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39	A	B	C	D	E

PRACTICE QUESTIONS

PEBC Demo Exams

PEXDE

Pharmacist Evaluating Examination Demo - Version 1

EDITION

Demo

QUESTIONS

9

ISSUED

September 3, 2026

Before you begin

What this is

9 practice questions for PEBC Demo Exams **PEXDE**, each with its answer key and an explanation. Answer a question before you read its key — the explanation is worth more once you have committed.

If something looks wrong

Send us three things and we will check it:

- the exam code, **PEXDE**
- the question number
- the email address on your order

Write to **support@mometrix.net**. We reply within one business day.

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QUESTION 1

SINGLE CHOICE

A patient is started on lisinopril for hypertension. By what primary mechanism does lisinopril lower blood pressure?

- ☐ A Inhibition of the sodium-potassium ATPase pump in the renal tubules
- ☐ B Competitive antagonism of angiotensin II at the AT1 receptor
- ☒ C Inhibition of angiotensin-converting enzyme (ACE), reducing conversion of angiotensin I to angiotensin II
- ☐ D Blockade of beta-1 adrenergic receptors in the myocardium

ANSWER C

WHY Lisinopril is an angiotensin-converting enzyme (ACE) inhibitor. It lowers blood pressure by blocking ACE, which decreases the conversion of angiotensin I to angiotensin II and also reduces bradykinin degradation. Option A describes loop or thiazide diuretics, B describes ARBs such as losartan, and D describes beta-blockers such as metoprolol.

QUESTION 2

SINGLE CHOICE

A patient taking warfarin is initiated on trimethoprim-sulfamethoxazole. Which of the following best describes the most likely interaction?

- ☐ A Trimethoprim-sulfamethoxazole induces CYP3A4, increasing warfarin metabolism and decreasing INR
- ☒ B Trimethoprim-sulfamethoxazole inhibits CYP2C9 and displaces warfarin from albumin, increasing INR and bleeding risk
- ☐ C Trimethoprim-sulfamethoxazole chelates warfarin in the gut, reducing its absorption and decreasing INR
- ☐ D Trimethoprim-sulfamethoxazole has no clinically meaningful interaction with warfarin

ANSWER B

WHY TMP-SMX is a well-known potentiator of warfarin's effect. The sulfonamide component inhibits CYP2C9 (the main enzyme that metabolizes the more potent S-warfarin) and can also displace warfarin from plasma protein binding, leading to an elevated INR and increased bleeding risk. INR should be monitored closely when TMP-SMX is started or stopped.

QUESTION 3 SINGLE CHOICE

A drug has a high first-pass hepatic extraction ratio. Which statement about its oral bioavailability is most accurate?

- ☐ A Oral bioavailability will be high because absorption from the gut is complete
- ☐ B Oral bioavailability will be low and will be increased significantly by enzyme induction
- ☒ C Oral bioavailability will be low and will be increased by liver disease that reduces hepatic metabolism
- ☐ D Oral bioavailability is unaffected by hepatic blood flow

ANSWER C

WHY For drugs with a high hepatic extraction ratio, bioavailability is limited primarily by first-pass metabolism. Conditions that reduce hepatic metabolic capacity (such as cirrhosis or enzyme inhibition) or reduce hepatic blood flow can substantially increase oral bioavailability. Enzyme induction would further reduce bioavailability by increasing metabolism.

QUESTION 4 SINGLE CHOICE

A patient starting metformin therapy should be counseled about which of the following as the most common dose-limiting adverse effect?

- ☐ A Lactic acidosis
- ☐ B Hypoglycemia
- ☒ C Gastrointestinal upset such as nausea and diarrhea
- ☐ D Weight gain

ANSWER C

WHY Gastrointestinal effects (nausea, diarrhea, abdominal discomfort) are the most common adverse effects of metformin and the most frequent reason patients discontinue therapy. They are minimized by taking metformin with meals and titrating the dose slowly. Lactic acidosis is rare but serious; hypoglycemia is uncommon when metformin is used without insulin or sulfonylureas; metformin is typically weight-neutral or associated with modest weight loss.

QUESTION 5

SINGLE CHOICE

A physician orders gentamicin 1.5 mg/kg IV for a patient who weighs 70 kg. Gentamicin is available as 40 mg/mL in a 2 mL vial. How many millilitres should be administered?

