunologic phenomena (such as Osler nodes and Roth spots), and predisposing conditions like IV drug use are all minor criteria, not major ones. Distinguishing major from minor criteria is essential since the diagnosis requires specific combinations of these categories.

Q89. A 65-year-old man is found to have *Streptococcus gallolyticus* (formerly *S. bovis*) bacteremia and a new aortic valve vegetation on echocardiography. Beyond treating the endocarditis, which of the following is the most appropriate next step in management?

A. Bone marrow biopsy B. Screening colonoscopy C. CT scan of the chest D. Testicular ultrasound E. Upper endoscopy only

Correct Answer: B

Explanation: *Streptococcus gallolyticus* bacteremia and endocarditis have a well-established association with colorectal malignancy (and other colonic mucosal lesions), so a screening colonoscopy is warranted in all such patients regardless of age or prior screening history. Upper endoscopy, chest CT, testicular ultrasound, and bone marrow biopsy are not the established workup linked to this specific organism. This organism-disease association is a classic, frequently tested clinical pearl.

Q90. A 45-year-old woman with a mechanical aortic valve is scheduled for a routine dental cleaning that will involve gingival manipulation. Which of the following is the most appropriate approach regarding antibiotic prophylaxis for infective endocarditis?

A. Prophylaxis is only indicated if she has a fever at the time of the procedure B. Prophylaxis is indicated for all patients undergoing any dental procedure C. Prophylaxis should be given only if she has a documented penicillin allergy D. Prophylaxis is not indicated because dental cleanings carry negligible bacteremia risk E. Prophylaxis should be given because she has a prosthetic heart valve

Correct Answer: E

Explanation: Antibiotic prophylaxis before dental procedures involving gingival manipulation is recommended only for high-risk cardiac conditions, which include prosthetic heart valves (including transcatheter valves), a prior history of infective endocarditis, unrepaired cyanotic congenital heart disease, and cardiac transplant recipients with valvulopathy. This patient's mechanical aortic valve places her squarely in the high-risk category warranting prophylaxis. Prophylaxis is not recommended for most other structural conditions, such as isolated mitral valve prolapse without regurgitation, and is not a blanket recommendation for all dental patients.

Q91. A patient with subacute infective endocarditis is found to have painful, violaceous, tender nodules on the pulp of several fingers. This finding is best explained by which of the following mechanisms?

A. Linear microvascular hemorrhage under the nail bed B. Immune complex deposition C. Septic microemboli causing painless macules D. Retinal artery vasculitis E. Direct bacterial invasion of the epidermis

Correct Answer: B

Explanation: The findings described are Osler nodes — painful, tender nodules on the finger or toe pads caused by immune complex deposition, a classic feature of subacute infective endocarditis. This is in contrast to Janeway lesions, which are painless, flat,

erythematous macules on the palms or soles resulting from septic microemboli (option C describes Janeway lesions, not the finding in this vignette). Roth spots are retinal hemorrhages with pale centers (not a finger finding), and splinter hemorrhages are the linear reddish-brown streaks under the nails described in option A.

Section 13: Cardiovascular Pharmacology and Therapeutics — Q92-Q100

Q92. An 83-year-old woman (weight 58 kg, serum creatinine 1.6 mg/dL) with newly diagnosed atrial fibrillation is being started on apixaban for stroke prevention. Which of the following is the correct dosing regimen for her?

A. Apixaban 5 mg twice daily B. Apixaban 10 mg once daily C. Apixaban 5 mg once daily D. Apixaban 2.5 mg twice daily E. Apixaban 2.5 mg once daily

Correct Answer: D

Explanation: Apixaban dose reduction to 2.5 mg twice daily is required when a patient meets at least 2 of the following 3 criteria: age ≥ 80 years, weight ≤ 60 kg, and serum creatinine ≥ 1.5 mg/dL. This patient meets all three criteria (age 83, weight 58 kg, creatinine 1.6 mg/dL), so the reduced dose of 2.5 mg twice daily is correct rather than the standard 5 mg twice daily dose. Apixaban is dosed twice daily, not once daily, which eliminates the once-daily options.

Q93. A 58-year-old man with a mechanical mitral valve is maintained on warfarin for anticoagulation. What is his target INR range?

A. 1.5–2.0 B. 2.0–3.0 C. 2.5–3.5 D. 3.0–4.5 E. 3.5–4.5

Correct Answer: C

Explanation: Mechanical mitral valves (and other high-risk mechanical valves) require a higher target INR of 2.5–3.5 due to their greater thrombogenicity compared with other indications. Standard indications for warfarin, such as atrial fibrillation or venous thromboembolism, target an INR of 2.0–3.0, which would be inadequate for this higher-risk mechanical valve. INR ranges above 3.5 are not a standard target range and would carry excessive bleeding risk without added benefit.

