

ANSWER KEY WITH EXPLANATIONS FOR CHAPTER 25

Skill Drill Answers

SD 25.1: Drug Class Matching

1. **A** — Omeprazole is a PPI
 2. **B** — Famotidine is an H₂ receptor antagonist
 3. **D** — Metoclopramide is a prokinetic
 4. **E** — Ondansetron is a 5-HT₃ antagonist
 5. **F** — Mesalamine is an aminosalicylate
 6. **C** — Calcium carbonate is an antacid
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SD 25.2: True/False with Correction

1. **F** — PPIs should be taken 30-60 minutes BEFORE meals for maximum efficacy (only inhibit active pumps).
 2. **F** — PPIs are MORE effective than H₂ blockers (>90% vs ~70% acid suppression).
 3. **T** — Metoclopramide can cause tardive dyskinesia, prompting a black box warning limiting use to ≤12 weeks.
 4. **F** — Loperamide does NOT readily cross the blood-brain barrier at usual doses, so it lacks CNS effects.
 5. **F** — Aminosalicylates have limited efficacy in Crohn's disease; biologics or immunomodulators are used for severe disease.
 6. **T** — H. pylori develops resistance to single antibiotics; combination therapy is required for eradication.
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SD 25.3: Fill in the Blank

1. **proton pump**
 2. **14**
 3. **acidifies**
 4. **NK1**
 5. **acidic**
 6. **$\alpha 4\beta 7$**
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SD 25.4: Acid-Suppression Comparison

1. **P** — Only PPIs irreversibly inhibit the proton pump
 2. **H** — H2 blockers develop tolerance (tachyphylaxis)
 3. **P** — PPIs should be taken before meals (H2 blockers timing less critical)
 4. **P** — PPIs achieve >90% suppression; H2 blockers ~70%
 5. **B** — Both are used for stress ulcer prophylaxis
 6. **H** — Only H2 blockers competitively block histamine receptors
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SD 25.5: Laxative Mechanism Matching

1. **A** — Psyllium is bulk-forming
 2. **B** — PEG is osmotic
 3. **C** — Senna is a stimulant
 4. **D** — Docusate is a stool softener
 5. **E** — Lubiprostone is a chloride channel activator
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SD 25.6: Rapid Recall

1. **PPIs (proton pump inhibitors)**
2. **Cimetidine**
3. **Misoprostol**
4. **5-HT₃ antagonists (setrons)**

5. **Loperamide**
 6. **Aminosalicylates**
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SD 25.7: Complete the Sentence

1. **prodrugs**
 2. **12**
 3. **clarithromycin**
 4. **fever and bloody stools**
 5. **sulfapyridine**
 6. **first-pass metabolism**
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SD 25.8: Antiemetic Receptor Matching

1. **B** — Ondansetron blocks 5-HT₃ receptors
 2. **A** — Prochlorperazine blocks D₂ receptors
 3. **C** — Aprepitant blocks NK₁ receptors
 4. **D** — Meclizine blocks H₁ receptors
 5. **E** — Scopolamine blocks muscarinic receptors
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SD 25.9: IBD Drug Classification

1. **A** — Mesalamine is an aminosalicylate
 2. **B** — Infliximab is a TNF- α inhibitor
 3. **C** — Vedolizumab is an anti-integrin (gut-selective)
 4. **D** — Ustekinumab is an IL-12/23 inhibitor
 5. **E** — Tofacitinib is a JAK inhibitor
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SD 25.10: H. pylori Regimen Components

1. **YES** — PPI is included
2. **YES** — Bismuth is included

3. **YES** — Metronidazole is included
 4. **NO** — Clarithromycin is NOT in bismuth quadruple therapy
 5. **YES** — Tetracycline is included
 6. **NO** — Amoxicillin is NOT in bismuth quadruple therapy
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SD 25.11: Adverse Effect Identification

1. **T** — Long-term PPI use may increase *C. difficile* risk by reducing gastric acid barrier
 2. **F** — Ondansetron commonly causes CONSTIPATION, not diarrhea
 3. **F** — Misoprostol is CONTRAINDICATED in pregnancy (causes uterine contractions, abortion)
 4. **T** — Cimetidine has anti-androgenic effects causing gynecomastia
 5. **T** — TNF inhibitors require TB screening due to reactivation risk
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SD 25.12: Clinical Scenario Drug Selection

