

ANSWER KEY WITH EXPLANATIONS FOR CHAPTER 13

Skill Drill Answers

SD 13.1: Substance-Mechanism Matching

1. **A** — Cocaine blocks DAT
2. **C** — Heroin is a mu agonist
3. **E** — Alcohol enhances GABA-A and inhibits NMDA
4. **D** — THC is a CB1 partial agonist
5. **B** — Methamphetamine reverses DAT
6. **C** — Fentanyl is a mu agonist

SD 13.2: True/False with Correction

1. **F** — Alcohol withdrawal IS life-threatening; can cause seizures and delirium tremens.
2. **T** — Cocaine blocks DAT, preventing dopamine reuptake.
3. **F** — Methadone is a FULL agonist; BUPRENORPHINE is the partial agonist.
4. **T** — Buprenorphine can precipitate withdrawal by displacing full agonists.
5. **F** — Disulfiram blocks ALDEHYDE DEHYDROGENASE; NALTREXONE blocks opioid receptors.
6. **F** — ALCOHOL and BENZODIAZEPINE withdrawal cause fatal seizures; opioid withdrawal is uncomfortable but rarely dangerous.

SD 13.3: Fill in the Blank

1. **nucleus accumbens**

2. **alcohol dehydrogenase (ADH)**
 3. **disulfiram (Antabuse)**
 4. **naltrexone (Vivitrol)**
 5. **CB1 (cannabinoid)**
 6. **24-48**
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SD 13.4: MAT Drug Classification

1. **A** — Methadone is a full mu agonist
 2. **B** — Buprenorphine is a partial mu agonist
 3. **C** — Naltrexone is a mu antagonist
 4. **C** — Naloxone is a mu antagonist
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SD 13.5: Withdrawal Syndrome Matching

1. **A** — Alcohol withdrawal is life-threatening
 2. **D** — Stimulant withdrawal causes dysphoria and fatigue
 3. **B** — Opioid withdrawal causes lacrimation, diarrhea, muscle aches
 4. **C** — Benzodiazepine withdrawal resembles alcohol withdrawal
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SD 13.6: Rapid Recall

1. **Dopamine**
 2. **Aldehyde dehydrogenase (ALDH)**
 3. **Buprenorphine**
 4. **CB1**
 5. **Delirium tremens**
 6. **Naltrexone**
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SD 13.7: Complete the Sentence

1. **blocks**

2. **reverse**
 3. **higher**
 4. **more**
 5. **complete detox**
 6. **long**
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SD 13.8: Dopamine Mechanism Comparison

Substance	Effect on DA Mechanism
Cocaine	+ Blocks DAT (prevents reuptake)
Heroin	+ Disinhibits VTA neurons
Amphetamine	+ Reverses DAT (causes efflux)

SD 13.9: Alcohol Use Disorder Treatment Matching

1. **C** — Disulfiram inhibits ALDH
 2. **A** — Naltrexone blocks opioid receptors
 3. **B** — Acamprosate modulates glutamate/GABA
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SD 13.10: Withdrawal Timeline Ordering

Tremor and anxiety: **1** (6-24 hours)

Hallucinations: **2** (12-48 hours)

Seizures: **3** (24-48 hours)

Delirium tremens: **4** (48-96 hours)

SD 13.11: Buprenorphine Characteristics

1. **F** — Buprenorphine is a PARTIAL agonist
2. **T** — It has a ceiling effect for respiratory depression
3. **T** — Must start when patient is in withdrawal
4. **T** — Suboxone contains naloxone to deter IV abuse

5. **F** — Can be prescribed office-based (not clinic-only)
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SD 13.12: Clinical Scenario Drug Selection

1. **B** — Naltrexone reduces craving by blocking reward
2. **B** — Buprenorphine can be prescribed office-based
3. **C** — Lorazepam (benzodiazepine) for alcohol withdrawal

Case Study Answers

Case 13.1: Opioid Use Disorder Treatment

Question 1. Detoxification alone has very high relapse rates (>90%) because it addresses only physical dependence, not the neuroplastic changes in reward circuits that drive craving and drug-seeking. Without medication to stabilize these circuits, relapse is almost inevitable.

Question 2. The three FDA-approved medications are: (1) Methadone—full agonist, prevents withdrawal and craving, requires daily clinic visits; (2) Buprenorphine—partial agonist, ceiling effect makes it safer, can be prescribed office-based; (3) Naltrexone—antagonist, blocks opioid effects, requires complete detox first, monthly injection improves adherence.

Question 3. Buprenorphine has very high receptor affinity but is only a partial agonist. If given while full agonist (fentanyl) occupies receptors, buprenorphine displaces it and provides less receptor activation, precipitating immediate withdrawal. Waiting until mild withdrawal ensures full agonist has dissipated.

Question 4. Suboxone contains buprenorphine plus naloxone. When taken sublingually as intended, naloxone has poor bioavailability and doesn't affect the patient. If crushed and injected, naloxone becomes active and precipitates withdrawal, deterring IV abuse.

Case 13.2: Alcohol Withdrawal

Question 1. The diagnosis is alcohol withdrawal syndrome. Chronic alcohol use causes GABA-A receptor downregulation and glutamate (NMDA) receptor upregulation. When alcohol stops, the inhibitory effect is removed, causing CNS hyperexcitability manifesting as tremor, anxiety, and autonomic hyperactivity.

