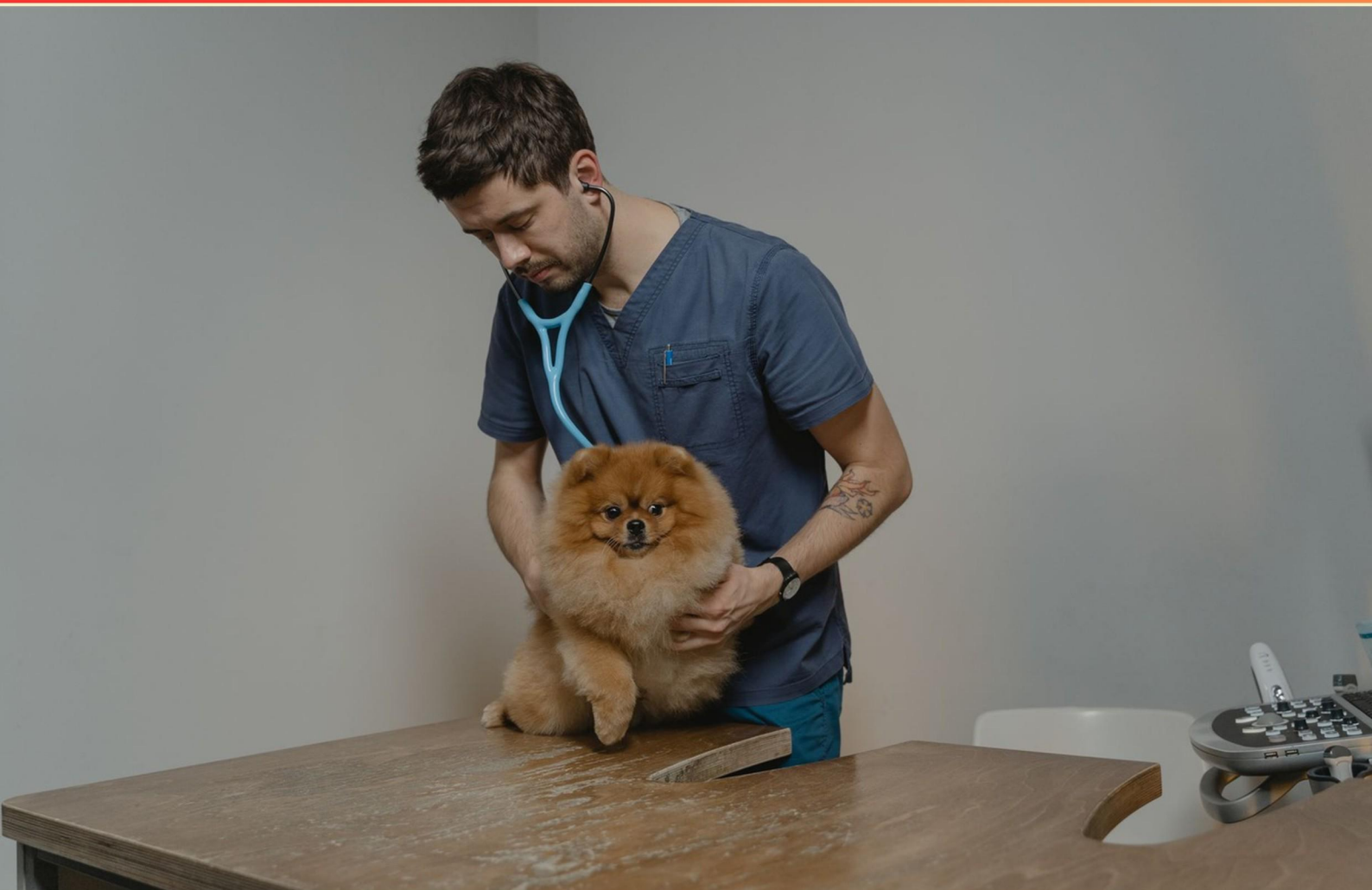


NBME FORM 27 PRACTICE TEST (SAMPLE)

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.

