

# Cardiac Medicine Certification (CMC) Practice Exam (Sample)

## Study Guide



**Everything you need from our exam experts!**

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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. Name the three classic symptoms of a myocardial infarction.**
  - A. Chest pain, nausea, and dizziness**
  - B. Chest pain, shortness of breath, and sweating**
  - C. Fatigue, palpitations, and anxiety**
  - D. Arm pain, jaw pain, and headache**
- 2. The area from the end of the S wave to the end of the T wave on an ECG represents:**
  - A. Atrial depolarization**
  - B. Ventricular depolarization**
  - C. Ventricular repolarization**
  - D. Resting potential**
- 3. What is the advantage of using dual-antiplatelet therapy in post-stenting patients?**
  - A. Increases blood pressure**
  - B. Reduces risk of thrombosis and recurrent myocardial infarction**
  - C. Enhances cholesterol levels**
  - D. Promotes faster healing of the artery**
- 4. How is a myocardial infarction classically diagnosed?**
  - A. Chest X-ray findings**
  - B. Elevated cardiac enzymes and an ECG showing ST-segment elevation**
  - C. Coronary angiography results**
  - D. Physical examination findings**
- 5. What lifestyle modification is crucial for patients with heart failure?**
  - A. Increased exercise**
  - B. Sodium restriction**
  - C. High-protein diet**
  - D. Frequent small meals**

- 6. Which of the following is a potential cause of ventricular tachycardia?**
- A. Hypertension and hyperglycemia**
  - B. Hypokalemia and myocardial infarction**
  - C. Chronic anxiety and sleep apnea**
  - D. Low calcium and high sodium levels**
- 7. What medications are commonly used to lower cardiac workload and prevent reinfarction in patients with STEMI?**
- A. Aspirin and Plavix**
  - B. Beta-blockers and ACE inhibitors**
  - C. Nitrates and statins**
  - D. Calcium channel blockers and anticoagulants**
- 8. During an anteroseptal wall myocardial infarction, which ECG changes are most commonly observed?**
- A. ST elevation in V1-V3**
  - B. ST depression in V5-V6**
  - C. ST elevation in II, III, and aVF**
  - D. No significant ECG changes**
- 9. How is ventricular tachycardia characterized?**
- A. Heart rate between 60 and 100 bpm**
  - B. Multiple PVCs at a rate of 100 to 250 bpm**
  - C. Regular p-wave to QRS ratio**
  - D. Presence of sinus bradycardia**
- 10. In 2nd degree AV block Type I (Wenckebach), how is the PR interval affected?**
- A. It remains constant throughout the rhythm.**
  - B. It gradually lengthens before a dropped QRS complex.**
  - C. It is shorter than 0.12 seconds.**
  - D. It is consistently longer than 0.20 seconds.**



## **Answers**

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

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## **Explanations**

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**1. Name the three classic symptoms of a myocardial infarction.**

- A. Chest pain, nausea, and dizziness
- B. Chest pain, shortness of breath, and sweating**
- C. Fatigue, palpitations, and anxiety
- D. Arm pain, jaw pain, and headache

The three classic symptoms of a myocardial infarction, commonly known as a heart attack, are chest pain, shortness of breath, and sweating. Chest pain is typically described as a feeling of pressure, squeezing, or fullness in the chest and is often the most recognized symptom. Shortness of breath may occur alongside or independently from chest pain, indicating that the heart is not pumping effectively. Sweating, particularly cold sweats, is also a common response during a myocardial infarction, driven by stress on the body and the autonomic nervous system response to the acute event. Other symptoms, such as fatigue, palpitations, anxiety, arm pain, jaw pain, and headache, can also be associated with heart conditions, but they do not represent the classic triad typically used to identify myocardial infarction. Recognizing the classic symptoms is crucial for timely medical intervention and can significantly affect patient outcomes.

**2. The area from the end of the S wave to the end of the T wave on an ECG represents:**

- A. Atrial depolarization
- B. Ventricular depolarization
- C. Ventricular repolarization**
- D. Resting potential

The area from the end of the S wave to the end of the T wave on an ECG represents ventricular repolarization. This interval is defined as the ST segment and the T wave, which together signify the recovery phase of the ventricles after they have contracted. During this phase, the ventricles are in the process of returning to their resting state after depolarization and contraction. The S wave marks the end of ventricular depolarization, while the T wave signifies the completion of the repolarization process. Understanding this phase is critical, as it can provide insights into the heart's electrical activity and how well it is functioning. Additionally, the other areas mentioned in the choices are distinct and do not overlap with the interval representing ventricular repolarization. Atrial depolarization occurs prior to the onset of the QRS complex, while ventricular depolarization itself is represented by the entire QRS complex. Resting potential refers to the state of the cardiac cells when they are not conducting electrical impulses, which is not specifically represented by any particular segment on the ECG. Thus, recognizing that the time from the end of the S wave to the end of the T wave specifically indicates the ventricular repolarization phase is key.

