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.

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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!

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Questions

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1. What term describes the small vessels that supply the walls of large arteries and veins?

A. Lymphatics

B. Vasa vasorum

C. Capillaries

D. Venules

2. Benign hypertension is most commonly caused by which vascular change?

A. Hyaline arteriolosclerosis

B. Hyperplastic arteriolosclerosis

C. Atherosclerosis of large arteries

D. Cerebral atherosclerosis

3. The vegetation in infective endocarditis is composed of which components?

A. Bacteria alone

B. Platelets, fibrin, bacteria, and leukocytes

C. Myocytes

D. Lipids

4. Contractile cells include which components?

A. Atrial muscles, ventricular muscles, Purkinje system

B. SA node and AV node

C. Nodal tissue

D. Pacemaker cells

5. In edema, inflammatory edema is characterized by which vascular change?

A. Decreased capillary permeability

B. Increased capillary permeability

C. Increased lymphatic drainage

D. Venous thrombosis

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6. 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

7. In mitral stenosis, which two findings are classically described as opening snap and diastolic rumbling?

A. Opening snap and diastolic rumbling

B. S3 and diastolic rumbling

C. Opening snap and systolic murmur

D. S4 and continuous murmur

8. Phenylephrine is a selective alpha-1 agonist. Which best describes its hemodynamic effect?

A. Decreases pulse pressure

B. Increases heart rate

C. Increases SVR and diastolic pressure without affecting heart rate or contractility

D. Decreases SVR and lowers diastolic pressure

9. Pathophysiology of Tet spells includes which of the following?

A. Decrease vascular resistance with no change in pulmonary vascular resistance

B. Arterial dilation

C. Increase in aorta flow

D. Produced by heavy exercise

10. Which feature supplies perfusion to the wall of large arteries via the adventitia?

A. Vaso vasorum in the adventitia

B. Endothelial tight junctions

C. Internal elastic lamina

D. Tunica media smooth muscle

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Answers

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

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Explanations

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1. What term describes the small vessels that supply the walls of large arteries and veins?

A. Lymphatics

B. Vasa vasorum

C. Capillaries

D. Venules

The term is vasa vasorum. Large arteries and veins have thick walls, and diffusion from the blood inside the lumen can't reach all the outer layers, so a tiny network of vessels runs within the vessel wall itself to nourish it. These microvessels—the vasa vasorum—supply oxygen and nutrients to the media and adventitia, keeping the wall healthy. Lymphatics carry lymph, not blood to vessel walls, so they don't nourish the walls. Capillaries are the exchange vessels that feed surrounding tissues, but they don't serve the walls of large vessels. Venules are small veins that drain capillary beds, not the vessels that supply the vessel walls.

2. Benign hypertension is most commonly caused by which vascular change?

A. Hyaline arteriolosclerosis

B. Hyperplastic arteriolosclerosis

C. Atherosclerosis of large arteries

D. Cerebral atherosclerosis

In benign essential hypertension, the vessels most characteristically show hyaline arteriolosclerosis. This involves small arteries and arterioles developing a homogeneous, glassy pink thickening of the wall due to leakage of plasma proteins into the vessel wall and increased production of extracellular matrix by smooth muscle cells. The result is a narrowed lumen and higher vascular resistance, which is a hallmark of long-standing, nonmalignant hypertension. This microvascular change helps explain why prolonged hypertension can lead to target-organ damage such as nephrosclerosis and retinopathy. By contrast, hyperplastic arteriolosclerosis with onion-skinning is associated with malignant hypertension, while atherosclerosis of large arteries or cerebral arteries involves larger vessels and is not the typical change seen in benign hypertension.

3. The vegetation in infective endocarditis is composed of which components?

A. Bacteria alone

B. Platelets, fibrin, bacteria, and leukocytes

C. Myocytes

D. Lipids

Infective endocarditis vegetations are composite masses made up of a fibrin-platelet scaffold that traps bacteria and inflammatory cells. Endothelial injury on a valve starts the process by promoting deposition of fibrin and platelets, forming a sterile thrombus. When bacteria enter the bloodstream, they adhere to this thrombus and proliferate, while immune cells (leukocytes) are recruited to the site. The result is a friable mass consisting of platelets, fibrin, bacteria, and leukocytes. Lipids or heart muscle cells don't form the main structure of these vegetations, and bacteria alone wouldn't explain the thrombotic framework and immune cell presence.

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4. Contractile cells include which components?

