OME CARDIAC MODULES PRACTICE TEST (SAMPLE)

STUDY GUIDE

SAMPLE STUDY GUIDE WITH LIMITED QUESTIONS

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Introduction

Preparing for a certification exam can feel overwhelming, but with the right tools, it becomes an opportunity to build confidence, sharpen your skills, and move one step closer to your goals. At Examzify, we believe that effective exam preparation isn't just about memorization, it's about understanding the material, identifying knowledge gaps, and building the test-taking strategies that lead to success.

This guide was designed to help you do exactly that.

Whether you're preparing for a licensing exam, professional certification, or entry-level qualification, this book offers structured practice to reinforce key concepts. You'll find a wide range of multiple-choice questions, each followed by clear explanations to help you understand not just the right answer, but why it's correct.

The content in this guide is based on real-world exam objectives and aligned with the types of questions and topics commonly found on official tests. It's ideal for learners who want to:

- Practice answering questions under realistic conditions,
- Improve accuracy and speed,
- Review explanations to strengthen weak areas, and
- Approach the exam with greater confidence.

We recommend using this book not as a stand-alone study tool, but alongside other resources like flashcards, textbooks, or hands-on training. For best results, we recommend working through each question, reflecting on the explanation provided, and revisiting the topics that challenge you most.

Remember: successful test preparation isn't about getting every question right the first time, it's about learning from your mistakes and improving over time. Stay focused, trust the process, and know that every page you turn brings you closer to success.

Let's begin.

How to Use This Guide

This guide is designed to help you study more effectively and approach your exam with confidence. Whether you're reviewing for the first time or doing a final refresh, here's how to get the most out of your Examzify study guide:

1. Start with a Diagnostic Review

Skim through the questions to get a sense of what you know and what you need to focus on. Your goal is to identify knowledge gaps early.

2. Study in Short, Focused Sessions

Break your study time into manageable blocks (e.g. 30 – 45 minutes). Review a handful of questions, reflect on the explanations.

3. Learn from the Explanations

After answering a question, always read the explanation, even if you got it right. It reinforces key points, corrects misunderstandings, and teaches subtle distinctions between similar answers.

4. Track Your Progress

Use bookmarks or notes (if reading digitally) to mark difficult questions. Revisit these regularly and track improvements over time.

5. Simulate the Real Exam

Once you're comfortable, try taking a full set of questions without pausing. Set a timer and simulate test-day conditions to build confidence and time management skills.

6. Repeat and Review

Don't just study once, repetition builds retention. Re-attempt questions after a few days and revisit explanations to reinforce learning. Pair this guide with other Examzify tools like flashcards, and digital practice tests to strengthen your preparation across formats.

There's no single right way to study, but consistent, thoughtful effort always wins. Use this guide flexibly, adapt the tips above to fit your pace and learning style. You've got this!

