

ANSWER KEY WITH EXPLANATIONS FOR CHAPTER 6

Skill Drill Answers

SD 6.1: Drug-Receptor Matching

1. **B** — Atropine is a muscarinic receptor antagonist
 2. **D** — Neostigmine is an acetylcholinesterase inhibitor
 3. **C** — Rocuronium is a nicotinic Nm receptor blocker
 4. **A** — Pilocarpine is a muscarinic receptor agonist
 5. **C** — Succinylcholine acts at nicotinic Nm receptors (depolarizing blocker)
 6. **A** — Bethanechol is a muscarinic receptor agonist
 7. **B** — Ipratropium is a muscarinic receptor antagonist
 8. **D** — Pyridostigmine is an acetylcholinesterase inhibitor
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SD 6.2: True/False with Correction

1. **T** — Atropine blocks muscarinic receptors, reversing muscarinic effects of organophosphate poisoning.
 2. **F** — Succinylcholine is a **DEPOLARIZING** neuromuscular blocker.
 3. **F** — Neostigmine cannot reverse succinylcholine; it would prolong its effect by inhibiting plasma cholinesterase.
 4. **F** — Pyridostigmine is a quaternary amine and does **NOT** cross the blood-brain barrier readily.
 5. **F** — Pralidoxime reactivates acetylcholinesterase; it does not block muscarinic receptors.
 6. **F** — Ipratropium is a quaternary amine with minimal systemic absorption when inhaled.
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SD 6.3: Fill in the Blank

1. **retention**
 2. **acetylcholinesterase**
 3. **irreversible**
 4. **pralidoxime (2-PAM)**
 5. **fasciculations**
 6. **neostigmine (or anticholinesterases)**
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SD 6.4: Direct vs. Indirect Acting Classification

1. **D** — Bethanechol directly activates muscarinic receptors
 2. **I** — Neostigmine inhibits AChE (indirect)
 3. **D** — Pilocarpine directly activates muscarinic receptors
 4. **I** — Donepezil inhibits AChE (indirect)
 5. **D** — Carbachol directly activates receptors
 6. **I** — Pyridostigmine inhibits AChE (indirect)
 7. **I** — Physostigmine inhibits AChE (indirect)
 8. **D** — Methacholine directly activates muscarinic receptors
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SD 6.5: Depolarizing vs. Non-depolarizing Sorting

1. **D** — Succinylcholine is depolarizing
 2. **ND** — Rocuronium is non-depolarizing
 3. **ND** — Vecuronium is non-depolarizing
 4. **ND** — Cisatracurium is non-depolarizing
 5. **ND** — Pancuronium is non-depolarizing
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SD 6.6: Rapid Recall

1. **Ipratropium or Tiotropium**
2. **Physostigmine**

3. **Succinylcholine**
 4. **Sugammadex**
 5. **Pilocarpine**
 6. **Pyridostigmine**
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SD 6.7: Complete the Sentence

1. **crosses**
 2. **plasma cholinesterase**
 3. **muscarinic**
 4. **antagonists**
 5. **before**
 6. **overactive bladder**
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SD 6.8: Antidote Pairing

1. **B** — Physostigmine for anticholinergic toxicity
 2. **A** — Atropine for muscarinic effects of organophosphates
 3. **C** — Pralidoxime for nicotinic effects (reactivates AChE)
 4. **D** — Neostigmine reverses non-depolarizing blockade
 5. **E** — Sugammadex specifically reverses rocuronium
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SD 6.9: Duration of Action Ranking

Edrophonium: **1** (shortest, minutes)

Neostigmine: **2**

Pyridostigmine: **3**

Organophosphates: **4** (irreversible, permanent until new enzyme synthesized)

SD 6.10: Reversal Agent Selection

1. **B** — Sugammadex provides rapid, complete reversal of rocuronium

2. **D** — No pharmacologic reversal for succinylcholine; supportive care until recovery
 3. **B** — Both options work, but sugammadex is effective at any level of blockade
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SD 6.11: Adverse Effect Clustering

1. **B** — Muscarinic antagonist toxicity (blind, dry, mad, hot)
 2. **C** — Depolarizing neuromuscular blocker (succinylcholine)
 3. **D** — Irreversible anticholinesterase toxicity (organophosphate poisoning)
 4. **A** — Muscarinic agonist toxicity (excessive parasympathetic effects)
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SD 6.12: Clinical Indication Matching

1. **A** — Pyridostigmine for myasthenia gravis
2. **C** — Tiotropium for COPD
3. **E** — Donepezil for Alzheimer's disease
4. **B** — Pilocarpine for glaucoma
5. **D** — Oxybutynin for overactive bladder
6. **F** — Pralidoxime + Atropine for organophosphate poisoning

Case Study Answers

Case 6.1: Myasthenia Gravis Management

Question 1. Myasthenia gravis is an autoimmune disease where antibodies attack nicotinic acetylcholine receptors at the neuromuscular junction, reducing functional receptors. With fewer receptors, normal ACh release produces inadequate muscle stimulation. Symptoms worsen with activity because ACh is depleted with repeated stimulation, and fewer receptors are available to respond.

Question 2. Pyridostigmine inhibits acetylcholinesterase, preventing ACh breakdown. More ACh accumulates at the neuromuscular junction, increasing the chance of activating the remaining receptors and improving muscle contraction.

Question 3. GI adverse effects (cramping, diarrhea) result from increased ACh at muscarinic M3 receptors in the GI tract, stimulating motility and secretions. Management options include dose reduction, taking medication with food, or adding an anticholinergic like glycopyrrolate (which does not cross the BBB and therefore won't reverse muscle benefits).