Question 2. Timeline: Minor withdrawal (tremor, anxiety, tachycardia) at 6-24 hours → Hallucinations (visual, tactile) at 12-48 hours → Withdrawal seizures at 24-48 hours → Delirium tremens (confusion, severe autonomic instability, hallucinations) at 48-96 hours. Not all patients progress through all stages.

Question 3. First-line treatment is benzodiazepines (lorazepam, diazepam, chlordiazepoxide). They cross-react with alcohol at GABA-A receptors, replacing alcohol's effect and preventing withdrawal progression. Symptom-triggered protocols (CIWA-Ar scale) are preferred over fixed schedules.

Question 4. Thiamine (vitamin B1) supplementation is essential. Chronic alcoholics are thiamine-depleted, and glucose metabolism consumes thiamine. Without supplementation, patients can develop Wernicke encephalopathy (confusion, ataxia, ophthalmoplegia) or permanent Korsakoff syndrome (anterograde amnesia). Give thiamine BEFORE glucose.

Diagram/Table Exercise Answers

DT 13.1: Reward Pathway and Drug Mechanisms

1. Cocaine increases dopamine by blocking: **Dopamine transporter (DAT)**
2. Amphetamines increase dopamine by reversing: **Dopamine transporter (DAT)**
3. Opioids increase dopamine by disinhibiting: **VTA dopamine neurons (via GABAergic interneurons)**
4. Alcohol enhances **GABA-A** and blocks **NMDA (glutamate)**

DT 13.2: Alcohol Metabolism Pathway

1. Enzyme 1: **Alcohol dehydrogenase (ADH)**
2. Enzyme 2: **Aldehyde dehydrogenase (ALDH)**
3. Disulfiram inhibits: **Aldehyde dehydrogenase (ALDH)**
4. Acetaldehyde accumulation causes: **Flushing, nausea, vomiting, headache**

DT 13.3: MAT Comparison Table

Medication	Mechanism	Dispensing	Key Features
Methadone	Full mu agonist	OTP clinic only	Long half-life, prevents withdrawal/craving
Buprenorphine	Partial mu agonist	Office-based	Ceiling effect, precipitated withdrawal risk
Naltrexone	Mu antagonist	Any prescriber	Requires complete detox, monthly injection available

DT 13.4: Withdrawal Syndrome Comparison Table

Substance	Onset	Key Symptoms	Danger Level	Treatment
Alcohol	6-96 hours	Tremor, seizures, DTs	Life-threatening	Benzodiazepines
Opioids	12-72 hours	Lacrimation, rhinorrhea, diarrhea, muscle aches	Uncomfortable, rarely dangerous	Buprenorphine, clonidine
Benzodiazepines	Days-weeks	Similar to alcohol	Potentially life-threatening	Slow taper
Stimulants	Hours-days	Dysphoria, fatigue, hypersomnia	Not dangerous	Supportive care

Multiple-Choice Question Answers

Question 1. Answer: C

Dopamine is the primary neurotransmitter in the mesolimbic reward pathway.

Question 2. Answer: C

Cocaine blocks the dopamine transporter (DAT), preventing reuptake and increasing synaptic dopamine.

Question 3. Answer: C

Disulfiram inhibits ALDH, causing acetaldehyde accumulation and aversive symptoms with alcohol.

Question 4. Answer: B

THC acts as a partial agonist at CB1 cannabinoid receptors, primarily located in the CNS.

Question 5. Answer: D

Delirium tremens typically occurs 48-96 hours after the last drink, the latest and most severe manifestation.

Question 6. Answer: B

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

Question 7. Answer: B

Naltrexone blocks opioid receptors. Standard opioid doses won't provide analgesia; non-opioid strategies are needed.

Question 8. Answer: B

Alcohol enhances GABA-A receptor function, producing sedation and anxiolysis. It also inhibits NMDA receptors.

Question 9. Answer: C

Alcohol withdrawal can cause seizures and delirium tremens with 5-15% mortality. Opioid and stimulant withdrawal are uncomfortable but not life-threatening.

Question 10. Answer: B

Amphetamines reverse the dopamine transporter, causing dopamine efflux. Cocaine blocks the transporter.

Question 11. Answer: B

Buprenorphine should be started when the patient is in mild withdrawal (COWS ≥ 8 -12) to avoid precipitating withdrawal.

Question 12. Answer: B

Buprenorphine's high affinity but partial efficacy means it displaces the full agonist and provides less activation, precipitating withdrawal.

Question 13. Answer: B

Fentanyl is 50-100x more potent than morphine, making accidental overdose common when users can't accurately dose.

Question 14. Answer: B

Disulfiram inhibits aldehyde dehydrogenase, causing toxic acetaldehyde accumulation when alcohol is consumed.

Question 15. Answer: C

The mesolimbic pathway projects from VTA to nucleus accumbens and mediates both drug reward and natural rewards.

Question 16. Answer: C

Benzodiazepines are first-line for alcohol withdrawal; they cross-react at GABA-A receptors and prevent progression.