INBDE PHARMACOLOGY 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.

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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!

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Questions

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1. Which insulin is classified as intermediate-acting?

A. NPH

B. Lispro

C. Glargine

D. Detemir

2. Which medication is an antihistamine used in asthma management focusing on allergic triggers?

A. Diphenhydramine

B. Albuterol

C. Budesonide

D. Montelukast

3. Type 2 antiarrhythmics are which general category?

A. Beta blockers

B. Sodium channel blockers

C. Calcium channel blockers

D. Potassium channel blockers

4. Which drug class is listed as a treatment for congestive heart failure in addition to glycosides?

A. ACE inhibitors

B. ARBs

C. Diuretics

D. Calcium channel blockers

5. Which of the following statements correctly compares the two max lidocaine doses listed in the material?

A. With epi max is 4.4 mg/kg and without epi max is 7.0 mg/kg

B. With epi max is 7.0 mg/kg and without epi max is 4.4 mg/kg

C. Both max doses are 5 mg/kg

D. No data given about max doses

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6. Which drug is a SGLT2 inhibitor?

A. Metformin

B. Sitagliptin

C. Canagliflozin

D. Acarbose

7. Lidocaine treats which type of arrhythmias?

A. Atrial fibrillation

B. Ventricular arrhythmias

C. Digitalis-induced arrhythmias

D. Torsades de pointes

8. Diflunisal differs from Aspirin, Acetaminophen, and Ibuprofen in having which characteristic?

A. More potent analgesic

B. Longer Half-Life

C. Higher risk of GI bleeding than aspirin

D. No anti-inflammatory effect

9. Which two drugs are listed as inhibitors of norepinephrine release?

A. Guanethidine and Reserpine

B. Propranolol and Metoprolol

C. Celecoxib and Meloxicam

D. Diuretics and ACE inhibitors

10. Which of the following is NOT a typical side effect of systemic corticosteroid therapy?

A. Osteoporosis

B. Immunosuppression

C. Redistribution of body fat

D. Decreased risk of infection

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Answers

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

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Explanations

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1. Which insulin is classified as intermediate-acting?

A. NPH

B. Lispro

C. Glargine

D. Detemir

Insulins are grouped by how long they act. Intermediate-acting insulin is designed to act more slowly than rapid-acting or short-acting insulins but not as long as the long-acting ones. This slower action comes from forming a complex with protamine (in neutral protamine Hagedorn, or NPH), which delays absorption after injection. The result is a onset of about 1–2 hours, a peak around 6–12 hours, and a total duration roughly 14–24 hours. That mid-range profile fits between the quick-acting insulins used for meals and the steady, long-acting insulins that provide basal coverage. So the insulin that is intermediate-acting is NPH.

2. Which medication is an antihistamine used in asthma management focusing on allergic triggers?

A. Diphenhydramine

B. Albuterol

C. Budesonide

D. Montelukast

Allergic triggers in asthma involve histamine release from mast cells, which can worsen airway inflammation and related symptoms. An antihistamine blocks histamine H1 receptors, reducing those histamine-driven effects and helping ease allergic symptoms that can aggravate asthma. Diphenhydramine is a classic H1 antihistamine, so it fits as the option addressing the allergic component of asthma management. The other drugs serve different roles: albuterol is a bronchodilator that relieves acute bronchospasm, budesonide is an inhaled anti-inflammatory corticosteroid for long-term control, and montelukast blocks leukotriene receptors to reduce inflammation and bronchoconstriction. While antihistamines aren't a primary asthma controller, they can help with allergic symptoms that contribute to asthma control.

3. Type 2 antiarrhythmics are which general category?

A. Beta blockers

B. Sodium channel blockers

C. Calcium channel blockers

D. Potassium channel blockers

Type 2 antiarrhythmics are beta-adrenergic blockers. They work by blocking beta receptors in the heart, which reduces sympathetic stimulation. This lowers the slope of automaticity in the SA node and slows conduction through the AV node, leading to a slower heart rate and slower AV conduction—useful for controlling tachyarrhythmias with rapid ventricular response. This differs from the other classes: sodium channel blockers (Type I) affect depolarization in atrial/ventricular tissue; calcium channel blockers (Type IV) mainly slow nodal conduction by inhibiting L-type calcium channels; potassium channel blockers (Type III) prolong repolarization and the QT interval. Examples of Type II agents include propranolol, metoprolol, and esmolol.

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4. Which drug class is listed as a treatment for congestive heart failure in addition to glycosides?

A. ACE inhibitors

B. ARBs

C. Diuretics

D. Calcium channel blockers

Managing congestive heart failure involves counteracting the harmful neurohormonal activation that drives progression. ACE inhibitors block the enzyme that forms angiotensin II, leading to vasodilation, reduced aldosterone release, and decreased sodium and water retention. This lowers both afterload and preload and helps curb cardiac remodeling, which translates into better symptoms and improved survival for many patients. While glycosides (digoxin) can improve symptoms by increasing cardiac contractility, they don't modify the disease process or long-term outcomes. Diuretics relieve fluid overload but don't change prognosis, and certain calcium channel blockers can worsen systolic heart failure in some patients. ARBs are an alternative if ACE inhibitors aren't tolerated, but the class commonly listed alongside glycosides for long-term management is ACE inhibitors.

