

ANSWER KEY WITH EXPLANATIONS FOR CHAPTER 5

Skill Drill Answers

SD 5.1: ANS Division Matching

1. **S** — Thoracolumbar origin is sympathetic
 2. **P** — "Rest and digest" is parasympathetic
 3. **P** — Parasympathetic has long preganglionic, short postganglionic
 4. **S** — Norepinephrine is released by sympathetic postganglionic neurons
 5. **P** — Craniosacral origin is parasympathetic
 6. **S** — "Fight or flight" is sympathetic
 7. **P** — Parasympathetic ganglia are near/within effector organs
 8. **S** — Sympathetic has short preganglionic, long postganglionic
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SD 5.2: True/False with Correction

1. **F** — All preganglionic neurons release acetylcholine, not norepinephrine.
 2. **T** — Parasympathetic originates from cranial nerves (III, VII, IX, X) and sacral segments (S2–S4).
 3. **T** — Nicotinic receptors are found at all autonomic ganglia.
 4. **F** — Sympathetic neurons to sweat glands are an exception; they release acetylcholine.
 5. **F** — Acetylcholine is degraded by acetylcholinesterase, not MAO.
 6. **T** — The adrenal medulla releases approximately 80% epinephrine and 20% norepinephrine.
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SD 5.3: Fill in the Blank

1. **parasympathetic**

2. **acetylcholine**
 3. **choline acetyltransferase**
 4. **tyrosine hydroxylase**
 5. **heart**
 6. **reuptake**
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SD 5.4: Receptor-Effector Organ Pairing

1. **A** — β_1 receptors are in the heart
 2. **B** — α_1 receptors cause vasoconstriction
 3. **F** — M3 receptors in GI tract cause contraction
 4. **C** — β_2 receptors cause bronchodilation
 5. **D** — Nn receptors are at autonomic ganglia
 6. **E** — α_1 receptors in the eye dilate the pupil
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SD 5.5: Fight-or-Flight vs. Rest-and-Digest Sorting

1. **S** — Sympathetic increases heart rate
 2. **P** — Parasympathetic causes miosis
 3. **S** — Sympathetic causes bronchodilation
 4. **P** — Parasympathetic increases GI motility
 5. **S** — Sympathetic causes mydriasis
 6. **P** — Parasympathetic decreases heart rate
 7. **P** — Parasympathetic increases salivation
 8. **S** — Sympathetic stimulates glycogenolysis
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SD 5.6: Rapid Recall

1. **Acetylcholine**
2. **Nicotinic (Nn)**
3. **Acetylcholinesterase**

4. **Thoracolumbar (T1–L2)**
 5. **β 1**
 6. **Epinephrine**
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SD 5.7: Complete the Sentence

1. **short**
 2. **Gi**
 3. **contraction**
 4. **relaxation**
 5. **tyrosine**
 6. **acetylcholine**
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SD 5.8: Neurotransmitter Pathway Sequencing

Norepinephrine Synthesis:

- Tyrosine is transported into the nerve terminal: **1**
- Tyrosine is converted to DOPA by tyrosine hydroxylase: **2**
- DOPA is converted to dopamine by DOPA decarboxylase: **3**
- Dopamine is converted to norepinephrine by dopamine β -hydroxylase: **4**

Acetylcholine Lifecycle:

- ACh is synthesized from choline and acetyl-CoA: **1**
 - ACh is stored in vesicles: **2**
 - ACh is released by exocytosis: **3**
 - ACh binds to postsynaptic receptor: **4**
 - ACh is hydrolyzed by acetylcholinesterase: **5**
 - Choline is transported back into nerve terminal: **6**
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SD 5.9: Receptor Subtype Classification

1. **N** — Nicotinic Nm at neuromuscular junction

2. β — β_2 causes bronchodilation
 3. **M** — M2 decreases heart rate
 4. **N** — Nicotinic Nn at ganglia
 5. α — α_1 causes vasoconstriction
 6. β — β_1 increases heart rate and contractility
 7. **M** — M3 in GI smooth muscle
 8. α — α_2 presynaptic inhibits NE release
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SD 5.10: Autonomic Reflex Analysis

1. Sympathetic outflow to the heart increases to raise heart rate and restore blood pressure (baroreceptor reflex).
 2. Parasympathetic division is activated to stimulate digestion.
 3. Heart rate increases because parasympathetic (vagal) tone is blocked, leaving sympathetic tone unopposed.
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SD 5.11: Ganglionic vs. Postganglionic Identification

1. Sympathetic ganglion: **Acetylcholine, Nicotinic (Nn)**
 2. Parasympathetic ganglion: **Acetylcholine, Nicotinic (Nn)**
 3. Sympathetic neuroeffector junction: **Norepinephrine, α or β (varies by tissue)**
 4. Parasympathetic neuroeffector junction: **Acetylcholine, Muscarinic (M2, M3)**
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SD 5.12: Response Prediction

1. β_1 receptor activation: **Increased heart rate and contractility**
2. M3 receptor activation in bronchial smooth muscle: **Bronchoconstriction**
3. α_1 receptor blockade: **Vasodilation, decreased blood pressure**
4. M2 receptor blockade: **Increased heart rate (removes vagal inhibition)**

Case Study Answers

Case 5.1: Autonomic Dysfunction Assessment

Question 1. Standing causes blood to pool in the legs, reducing venous return and blood pressure. Baroreceptors detect this and trigger sympathetic activation: increased heart rate (β_1), vasoconstriction (α_1), and maintained blood pressure.

