

Cardiac Electrophysiology Practice Test (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

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- 1. Abnormal pacemaker sites such as ectopic foci illustrate which kind of conduction issue?**
 - A. Normal conduction**
 - B. Functional abnormalities**
 - C. Anatomical abnormalities**
 - D. Electrical remodeling without functional impact**

- 2. T-type channel is described as**
 - A. Transient current; contributes to early phase 4 pacemaker currents in SA and AV nodal cells**
 - B. Continuous current for plateau**
 - C. Inactivated during depolarization**
 - D. Only in ventricular myocytes**

- 3. What is the effect of sympathetic tone on heart rate?**
 - A. Increases HR**
 - B. Decreases HR**
 - C. No effect on HR**
 - D. Causes irregular rhythm**

- 4. The Na/Ca exchanger operates with a stoichiometry of 1 calcium to 3 sodium and can move ions in either direction. Which statement best describes this?**
 - A. 1 Ca²⁺ per 3 Na⁺ in either direction**
 - B. 1 Ca²⁺ per 2 Na⁺ in either direction**
 - C. 2 Ca²⁺ per 3 Na⁺ in either direction**
 - D. 1 Ca²⁺ per 4 Na⁺ in either direction**

- 5. Transient outward channels contribute to which phase of non-pacemaker APs?**
 - A. Phase 1**
 - B. Phase 2**
 - C. Phase 3**
 - D. Phase 0**

- 6. Which stimulus increases ERP in AV nodal tissue?**
- A. Vagal activation**
 - B. Sympathetic activation**
 - C. Ischemia**
 - D. Exercise**
- 7. Abnormal conduction can be caused by which mechanism?**
- A. Reentry**
 - B. Automaticity**
 - C. Atrioventricular block**
 - D. Sinus node dysfunction**
- 8. Changes in autonomic function can do what to reentry?**
- A. Initiate or stop reentry**
 - B. Always increase reentry**
 - C. Have no effect**
 - D. Cause immediate arrest**
- 9. Which of the following is an example of an anatomic abnormality that can cause abnormal conduction?**
- A. Congenital accessory pathways**
 - B. Degenerative disease**
 - C. Atherosclerotic plaque**
 - D. Postoperative scar**
- 10. Sympathetic activation produces positive chronotropy. What does this mean for heart rate?**
- A. Increases heart rate**
 - B. Decreases heart rate**
 - C. No change in heart rate**
 - D. Increases contractility (inotropy)**

Answers

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

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Explanations

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1. Abnormal pacemaker sites such as ectopic foci illustrate which kind of conduction issue?

A. Normal conduction

B. Functional abnormalities

C. Anatomical abnormalities

D. Electrical remodeling without functional impact

Ectopic pacemaker sites show the heart's electrical system behaving abnormally in its function rather than due to a fixed structure. These foci generate impulses outside the normal SA node because of altered automaticity or triggered activity, a reversible and dynamic change in conduction properties. That's why this represents a functional abnormality: the issue lies in how the tissue conducts and generates impulses, not in an inherent anatomical lesion. Normal conduction would have impulses strictly from the SA node; anatomical abnormalities would imply a structural lesion; remodeling without functional impact would not cause a change in impulse generation or conduction behavior.

2. T-type channel is described as

A. Transient current; contributes to early phase 4 pacemaker currents in SA and AV nodal cells

B. Continuous current for plateau

C. Inactivated during depolarization

D. Only in ventricular myocytes

T-type calcium channels are low-voltage-activated, transient calcium channels. They open with small depolarizations near the resting membrane potential and then inactivate quickly, producing a brief inward Ca^{2+} current. This transient current contributes to the early phase of diastolic depolarization (phase 4) in sinoatrial and atrioventricular nodal cells, helping to bring the membrane potential toward threshold and support automaticity. This distinguishes them from the L-type calcium channels, which sustain the plateau phase and require a higher depolarization to activate. They are not exclusive to ventricular myocytes, as they also appear in nodal tissue. Thus, describing T-type channels as a transient, low-voltage-activated current that contributes to early phase 4 pacemaker currents in SA and AV nodal cells is the best fit.

3. What is the effect of sympathetic tone on heart rate?

A. Increases HR

B. Decreases HR

C. No effect on HR

D. Causes irregular rhythm

Sympathetic tone produces a positive chronotropic effect, meaning it increases heart rate. It does this by activating beta-1 adrenergic receptors in the SA node, which raises cAMP and enhances the funny current and calcium entry. This speeds up the rate of spontaneous depolarization and increases conduction through the AV node, resulting in a faster heart rate. In contrast, parasympathetic activity slows the rate, and sympathetic stimulation does not normally cause no effect or an irregular rhythm.

4. The Na/Ca exchanger operates with a stoichiometry of 1 calcium to 3 sodium and can move ions in either direction. Which statement best describes this?

- A. 1 Ca²⁺ per 3 Na⁺ in either direction**
- B. 1 Ca²⁺ per 2 Na⁺ in either direction**
- C. 2 Ca²⁺ per 3 Na⁺ in either direction**
- D. 1 Ca²⁺ per 4 Na⁺ in either direction**

The important concept is that the Na/Ca exchanger moves calcium and sodium in a fixed 3:1 ratio: for every Ca²⁺ exchanged, 3 Na⁺ move in or out. This coupling stays the same whether the exchanger is removing Ca²⁺ from the cell (forward mode) or bringing Ca²⁺ into the cell (reverse mode). In cardiac cells, this exchanger typically extrudes Ca²⁺ during relaxation, using 3 Na⁺ entry for each Ca²⁺ leaving. It can reverse when the gradients change, such as high intracellular Na⁺ or a depolarized membrane, causing Ca²⁺ to enter in exchange for Na⁺ leaving, but the ratio remains 3 Na⁺ per 1 Ca²⁺. The other proposed ratios don't match this established 3:1 coupling, so they aren't correct.