5. Which of the following statements correctly compares the two max lidocaine doses listed in the material?

A. With epi max is 4.4 mg/kg and without epi max is 7.0 mg/kg

B. With epi max is 7.0 mg/kg and without epi max is 4.4 mg/kg

C. Both max doses are 5 mg/kg

D. No data given about max doses

The main idea here is how the presence of a vasoconstrictor like epinephrine changes the stated maximum lidocaine dose per kilogram. Epinephrine slows local blood flow, which can reduce how quickly lidocaine enters the bloodstream and thus affects the safe upper limit per kilogram. According to the material you're using, the listed maximums are 4.4 mg/kg when epinephrine is included and 7.0 mg/kg when it is not. So the choice that matches that material is that the max dose with epinephrine is 4.4 mg/kg and the max dose without epinephrine is 7.0 mg/kg. In practice, many standard references describe the opposite pattern—epinephrine allows a higher per-kilogram maximum because it reduces systemic absorption—so when studying, always rely on the specific dose table your material provides and check for context (adult vs. pediatric dosing, formulation, and concentration).

6. Which drug is a SGLT2 inhibitor?

A. Metformin

B. Sitagliptin

C. Canagliflozin

D. Acarbose

SGLT2 inhibitors lower blood glucose by blocking the sodium-glucose cotransporter 2 in the proximal tubule of the kidney, which reabsorbs filtered glucose back into the bloodstream. When SGLT2 is inhibited, more glucose is excreted in the urine (glucosuria), reducing plasma glucose independently of insulin. This mechanism is exemplified by canagliflozin, an SGLT2 inhibitor. Metformin works mainly by reducing hepatic glucose production and improving insulin sensitivity, not by inhibiting glucose reabsorption in the kidney. Sitagliptin is a DPP-4 inhibitor that raises incretin levels to boost insulin release after meals. Acarbose is an alpha-glucosidase inhibitor that delays carbohydrate absorption in the gut.

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7. Lidocaine treats which type of arrhythmias?

A. Atrial fibrillation

B. Ventricular arrhythmias

C. Digitalis-induced arrhythmias

D. Torsades de pointes

Lidocaine is a class IB antiarrhythmic, which means it blocks fast sodium channels with rapid kinetics. It preferentially binds to inactivated sodium channels in depolarized, ischemic ventricular tissue, shortening the action potential duration there and decreasing the slope of phase 0. This reduces excitability and automaticity in the ventricles, helping to suppress ventricular ectopy and ventricular tachyarrhythmias, especially in the setting of ischemia or myocardial infarction. Because atrial tissue is less affected by lidocaine and ventricular-specific mechanisms drive its antiarrhythmic effect, it is not effective for atrial fibrillation. Torsades de pointes is best treated with magnesium, and digitalis-induced ventricular arrhythmias are not the primary target of lidocaine. So its clinical utility is strongest for ventricular arrhythmias.

8. Diflunisal differs from Aspirin, Acetaminophen, and Ibuprofen in having which characteristic?

A. More potent analgesic

B. Longer Half-Life

C. Higher risk of GI bleeding than aspirin

D. No anti-inflammatory effect

The main idea here is how long a drug stays active in the body, its half-life, which directly affects how long its effects last. Diflunisal is studied for its notably longer elimination half-life compared with aspirin, acetaminophen, and ibuprofen. This means diflunisal remains in the bloodstream and keeps producing analgesic and anti-inflammatory effects for a much longer period, allowing less frequent dosing. This extended half-life is influenced by its pharmacokinetics: diflunisal binds very strongly to plasma proteins and is cleared more slowly after hepatic metabolism, with enterohepatic recirculation contributing to prolonged presence in the body. In contrast, the other drugs in question are cleared more quickly—acetaminophen and ibuprofen have relatively short half-lives, and aspirin's analgesic half-life is shorter and dose-dependent. Because of these differences in duration, the distinguishing characteristic is the longer half-life of diflunisal.

9. Which two drugs are listed as inhibitors of norepinephrine release?

A. Guanethidine and Reserpine

B. Propranolol and Metoprolol

C. Celecoxib and Meloxicam

D. Diuretics and ACE inhibitors

Inhibitors of norepinephrine release work by reducing how much NE is available for signaling at sympathetic nerve endings. Guanethidine acts as a false transmitter that is taken up into sympathetic neurons and into vesicles, where it displaces NE and ultimately prevents its release. Reserpine blocks the vesicular monoamine transporter, so NE cannot be packaged into vesicles and is depleted from nerve terminals, leading to a diminished NE release. The other options don't target NE release. Propranolol and metoprolol block beta-adrenergic receptors, not the release of NE. Celecoxib and meloxicam are NSAIDs, and diuretics with ACE inhibitors affect blood pressure through other mechanisms rather than NE release.

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10. Which of the following is NOT a typical side effect of systemic corticosteroid therapy?

A. Osteoporosis

B. Immunosuppression

C. Redistribution of body fat

D. Decreased risk of infection

Systemic corticosteroids have wide-ranging effects on both the immune system and body metabolism. They blunt immune defenses by reducing cytokine production, inhibiting T-cell proliferation, and impairing leukocyte migration and macrophage function. This leads to increased susceptibility to infections and can mask signs of infection, rather than protecting against them. They also disrupt bone remodeling, as osteoblast activity is decreased and bone resorption increased, contributing to osteoporosis. Additionally, fat distribution changes occur with central adiposity and features like moon facies or a buffalo hump due to altered fat metabolism. Because of these mechanisms, a decreased risk of infection is not typical; in fact, infection risk is increased with systemic corticosteroid therapy.

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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:

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

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