### 3. What is the advantage of using dual-antiplatelet therapy in post-stenting patients?

- A. Increases blood pressure
- B. Reduces risk of thrombosis and recurrent myocardial infarction**
- C. Enhances cholesterol levels
- D. Promotes faster healing of the artery

Using dual-antiplatelet therapy in post-stenting patients significantly reduces the risk of thrombosis and recurrent myocardial infarction. This is due to the combined action of two antiplatelet medications, which work together to inhibit platelet aggregation more effectively than a single agent. After a stent placement, there is a heightened risk of clot formation at the site, which can lead to serious complications such as stent thrombosis or another myocardial infarction. By employing dual-antiplatelet therapy, typically consisting of aspirin and a P2Y12 inhibitor, clinicians aim to maintain vascular patency and minimize these risks, which is crucial for the long-term success of the stent and the overall health of the patient. The other options, while relevant to different aspects of cardiac health, do not reflect the specific benefit of dual-antiplatelet therapy in this context. Enhancing cholesterol levels or blood pressure management are not direct actions of dual-antiplatelet therapy, nor does this therapeutic approach directly promote faster healing of the artery. Instead, the primary focus of dual-antiplatelet therapy is to prevent thrombotic events that could arise following the placement of a stent. Thus, option B rightly highlights the purpose and advantage of this important post

### 4. How is a myocardial infarction classically diagnosed?

- A. Chest X-ray findings
- B. Elevated cardiac enzymes and an ECG showing ST-segment elevation**
- C. Coronary angiography results
- D. Physical examination findings

A myocardial infarction, commonly referred to as a heart attack, is classically diagnosed through a combination of clinical features, elevated cardiac enzymes, and specific changes on an electrocardiogram (ECG). The most telling indicators are elevated cardiac biomarkers, such as troponins, which indicate damage to heart muscle, along with an ECG that shows ST-segment elevation. This ST-segment elevation is indicative of acute myocardial ischemia, typically associated with a more serious form of heart attack known as ST-elevation myocardial infarction (STEMI). While chest X-ray findings may show cardiac silhouette or signs of heart failure, they do not provide definitive information about myocardial ischemia. Coronary angiography, although valuable for confirming the presence and extent of coronary artery disease, is not used as a primary diagnostic tool for an acute myocardial infarction. It generally follows laboratory and ECG findings for further evaluation or intervention. Similarly, while physical examination findings can provide clues - such as signs of distress or abnormal heart sounds - they are not definitive for diagnosing a myocardial infarction. Therefore, the combination of elevated cardiac enzymes and an ECG showing ST-segment elevation is the classic diagnostic approach for myocardial infarction, as it directly indicates myocardial damage and the urgency for treatment

**5. What lifestyle modification is crucial for patients with heart failure?**

- A. Increased exercise**
- B. Sodium restriction**
- C. High-protein diet**
- D. Frequent small meals**

Sodium restriction is a crucial lifestyle modification for patients with heart failure because it helps to manage fluid retention and control blood pressure. In heart failure, the heart's ability to pump blood is compromised, leading to congestion and swelling in the body. Excess sodium can increase fluid retention, worsening symptoms like edema and shortness of breath. By reducing sodium intake, patients can help decrease the workload on the heart, improve symptoms, and enhance their overall quality of life. Managing sodium intake is essential in creating a dietary plan that supports the treatment of heart failure. Health care providers often recommend a low-sodium diet, generally suggesting less than 2,000 milligrams of sodium per day. This dietary approach aids in preventing hospitalizations due to fluid overload and promotes a more effective management of the condition. The other lifestyle modifications, while potentially beneficial to overall health, do not address the immediate concerns associated with heart failure as directly as sodium restriction does. Increased exercise may not be appropriate for all heart failure patients, particularly those with severe symptoms. A high-protein diet is not a standard recommendation for heart failure unless warranted by other conditions. Frequent small meals may help some patients manage symptoms but do not have the same impact on fluid retention and heart workload as sodium restriction.

**6. Which of the following is a potential cause of ventricular tachycardia?**

- A. Hypertension and hyperglycemia**
- B. Hypokalemia and myocardial infarction**
- C. Chronic anxiety and sleep apnea**
- D. Low calcium and high sodium levels**

Ventricular tachycardia (VT) is a potentially life-threatening arrhythmia characterized by a fast and abnormal heart rhythm originating from the ventricles. Understanding the causes of VT is essential for managing patients at risk. The presence of hypokalemia — a condition characterized by low potassium levels in the blood — is known to predispose individuals to arrhythmias, including VT. Potassium is crucial for maintaining the electrical stability of cardiac cells, and deficiencies can lead to disturbances in the heart's rhythm. Myocardial infarction, or heart attack, is another significant risk factor for the development of VT. Damage to the heart muscle can disrupt the normal electrical conduction pathways, leading to altered depolarization and increased susceptibility to arrhythmias. The scarred tissue from a myocardial infarction can create areas of re-entry, resulting in VT. While hypertension and hyperglycemia, chronic anxiety and sleep apnea, as well as low calcium and high sodium levels can affect heart health, they are not as directly linked to the acute induction of ventricular tachycardia as hypokalemia and myocardial infarction are. Understanding these associations helps medical professionals monitor and treat patients effectively to prevent serious complications related to arrhythmias.