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. What clinical finding is associated with the glucocorticoid effects of cortisol?**
 - A. Weight loss
 - B. Increased muscle mass
 - C. Increased sodium and water reabsorption
 - D. Decreased blood pressure
- 2. Which of the following is a common sign of fetal alcohol syndrome?**
 - A. Macrocephaly
 - B. Intellectual disability and developmental delays
 - C. Excessive weight gain
 - D. Hyperactivity
- 3. Which is a common side effect of beta-2 adrenergic agonists?**
 - A. Tachycardia
 - B. Hypotension
 - C. Angioedema
 - D. Excessive salivation
- 4. How do tetracyclines disrupt bacterial protein synthesis?**
 - A. By inhibiting DNA replication
 - B. By reversibly binding to bacterial ribosomal 30S subunit
 - C. By interfering with cell wall synthesis
 - D. By preventing RNA polymerase activity
- 5. Which of the following is NOT a typical sign of acute inflammatory nephritis (AIN)?**
 - A. Rash
 - B. Hematuria
 - C. Dehydration
 - D. Hypotension

- 6. What happens to the endplate potential when botulinum toxin is present?**
- A. It increases significantly
 - B. It decreases
 - C. It remains the same
 - D. It becomes erratic
- 7. What is the mechanism of action of fluoroquinolones?**
- A. Inhibit protein synthesis by binding to ribosomes
 - B. Inhibit DNA gyrase and topoisomerase IV
 - C. Inhibit cell wall synthesis
 - D. Inhibit folic acid synthesis
- 8. What neurological deficit is most commonly observed in a thrombotic stroke affecting the non-dominant MCA territory?**
- A. Contralateral hemineglect
 - B. Global aphasia
 - C. Visual agnosia
 - D. Pure motor hemiplegia
- 9. What is the most common condition causing bloody stools and abdominal distension in premature infants?**
- A. Necrotizing enterocolitis
 - B. Meconium ileus
 - C. Hypoxic-ischemic enterocolitis
 - D. Infections of the bowel
- 10. What condition may present with an increase in sarcomeres coupled with decreased abundance of F-actin?**
- A. Hypertrophic cardiomyopathy
 - B. Concentric hypertrophy
 - C. Heart failure
 - D. Aortic stenosis

Answers

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

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Explanations

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1. What clinical finding is associated with the glucocorticoid effects of cortisol?

- A. Weight loss
- B. Increased muscle mass
- C. Increased sodium and water reabsorption**
- D. Decreased blood pressure

Cortisol, a glucocorticoid hormone produced by the adrenal cortex, plays a significant role in various physiological processes, including metabolism, immune response, and fluid balance. One of the primary effects of cortisol is its influence on sodium and water reabsorption in the kidneys. Cortisol acts on the distal tubules and collecting ducts of the nephron, promoting the reabsorption of sodium. This mechanism leads to an increase in sodium levels in the bloodstream. When sodium is reabsorbed, water follows osmotically, resulting in increased blood volume and, consequently, increased blood pressure. This is a critical adaptation for maintaining homeostasis during stress. The other options presented don't align with the well-documented physiological effects of cortisol. Weight loss is not typically associated with glucocorticoids; instead, prolonged exposure can lead to weight gain due to increased appetite and fat deposition. Increased muscle mass is contrary to the catabolic effects of cortisol, which can lead to muscle breakdown. Lastly, cortisol is associated with increased blood pressure rather than decreased blood pressure due to its effects on fluid retention and vasoconstriction. In summary, the reabsorption of sodium and water by the kidneys, influenced by cortisol, explains why the choice

2. Which of the following is a common sign of fetal alcohol syndrome?

- A. Macrocephaly
- B. Intellectual disability and developmental delays**
- C. Excessive weight gain
- D. Hyperactivity

Intellectual disability and developmental delays are hallmark characteristics of fetal alcohol syndrome (FAS). This condition arises from prenatal exposure to alcohol, which can severely affect brain development and function. Children with FAS often exhibit a spectrum of intellectual impairments that can range from mild learning disabilities to more profound intellectual disabilities. Developmental delays can manifest in various ways, including delays in speech and motor skills, social interactions, and adaptive functioning. The neurotoxic effects of alcohol on the developing fetus can impair the formation of neural connections and the overall structure of the brain, leading to these cognitive and developmental challenges. While other symptoms might be associated with FAS, including growth deficiencies or behavioral issues, intellectual disability and developmental delays are the most consistent and recognizable signs that clinicians look for when diagnosing this syndrome.