Questions

1. Necrotic lipid core of a plaque contains which components?

A. HDL and phospholipids

B. LDL, cholesterol, dead foam cells, oxidized LDL

C. Smooth muscle cells and collagen

D. Calcium deposits

2. In heart failure management, what is the effect of dobutamine or milrinone?

A. Increase contractility and decrease systemic vascular resistance (inodilator)

B. Decrease contractility and increase systemic vascular resistance

C. Decrease heart rate with increased afterload

D. No effect on vascular resistance

3. Stage 10 of plaque formation is associated with which event leading to artery occlusion?

A. Rupture and thrombosis

B. Plaque calcification

C. Endothelial dilation

D. Systemic hypertension

4. Phase 0 of the SA node action potential occurs due to entry of which ion?

A. Calcium enters

B. Sodium enters

C. Potassium leaves

D. Chloride enters

5. Which valve is between the right ventricle and the pulmonary artery?

A. Aortic valve

B. Tricuspid valve

C. Pulmonary valve

D. Mitral valve

6. PCSK9 inhibitors such as Alirocumab and Evolocumab work by which mechanism?

A. Inhibits HMG-CoA reductase

B. Blocks destruction of LDL receptors

C. Blocks absorption of cholesterol in the gut

D. Increases LDL receptor degradation

7. Which cells become foam cells as oxidized LDL is ingested but not digested?

A. Macrophages

B. T-lymphocytes

C. Endothelial cells

D. Platelets

8. Constrictive pericarditis results from

A. Increased diastolic filling

B. Pericardial inflammation with effusion

C. Fibrous pericardium becomes inelastic, restricting diastolic filling

D. Resolution with NSAIDs

9. Which statement best describes the Frank-Starling mechanism?

A. Increase in afterload

B. Increase in end-diastolic volume

C. Decrease in preload

D. Increase in heart rate

10. Atropine increases heart rate by blocking which receptor?

A. Beta-1 receptor

B. M2 receptor

C. Alpha-1 receptor

D. D2 receptor

Answers

1. B
2. A
3. A
4. A
5. C
6. B
7. A
8. D
9. B
10. B

Explanations

1. Necrotic lipid core of a plaque contains which components?

A. HDL and phospholipids

B. LDL, cholesterol, dead foam cells, oxidized LDL

C. Smooth muscle cells and collagen

D. Calcium deposits

The necrotic lipid core in an atherosclerotic plaque is made up of cholesterol-rich debris derived from modified LDL taken up by macrophages, which become foam cells. When these foam cells die, they release cholesterol, cholesterol esters, and other lipid-rich material into the plaque center. Oxidized LDL is a key player, promoting more uptake by macrophages and sustaining inflammation that enlarges the core. HDL, smooth muscle cells with collagen, and calcium deposits play other roles in the plaque structure or its stability, but they are not the defining contents of the necrotic lipid core.

2. In heart failure management, what is the effect of dobutamine or milrinone?

A. Increase contractility and decrease systemic vascular resistance (inodilator)

B. Decrease contractility and increase systemic vascular resistance

C. Decrease heart rate with increased afterload

D. No effect on vascular resistance

These drugs work as inodilators in heart failure. They boost the heart's pumping strength (positive inotropy) and also widen the peripheral vessels (vasodilation), which lowers the afterload the heart has to push against. Dobutamine mainly stimulates beta-1 receptors to increase contractility, with some beta-2–mediated vasodilation that lowers systemic vascular resistance. Milrinone inhibits PDE-3, raising cAMP in both heart muscle and vascular smooth muscle, which directly increases contractility and causes vasodilation. The net result is higher cardiac output with reduced systemic vascular resistance, fitting the description of increasing contractility and decreasing afterload. Other options describe either reduced contractility, increased afterload, or no change in vascular resistance, which don't match how these drugs work. These agents are typically used short-term in acute settings, with attention to potential tachycardia or hypotension.

3. Stage 10 of plaque formation is associated with which event leading to artery occlusion?

A. Rupture and thrombosis

B. Plaque calcification

C. Endothelial dilation

D. Systemic hypertension

Rupture of a vulnerable plaque with subsequent thrombosis is the event that directly leads to artery occlusion at this stage. When an atherosclerotic plaque becomes unstable, its fibrous cap thins and the underlying lipid-rich core is exposed to flowing blood. Inflammatory processes and enzymes weaken the cap, and a rupture allows platelets to adhere and the coagulation cascade to activate, forming a thrombus. This clot can rapidly grow and block the vessel, cutting off blood flow and causing acute ischemia or infarction. Calcification, while a marker of advanced disease, tends to stiffen plaques and is not the immediate cause of occlusion. Endothelial dilation is not the characteristic event driving occlusion at this stage, and systemic hypertension is a risk factor that accelerates atherosclerosis but does not itself cause the acute occlusive event.

4. Phase 0 of the SA node action potential occurs due to entry of which ion?

A. Calcium enters

B. Sodium enters

C. Potassium leaves

D. Chloride enters

Phase 0 in the SA node is driven by calcium entry through L-type calcium channels. Pacemaker cells rely on Ca2+ for the rapid depolarization because they lack the fast Na+ current that drives phase 0 in ventricular tissue. The Ca2+ influx raises the membrane potential to initiate the upstroke. Sodium entering would mainly depolarize in non-pacemaker heart tissue, potassium leaving would hyperpolarize/repolarize, and chloride influx isn't responsible for this rapid depolarization.

5. Which valve is between the right ventricle and the pulmonary artery?

A. Aortic valve

B. Tricuspid valve

C. Pulmonary valve

D. Mitral valve

The valve between the right ventricle and the pulmonary artery is the pulmonary valve. It is a semilunar valve located at the outflow tract from the right ventricle, and it opens to allow blood to flow into the pulmonary artery during systole. When the right ventricle relaxes (diastole), the valve closes to prevent blood from flowing back into the ventricle. This helps direct blood toward the lungs for oxygenation. In contrast, the aortic valve sits between the left ventricle and the aorta, the tricuspid valve lies between the right atrium and right ventricle, and the mitral valve lies between the left atrium and left ventricle.

