

ANSWER KEY WITH EXPLANATIONS FOR CHAPTER 12

Skill Drill Answers

SD 12.1: Drug Class Matching

1. **D** — Propofol is an intravenous anesthetic
 2. **B** — Zolpidem is a Z-drug
 3. **C** — Sevoflurane is an inhaled anesthetic
 4. **F** — Morphine is an opioid agonist
 5. **A** — Midazolam is a benzodiazepine
 6. **E** — Lidocaine is a local anesthetic
 7. **F** — Fentanyl is an opioid agonist
 8. **D** — Ketamine is an intravenous anesthetic
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SD 12.2: True/False with Correction

1. **F** — Flumazenil reverses BENZODIAZEPINES only; NALOXONE reverses opioids.
 2. **T** — Local anesthetics block voltage-gated sodium channels.
 3. **F** — Naloxone has a SHORTER half-life than most opioids, requiring repeat dosing.
 4. **F** — ETOMIDATE is preferred for hemodynamically unstable patients; propofol causes hypotension.
 5. **T** — Mu receptors mediate both analgesia and respiratory depression.
 6. **F** — Buprenorphine is a PARTIAL agonist with a ceiling effect.
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SD 12.3: Fill in the Blank

1. **flumazenil**

2. **inhaled (volatile)**
 3. **naloxone**
 4. **lipid**
 5. **dantrolene**
 6. **mu (μ)**
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SD 12.4: Opioid Classification

1. **FA** — Morphine is a full agonist
 2. **AN** — Naloxone is an antagonist
 3. **PA** — Buprenorphine is a partial agonist
 4. **FA** — Fentanyl is a full agonist
 5. **AN** — Naltrexone is an antagonist
 6. **FA** — Methadone is a full agonist
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SD 12.5: Local Anesthetic Classification

1. **A** — Lidocaine is an amide
 2. **E** — Procaine is an ester
 3. **A** — Bupivacaine is an amide
 4. **E** — Tetracaine is an ester
 5. **A** — Ropivacaine is an amide
 6. **E** — Benzocaine is an ester
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SD 12.6: Rapid Recall

1. **Zolpidem**
2. **Etomidate**
3. **Naloxone**
4. **Bupivacaine**
5. **Propofol**

6. Fentanyl

SD 12.7: Complete the Sentence

1. **frequency**
 2. **uncharged**
 3. **before**
 4. **shorter**
 5. **NMDA blockade**
 6. **ceiling**
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SD 12.8: Opioid Adverse Effect Matching

1. **A** — Respiratory depression from brainstem mu activation
 2. **B** — Constipation from GI tract mu activation
 3. **C** — Miosis from Edinger-Westphal mu activation
 4. **D** — Euphoria from reward pathway mu activation
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SD 12.9: Anesthetic Agent Comparison

1. **B** — Etomidate for hemodynamically unstable patients
 2. **C** — Ketamine causes emergence delirium
 3. **A** — Propofol is most common, causes hypotension
 4. **D** — Sevoflurane is non-irritating for mask induction
 5. **C** — Ketamine is an NMDA antagonist with analgesia
 6. **B** — Etomidate causes adrenal suppression
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SD 12.10: Local Anesthetic Toxicity Sequence

Circumoral numbness and tinnitus: **1**

Seizures: **2**

Cardiovascular collapse: **3**

SD 12.11: Multimodal Analgesia Components

1. **C** — Ketamine reduces central sensitization
 2. **B** — Ketorolac inhibits COX
 3. **A** — Acetaminophen has hepatic toxicity risk
 4. **D** — Gabapentin for neuropathic pain
 5. **E** — Lidocaine patch for local analgesia
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SD 12.12: Clinical Scenario Drug Selection

1. **B** — Etomidate (or D-Ketamine also acceptable) for hypotensive patient
2. **B** — Naloxone reverses opioid respiratory depression
3. **B** — IV lipid emulsion for bupivacaine cardiotoxicity

Case Study Answers

Case 12.1: Opioid Overdose

Question 1. The classic opioid overdose triad is: (1) Respiratory depression (rate <12, often <8), (2) Decreased level of consciousness (sedation to coma), (3) Miosis (pinpoint pupils). All three result from mu receptor activation.

Question 2. Naloxone is a competitive antagonist at opioid receptors. It displaces opioids from mu receptors, rapidly reversing respiratory depression, sedation, and miosis. It has higher affinity but no intrinsic activity.

Question 3. Naloxone has a shorter half-life (30-90 minutes) than oxycodone and other opioids. As naloxone is metabolized, opioid effects return because the drug is still present. This patient needs continued monitoring and possibly repeat naloxone doses or infusion.

Question 4. Essential discharge planning includes: naloxone kit prescription and training for patient and family; substance abuse treatment referral; harm reduction counseling; medication-assisted treatment discussion (buprenorphine or methadone); overdose prevention education.