3. Which is a common side effect of beta-2 adrenergic agonists?

- A. Tachycardia**
- B. Hypotension
- C. Angioedema
- D. Excessive salivation

Beta-2 adrenergic agonists are primarily used as bronchodilators in the treatment of conditions like asthma and chronic obstructive pulmonary disease (COPD). One of the well-known side effects of these medications is tachycardia, which refers to an increased heart rate. The mechanism behind this side effect is linked to the action of beta-2 receptors, which are primarily found in the lungs but also have some presence in the cardiovascular system. When these agonists stimulate beta-2 receptors, they can lead to a reflex increase in heart rate as a result of peripheral vasodilation and the body's compensatory mechanisms to maintain blood pressure and cardiac output. While beta-2 agonists are less commonly associated with significant cardiovascular effects compared to beta-1 agonists, tachycardia can still occur, especially with high doses or systemic administration. This side effect is particularly important for clinicians to consider when prescribing these medications, as it can be more pronounced in patients with pre-existing cardiovascular conditions.

4. How do tetracyclines disrupt bacterial protein synthesis?

- A. By inhibiting DNA replication
- B. By reversibly binding to bacterial ribosomal 30S subunit**
- C. By interfering with cell wall synthesis
- D. By preventing RNA polymerase activity

Tetracyclines disrupt bacterial protein synthesis by reversibly binding to the bacterial ribosomal 30S subunit. This binding interferes with the attachment of aminoacyl-tRNA to the mRNA-ribosomal complex during the translation process, which is crucial for synthesizing proteins. The inhibition of this step ultimately leads to a decrease in the production of essential proteins needed for bacterial growth and replication. This mechanism specifically targets the 30S subunit, which is a distinct feature of prokaryotic ribosomes, allowing tetracyclines to selectively inhibit bacterial protein synthesis without affecting eukaryotic ribosomes significantly. This selectivity is what makes tetracyclines effective antibiotics for treating a variety of bacterial infections. The correct mechanism highlights how tetracyclines disrupt protein synthesis directly at the ribosomal level, which is fundamental to their action as antibiotics.

5. Which of the following is NOT a typical sign of acute inflammatory nephritis (AIN)?

- A. Rash
- B. Hematuria
- C. Dehydration**
- D. Hypotension

Acute inflammatory nephritis (AIN), commonly associated with various conditions such as drug reactions, infections, or autoimmune diseases, typically presents with symptoms that reflect inflammation and damage to the renal tubules and interstitium. A rash can often be seen in cases of AIN, especially if it is related to hypersensitivity reactions to medications or infections that lead to a systemic response. Hematuria, or the presence of blood in the urine, is a common finding as well, resulting from glomerular or tubular damage during the inflammatory process. Hypotension may arise due to fluid shifts or fluid loss if renal function is compromised significantly, leading to inadequate blood volume or renal perfusion. Dehydration is not a distinct or typical sign of AIN itself. While patients may become dehydrated due to vomiting, diarrhea, or other underlying conditions that cause fluid loss, dehydration is not an inherent feature of the inflammatory processes occurring in the kidneys. AIN primarily manifests through renal-related symptoms rather than the systemic effects of dehydration, making it the least characteristic among the listed signs.

6. What happens to the endplate potential when botulinum toxin is present?

- A. It increases significantly
- B. It decreases**
- C. It remains the same
- D. It becomes erratic

Botulinum toxin functions by blocking the release of acetylcholine at the neuromuscular junction. When this toxin binds to the presynaptic terminals, it prevents the fusion of neurotransmitter-filled vesicles with the presynaptic membrane, effectively inhibiting the exocytosis of acetylcholine. Consequently, with less acetylcholine being released into the synaptic cleft, there are fewer molecules available to bind to the nicotinic receptors on the postsynaptic membrane. This reduction in acetylcholine leads to a decrease in the endplate potential, which is the change in membrane potential due to the influx of sodium ions through the acetylcholine receptors. If the endplate potential is insufficient to reach the threshold for action potentials in the muscle fiber, it results in muscle weakness or paralysis. Thus, the presence of botulinum toxin directly correlates with a decrease in the endplate potential.

