Vasopressors & Inotropes Practice Test (Sample)

Study Guide

BY EXAMZIFY

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. What is the B1 effect of ephedrine?
   A. Decrease heart rate and contractility
   B. No change in heart rate or contractility
   C. Increase heart rate only
   D. Increase heart rate and contractility

2. Which form of calcium is physiologically active?
   A. Bound calcium
   B. Calcium bound to proteins
   C. Ionized calcium (unbound)
   D. Total calcium

3. Which statement best describes the beta-2 receptor involvement differentiating epinephrine from norepinephrine?
   A. Beta-2 stimulation is inhibited by norepinephrine
   B. Both stimulate beta-2 equally
   C. Neither stimulates beta-2 receptors
   D. Epinephrine stimulates beta-2 receptors; norepinephrine does not

4. Milrinone MOA involves which mechanism?
   A. PDE-3 inhibition leading to increased cAMP
   B. Beta-adrenergic receptor blockade
   C. Calcium chelation
   D. ACE inhibition

5. Which mediator decreases norepinephrine release from presynaptic sympathetic neurons?
   A. Nitric oxide
   B. Norepinephrine
   C. Epinephrine
   D. Angiotensin II

6. What is the primary effect of alpha-1 agonism?

- **A. Vasodilation**
- **B. Vasoconstriction**
- **C. Bronchodilation**
- **D. Decreased heart rate**

7. Which statement about phenylephrine's effect on heart rate is most accurate?

- **A. No effect on HR**
- **B. Reflex bradycardia**
- **C. Reflex tachycardia**
- **D. Random HR changes**

8. Vasopressin can cause bronchoconstriction through stimulation of which receptors on bronchial smooth muscle?

- **A. V2 receptors**
- **B. V1 receptors**
- **C. Alpha-1 receptors**
- **D. Beta-2 receptors**

9. Which two drugs should not be given with ephedrine, and why?

- **A. MAO inhibitors and cocaine**
- **B. Beta blockers and nitrates**
- **C. ACE inhibitors and ARBs**
- **D. Diuretics and benzodiazepines**

10. Which receptor mediates vasodilation in the pulmonary vasculature at low epinephrine concentrations?

- **A. Beta2**
- **B. Alpha1**
- **C. Beta1**
- **D. Alpha2**

Answers

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

Explanations

1. What is the B1 effect of ephedrine?

A. Decrease heart rate and contractility

B. No change in heart rate or contractility

C. Increase heart rate only

D. Increase heart rate and contractility

Beta-1 stimulation increases both heart rate (chronotropy) and contractility (inotropy). Ephedrine is a mixed-acting sympathomimetic that directly stimulates alpha and beta receptors and also increases norepinephrine release. The beta-1 effects raise heart rate and strengthen each beat by increasing cAMP in cardiac cells, which boosts calcium availability and conduction through the AV node as well. So the net B1 effect is to increase both heart rate and contractility. The other options don't fit because beta-1 activation does not produce only one of those effects, nor no change, nor a decrease.

2. Which form of calcium is physiologically active?

A. Bound calcium

B. Calcium bound to proteins

C. Ionized calcium (unbound)

D. Total calcium

The physiologically active form of calcium in the blood is the free, ionized calcium (Ca2+). This is the portion that is unbound from proteins and not tied up in complexes, and it's the form that directly participates in essential bodily functions. Ionized calcium is what enables muscle contraction, supports neurotransmitter release, acts as a cofactor for many enzymes, and drives blood coagulation processes. The protein-bound and complexed calcium serve mainly as a reservoir and are not readily available to cells, so they don't reflect immediate calcium activity. That's why total calcium can be misleading in some clinical situations; only the ionized fraction truly represents functional calcium status. Factors like albumin levels and blood pH can shift the balance between bound and ionized calcium, affecting the physiologically active pool even if the total calcium changes.

3. Which statement best describes the beta-2 receptor involvement differentiating epinephrine from norepinephrine?

A. Beta-2 stimulation is inhibited by norepinephrine

B. Both stimulate beta-2 equally

C. Neither stimulates beta-2 receptors

D. Epinephrine stimulates beta-2 receptors; norepinephrine does not

Beta-2 receptor stimulation produces smooth muscle relaxation, including bronchodilation and some vascular effects in skeletal muscle. Epinephrine activates beta-2 receptors strongly, giving bronchodilation and certain vasodilatory effects, while norepinephrine has little affinity for beta-2 receptors and thus does not produce those beta-2-mediated effects at typical concentrations. That makes the statement that epinephrine stimulates beta-2 receptors while norepinephrine does not the best description of their differences. The other ideas aren't correct because norepinephrine mainly targets alpha-1 (and beta-1) with negligible beta-2 activity, so it doesn't produce the beta-2-driven responses seen with epinephrine.

4. Milrinone MOA involves which mechanism?

A. PDE-3 inhibition leading to increased cAMP

B. Beta-adrenergic receptor blockade

C. Calcium chelation

D. ACE inhibition

Milrinone's main mechanism is inhibition of phosphodiesterase-3, which normally breaks down cyclic AMP in cardiac and vascular smooth muscle. By blocking PDE-3, milrinone raises intracellular cAMP, activating protein kinase A. In heart cells, this increases calcium entry and release, boosting contractility (positive inotropy). In vascular smooth muscle, higher cAMP reduces calcium sensitivity and promotes relaxation, causing vasodilation and lower afterload. This dual inotropic and vasodilatory effect explains its use as an inodilator in acute heart failure. The other options don't fit: beta-blockade would decrease cAMP and contractility, calcium chelation would reduce calcium directly, and ACE inhibition lowers angiotensin II without raising cAMP.