1. **B** — PPIs are first-line for erosive esophagitis (superior healing)
 2. **C** — Three-drug regimen (5-HT₃ + NK₁ + steroid) for highly emetogenic chemo
 3. **A** — Oral + topical mesalamine for left-sided moderate UC
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Case Study Answers

Case 25.1: Peptic Ulcer Disease and *H. pylori*

Question 1. *H. pylori* is present in >90% of duodenal ulcers. The bacterium causes chronic gastritis and disrupts the normal protective mechanisms of the gastric and duodenal mucosa. It produces urease, which generates ammonia and creates a local alkaline environment allowing survival in the acidic stomach. The resulting inflammation and altered acid regulation lead to ulcer formation.

Question 2. *H. pylori* rapidly develops resistance to single antibiotics. Combination therapy with multiple antibiotics from different classes provides broader coverage and reduces the chance of resistance emerging during treatment. The PPI component raises gastric pH, which enhances antibiotic efficacy (some antibiotics work better at higher pH) and reduces bacterial load.

Question 3. For a treatment-naïve patient without allergies, appropriate first-line options include: (1) Bismuth quadruple therapy: PPI + bismuth + metronidazole + tetracycline for 14 days (preferred if

clarithromycin resistance rates are high locally), or (2) Concomitant therapy: PPI + clarithromycin + amoxicillin + metronidazole for 14 days (provides broader coverage than traditional triple therapy).

Question 4. Eradication should be confirmed at least 4 weeks after completing therapy using a urea breath test or stool antigen test. PPIs should be held for 1-2 weeks before testing to avoid false-negative results (PPIs suppress *H. pylori* without eradicating it). Successful eradication dramatically reduces ulcer recurrence risk.

Case 25.2: Inflammatory Bowel Disease Management

Question 1. Mesalamine is appropriate for mild-moderate ulcerative colitis but inadequate for severe flares. This patient has signs of severe colitis (8-10 bloody stools daily, fever, tachycardia, abdominal tenderness)—systemic inflammatory response requiring more potent immunosuppression. Aminosaliculates work topically with limited systemic anti-inflammatory effect, insufficient for controlling severe disease.

Question 2. Acute severe ulcerative colitis requires IV corticosteroids (methylprednisolone 40-60 mg daily or equivalent). If she were toxic-appearing or failing to improve, hospital admission with IV steroids, monitoring for toxic megacolon, and possible surgical consultation would be warranted. Supportive care includes IV fluids, correction of electrolyte abnormalities, and thromboprophylaxis (UC flares increase VTE risk).

Question 3. Corticosteroids effectively induce remission but do NOT prevent relapse when used for maintenance. Chronic steroid use causes serious adverse effects including osteoporosis, diabetes, adrenal suppression, increased infection risk, cataracts, and weight gain. The goal is always steroid-free remission maintained with safer agents (aminosalicylates, thiopurines, or biologics).

Question 4. If she fails to respond to IV steroids (steroid-refractory), biologic therapy is appropriate. Options include: TNF- α inhibitors (infliximab, adalimumab, golimumab—all approved for UC), vedolizumab (anti-integrin, gut-selective with favorable safety profile), or JAK inhibitors (tofacitinib, upadacitinib—oral option). Choice depends on patient preferences, comorbidities, and insurance/access considerations. All require appropriate screening (TB, hepatitis B) before initiation.

Diagram/Table Exercise Answers

DT 25.1: Gastric Acid Secretion and Drug Targets

1. PPIs irreversibly inhibit the **proton pump (H⁺/K⁺-ATPase)**, blocking the final common pathway.
2. H₂ receptor antagonists block stimulation by **histamine** from ECL cells.
3. All three stimulatory pathways (histamine, acetylcholine, gastrin) converge on the **proton pump**.
4. Antacids work by directly **neutralizing** acid in the gastric lumen.

