

ANSWER KEY WITH EXPLANATIONS FOR CHAPTER 7

Skill Drill Answers

SD 7.1: Receptor-Drug Matching

1. **A** — Phenylephrine is an α_1 agonist
2. **C** — Albuterol is a β_2 selective agonist
3. **D** — Epinephrine is non-selective (α and β)
4. **B** — Dobutamine is relatively β_1 selective
5. **E** — Propranolol is a non-selective β blocker
6. **F** — Metoprolol is a β_1 selective blocker
7. **D** — Norepinephrine activates α_1 , α_2 , and β_1 (minimal β_2)
8. **C** — Salmeterol is a long-acting β_2 selective agonist

SD 7.2: True/False with Correction

1. **T** — Tyrosine hydroxylase is the rate-limiting enzyme.
2. **F** — Norepinephrine has MINIMAL β_2 activity; it lacks significant bronchodilating effect.
3. **T** — β_1 -selective blockers have less effect on bronchial β_2 receptors.
4. **F** — α_1 activation causes VASOCONSTRICTION and INCREASED blood pressure.
5. **T** — Cocaine blocks NET, preventing NE reuptake.
6. **F** — Propranolol is CONTRAINDICATED in asthma due to β_2 blockade.

SD 7.3: Fill in the Blank

1. **reuptake**
 2. **bronchodilation**
 3. **tamsulosin**
 4. **agonists**
 5. **anaphylaxis**
 6. **esmolol**
-

SD 7.4: Selectivity Classification

1. **C** — Metoprolol is β_1 selective
 2. **A** — Epinephrine is non-selective
 3. **B** — Prazosin is α_1 selective
 4. **D** — Albuterol is β_2 selective
 5. **E** — Propranolol is non-selective β blocker
 6. **D** — Terbutaline is β_2 selective
 7. **C** — Atenolol is β_1 selective
 8. **B** — Tamsulosin is α_1 selective (specifically α_{1A})
-

SD 7.5: Direct vs. Indirect Acting Sorting

1. **D** — Epinephrine directly binds receptors
 2. **I** — Amphetamine releases stored NE
 3. **D** — Phenylephrine directly binds α_1 receptors
 4. **I** — Cocaine blocks NE reuptake
 5. **D** — Albuterol directly binds β_2 receptors
 6. **I** — Tyramine releases stored NE
 7. **D** — Dobutamine directly binds receptors
 8. **D** — Norepinephrine directly binds receptors
-

SD 7.6: Rapid Recall

1. Dopamine β -hydroxylase
 2. β_2 (Beta-2)
 3. Epinephrine
 4. Esmolol
 5. Phenoxybenzamine
 6. Norepinephrine
-

SD 7.7: Complete the Sentence

1. contraction
 2. relaxation
 3. β_1
 4. negative
 5. reuptake
 6. asthma
-

SD 7.8: Hemodynamic Effect Prediction

1. Norepinephrine: HR: $\downarrow$ (reflex) BP: $\uparrow$ PR: $\uparrow$
 2. Isoproterenol: HR: $\uparrow$ BP: $\downarrow$ (diastolic) PR: $\downarrow$
 3. Phenylephrine: HR: $\downarrow$ (reflex) BP: $\uparrow$ PR: $\uparrow$
 4. Propranolol: HR: $\downarrow$ BP: $\downarrow$ PR: $\uparrow$ (initially, due to β_2 blockade)
-

SD 7.9: Beta-Blocker Property Matching

1. A — Propranolol is non-selective, lipophilic, crosses BBB
2. B — Metoprolol is β_1 selective, used in heart failure
3. C — Esmolol is ultra-short acting
4. D — Carvedilol has combined α and β blockade
5. E — Pindolol has ISA
6. B — Metoprolol has long-acting (succinate) formulation

