

ANSWER KEY WITH EXPLANATIONS FOR CHAPTER 17

Skill Drill Answers

SD 17.1: Vaughan-Williams Classification Matching

1. **E** — Amiodarone is Class III (primarily)
2. **B** — Lidocaine is Class Ib
3. **C** — Flecainide is Class Ic
4. **D** — Metoprolol is Class II
5. **F** — Verapamil is Class IV
6. **A** — Quinidine is Class Ia
7. **E** — Sotalol is Class III (with Class II effects)
8. **C** — Propafenone is Class Ic

SD 17.2: True/False with Correction

1. **F** — Class Ic drugs are CONTRAINDICATED in structural heart disease (CAST trial).
2. **T** — Amiodarone has effects in all four Vaughan-Williams classes.
3. **F** — Class III drugs PROLONG the QT interval.
4. **F** — Lidocaine is IV-only; MEXILETINE is the oral Class Ib drug.
5. **T** — IV magnesium is first-line treatment for torsades.
6. **F** — Beta-blockers do NOT cause QT prolongation and are very safe.

SD 17.3: Fill in the Blank

1. **sodium**

2. **potassium**
 3. **torsades de pointes**
 4. **Ic**
 5. **thyroid**
 6. **accessory pathway**
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SD 17.4: Action Potential Phase Matching

1. **A** — Phase 0 is Na^+ influx
 2. **B** — Phase 2 is Ca^{2+} influx balanced by K^+ efflux
 3. **C** — Phase 3 is K^+ efflux
 4. **D** — Phase 4 is stable resting potential
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SD 17.5: Class I Subdivision Comparison

1. **A** — Class Ia prolongs APD
 2. **B** — Class Ib shortens APD
 3. **C** — Class Ic has no effect on APD
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SD 17.6: Rapid Recall

1. **Class III (or "all classes")**
 2. **Lidocaine**
 3. **Magnesium**
 4. **CAST trial**
 5. **Beta-blockers**
 6. **Pulmonary (fibrosis)**
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SD 17.7: Complete the Sentence

1. **sodium**
2. **prolong**

3. **inactivated**
 4. **beta-blocking**
 5. **contraindicated**
 6. **long**
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SD 17.8: Drug-Induced Torsades Risk

1. **HIGH** — Quinidine (Class Ia)
 2. **LOW** — Amiodarone (despite QT prolongation)
 3. **MINIMAL** — Lidocaine (shortens APD)
 4. **HIGH** — Sotalol
 5. **MINIMAL** — Metoprolol (beta-blocker)
 6. **HIGH** — Dofetilide
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SD 17.9: Antiarrhythmic Adverse Effect Matching

1. **A** — Amiodarone causes pulmonary fibrosis
 2. **C** — Procainamide causes drug-induced lupus
 3. **B** — Quinidine causes cinchonism
 4. **E** — Lidocaine causes CNS toxicity
 5. **D** — Flecainide increases mortality in structural heart disease
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SD 17.10: QT Prolongation Risk Factors

1. ↑ — Hypokalemia increases risk
 2. ↑ — Bradycardia increases risk
 3. ↓ — Normal magnesium decreases risk
 4. ↑ — Female sex increases risk
 5. ↑ — Congenital long QT increases risk
 6. ↓ — Tachycardia decreases risk
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SD 17.11: Antiarrhythmic Selection

1. **T** — Beta-blockers are the safest and reduce mortality
 2. **F** — Sotalol requires IN-HOSPITAL initiation with monitoring
 3. **T** — Amiodarone is effective for both atrial and ventricular arrhythmias
 4. **F** — Verapamil is CONTRAINDICATED in WPW with AFib
 5. **F** — Dronedarone is CONTRAINDICATED in permanent AFib (PALLAS trial)
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SD 17.12: Clinical Scenario Drug Selection

1. **B** — IV lidocaine for acute VT in MI (Class Ib, preferential ischemic binding)
2. **B** — Metoprolol (beta-blocker) for rate control
3. **D** — Any may be appropriate in absence of structural heart disease

Case Study Answers

Case 17.1: Drug-Induced QT Prolongation

Question 1. The patient has torsades de pointes, a polymorphic VT associated with QT prolongation. It was caused by sotalol (Class III drug that prolongs QT) combined with hypokalemia ($K^+ 3.2$). Sotalol blocks IK_r potassium channels, prolonging repolarization and creating conditions for early afterdepolarizations.

Question 2. Risk factors present: (1) QT-prolonging drug (sotalol), (2) Hypokalemia ($K^+ 3.2$), (3) Female sex, (4) QTc prolongation (540 ms vs. baseline 440 ms). The combination of these factors markedly increased her torsades risk.

Question 3. Immediate treatment includes: (1) IV magnesium sulfate 1-2 g (first-line), (2) Stop sotalol immediately, (3) Correct hypokalemia aggressively (target $K^+ >4.0$), (4) If unstable or magnesium fails: overdrive pacing or IV isoproterenol to increase heart rate (shortens QT), (5) If pulseless: defibrillation.

Question 4. Alternatives for AFib include: (1) Rate control with beta-blockers or non-DHP CCBs (no QT prolongation), (2) If rhythm control needed: flecainide or propafenone if she has no structural heart disease, (3) Catheter ablation, (4) If antiarrhythmic needed and prior torsades: amiodarone (lower torsades risk despite QT prolongation) with careful monitoring.