**7. What medications are commonly used to lower cardiac workload and prevent reinfarction in patients with STEMI?**

**A. Aspirin and Plavix**

**B. Beta-blockers and ACE inhibitors**

**C. Nitrates and statins**

**D. Calcium channel blockers and anticoagulants**

In patients with ST-Elevation Myocardial Infarction (STEMI), the management aims to lower cardiac workload and prevent reinfarction. Beta-blockers are commonly used in these cases due to their ability to reduce heart rate and myocardial oxygen demand, which alleviates stress on the heart. By blocking the effects of adrenaline and norepinephrine on the heart, beta-blockers help to diminish the risk of arrhythmias and subsequent cardiac events. ACE inhibitors play a crucial role as well, especially in patients with left ventricular dysfunction or heart failure. They help relax blood vessels, reducing preload and afterload on the heart, and consequently, they decrease myocardial oxygen demand. Furthermore, ACE inhibitors provide renal protection and have been shown to improve post-infarction outcomes, thereby further preventing reinfarction. In contrast, while other options may play important roles in the overall management of STEMI, they do not primarily focus on lowering cardiac workload in the same manner as beta-blockers and ACE inhibitors. Aspirin and Plavix primarily function to prevent platelet aggregation and thus reduce thrombus formation, while nitrates also help alleviate chest pain but are not the mainstay for long-term management in STEMI. Calcium channel blockers and anticoagulants have

**8. During an anteroseptal wall myocardial infarction, which ECG changes are most commonly observed?**

**A. ST elevation in V1-V3**

**B. ST depression in V5-V6**

**C. ST elevation in II, III, and aVF**

**D. No significant ECG changes**

During an anteroseptal wall myocardial infarction, the most commonly observed ECG changes are characterized by ST elevation in the precordial leads, particularly in leads V1-V3. These leads are positioned over the anterior part of the heart, where the left anterior descending artery (LAD) supplies blood. Anteroseptal infarctions usually occur due to occlusion of this artery, leading to ischemia and subsequent elevation of the ST segment in the affected leads. In detail, the presence of ST elevation in V1-V3 indicates that myocardial injury is occurring in the anterior and septal portions of the left ventricle. This finding is critical in diagnosing the specific type of myocardial infarction and guiding treatment. Medically, recognizing these changes can prompt immediate interventions, such as coronary angioplasty or thrombolysis, to restore blood flow and minimize damage to the heart muscle. The other options do not correspond to the typical ECG changes observed in an anteroseptal myocardial infarction. ST depression in leads V5-V6 is typically associated with lateral wall ischemia rather than anteroseptal involvement. ST elevation in II, III, and aVF indicates an inferior wall MI, usually due to right coronary artery oc

## 9. How is ventricular tachycardia characterized?

- A. Heart rate between 60 and 100 bpm
- B. Multiple PVCs at a rate of 100 to 250 bpm**
- C. Regular p-wave to QRS ratio
- D. Presence of sinus bradycardia

Ventricular tachycardia is characterized primarily by the presence of multiple premature ventricular contractions (PVCs) occurring at a rapid rate, specifically between 100 and 250 beats per minute. This arrhythmia originates from the ventricles, and the rapid firing of the ventricular ectopic pacemakers leads to this elevated heart rate. The distinguishing feature of ventricular tachycardia is that it often has a wide QRS complex, reflecting the abnormal conduction pathway through the ventricles due to the ectopic focus. When observing patients with this tachycardia, one may notice that the rhythm is typically regular, and the QRS complexes are usually wider than those seen in normal sinus rhythm. In contrast, the other options provided do not accurately represent ventricular tachycardia. A heart rate between 60 and 100 beats per minute indicates a normal sinus rhythm rather than tachycardia. The regular p-wave to QRS ratio is typically present in normal rhythms but is absent in ventricular tachycardia, where atrial activity may be dissociated from ventricular contractions. Lastly, sinus bradycardia signifies a slower than normal heart rate, which is the opposite of what is seen in ventricular tachycardia. Therefore,

## 10. In 2nd degree AV block Type I (Wenckebach), how is the PR interval affected?

- A. It remains constant throughout the rhythm.
- B. It gradually lengthens before a dropped QRS complex.**
- C. It is shorter than 0.12 seconds.
- D. It is consistently longer than 0.20 seconds.

In 2nd degree AV block Type I, also known as Wenckebach, the defining characteristic is that the PR interval progressively lengthens with each successive heartbeat until a QRS complex is finally dropped. This phenomenon is referred to as "grouped beating" or "progressive block." The gradual elongation of the PR interval is a reflection of the increasing delay in conduction through the AV node. As the cycle continues, this progressive lengthening continues until the electrical impulse fails to transmit, leading to a missed or dropped QRS complex. Following this, the cycle resets, and the pattern repeats. This pattern of progressively increasing intervals followed by a dropped beat is what differentiates Wenckebach from other types of AV block, making it crucial to recognize the specific behavior of the PR interval in this scenario.



## 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](mailto:hello@examzify.com).**

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

**<https://cmc-certification.examzify.com>**

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