6. PCSK9 inhibitors such as Alirocumab and Evolocumab work by which mechanism?

A. Inhibits HMG-CoA reductase

B. Blocks destruction of LDL receptors

C. Blocks absorption of cholesterol in the gut

D. Increases LDL receptor degradation

PCSK9 inhibitors work by preserving LDL receptors on liver cells. Normally, LDL receptors bind LDL and recycle to the cell surface to keep clearing LDL from the blood, but PCSK9 binds these receptors and sends them to degradation, reducing their numbers. Alirocumab and evolocumab are antibodies that bind PCSK9, blocking its interaction with the receptor. This prevents receptor degradation, increases the amount of LDL receptor on the liver surface, and enhances LDL clearance from the bloodstream, lowering LDL cholesterol. Inhibiting HMG-CoA reductase is how statins work, blocking intestinal cholesterol absorption is the action of ezetimibe, and increasing LDL receptor degradation would decrease receptor availability—opposite of what PCSK9 inhibitors achieve.

7. Which cells become foam cells as oxidized LDL is ingested but not digested?

A. Macrophages

B. T-lymphocytes

C. Endothelial cells

D. Platelets

Foam cells arise when macrophages take up oxidized LDL through scavenger receptors in an unregulated way. Oxidized LDL is not efficiently processed and cleared, so cholesterol esters accumulate inside the macrophage, filling it with lipid droplets and giving it a foamy appearance. This lipid-laden state is a hallmark of early atherosclerotic plaques. Other immune cells like T-lymphocytes are involved in inflammation but don't become foam cells by ingesting LDL. Endothelial cells can take up some lipids and participate in vascular function, but they don't turn into foam cells. Platelets are involved in clotting, not lipid storage. Thus, macrophages are the cells that become foam cells.

8. Constrictive pericarditis results from

A. Increased diastolic filling

B. Pericardial inflammation with effusion

C. Fibrous pericardium becomes inelastic, restricting diastolic filling

D. Resolution with NSAIDs

Constrictive pericarditis occurs when the pericardium becomes thickened and scarred, turning it into an inelastic shell. That rigid pericardium can't expand during diastole, so diastolic filling is restricted and pressures rise early in filling. This leads to signs of venous congestion and the characteristic hemodynamics of constriction. NSAIDs address inflammation in acute or inflammatory pericarditis, but they don't reverse established fibrous constriction. A pericardial effusion causing tamponade is a different problem, where fluid compresses the heart rather than a stiffened pericardium restricting filling. The defining feature here is the inelastic pericardium that limits diastolic filling, which is why this mechanism best explains constrictive physiology.

9. Which statement best describes the Frank-Starling mechanism?

A. Increase in afterload

B. Increase in end-diastolic volume

C. Decrease in preload

D. Increase in heart rate

Frank-Starling describes how the heart's stroke volume increases as the ventricle fills more during diastole. When the end-diastolic volume rises, the ventricular muscle stretches more, bringing actin and myosin to a length that produces a stronger contraction. That stronger contraction ejects more blood, increasing stroke volume and cardiac output within physiological limits. This mechanism centers on preload—the amount of filling measured by end-diastolic volume. Increasing afterload would make it harder to eject blood and can lower stroke volume; decreasing preload reduces stretch and weakens contraction; and heart rate changes involve separate control mechanisms rather than the intrinsic stretch–contraction relationship of the Frank-Starling mechanism.

10. Atropine increases heart rate by blocking which receptor?

A. Beta-1 receptor

B. M2 receptor

C. Alpha-1 receptor

D. D2 receptor

Atropine increases heart rate by blocking parasympathetic signaling at the muscarinic receptors in the heart, specifically the M2 subtype. Normally, acetylcholine from the vagus nerve binds M2 receptors on the SA and AV nodes and slows heart rhythm. By blocking these receptors, atropine removes this braking effect, allowing the heart to beat faster. The other receptors listed aren't responsible for this action: blocking beta-1 would tend to reduce heart rate, while alpha-1 and D2 don't directly control cardiac pacemaker activity.

Next Steps

Congratulations on reaching the final section of this guide. You've taken a meaningful step toward passing your certification exam and advancing your career.

As you continue preparing, remember that consistent practice, review, and self-reflection are key to success. Make time to revisit difficult topics, simulate exam conditions, and track your progress along the way.

If you need help, have suggestions, or want to share feedback, we'd love to hear from you. Reach out to our team at hello@examzify.com.

Or visit your dedicated course page for more study tools and resources:

https://omecardiacmodules.examzify.com

We wish you the very best on your exam journey. You've got this!