Q94. A 61-year-old man is undergoing PCI with stent placement after an NSTEMI. His history includes an ischemic stroke 2 years ago. Which P2Y₁₂ inhibitor should be avoided in this patient due to a contraindication?

A. Aspirin B. Cangrelor C. Clopidogrel D. Prasugrel E. Ticagrelor

Correct Answer: D

Explanation: Prasugrel is contraindicated in patients with a prior history of stroke or transient ischemic attack because of a significantly increased risk of intracranial and other major bleeding demonstrated in clinical trials. Clopidogrel and ticagrelor do not carry this specific contraindication and are appropriate alternative P2Y₁₂ inhibitors for dual

antiplatelet therapy after PCI in this patient. Aspirin is not a P2Y12 inhibitor, and cangrelor is an IV agent used periprocedurally, not the focus of this contraindication.

Q95. A patient with HFrEF is being transitioned from an ACE inhibitor to sacubitril-valsartan (an ARNI) due to persistent symptoms despite guideline-directed therapy. To minimize the risk of angioedema, how long should the ACE inhibitor be stopped before starting the ARNI?

A. At least 12 hours B. At least 36 hours C. At least 7 days D. No washout period is needed E. At least 24 hours

Correct Answer: B

Explanation: A washout period of at least 36 hours is required between stopping an ACE inhibitor and starting sacubitril-valsartan because concurrent inhibition of ACE and neprilysin markedly increases the risk of life-threatening angioedema. Shorter intervals (12 or 24 hours) do not provide adequate clearance of the ACE inhibitor to safely mitigate this risk, and a 7-day washout is unnecessarily long and not the established recommendation. This washout is not required when switching from an ARB to an ARNI in the same manner, but it is required from an ACE inhibitor specifically.

Q96. Which of the following beta-blockers has NOT been shown to reduce mortality in patients with heart failure with reduced ejection fraction (HFrEF)?

A. Metoprolol tartrate (immediate-release) B. Carvedilol C. Metoprolol succinate (extended-release) D. None of the above; all beta-blockers reduce mortality equally in HFrEF E. Bisoprolol

Correct Answer: A

Explanation: Only three beta-blockers have demonstrated a proven mortality benefit in HFrEF: carvedilol, metoprolol succinate (extended-release), and bisoprolol. Metoprolol tartrate, the immediate-release formulation, was studied in the MDC trial and did not show the same mortality benefit and is not recommended for HFrEF management. This is a key distinction, as not all beta-blockers — and not all formulations of the same drug — are interchangeable in heart failure therapy.

Q97. A 70-year-old man with HFrEF on dapagliflozin is admitted with acute gastroenteritis, vomiting, and poor oral intake. Which of the following is the most appropriate management of his SGLT2 inhibitor during this acute illness?

A. Continue dapagliflozin at the same dose without interruption B. Permanently discontinue dapagliflozin and avoid SGLT2 inhibitors in the future C. Double the dapagliflozin dose to compensate for volume loss D. Switch immediately to insulin therapy E. Temporarily hold dapagliflozin due to risk of euglycemic diabetic ketoacidosis

Correct Answer: E

Explanation: SGLT2 inhibitors such as dapagliflozin and empagliflozin should be temporarily held during acute illness or periods of reduced oral intake (“sick day rules”)

because of the risk of euglycemic diabetic ketoacidosis, which can occur even with normal or near-normal blood glucose levels. This benefit-risk consideration applies across the ejection fraction spectrum and regardless of diabetes status. There is no indication to permanently discontinue the drug or to increase the dose; once the acute illness resolves and oral intake normalizes, the medication can typically be resumed.

Q98. A patient with established atherosclerotic cardiovascular disease is being started on statin therapy with a goal of achieving at least a 50% reduction in LDL-cholesterol. Which of the following regimens meets the definition of high-intensity statin therapy?

A. Simvastatin 80 mg daily B. Atorvastatin 10 mg daily C. Pravastatin 40 mg daily D. Atorvastatin 40 mg daily E. Rosuvastatin 5 mg daily

Correct Answer: D

Explanation: High-intensity statin therapy, defined as achieving an average LDL-C reduction of $\geq 50\%$, includes atorvastatin 40–80 mg daily or rosuvastatin 20–40 mg daily. Atorvastatin 40 mg daily meets this definition, while atorvastatin 10 mg, rosuvastatin 5 mg, and pravastatin 40 mg are moderate- or low-intensity regimens. Simvastatin 80 mg is specifically avoided due to an increased risk of myopathy and rhabdomyolysis at that dose, making it an inappropriate choice regardless of intensity classification.

Q99. A patient on long-term amiodarone therapy for recurrent ventricular tachycardia presents for routine follow-up. Which of the following surveillance strategies is most appropriate given amiodarone's toxicity profile?