5. Which mediator decreases norepinephrine release from presynaptic sympathetic neurons?

A. Nitric oxide

B. Norepinephrine

C. Epinephrine

D. Angiotensin II

Presynaptic inhibition of sympathetic transmission can be produced by nitric oxide, a gaseous signaling molecule. It diffuses to the presynaptic terminal and activates soluble guanylate cyclase, raising cGMP levels. This shift reduces calcium influx and vesicle fusion, so less norepinephrine is released into the synapse. Nitric oxide acts as a local brake on transmitter release, helping dampen sympathetic signaling. The other substances listed don't serve as local presynaptic inhibitors in this context: norepinephrine is the transmitter itself, epinephrine mainly acts systemically rather than as a presynaptic brake in sympathetic terminals, and angiotensin II tends to enhance sympathetic activity rather than inhibit NE release.

6. What is the primary effect of alpha-1 agonism?

A. Vasodilation

B. Vasoconstriction

C. Bronchodilation

D. Decreased heart rate

Activation of alpha-1 receptors on vascular smooth muscle causes contraction, leading to vasoconstriction. This constricts blood vessels, increases systemic vascular resistance, and raises blood pressure. The higher pressure can trigger a reflex slowing of the heart, but the direct, primary effect of alpha-1 agonism is narrowing of vessels, not a decrease in heart rate. Bronchodilation is mediated by beta-2 receptors in the airways, and a direct decrease in heart rate is not the primary action of alpha-1 stimulation. So the main effect is vasoconstriction.

7. Which statement about phenylephrine's effect on heart rate is most accurate?

A. No effect on HR

B. Reflex bradycardia

C. Reflex tachycardia

D. Random HR changes

Phenylephrine raises blood pressure by constricting peripheral vessels through alpha-1 stimulation. That rise in arterial pressure activates the baroreceptor reflex, which increases vagal (parasympathetic) activity to the heart and lowers sympathetic outflow. The net effect is a slower heart rate—reflex bradycardia—rather than a direct increase in rate. So, the most accurate statement is that it causes reflex bradycardia. Some patients may have minimal or variable HR responses depending on autonomic tone, but bradycardia is the classic reflex outcome with phenylephrine.

8. Vasopressin can cause bronchoconstriction through stimulation of which receptors on bronchial smooth muscle?

A. V2 receptors

B. V1 receptors

C. Alpha-1 receptors

D. Beta-2 receptors

Vasopressin can cause bronchoconstriction by acting on V1 receptors on bronchial smooth muscle. When these V1 receptors are stimulated, they couple to a Gq protein, which activates phospholipase C, leading to increased inositol triphosphate (IP3) and diacylglycerol (DAG). This raises intracellular calcium and promotes contraction of the airway smooth muscle, narrowing the airways. V2 receptors, in contrast, are mainly in the kidneys and regulate water reabsorption, not airway tone. Beta-2 receptors produce bronchodilation when stimulated, not constriction. Alpha-1 receptors are adrenergic and are not the receptors through which vasopressin acts to affect the bronchial smooth muscle in this context. So the bronchoconstrictive effect of vasopressin is best explained by stimulation of V1 receptors on bronchial smooth muscle.

9. Which two drugs should not be given with ephedrine, and why?

A. MAO inhibitors and cocaine

B. Beta blockers and nitrates

C. ACE inhibitors and ARBs

D. Diuretics and benzodiazepines

The main idea is that ephedrine can provoke a strong sympathetic response by both directly stimulating receptors and indirectly increasing norepinephrine release. When paired with drugs that dramatically raise catecholamine activity or block its breakdown, this response can become dangerous. With monoamine oxidase inhibitors, the breakdown of norepinephrine is inhibited. Ephedrine's indirect mechanism increases norepinephrine release, so combining the two leads to a much higher level of adrenergic stimulation. That can trigger a hypertensive crisis, tachyarrhythmias, and potential organ injury due to excessive vasoconstriction and cardiac workload. With cocaine, the drug blocks reuptake of norepinephrine (and other monoamines), leaving more not only at the synapse but also enhancing sympathetic tone. Adding ephedrine further increases norepinephrine release and receptor stimulation, again risking severe hypertension, arrhythmias, myocardial ischemia, or stroke from excessive vasoconstriction and cardiac strain. Other options don't carry this same direct, high-risk interaction profile with ephedrine, so the combination of these two classes remains the dangerous pairing to avoid.

10. Which receptor mediates vasodilation in the pulmonary vasculature at low epinephrine concentrations?

A. Beta2

B. Alpha1

C. Beta1

D. Alpha2

Vasodilation in the pulmonary circulation at low epinephrine concentrations is driven by β2-adrenergic receptors on pulmonary arterial smooth muscle. When epinephrine activates these receptors, it stimulates Gs proteins to raise cAMP, causing smooth muscle relaxation and thus vessel dilation, which lowers pulmonary vascular resistance. β1 receptors mainly affect heart rate and contractility, not pulmonary vessel tone, while α1 receptors cause vasoconstriction and require higher epinephrine levels to become influential. Therefore, the β2 receptor mediates the observed vasodilation at low epinephrine concentrations.

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://vasopressorsinotropes.examzify.com

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