Case 12.2: Local Anesthetic Toxicity

Question 1. Local anesthetic systemic toxicity progresses from CNS to cardiovascular: (1) Prodromal symptoms—circumoral numbness, metallic taste, tinnitus, dizziness; (2) CNS excitation—confusion, agitation, seizures; (3) CNS depression—unconsciousness; (4) Cardiovascular toxicity—hypotension, arrhythmias, cardiac arrest.

Question 2. Bupivacaine is the most cardiotoxic local anesthetic. It binds cardiac sodium channels with high affinity and slow dissociation ("fast-in, slow-out"), causing refractory arrhythmias and cardiovascular collapse that is difficult to resuscitate.

Question 3. IV lipid emulsion (Intralipid 20%) is the specific treatment. Give 1.5 mL/kg bolus followed by infusion. It creates a "lipid sink" that sequesters lipophilic bupivacaine, reducing tissue binding. Continue CPR and standard ACLS; avoid vasopressin and calcium channel blockers.

Question 4. Prevention includes: aspirating before injection to check for blood return; using test doses with epinephrine marker; incremental dosing (not entire dose at once); using ultrasound guidance; maintaining verbal contact with patient; using lowest effective dose; considering less cardiotoxic alternatives (ropivacaine).

Diagram/Table Exercise Answers

DT 12.1: Opioid Receptor Effects

1. Mu receptor activation produces: **Analgesia, respiratory depression, euphoria, constipation** (also: miosis, physical dependence)
2. All opioid receptors couple to **Gi** proteins.
3. The receptor most responsible for respiratory depression is: **Mu**
4. Kappa receptor activation produces analgesia but also: **Dysphoria**

DT 12.2: Local Anesthetic Mechanism

1. Local anesthetics block **voltage-gated sodium** channels.
2. The uncharged form crosses the membrane; the **charged** form binds the channel.
3. Small fibers (pain) are blocked **before** large fibers (motor).
4. Severe toxicity is treated with IV **lipid** emulsion.

DT 12.3: Sedative-Hypnotic Comparison Table

Drug Class	Mechanism	Examples	Key Adverse Effects
Benzodiazepines	GABA-A enhancement (↑ frequency)	Diazepam, midazolam	Tolerance, dependence, respiratory depression
Z-drugs	GABA-A ($\alpha 1$ selective)	Zolpidem, zaleplon, eszopiclone	Complex sleep behaviors
Reversal agent	Competitive antagonist at BZ site	Flumazenil	May precipitate seizures

DT 12.4: Opioid Comparison Table

Drug	Classification	Relative Potency	Special Features
Morphine	Full agonist	1 (reference)	Histamine release, active metabolites
Fentanyl	Full agonist	100x morphine	Rapid onset, lipophilic
Buprenorphine	Partial agonist	High affinity	Ceiling effect for respiratory depression
Naloxone	Antagonist	N/A	Short half-life, may need repeat dosing
Methadone	Full agonist	Variable	Long half-life, NMDA antagonism, QT prolongation

Multiple-Choice Question Answers

Question 1. Answer: B

Benzodiazepines increase the frequency of GABA-A channel opening; barbiturates increase duration.

Question 2. Answer: A

Mu receptors are the primary mediators of both analgesia and respiratory depression.

Question 3. Answer: B

Flumazenil is a benzodiazepine antagonist; naloxone is for opioids.

Question 4. Answer: C

Local anesthetics block voltage-gated sodium channels, preventing action potential propagation.

Question 5. Answer: C

Etomidate causes minimal cardiovascular depression, making it preferred for unstable patients.

Question 6. Answer: B

Naloxone's half-life (30-90 min) is shorter than most opioids, allowing re-sedation as it wears off.

Question 7. Answer: C

Bupivacaine is most cardiotoxic due to slow dissociation from cardiac sodium channels.

Question 8. Answer: B

Buprenorphine is a partial agonist with ceiling effect for respiratory depression, making it safer in overdose.

Question 9. Answer: C

IV lipid emulsion is rescue therapy for local anesthetic systemic toxicity (LAST).

Question 10. Answer: C

Dantrolene treats malignant hyperthermia by blocking ryanodine receptors and reducing calcium release from sarcoplasmic reticulum.

Question 11. Answer: B

Ketamine is an NMDA receptor antagonist, producing dissociative anesthesia.

Question 12. Answer: B

Buprenorphine has high affinity but partial efficacy. It displaces full agonists, precipitating withdrawal in dependent patients.

Question 13. Answer: B

Z-drugs selectively bind the $\alpha 1$ subunit of GABA-A receptors, providing hypnotic effects with less anxiolytic/muscle relaxant activity.

Question 14. Answer: B

Small pain fibers ($A\delta$, C) are blocked before large motor fibers ($A\alpha$) due to differences in fiber size and myelination.

Question 15. Answer: C

Naltrexone blocks opioid receptors; standard opioid doses won't work, and high doses are dangerous. Use non-opioid multimodal analgesia.

Question 16. Answer: C

Multimodal analgesia improves pain control but does not eliminate all pain—complete pain elimination is unrealistic.