SD 7.10: Catecholamine Metabolism Pathway

Tyrosine → DOPA: 1

DOPA → Dopamine: 2

Dopamine → Norepinephrine: 3

Norepinephrine → Epinephrine: 4

SD 7.11: Contraindication Identification

1. **B** — Propranolol is contraindicated in asthma
 2. **B** — Prazosin worsens orthostatic hypotension
 3. **C** — Albuterol can worsen tachycardia and hypertension
-

SD 7.12: Pheochromocytoma Drug Selection

1. **B** — Alpha receptors must be blocked first
2. Without alpha-blockade, blocking β_2 -mediated vasodilation leaves α -mediated vasoconstriction unopposed, potentially causing hypertensive crisis.
3. **C** — Phenoxybenzamine (irreversible α -blocker) is used preoperatively

Case Study Answers

Case 7.1: Anaphylaxis Epinephrine Administration

Question 1. β_1 activation increases heart rate and contractility, supporting cardiac output against hypotension. β_2 activation relaxes bronchial smooth muscle (reversing bronchospasm), stabilizes mast cells (reducing further mediator release), and causes vasodilation in skeletal muscle beds. α_1 activation causes vasoconstriction (reversing hypotension) and reduces vascular permeability (decreasing edema in airways and skin).

Question 2. IM injection into the lateral thigh provides faster absorption due to greater vascularity compared to subcutaneous injection. The lateral thigh has consistent muscle mass for reliable delivery. SC absorption is slower and less predictable, especially in hypotensive patients with peripheral vasoconstriction.

Question 3. Propranolol blocks both β_1 and β_2 receptors. This may blunt epinephrine's cardiac stimulation and bronchodilation, making the anaphylaxis more difficult to reverse. Higher doses of epinephrine may be needed. Glucagon can be used as an alternative since it increases cardiac output through a non-adrenergic mechanism.

Question 4. Additional treatments include IV fluids for volume resuscitation, H1 antihistamines (diphenhydramine), H2 antihistamines (ranitidine), corticosteroids (to prevent biphasic reactions), and nebulized albuterol for persistent bronchospasm. If hypotension persists despite epinephrine, vasopressors (norepinephrine) may be needed.

Case 7.2: Beta-Blocker Selection in Heart Failure

Question 1. Carvedilol or metoprolol succinate would be most appropriate. Both have proven mortality benefit in HFrEF. Carvedilol's additional α_1 -blocking activity provides vasodilation that may help offset negative inotropic effects. Either is acceptable; carvedilol may be preferred given the patient's diabetes (better metabolic profile) and COPD (α -blockade may partially offset bronchospasm concerns).

Question 2. Heart failure patients depend on sympathetic drive to maintain cardiac output. Abrupt beta-blockade can precipitate acute decompensation. Starting at very low doses allows gradual adaptation while gaining long-term benefits of neurohormonal blockade and reverse remodeling.

Question 3. COPD makes β_1 -selective blockers preferable since they have less effect on bronchial β_2 receptors. However, carvedilol (non-selective) has been safely used in patients with COPD in clinical trials. The benefit in heart failure generally outweighs the risk in well-controlled COPD. Severe or poorly controlled COPD would favor metoprolol succinate.

Question 4. In the short term, beta-blockers do reduce contractility. However, chronic sympathetic activation in heart failure is harmful—it causes toxic effects on the heart muscle, worsens remodeling, and increases mortality. By blocking this harmful stimulation, beta-blockers allow the heart to rest and gradually recover. Over months, ejection fraction improves, symptoms decrease, and survival increases. Multiple large trials have proven mortality reduction.