7. What is the mechanism of action of fluoroquinolones?

- A. Inhibit protein synthesis by binding to ribosomes
- B. Inhibit DNA gyrase and topoisomerase IV**
- C. Inhibit cell wall synthesis
- D. Inhibit folic acid synthesis

Fluoroquinolones exert their antibacterial effects primarily through the inhibition of bacterial enzymes known as DNA gyrase and topoisomerase IV. These enzymes play crucial roles in the processes of DNA replication, transcription, and repair. DNA gyrase is involved in introducing negative supercoils into DNA, which is essential for relieving torsional stress that occurs ahead of the replication fork during DNA synthesis. Topoisomerase IV is vital for separating the interlinked daughter DNA strands that result from replication. By inhibiting these enzymes, fluoroquinolones effectively disrupt the proper function of bacterial DNA, leading to bacterial growth inhibition and eventual cell death. This precise mechanism highlights the fluoroquinolones' role as broad-spectrum antibiotics against a variety of Gram-negative and Gram-positive bacteria. Their ability to target critical pathways in bacterial DNA handling distinguishes them from other classes of antibiotics, such as those that inhibit protein synthesis or cell wall synthesis.

8. What neurological deficit is most commonly observed in a thrombotic stroke affecting the non-dominant MCA territory?

- A. Contralateral hemineglect**
- B. Global aphasia
- C. Visual agnosia
- D. Pure motor hemiplegia

In cases of a thrombotic stroke affecting the non-dominant middle cerebral artery (MCA) territory, the most commonly observed neurological deficit is contralateral hemineglect. This phenomenon occurs when there is damage to regions of the brain that are critical for attention and spatial awareness, particularly in the right hemisphere, which is typically dominant for these functions in individuals who are left hemisphere dominant (right-handed). Contralateral hemineglect manifests as a failure to attend to or respond to stimuli on the side of the body opposite to the stroke. For example, if the right MCA territory is affected, the patient may ignore objects or even the entire left side of their visual field. This neglect is not due to a visual field defect or a motor impairment but rather arises from an impairment in the brain's ability to process and attend to spatial information. Understanding this provides valuable insight into the clinical presentations of strokes in different brain regions and their correlation with neurological deficits. In contrast, other deficits, such as global aphasia, visual agnosia, or pure motor hemiplegia, are typically associated with strokes in the dominant hemisphere or specific pathways, rather than the non-dominant MCA territory.

9. What is the most common condition causing bloody stools and abdominal distension in premature infants?

- A. Necrotizing enterocolitis**
- B. Meconium ileus
- C. Hypoxic-ischemic enterocolitis
- D. Infections of the bowel

Necrotizing enterocolitis (NEC) is the most common condition leading to bloody stools and abdominal distension in premature infants. This serious gastrointestinal condition primarily affects the intestines of premature newborns, whose immature intestines are more susceptible to injury and infection. In NEC, there is inflammation, and in severe cases, necrosis of the intestinal tissue. One of the hallmark signs of this condition is the presence of bloody stools, which occurs due to damage to the intestinal mucosa. Along with bloody stools, these infants may exhibit abdominal distension, which can arise from both the accumulation of gas and fluid in the intestines and from the inability to effectively process feedings. Premature infants have a higher risk of developing NEC due to their underdeveloped gastrointestinal systems, making it a critical condition to monitor for in neonatal care units. Understanding the signs, symptoms, and risks associated with NEC is crucial for prompt diagnosis and management to improve outcomes in affected infants.

10. What condition may present with an increase in sarcomeres coupled with decreased abundance of F-actin?

- A. Hypertrophic cardiomyopathy**
- B. Concentric hypertrophy
- C. Heart failure
- D. Aortic stenosis

Hypertrophic cardiomyopathy is characterized by an increase in the number of sarcomeres, leading to thickened heart muscle, particularly affecting the ventricular walls. This condition arises from genetic mutations that affect the proteins in the cardiac sarcomere, causing cardiac muscle fibers to proliferate and grow more densely. The decrease in F-actin abundance observed in this context may be related to the reorganization of the cytoskeletal elements as the thickening of the myocardium occurs. In hypertrophic cardiomyopathy, the reconfiguration of the myofilament structure, including alterations in actin levels, can contribute to the overall contractility and functional alterations seen in the heart. Understanding this mechanism is crucial, as it reflects how the structural changes inherent to hypertrophic cardiomyopathy impact heart function and contributes to symptoms such as dyspnea and arrhythmias. The increase in sarcomeres combines with the altered composition of the cytoskeletal components, leading to the distinctive pathophysiological features of the disease.

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://nbmeform27.examzify.com>

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

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