Question 2. Diabetic autonomic neuropathy damages the sympathetic neurons that normally increase heart rate in response to hypotension. The baroreceptor reflex arc is impaired, preventing compensatory tachycardia.

Question 3. Parasympathetic division stimulates detrusor contraction (M3 receptors), enabling bladder emptying.

Question 4. Diabetic autonomic neuropathy causes gradual, progressive nerve damage affecting both divisions variably. Ganglionic blockers cause acute, complete blockade of both divisions simultaneously with more predictable, immediate effects.

Case 5.2: Organophosphate Poisoning

Question 1. Organophosphates irreversibly inhibit acetylcholinesterase, preventing ACh breakdown. ACh accumulates at all cholinergic synapses, causing excessive stimulation of muscarinic receptors (SLUD, bradycardia, miosis, bronchospasm) and nicotinic receptors (muscle fasciculations, weakness).

Question 2. SLUD symptoms reflect excessive muscarinic (M3) receptor stimulation at glands and smooth muscle: salivary glands, lacrimal glands, bladder detrusor, and GI smooth muscle.

Question 3. Bradycardia results from excessive ACh at cardiac M2 receptors, which decrease heart rate through Gi-mediated reduction in cAMP and increased K^+ conductance.

Question 4. Atropine is a muscarinic antagonist that blocks ACh at muscarinic receptors. It reverses SLUD symptoms, bronchospasm, and bradycardia but does NOT reverse nicotinic effects (skeletal muscle weakness, fasciculations).

Diagram/Table Exercise Answers

DT 5.1: Autonomic Pathway Diagram

1. All preganglionic neurons release: **Acetylcholine**
2. The receptor at all autonomic ganglia is: **Nicotinic (Nn)**

- Sympathetic postganglionic neurons typically release: **Norepinephrine**
- Parasympathetic postganglionic neurons release: **Acetylcholine**

DT 5.2: Receptor Distribution Table

Receptor	Primary Location	G-Protein	Effect of Activation
$\alpha 1$	Vascular smooth muscle	Gq	Vasoconstriction
$\alpha 2$	Presynaptic terminals	Gi	Decreased NE release
$\beta 1$	Heart	Gs	Increased HR and contractility
$\beta 2$	Bronchial smooth muscle	Gs	Bronchodilation
M2	Heart	Gi	Decreased heart rate
M3	GI smooth muscle, glands	Gq	Contraction, secretion

DT 5.3: Neurotransmitter Comparison

Property	Acetylcholine	Norepinephrine
Precursor	Choline + Acetyl-CoA	Tyrosine
Synthesizing enzyme	Choline acetyltransferase	Tyrosine hydroxylase (rate-limiting)
Termination mechanism	Enzymatic hydrolysis	Reuptake (uptake-1)
Degrading enzyme	Acetylcholinesterase	MAO and COMT

DT 5.4: Organ Response Chart

Organ	Sympathetic Effect	Receptor	Parasympathetic Effect	Receptor
Heart (rate)	Increased	$\beta 1$	Decreased	M2
Bronchioles	Dilation	$\beta 2$	Constriction	M3
Pupil	Dilation (mydriasis)	$\alpha 1$	Constriction (miosis)	M3
GI motility	Decreased	$\alpha 2, \beta 2$	Increased	M3
Blood vessels	Constriction	$\alpha 1$	Little effect	—

Multiple-Choice Question Answers

Question 1. Answer: C

All preganglionic autonomic neurons release acetylcholine.

Question 2. Answer: B

The sympathetic division originates from thoracolumbar spinal cord (T1–L2).

Question 3. Answer: B

Nicotinic receptors (Nn subtype) are found at all autonomic ganglia.

Question 4. Answer: B

β_1 receptor activation increases heart rate and contractility.

Question 5. Answer: C

Acetylcholinesterase degrades acetylcholine in the synaptic cleft.

Question 6. Answer: B

Pupil dilation, increased heart rate, and bronchodilation are sympathetic ("fight or flight") effects.

Question 7. Answer: B

Parasympathetic division has long preganglionic neurons and short postganglionic neurons because ganglia are located near effector organs.

Question 8. Answer: B

The adrenal medulla releases approximately 80% epinephrine and 20% norepinephrine.

Question 9. Answer: B

M2 receptors couple to Gi protein, decreasing cAMP and heart rate.

Question 10. Answer: C

Alpha-1 (α_1) receptor activation causes vasoconstriction in most vascular beds.

Question 11. Answer: C

Sympathetic neurons to sweat glands are an exception—they release acetylcholine, not norepinephrine.

Question 12. Answer: B

At rest, parasympathetic (vagal) tone dominates at the heart, keeping heart rate low. Blocking ganglionic transmission removes this vagal tone, resulting in increased heart rate.

Question 13. Answer: C

Tyrosine hydroxylase catalyzes the rate-limiting step: tyrosine $\rightarrow$ DOPA.

Question 14. Answer: A

Organophosphate poisoning causes accumulation of acetylcholine, excessively activating both nicotinic and muscarinic receptors.

Question 15. Answer: B

Presynaptic α_2 receptors provide negative feedback, inhibiting further norepinephrine release when activated.

Question 16. Answer: B

With beta receptors blocked, epinephrine's alpha effects (vasoconstriction) are unopposed, causing severe hypertension. This is why non-selective beta-blockers are contraindicated with epinephrine in some situations.