A. Atrial muscles, ventricular muscles, Purkinje system

B. SA node and AV node

C. Nodal tissue

D. Pacemaker cells

The main idea is distinguishing the heart's pumping muscle from its electrical wiring. Contractile cells are the muscle fibers that actually shorten to push blood—the atrial and ventricular myocardium. The Purkinje network is a specialized conducting system that rapidly carries electrical impulses to coordinate timing of contraction, but it does not itself contract. The SA node, AV node, nodal tissue, and pacemaker cells are all part of this electrical wiring, not the contracting muscle. So, contractile cells comprise the atrial and ventricular muscles, while the Purkinje system and other nodal/pacemaker tissues serve to control the rhythm and propagation of the heartbeat.

5. In edema, inflammatory edema is characterized by which vascular change?

A. Decreased capillary permeability

B. Increased capillary permeability

C. Increased lymphatic drainage

D. Venous thrombosis

Inflammatory edema is driven by a rise in capillary permeability. Inflammation releases mediators that make the endothelial cells that line capillaries separate and form gaps, allowing fluid and proteins to leak into the surrounding tissue. This protein-rich exudate swelling is the hallmark of inflammatory edema. Increased lymphatic drainage, while helpful for clearing excess fluid, is not the vascular change that defines this type of edema, and venous thrombosis causes edema through impaired venous return rather than the inflammatory permeability change.

6. 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.

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7. In mitral stenosis, which two findings are classically described as opening snap and diastolic rumbling?

A. Opening snap and diastolic rumbling

B. S3 and diastolic rumbling

C. Opening snap and systolic murmur

D. S4 and continuous murmur

Mitral stenosis narrows the valve so blood flow from the left atrium to the left ventricle during diastole becomes turbulent and the valve leaflets are stiff. After S2, the valve opens abruptly, producing an opening snap as the stiff leaflets finally give way. The ongoing diastolic flow across the narrowed orifice then creates a low-pitched, rumbling murmur best heard at the apex in the left lateral decubitus position, often peaking toward the end of diastole. This pairing of an opening snap with a diastolic rumble is classic for mitral stenosis. Other findings described in the distractors—such as S3 (volume overload), S4 (stiff ventricle), systolic murmurs (often indicating regurgitation), or continuous murmurs (as with PDA or other shunts)—do not define mitral stenosis in the same way.

8. Phenylephrine is a selective alpha-1 agonist. Which best describes its hemodynamic effect?

A. Decreases pulse pressure

B. Increases heart rate

C. Increases SVR and diastolic pressure without affecting heart rate or contractility

D. Decreases SVR and lowers diastolic pressure

Phenylephrine acts mainly on vascular smooth muscle via alpha-1 receptors, causing widespread vasoconstriction. This raises systemic vascular resistance and increases arterial tone, especially diastolic pressure. Because it has no beta-adrenergic activity, it does not directly increase heart rate or myocardial contractility. Any heart-rate change is usually a reflex response to the higher blood pressure rather than a direct effect of the drug. So the best description is that it increases SVR and diastolic pressure without directly affecting heart rate or contractility. The other statements conflict with its lack of direct cardiac stimulation and its primary vasoconstrictive action.

9. Pathophysiology of Tet spells includes which of the following?

A. Decrease vascular resistance with no change in pulmonary vascular resistance

B. Arterial dilation

C. Increase in aorta flow

D. Produced by heavy exercise

Tet spells occur when there is a sudden increase in right-to-left shunting across the ventricular septal defect in Tetralogy of Fallot. This is driven mainly by a drop in systemic vascular resistance (afterload) with little or no change in pulmonary vascular resistance. When SVR falls, the pressure balance across the VSD shifts to favor deoxygenated blood shunting into the systemic circulation, especially given the right ventricular outflow tract obstruction that already raises RV pressure. Since pulmonary vascular resistance stays the same, there isn't an accompanying increase in pulmonary blood flow to oxygenate that blood, so systemic hypoxemia worsens during the spell.

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10. Which feature supplies perfusion to the wall of large arteries via the adventitia?

A. Vaso vasorum in the adventitia

B. Endothelial tight junctions

C. Internal elastic lamina

D. Tunica media smooth muscle

Perfusion of the wall in large arteries is provided by the vasa vasorum, tiny vessels that run through the adventitia and often into the outer layers of the media. Because the wall is thick, diffusion from the lumen can't nourish the outer regions, so these small vessels deliver oxygen and nutrients directly to the outer media and adventitia. The endothelial tight junctions belong to the barrier between the bloodstream and tissue and aren't responsible for nourishing the wall. The internal elastic lamina is a structural boundary between the intima and media, not a blood-supplying network. The tunica media's smooth muscle forms the muscular layer that regulates diameter but does not itself supply blood to the wall.

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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!

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