DT 25.2: Antiemetic Drug Targets

1. 5-HT₃ antagonists (ondansetron) block **serotonin (5-HT₃)** receptors in the CTZ.
 2. NK₁ antagonists (aprepitant) block **substance P (NK₁)** receptors.
 3. Motion sickness is best treated with **antihistamines** or anticholinergics.
 4. The three-drug regimen for highly emetogenic chemotherapy combines 5-HT₃ antagonist + **NK₁ antagonist (aprepitant)** + dexamethasone.
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DT 25.3: Acid-Suppression Drug Comparison Table

Drug Class	Mechanism	Acid Suppression	Key Consideration
PPIs	Irreversible proton pump inhibition	>90%	Take 30-60 min before meals
H₂ blockers	Competitive H₂ receptor blockade	~70%	Tolerance develops
Antacids	Neutralize gastric acid	Variable, short-lived	Drug interactions (separate by 2 hrs)
Sucralfate	Forms protective barrier	None (mucosal protection)	Requires acidic environment

DT 25.4: IBD Medication Comparison Table

Drug Class	Example	Mechanism	Role in IBD
Aminosalicylate	Mesalamine	Topical anti-inflammatory	First-line for mild-moderate UC
Corticosteroid	Prednisone	Broad immunosuppression	Induction only (not maintenance)
Thiopurine	Azathioprine	Inhibits purine synthesis	Steroid-sparing maintenance
TNF inhibitor	Infliximab	Blocks TNF-α cytokine	Moderate-severe IBD
Anti-integrin	Vedolizumab	Blocks gut lymphocyte trafficking	Moderate-severe IBD (gut-selective)

Multiple-Choice Question Answers

Question 1. Answer: C

PPIs work by irreversibly inhibiting the H⁺/K⁺-ATPase (proton pump) on parietal cells. This is the final common pathway for acid secretion, making PPIs the most effective acid-suppressing agents.

Question 2. Answer: C

PPIs should be taken 30-60 minutes before a meal. They only inhibit actively secreting proton pumps, and meal stimulation activates pumps, enhancing drug efficacy.

Question 3. Answer: C

5-HT₃ antagonists (ondansetron, granisetron) are the most effective agents for chemotherapy-induced nausea and vomiting. They block serotonin receptors in the CTZ and vagal afferents.

Question 4. Answer: C

Standard H. pylori eradication regimens are 14 days. Shorter courses (7-10 days) have lower eradication rates due to increasing antibiotic resistance.

Question 5. Answer: B

Aminosalicylates (mesalamine, sulfasalazine) are first-line therapy for mild-moderate ulcerative colitis, both for induction and maintenance of remission.

Question 6. Answer: C

Cimetidine inhibits multiple CYP450 enzymes (causing drug interactions) and has anti-androgenic effects (causing gynecomastia and impotence). Famotidine is preferred due to lack of these issues.

Question 7. Answer: B

Metoclopramide's black box warning is for tardive dyskinesia—an often irreversible movement disorder. Use is limited to ≤12 weeks.

Question 8. Answer: C

Loperamide should be avoided in diarrhea with fever and bloody stools, which suggest invasive bacterial infection. Slowing motility may prolong pathogen exposure and worsen outcomes.

Question 9. Answer: B

Misoprostol, a prostaglandin E1 analogue, induces uterine contractions and is an abortifacient. It is absolutely contraindicated in pregnancy (black box warning).

Question 10. Answer: B

Lactulose is metabolized by colonic bacteria to organic acids, acidifying the colon. This converts ammonia (NH₃) to ammonium (NH₄⁺), which is trapped in the colon and excreted, reducing systemic ammonia levels in hepatic encephalopathy.

Question 11. Answer: B

Vedolizumab blocks $\alpha4\beta7$ integrin, which is specifically involved in lymphocyte trafficking to the gut. This "gut-selective" mechanism provides efficacy in IBD with less systemic immunosuppression.

Question 12. Answer: B

PPI-associated hypomagnesemia is a recognized complication. Management includes considering PPI discontinuation if possible, switching to an H₂ blocker for less acid suppression needs, or magnesium supplementation if PPI must continue.

Question 13. Answer: C

Bismuth quadruple therapy includes PPI + bismuth + metronidazole + tetracycline. It does NOT include clarithromycin, making it preferred when clarithromycin resistance is suspected.

Question 14. Answer: B

The standard three-drug antiemetic regimen for highly emetogenic chemotherapy is ondansetron (5-HT₃ antagonist) + aprepitant (NK1 antagonist) + dexamethasone.

Question 15. Answer: C

Corticosteroids are effective for inducing remission in IBD flares but NOT for maintenance therapy—they don't prevent relapse and cause serious adverse effects with chronic use.

Question 16. Answer: B

TNF inhibitors (infliximab, adalimumab, etc.) can reactivate latent tuberculosis. TB screening (tuberculin skin test or interferon-gamma release assay) is required before initiating therapy.