A. Annual complete blood count only, as amiodarone's main toxicity is bone marrow suppression B. No routine monitoring is required once therapy is established C. Periodic renal function testing only D. Periodic serum potassium monitoring only E. Periodic thyroid function tests, liver function tests, and chest imaging/pulmonary function testing

Correct Answer: E

Explanation: Amiodarone has multiple organ toxicities, including thyroid dysfunction (both hypo- and hyperthyroidism), pulmonary fibrosis, hepatotoxicity, corneal microdeposits, and skin discoloration, so baseline and periodic monitoring of thyroid function tests, liver function tests, and chest imaging or pulmonary function tests is recommended. Renal function and bone marrow suppression are not characteristic amiodarone toxicities, making options B and C incorrect. Given the extensive toxicity profile, ongoing monitoring throughout therapy is required rather than none at all.

Q100. A 76-year-old man on digoxin and furosemide for heart failure presents with nausea, visual halos around lights, and new premature ventricular contractions. Laboratory studies reveal a potassium level of 2.9 mEq/L. In addition to correcting the potassium, which of the following is used to reverse life-threatening digoxin toxicity?

A. Idarucizumab B. Digoxin immune Fab (DigiFab) C. Vitamin K D. Protamine sulfate E. 4-factor prothrombin complex concentrate

Correct Answer: B

Explanation: This patient's presentation — nausea, visual disturbances (halos), and new ventricular ectopy in the setting of hypokalemia from diuretic use — is consistent with digoxin toxicity, since hypokalemia, hypomagnesemia, renal impairment, and concurrent amiodarone use all increase the risk of toxicity due to digoxin's narrow therapeutic index. Digoxin immune Fab (DigiFab) is the specific antidote used to reverse life-threatening digoxin toxicity by binding free digoxin. Idarucizumab reverses dabigatran, protamine reverses heparin, and 4-factor PCC and vitamin K are used for warfarin reversal — none of which are appropriate for this patient's digoxin toxicity. # Appendix A: Section Map and Answer Key

Section Map

Section	Topic	Questions
1	Cardiac Anatomy and Physiology	Q1-Q8
2	Hypertension	Q9-Q14
3	Preventive Cardiology and Lipid Management	Q15-Q20
4	ECG Basics, Rhythm, and Conduction Disorders	Q21-Q34
5	Emergency Cardiac Care and Resuscitation	Q35-Q40
6	Acute Coronary Syndromes and Ischemic Heart Disease	Q41-Q52
7	Heart Failure	Q53-Q60
8	Valvular Heart Disease	Q61-Q70
9	Cardiomyopathies	Q71-Q76
10	Pericardial Disease	Q77-Q80
11	Congenital Heart Disease	Q81-Q86
12	Infective Endocarditis and Cardiac Infections	Q87-Q91
13	Cardiovascular Pharmacology and Therapeutics	Q92-Q100

Compact Answer Key

Grouped in rows of 10 for quick self-scoring.

Q	1	2	3	4	5	6	7	8	9	10
Q1-10	C	B	B	E	A	A	B	A	A	D
Q11-20	D	A	A	B	C	A	D	C	B	C
Q21-30	A	B	C	B	A	E	D	B	C	D
Q31-40	D	C	E	D	E	E	D	C	A	C
Q41-50	B	D	E	A	D	B	B	A	C	E
Q51-60	E	B	C	B	B	D	D	D	C	C
Q61-70	E	A	B	C	C	E	E	D	A	A
Q71-80	A	A	C	D	D	E	A	B	E	C
Q81-90	E	E	C	D	C	E	E	A	B	E

Appendix B: Key References for Further Reading

- American College of Cardiology / American Heart Association / ACEP / NAEMSP / SCAI. *2025 Guideline for the Management of Patients With Acute Coronary Syndromes*. Circulation / JACC.
- American Heart Association / American College of Cardiology / Heart Failure Society of America. *Guideline for the Management of Heart Failure*. JACC.
- American Heart Association. *2025 Guidelines for CPR and Emergency Cardiovascular Care*. Circulation.
- American Heart Association / ACC / HRS. *Recommendations for the Standardization and Interpretation of the Electrocardiogram*. Circulation.
- European Society of Cardiology. *ESC Clinical Practice Guidelines* (escardio.org/Guidelines), including guidelines on valvular heart disease, cardiomyopathies, and infective endocarditis.
- Dr A M Thirugnanam. *Synopsis of Emergency Cardiac Care, Handy Drugs and Dosing Reference in Cardiology*, and *ECG Pocket Guide* — companion volumes in this series.

Readers are encouraged to consult the full primary guideline documents for complete detail beyond the scope of this self-assessment volume.

About the Author

Dr A M Thirugnanam, MD, MSICP, FSCAI, Ph.D., is a Senior Interventional Cardiologist and Founder of the Academy of Elite Doctors, based in Hyderabad, India. An educator and researcher, he has dedicated his career to advancing cardiology education for medical students, residents, and practicing physicians through structured courses, publications, and continuing medical education programs.