MEHLMAN CARDIOLOGY PRACTICE EXAM (SAMPLE)

STUDY GUIDE

SAMPLE STUDY GUIDE WITH LIMITED QUESTIONS

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Table of Contents

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. **A patient with STEMI two days ago now has a central friction rub. What is the most likely diagnosis?**

   A. Pericarditis Due to Autoimmune Disease

   B. Uremic Pericarditis

   C. Post-MI Fibrinous Pericarditis

   D. Dressler Syndrome

2. **In congenital rubella syndrome, which cardiovascular defect is classically associated?**

   A. ASD

   B. VSD

   C. TOF

   D. PDA

3. **Familial hypercholesterolemia is most commonly due to which defect?**

   A. LDL receptor deficiency

   B. Hypermastik

   C. Lipase deficiency

   D. Apolipoprotein E deficiency

4. **In a 40-year-old man with a history of rheumatic fever as a child and a rumbling diastolic murmur, which finding in childhood history is most likely?**

   A. Aortic stenosis

   B. Mitral regurgitation

   C. Pulmonary embolism

   D. Hypertrophic cardiomyopathy

5. **Which fluoroquinolone is commonly used for pneumonia, including HAP?**

   A. Ciprofloxacin

   B. Levofloxacin

   C. Moxifloxacin

   D. Ofloxacin

6. A 44-year-old man with hematuria and hemoptysis most strongly suggests which syndrome?

A. Goodpasture syndrome

B. Granulomatosis with polyangiitis

C. Churg-Strauss

D. Microscopic polyangiitis

7. Which combination among common heart failure medications is NOT shown to reduce mortality?

A. Digoxin and furosemide

B. ACE inhibitors and beta-blockers

C. ARBs and sacubitril

D. Spironolactone and eplerenone

8. Myositis and rhabdomyolysis risk increases when fibrates are used with statins due to which mechanism?

A. Inhibition of P450 enzymes

B. Increased hepatic uptake of statins

C. Induction of P-glycoprotein

D. Decreased statin bioavailability

9. Which drug is NOT a class III antiarrhythmic?

A. Amiodarone

B. Dofetilide

C. Sotalol

D. Lidocaine

10. Polyarteritis nodosa is best described as which type of vasculitis?

A. Large-vessel vasculitis

B. Small-vessel vasculitis with eosinophilia

C. Medium-vessel vasculitis characterized by immune complex deposition and fibrinoid necrosis

D. Capillaritis with alveolar hemorrhage

Answers

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

Explanations

1. A patient with STEMI two days ago now has a central friction rub. What is the most likely diagnosis?

A. Pericarditis Due to Autoimmune Disease

B. Uremic Pericarditis

C. Post-MI Fibrinous Pericarditis

D. Dressler Syndrome

Fibrinous pericarditis occurring after a myocardial infarction. After a STEMI, the inflammatory process can extend to the pericardium, causing sterile fibrinous exudate and a pericardial friction rub within the first day to a few days. This early post–MI pericarditis typically presents around 1–3 days after the infarct, which fits the scenario of symptoms appearing two days after the MI. Dressler syndrome is a later autoimmune pericarditis occurring weeks after the event, while uremic or autoimmune pericarditis is not tied to recent MI timing. So the timing and the finding point to post MI fibrinous pericarditis.

2. In congenital rubella syndrome, which cardiovascular defect is classically associated?

A. ASD

B. VSD

C. TOF

D. PDA

The key idea is that congenital rubella syndrome classically affects the ductus arteriosus, leaving it open after birth. Rubella infection during early pregnancy disrupts the development and closure mechanisms of the ductus arteriosus, so the vessel fails to constrict and remains patent. Clinically this gives a continuous machinery-type murmur and signs of a left-to-right shunt. While other heart defects like ASD or VSD can occur in rubella, they are not as characteristic as a persistently open ductus. Tetralogy of Fallot is not typically associated with congenital rubella. The classic rubella-associated cardiovascular finding is patent ductus arteriosus.

3. Familial hypercholesterolemia is most commonly due to which defect?

A. LDL receptor deficiency

B. Hypermastik

C. Lipase deficiency

D. Apolipoprotein E deficiency

Familial hypercholesterolemia most commonly results from defective LDL receptor function, which impairs clearance of LDL cholesterol from the bloodstream. When hepatocyte LDL receptors are reduced in number or work poorly due to LDLR gene mutations, LDL remains circulating at high levels from birth, driving very high LDL cholesterol and accelerating atherosclerosis. The body's normal route to remove LDL is via these receptors that bind apolipoprotein B-100 on LDL particles and internalize them into liver cells; if this pathway is compromised, LDL accumulates. That's why this defect is the classic cause of familial hypercholesterolemia. In contrast, a lipase deficiency tends to raise triglycerides, not LDL; apolipoprotein E deficiency leads to a different dyslipoproteinemia (type III) with abnormal remnants, and "hypermastik" isn't a recognized mechanism for FH.

4. In a 40-year-old man with a history of rheumatic fever as a child and a rumbling diastolic murmur, which finding in childhood history is most likely?

A. Aortic stenosis

B. Mitral regurgitation

C. Pulmonary embolism

D. Hypertrophic cardiomyopathy

The key idea is that rheumatic heart disease often starts with mitral valve inflammation during the acute rheumatic fever episode, leading to mitral regurgitation. Over years this valve can progress to chronic scarring and fusion of the valve apparatus, resulting in mitral stenosis a long time later, which produces the rumbling diastolic murmur heard at the apex. So in someone who currently has a diastolic rumble (mitral stenosis) and a history of rheumatic fever in childhood, it's most likely that, during childhood, there was an episode of rheumatic fever with mitral valve involvement presenting as mitral regurgitation. The other conditions don't fit this pattern of rheumatic valve disease progression and don't explain the diastolic rumble.