Case 17.2: Antiarrhythmic Selection in Structural Heart Disease

Question 1. Class Ic drugs are contraindicated because the CAST trial demonstrated increased mortality in post-MI patients treated with these drugs. They have proarrhythmic effects in ischemic and scarred myocardium, where slow conduction combined with marked sodium channel blockade can promote reentrant ventricular arrhythmias.

Question 2. Amiodarone is the preferred antiarrhythmic for rhythm control in patients with structural heart disease. It is the most effective drug for maintaining sinus rhythm and has less proarrhythmic risk than other agents in this population. Dofetilide is an alternative (approved for AFib in heart failure) but requires in-hospital initiation.

Question 3. Amiodarone monitoring includes: (1) Baseline and periodic chest X-ray and pulmonary function tests (pulmonary fibrosis), (2) Thyroid function tests every 6 months (hypo- or hyperthyroidism), (3) Liver function tests (hepatotoxicity), (4) Annual eye exam (corneal deposits, rare optic neuropathy), (5) ECG (QT monitoring, bradycardia), (6) Skin exam (photosensitivity advice).

Question 4. Dofetilide precautions: (1) Must be initiated in hospital with continuous monitoring for minimum 3 days, (2) QTc must be <440 ms at baseline, (3) Must assess renal function and dose-adjust (contraindicated if CrCl <20), (4) Must avoid drugs that inhibit renal cation transport (cimetidine, trimethoprim, etc.), (5) Daily QTc monitoring; stop if QTc >500 ms or increases >15% from baseline.

Diagram/Table Exercise Answers

DT 17.1: Cardiac Action Potential and Drug Targets

1. Phase 0 involves rapid **sodium** influx and is blocked by Class **I** drugs.
2. Phase 2 (plateau) involves **calcium** influx and is blocked by Class **IV** drugs.
3. Phase 3 (repolarization) involves **potassium** efflux and is prolonged by Class **III** drugs.
4. Class II drugs reduce automaticity by affecting Phase **4** in pacemaker cells.

DT 17.2: Torsades de Pointes Mechanism

1. Class III drugs prolong QT by blocking **potassium** channels.
2. Prolonged repolarization allows **early afterdepolarizations** (EADs) to occur.
3. First-line treatment for torsades: IV **magnesium (sulfate)**
4. Two electrolyte abnormalities: **hypokalemia** and **hypomagnesemia**

DT 17.3: Class I Subdivision Comparison Table

Subclass	Effect on APD	Binding Kinetics	Examples	Clinical Notes
Class Ia	Prolongs	Intermediate	Quinidine, procainamide	QT prolongation, torsades risk
Class Ib	Shortens	Fast	Lidocaine, mexiletine	Preferred in ischemia
Class Ic	No change	Slow	Flecainide, propafenone	Contraindicated in structural HD

DT 17.4: Antiarrhythmic Drug Comparison Table

Drug	Class	Primary Use	QT Effect	Major Toxicities
Amiodarone	III (+ I, II, IV)	AFib, VT	Prolongs (low torsades risk)	Pulmonary fibrosis, thyroid, hepatic
Sotalol	III + II	AFib, VT	Prolongs (high torsades risk)	Torsades, bradycardia
Flecainide	Ic	SVT, AFib (no SHD)	No change	Proarrhythmia in structural HD
Lidocaine	Ib	Acute VT	No change	CNS toxicity
Metoprolol	II	Rate control, post-MI	No change	Bradycardia, hypotension

Multiple-Choice Question Answers

Question 1. Answer: B

Phase 0 rapid depolarization is caused by sodium influx through voltage-gated sodium channels.

Question 2. Answer: C

Class III drugs block potassium channels, prolonging Phase 3 repolarization.

Question 3. Answer: C

Amiodarone has effects in all four Vaughan-Williams classes.

Question 4. Answer: B

IV magnesium sulfate is first-line treatment for torsades de pointes.

Question 5. Answer: B

Class Ic drugs (CAST trial) are contraindicated in structural heart disease.

Question 6. Answer: B

Cinchonism (from quinidine—derived from cinchona bark) includes tinnitus, headache, and GI symptoms.

Question 7. Answer: B

Lidocaine preferentially binds inactivated sodium channels, which are more prevalent in ischemic tissue.

Question 8. Answer: C

Dofetilide requires in-hospital initiation with continuous QT monitoring for at least 3 days.

Question 9. Answer: B

Sotalol combines Class III (K⁺ channel blockade) with Class II (beta-blockade) effects.

Question 10. Answer: B

The PALLAS trial showed increased mortality with dronedarone in permanent AFib.

Question 11. Answer: B

Blocking the AV node in WPW can force conduction down the accessory pathway, potentially causing VFib.

Question 12. Answer: B

Procainamide causes drug-induced lupus, especially in slow acetylators with chronic use.

Question 13. Answer: B

Class Ic drugs caused proarrhythmia (increased sudden death) in the ischemic post-MI myocardium.

Question 14. Answer: C

Tachycardia actually DECREASES torsades risk; bradycardia allows more time for EADs.

Question 15. Answer: B

Amiodarone contains iodine and commonly causes thyroid dysfunction (hypothyroidism more common than hyperthyroidism).

Question 16. Answer: C

Beta-blockers are the only antiarrhythmics consistently shown to reduce mortality post-MI.