- ☐ A 1.75 mL
- ☐ B 2.00 mL
- ☒ C 2.63 mL
- ☐ D 3.50 mL

ANSWER C

WHY Required dose = $1.5 \text{ mg/kg} \times 70 \text{ kg} = 105 \text{ mg}$. Volume = $105 \text{ mg} \div 40 \text{ mg/mL} = 2.625 \text{ mL}$, rounded to 2.63 mL. Option A reflects dividing 70 by 40 (treating weight as the dose), and Option B reflects not dividing by the concentration correctly.

QUESTION 6

SINGLE CHOICE

Look-alike/sound-alike (LASA) drug names are a recognized cause of medication errors. Which of the following is the most appropriate strategy to reduce LASA-related errors in a community pharmacy?

- ☒ A Store LASA pairs on different shelves separated by at least one other product, and include both the brand and generic name on the prescription label
- ☐ B Keep all LASA pairs together in a designated high-traffic section to encourage double-checking
- ☐ C Use only the brand name on labels so patients become familiar with one name only
- ☐ D Rely on pharmacists alone to catch LASA errors without changes to workflow

ANSWER A

WHY Standard patient-safety guidance for LASA medications includes physical separation on shelves (often with a non-LASA product between them), use of tall-man lettering, and clear labeling using both brand and generic names. Grouping LASA products together, using only the brand name, and relying solely on individual vigilance without workflow changes are not recommended.

QUESTION 7

SINGLE CHOICE

Which of the following best describes the mechanism of action of proton pump inhibitors such as omeprazole?

- ☐ A Reversible competitive antagonism of the H₂ receptor on gastric parietal cells
- ☒ B Irreversible inhibition of the H⁺/K⁺-ATPase (proton pump) of gastric parietal cells, activated in acidic environments
- ☐ C Neutralization of gastric acid via a weak base reaction with HCl in the stomach lumen
- ☐ D Blockade of muscarinic M₃ receptors on gastric parietal cells

ANSWER B

WHY PPIs are prodrugs that are activated in the acidic canaliculi of parietal cells, where they form covalent disulfide bonds with cysteine residues of the H⁺/K⁺-ATPase, irreversibly inhibiting acid secretion. H₂ blockers (e.g., ranitidine) act on H₂ receptors; antacids neutralize existing acid; pirenzepine-type agents act on muscarinic receptors.

QUESTION 8

SINGLE CHOICE

A patient receiving gentamicin 7 mg/kg IV once daily has a calculated creatinine clearance of 25 mL/min. Which adjustment to therapy is most appropriate?

- ☐ A Continue the same dose and interval because once-daily dosing is independent of renal function
- ☐ B Increase the dose because renal impairment reduces aminoglycoside exposure
- ☒ C Reduce the dose, extend the dosing interval, or switch to an alternative agent; monitor serum concentrations
- ☐ D Discontinue therapy immediately because aminoglycosides are contraindicated in renal impairment

ANSWER C

WHY Aminoglycosides are renally cleared and nephrotoxic. With a creatinine clearance of 25 mL/min, dose reduction, interval extension, or substitution of a less nephrotoxic agent is appropriate, along with monitoring of serum concentrations (peak/trough for traditional dosing or random levels for extended-interval dosing). They are not absolutely contraindicated in renal impairment but require careful adjustment.

QUESTION 9

MULTIPLE CHOICE

Which of the following drugs are well-known CYP3A4 substrates with a high risk of clinically significant interactions when co-administered with strong CYP3A4 inhibitors such as ketoconazole or clarithromycin? (Choose two.)

- ☒ **A** Atorvastatin
- ☐ **B** Acetaminophen
- ☒ **C** Simvastatin
- ☐ **D** Amoxicillin

ANSWER A - C

WHY Both atorvastatin and simvastatin are CYP3A4 substrates; strong inhibitors like ketoconazole and clarithromycin can markedly increase their plasma concentrations and the risk of statin-associated myopathy/rhabdomyolysis. Acetaminophen is primarily metabolized by glucuronidation and sulfation, and amoxicillin is excreted largely unchanged, making clinically significant CYP3A4-mediated interactions unlikely for these two agents.