5. Which fluoroquinolone is commonly used for pneumonia, including HAP?

A. Ciprofloxacin

B. Levofloxacin

C. Moxifloxacin

D. Ofloxacin

Levofloxacin is commonly used for pneumonia, including hospital-acquired pneumonia, because it combines broad spectrum activity with excellent lung penetration and convenient pharmacokinetics. It effectively covers the usual pneumonia pathogens—Streptococcus pneumoniae, Haemophilus influenzae, and atypicals such as Mycoplasma and Legionella—and can provide activity against Pseudomonas in some settings, making it a versatile option for both community and hospital cases. Its high lung tissue concentrations and good oral bioavailability allow it to be used as monotherapy in appropriate cases or as step-down therapy from IV to oral. In contrast, ciprofloxacin, while strong against Gram-negatives like Pseudomonas, has weaker coverage of S. pneumoniae; moxifloxacin has strong Gram-positive and anaerobic activity but limited Pseudomonas coverage, and ofloxacin has less favorable pharmacokinetics and spectrum. Thus levofloxacin best fits the needs for pneumonia management among the fluoroquinolones.

6. A 44-year-old man with hematuria and hemoptysis most strongly suggests which syndrome?

A. Goodpasture syndrome

B. Granulomatosis with polyangiitis

C. Churg-Strauss

D. Microscopic polyangiitis

Hemoptysis with hematuria reflects autoimmune injury affecting both the lungs and kidneys. Goodpasture syndrome arises from anti-GBM antibodies that attack basement membranes in these two organs, causing pulmonary hemorrhage and rapidly progressive glomerulonephritis. This direct, dual-target mechanism makes this combination the strongest clue. Clinically, you'd expect anti-GBM antibodies in the blood and linear IgG deposition along the basement membranes on kidney biopsy, with urgent therapy using plasmapheresis and immunosuppression. While other vasculitides can involve both systems, their patterns (such as granulomatous features in GPA or eosinophilia and asthma in EGPA) and serologies are different, making Goodpasture the best fit for this presentation.

7. Which combination among common heart failure medications is NOT shown to reduce mortality?

A. Digoxin and furosemide

B. ACE inhibitors and beta-blockers

C. ARBs and sacubitril

D. Spironolactone and eplerenone

Not every heart failure medication reduces mortality; only certain drug classes have proven survival benefit. In systolic heart failure, mortality reduction is shown with ACE inhibitors or ARBs, beta-blockers, and aldosterone antagonists, and more recently with sacubitril/valsartan. Digoxin improves symptoms and can reduce hospitalizations, but it has not been shown to decrease mortality. Loop diuretics like furosemide relieve fluid overload and symptoms without a proven effect on survival. Therefore, the combination of digoxin and furosemide is not associated with reduced mortality, unlike the other pairings.

8. Myositis and rhabdomyolysis risk increases when fibrates are used with statins due to which mechanism?

A. Inhibition of P450 enzymes

B. Increased hepatic uptake of statins

C. Induction of P-glycoprotein

D. Decreased statin bioavailability

The increased risk of myositis and rhabdomyolysis when fibrates are combined with statins comes from a pharmacokinetic interaction that raises statin exposure. Gemfibrozil can inhibit hepatic uptake and metabolism of statins by blocking transporters like OATP1B1 and by interfering with glucuronidation pathways. This leads to higher circulating statin levels, which increases the amount of drug reaching skeletal muscle and raises the risk of muscle injury. Inducing P-glycoprotein would generally decrease drug exposure by promoting efflux, not increase it, so it wouldn't explain the heightened myopathy risk. The key idea is that reduced hepatic clearance and higher statin concentrations drive the muscle toxicity risk.

9. Which drug is NOT a class III antiarrhythmic?

A. Amiodarone

B. Dofetilide

C. Sotalol

D. Lidocaine

Class III antiarrhythmics slow repolarization by blocking potassium channels, lengthening the ventricular action potential and the QT interval. Lidocaine does not fit this mechanism. It is a class Ib sodium channel blocker that mainly affects sodium influx during depolarization, especially in ischemic tissue, and has little effect on the duration of the action potential in normal myocardium. Therefore, lidocaine is not a class III antiarrhythmic. The other drugs listed act by blocking potassium channels and prolonging repolarization, which is characteristic of class III agents.

10. Polyarteritis nodosa is best described as which type of vasculitis?

A. Large-vessel vasculitis

B. Small-vessel vasculitis with eosinophilia

C. Medium-vessel vasculitis characterized by immune complex deposition and fibrinoid necrosis

D. Capillaritis with alveolar hemorrhage

Medium-sized vessel vasculitis with immune complex–mediated transmural inflammation and fibrinoid necrosis describes polyarteritis nodosa best. In PAN, the disease targets medium-sized muscular arteries, causing transmural inflammation that can lead to aneurysms and stenoses in organs such as the kidneys, gut, skin, and nerves. The lungs are typically spared, and there is no capillaritis, which helps distinguish PAN from small-vessel diseases that can involve the lungs. This pattern—immune complex deposition with fibrinoid necrosis in medium-sized arteries—fits PAN more accurately than large-vessel vasculitis (which affects the aorta and major branches) or small-vessel eosinophilic vasculitis/capillaritis.

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://mehlmancardiology.examzify.com

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