Question 4. Cholinergic crisis results from excessive ACh due to pyridostigmine overdose, featuring SLUD symptoms plus muscle weakness from depolarization blockade at the NMJ. Myasthenic crisis results from insufficient ACh effect due to disease worsening, featuring muscle weakness without SLUD symptoms. Historically, edrophonium (Tensilon) was used to distinguish them—improvement suggests myasthenic crisis; worsening suggests cholinergic crisis.

Case 6.2: Anesthesia Neuromuscular Blockade

Question 1. Rocuronium is a non-depolarizing neuromuscular blocker that competitively antagonizes ACh at nicotinic Nm receptors. This prevents depolarization and muscle contraction, producing paralysis. It was chosen for its intermediate duration, rapid onset, and reversibility.

Question 2. TOF of 2/4 indicates moderate neuromuscular blockade—approximately 75-80% of receptors are still blocked. This is insufficient for safe extubation (which requires 4/4 with no fade) but indicates some recovery is occurring.

Question 3. Neostigmine increases ACh by inhibiting AChE, allowing it to compete with and displace the blocker. It requires adequate recovery (TOF $\geq 2/4$) and causes muscarinic side effects (bradycardia) requiring co-administration of glycopyrrolate. Sugammadex directly encapsulates rocuronium molecules, works at any depth of blockade, acts faster, and requires no anticholinergic. For rapid reversal, sugammadex is preferred.

Question 4. Neostigmine inhibits AChE at all cholinergic synapses, including the heart (M2 receptors), causing bradycardia. Glycopyrrolate is a muscarinic antagonist that blocks this cardiac effect without crossing the BBB (quaternary amine). It prevents the dangerous bradycardia that would otherwise occur.

Diagram/Table Exercise Answers

DT 6.1: Cholinergic Synapse Drug Targets

1. Site 1 (receptor activation): **Muscarinic agonists** (e.g., bethanechol, pilocarpine)
 2. Site 2 (enzyme inhibition): **Anticholinesterases** (e.g., neostigmine, pyridostigmine)
 3. Site 3 (receptor blockade): **Muscarinic antagonists** (e.g., atropine, ipratropium)
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DT 6.2: Anticholinesterase Mechanism

1. Anticholinesterases inhibit the enzyme: **Acetylcholinesterase (AChE)**
2. This causes ACh to: **Accumulate in the synapse**
3. The result is: **Increased** cholinergic signaling

4. Name one reversible anticholinesterase: **Neostigmine** (or pyridostigmine, physostigmine, donepezil)
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DT 6.3: Muscarinic Drug Comparison Table

Effect	Muscarinic Agonist	Muscarinic Antagonist
Heart rate	Decreased	Increased
Pupil	Miosis	Mydriasis
Bronchial smooth muscle	Constriction	Dilation
GI motility	Increased	Decreased
Secretions	Increased	Decreased
Example drug	Pilocarpine	Atropine

DT 6.4: Neuromuscular Blocker Comparison Table

Property	Succinylcholine	Rocuronium
Mechanism	Persistent depolarization	Competitive antagonism
Fasciculations	Yes	No
Duration	Ultra-short (5-10 min)	Intermediate
Reversal agent	None	Neostigmine or Sugammadex
Hyperkalemia risk	Yes	No

Multiple-Choice Question Answers

Question 1. Answer: B

Bethanechol is a direct-acting muscarinic agonist used for urinary retention and GI atony.

Question 2. Answer: C

Atropine is a muscarinic antagonist that **DECREASES** GI motility, not increases it.

Question 3. Answer: C

Pyridostigmine is first-line for myasthenia gravis due to its intermediate duration and oral bioavailability.

Question 4. Answer: B

Succinylcholine causes initial fasciculations due to depolarization before paralysis occurs.

Question 5. Answer: C

Physostigmine is a tertiary amine that crosses the BBB to reverse CNS anticholinergic toxicity.

Question 6. Answer: B

Tiotropium is a long-acting muscarinic antagonist allowing once-daily dosing with minimal systemic absorption.

Question 7. Answer: B

Pralidoxime reactivates acetylcholinesterase by cleaving the organophosphate-enzyme bond.

Question 8. Answer: C

SLUD symptoms with bradycardia indicate excessive cholinergic stimulation from too much pyridostigmine.

Question 9. Answer: C

Cisatracurium undergoes Hofmann elimination (spontaneous degradation) independent of renal or hepatic function.

Question 10. Answer: B

Burns cause upregulation of extrajunctional ACh receptors, and succinylcholine-induced depolarization releases massive amounts of potassium, causing fatal hyperkalemia.

Question 11. Answer: B

Donepezil inhibits AChE in the CNS, increasing acetylcholine levels to partially compensate for cholinergic neuron loss in Alzheimer's disease.

Question 12. Answer: C

Atropine blocks muscarinic receptors only. Muscle fasciculations are a nicotinic effect that requires pralidoxime (AChE reactivation) to reverse.

Question 13. Answer: B

At TOF 1/4, blockade is too profound for neostigmine. Sugammadex can reverse profound rocuronium blockade at any depth.

Question 14. Answer: B

Myasthenic patients have fewer functional ACh receptors and are very sensitive to non-depolarizing blockers, requiring much lower doses.

Question 15. Answer: C

After aging, the organophosphate-AChE bond becomes permanent, and pralidoxime cannot reactivate the enzyme.

Question 16. Answer: D

Succinylcholine has no pharmacologic reversal. With plasma cholinesterase deficiency, paralysis is prolonged but will eventually resolve with supportive ventilation.