5. Transient outward channels contribute to which phase of non-pacemaker APs?

- A. Phase 1**
- B. Phase 2**
- C. Phase 3**
- D. Phase 0**

Transient outward potassium currents shape the early repolarization of the non-pacemaker ventricular action potential. After the rapid upstroke driven by Na⁺ entry (Phase 0), these I_{to} channels open quickly and then inactivate, producing a brief outward K⁺ current that creates the notch right at the start of repolarization. This brief outward current defines Phase 1. The plateau phase (Phase 2) follows, governed by Ca²⁺ influx balanced by K⁺ efflux, and then Phase 3 is final repolarization dominated by other K⁺ currents. In non-pacemaker cells, the transient outward current is the key contributor to the Phase 1 notch.

6. Which stimulus increases ERP in AV nodal tissue?

- A. Vagal activation**
- B. Sympathetic activation**
- C. Ischemia**
- D. Exercise**

The AV node's refractory behavior is strongly shaped by autonomic tone. Parasympathetic (vagal) activation increases AV nodal refractoriness, meaning the effective refractory period gets longer and conduction through the AV node slows. Mechanistically, acetylcholine acting on M2 receptors opens GIRK (I_KACH) channels and reduces L-type calcium current, hyperpolarizing the cells and slowing conduction. This lengthens the time before the node can be re-excited, which is exactly what the ERP reflects in AV nodal tissue. In contrast, sympathetic activation raises cAMP, enhances calcium entry, and speeds conduction, effectively shortening the ERP. Exercise, which boosts sympathetic drive, would do the opposite of increasing ERP. Ischemia can disrupt conduction and refractoriness in a variable way, but it does not specifically and reliably increase AV nodal ERP like vagal activation does. So, the stimulus that increases AV nodal ERP is vagal (parasympathetic) activation.

7. Abnormal conduction can be caused by which mechanism?

- A. Reentry**
- B. Automaticity**
- C. Atrioventricular block**
- D. Sinus node dysfunction**

Reentry is the mechanism that explains abnormal conduction because it creates a self-sustaining loop of activation. An impulse travels through a pathway and, due to a unidirectional block and differences in conduction speed and tissue refractoriness, can re-enter tissue that has recovered excitability. This looping circuit keeps reactivating the heart tissue and produces a faster, abnormal rhythm. Automaticity describes new impulses arising from non-sinus tissue rather than a looping conduction pathway, while AV block and sinus node dysfunction are disorders of conduction or the primary pacemaker's performance, not the looping reentrant process. So reentry best accounts for abnormal conduction.

8. Changes in autonomic function can do what to reentry?

- A. Initiate or stop reentry**
- B. Always increase reentry**
- C. Have no effect**
- D. Cause immediate arrest**

Changes in autonomic tone shift conduction velocity and refractoriness in cardiac tissue, which in turn alters the likelihood of reentry. Reentry needs a circuit where the impulse can reexcite tissue after it has recovered; autonomic influences that shorten the refractory period or speed conduction shorten the wavelength of excitation, making reentry more likely to occur. Conversely, autonomic changes that lengthen refractoriness or slow conduction increase the wavelength, making it harder for a circulating impulse to persist and often terminating an existing reentrant circuit. Because these effects can either promote or prevent reentry depending on the specific tissue and circuit, autonomic changes can initiate or stop reentry rather than always increasing it or causing arrest.

9. Which of the following is an example of an anatomic abnormality that can cause abnormal conduction?

- A. Congenital accessory pathways**
- B. Degenerative disease**
- C. Atherosclerotic plaque**
- D. Postoperative scar**

Abnormal conduction often stems from an anatomic substrate that provides an alternate route for impulses. Congenital accessory pathways are classic examples: these are extra muscular connections that link atrial tissue directly to ventricular tissue, bypassing the normal AV node. Because impulses can travel through this bypass tract, conduction can occur outside the usual slow AV nodal pathway, leading to preexcitation and potentially reentrant tachyarrhythmias. This discrete, persistent anatomical connection from birth makes it a quintessential substrate for abnormal conduction. Degenerative disease of the conduction system involves gradual loss or impairment of the nodes or His-Purkinje tissue, which slows or blocks conduction but isn't an extra conduction pathway. Atherosclerotic plaque affects the vessels rather than the conduction tissue itself, so it alters perfusion rather than providing a direct conduction substrate. Postoperative scar is an anatomic change that can disrupt conduction, but the classic, congenital example of a discrete conduction pathway that causes abnormal conduction is the accessory pathway.

10. Sympathetic activation produces positive chronotropy. What does this mean for heart rate?

- A. Increases heart rate**
- B. Decreases heart rate**
- C. No change in heart rate**
- D. Increases contractility (inotropy)**

Positive chronotropy means the heart rate increases. When the sympathetic system is activated, norepinephrine stimulates beta-1 receptors in the sinoatrial (SA) node, boosting the automaticity of pacemaker cells. This speeds up the rate of spontaneous depolarization (a steeper phase 4), so the SA node fires more often and the heart beats faster. This concept is about rate; separate terms describe how hard the heart contracts (inotropy) or how quickly impulses pass through the conduction pathways (dromotropy). Parasympathetic activity, by contrast, slows the rate (negative chronotropy).

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://cardiacelectrophysio.examzify.com>

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

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