Diagram/Table Exercise Answers

DT 7.1: Adrenergic Receptor Effects

1. β_1 receptor activation in the heart causes: **Increased heart rate and contractility**
2. β_2 receptor activation in the lungs causes: **Bronchodilation**
3. α_1 receptor activation in blood vessels causes: **Vasoconstriction**
4. α_2 receptor activation on presynaptic terminals causes: **Decreased norepinephrine release**

DT 7.2: Catecholamine Termination

1. The primary mechanism for NE termination is: **Reuptake (uptake-1)**
 2. The enzyme in nerve terminal mitochondria that metabolizes NE is: **MAO (monoamine oxidase)**
 3. The extraneuronal enzyme that metabolizes catecholamines is: **COMT (catechol-O-methyltransferase)**
 4. Drugs that block reuptake (like cocaine) cause NE to: **Accumulate in the synapse**
-

DT 7.3: Adrenergic Receptor Distribution Table

Receptor	Primary Location	G-Protein	Effect of Activation
$\alpha 1$	Vascular smooth muscle	Gq	Vasoconstriction
$\alpha 2$	Presynaptic terminals	Gi	↓ NE release
$\beta 1$	Heart	Gs	↑ HR, ↑ contractility
$\beta 2$	Bronchial smooth muscle	Gs	Bronchodilation
$\beta 3$	Adipose tissue	Gs	Lipolysis

DT 7.4: Beta-Blocker Comparison Table

Drug	$\beta 1$ Selectivity	ISA	Additional Properties
Propranolol	Non-selective	No	Lipophilic, crosses BBB
Metoprolol	$\beta 1$ selective	No	Available IV and PO
Carvedilol	Non-selective	No	$\alpha 1$ blocking activity
Esmolol	$\beta 1$ selective	No	Ultra-short acting ($t_{1/2}$ ~9 min)
Pindolol	Non-selective	Yes	Less resting bradycardia

Multiple-Choice Question Answers

Question 1. Answer: C

Tyrosine hydroxylase is the rate-limiting enzyme, converting tyrosine to DOPA.

Question 2. Answer: B

β_2 receptor activation in bronchial smooth muscle causes bronchodilation through smooth muscle relaxation.

Question 3. Answer: C

Phenylephrine is a selective α_1 agonist used as a nasal decongestant, mydriatic, and vasopressor.

Question 4. Answer: B

Propranolol blocks β_2 receptors in bronchial smooth muscle, preventing bronchodilation and potentially causing bronchospasm.

Question 5. Answer: C

Epinephrine is first-line for anaphylaxis because it addresses all pathophysiologic components.

Question 6. Answer: B

Norepinephrine causes significant α_1 -mediated vasoconstriction, raising blood pressure. The baroreceptor reflex response to hypertension causes bradycardia that overcomes the direct β_1 stimulation.

Question 7. Answer: C

Esmolol has an ultra-short half-life (~9 minutes), allowing rapid titration and quick offset if adverse effects occur.

Question 8. Answer: B

Cocaine blocks the norepinephrine transporter (NET), preventing reuptake and prolonging NE action at receptors.

Question 9. Answer: B

Tyramine in aged cheese is normally metabolized by MAO. With MAO inhibited, tyramine enters nerve terminals and releases norepinephrine, causing hypertensive crisis.

Question 10. Answer: B

Carvedilol has both β -blocking and α 1-blocking activity, while metoprolol is a pure β 1-selective blocker.

Question 11. Answer: B

First-dose phenomenon refers to severe orthostatic hypotension that can occur with the initial dose of α 1-blockers like prazosin.

Question 12. Answer: B

Without α -blockade, β -blockers remove β 2-mediated vasodilation, leaving α 1-mediated vasoconstriction unopposed and potentially causing severe hypertensive crisis.

Question 13. Answer: B

Beta-blockers block the sympathetic response to hypoglycemia, masking warning symptoms like tachycardia and tremor, and can prolong hypoglycemia by impairing glycogenolysis.

Question 14. Answer: B

Isoproterenol activates β 2 receptors in vascular smooth muscle, causing vasodilation and decreased peripheral resistance, which lowers diastolic blood pressure.

Question 15. Answer: C

Dobutamine has relative β 1 selectivity, increasing cardiac contractility with less tachycardia and vasoconstriction than other agents.

Question 16. Answer: B

Tamsulosin can cause intraoperative floppy iris syndrome (IFIS), a complication during cataract surgery where the iris becomes flaccid and billows. Ophthalmologists should be informed to prepare for this.