

ANSWER KEY WITH EXPLANATIONS

FOR CHAPTER 16

Skill Drill Answers

SD 16.1: Drug Class Matching

1. **A** — Nitroglycerin is a nitrate
 2. **B** — Metoprolol is a beta-blocker
 3. **C** — Ranolazine inhibits late sodium current
 4. **D** — Sacubitril/valsartan is an ARNI
 5. **E** — Dapagliflozin is an SGLT2 inhibitor
 6. **F** — Milrinone is a PDE3 inhibitor
 7. **B** — Carvedilol is a beta-blocker
 8. **A** — Isosorbide mononitrate is a nitrate
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SD 16.2: True/False with Correction

1. **F** — Nitrates + PDE5 inhibitors cause SEVERE HYPOTENSION (contraindicated).
 2. **F** — Beta-blockers should be started at LOW doses and titrated slowly.
 3. **T** — SGLT2 inhibitors benefit HFrEF independent of diabetes status.
 4. **F** — Tolerance is prevented by a NITRATE-FREE INTERVAL (not continuous dosing).
 5. **F** — Digoxin reduces HOSPITALIZATIONS but NOT mortality.
 6. **T** — Carvedilol has β_1 , β_2 , and α_1 blocking activity.
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SD 16.3: Fill in the Blank

1. **wall tension**
 2. **nitric oxide (NO)**
 3. **nitrate-free (10-12 hour)**
 4. **SGLT2 inhibitors**
 5. **sodium (INa)**
 6. **neprilysin**
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SD 16.4: Oxygen Supply vs. Demand

1. **D** — Heart rate is demand
 2. **S** — Coronary blood flow is supply
 3. **D** — Contractility is demand
 4. **S** — Diastolic time is supply
 5. **D** — Wall tension is demand
 6. **S** — Coronary artery patency is supply
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SD 16.5: HFrEF Drug Mechanism Matching

1. **A** — Lisinopril blocks RAAS
 2. **B** — Carvedilol blocks sympathetic activation
 3. **C** — Spironolactone blocks aldosterone
 4. **D** — Sacubitril/valsartan inhibits neprilysin + blocks AT1
 5. **E** — Dapagliflozin promotes natriuresis
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SD 16.6: Rapid Recall

1. **PDE5 inhibitors (sildenafil, tadalafil)**
2. **Carvedilol, bisoprolol, metoprolol succinate**
3. **Milrinone**
4. **Potassium**
5. **If (funny) channel**

6. Digoxin

SD 16.7: Complete the Sentence

1. **veins**
 2. **diastolic**
 3. **maladaptive**
 4. **low**
 5. **superior**
 6. **β 1**
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SD 16.8: Inotrope/Vasopressor Classification

1. **A** — Dobutamine is a β 1 agonist
 2. **B** — Milrinone is a PDE3 inhibitor
 3. **C** — Phenylephrine is an α 1 agonist
 4. **D** — Dopamine is dose-dependent
 5. **A and C** — Norepinephrine is primarily α 1 with β 1 activity
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SD 16.9: Antianginal Mechanism Comparison

1. **A** — Beta-blockers decrease demand (HR, contractility)
 2. **B** — Nitrates decrease demand (preload reduction)
 3. **C** — Ranolazine decreases demand (late INa blockade)
 4. **D** — DHP CCBs vasodilate (can increase supply, reduce afterload)
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SD 16.10: Heart Failure Neurohormonal Activation

1. **B** — RAAS causes vasoconstriction and aldosterone release
2. **A** — SNS increases HR and promotes arrhythmias
3. **C** — ADH causes water retention and hyponatremia
4. **D** — Natriuretic peptides are counter-regulatory (beneficial)

SD 16.11: Drug Safety Identification

1. **T** — Nitrates + PDE5 inhibitors = fatal hypotension
 2. **F** — Beta-blockers should NOT be initiated during acute decompensation
 3. **F** — Digoxin has a NARROW therapeutic index
 4. **T** — Long-term inotropes increase mortality
 5. **F** — SGLT2 inhibitors benefit ALL HFrEF patients
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SD 16.12: Clinical Scenario Drug Selection

1. **B** — Dapagliflozin (SGLT2 inhibitor) is the fourth pillar
2. **B** — Ranolazine (no effect on HR, so safe with bradycardia)
3. **C** — Norepinephrine + dobutamine for cardiogenic shock

Case Study Answers

Case 16.1: Chronic Stable Angina

Question 1. First-line therapy is a beta-blocker. In a patient with prior MI, beta-blockers provide dual benefit: antianginal effect by reducing heart rate and contractility (decreasing oxygen demand), and mortality reduction post-MI by preventing remodeling and arrhythmias.

Question 2. Metoprolol reduces angina by: (1) Decreasing heart rate—reduces myocardial oxygen demand and increases diastolic time (improves supply); (2) Decreasing contractility—reduces oxygen consumption; (3) Reducing blood pressure—decreases afterload and wall tension. The net effect is improved oxygen supply-demand balance.

Question 3. Add sublingual nitroglycerin for acute relief (take at onset of angina or prophylactically before exertion). For prophylaxis, add a long-acting nitrate (isosorbide mononitrate) with a nitrate-free interval to prevent tolerance. Alternatively, add a DHP calcium channel blocker (amlodipine).

Question 4. If angina remains uncontrolled, add ranolazine. It works through a different mechanism (late sodium current inhibition) without affecting heart rate or blood pressure, making it useful as add-on therapy. If still uncontrolled, consider revascularization (PCI or CABG).

Case 16.2: Heart Failure with Reduced Ejection Fraction

Question 1. The four pillars are: (1) ACE inhibitor/ARB/ARNI—blocks RAAS, reduces afterload, prevents remodeling; (2) Beta-blocker—blocks toxic sympathetic activation, prevents remodeling, reduces arrhythmias; (3) MRA (spironolactone/eplerenone)—blocks aldosterone-mediated fibrosis and sodium retention; (4) SGLT2 inhibitor—reduces preload through natriuresis, improves cardiac metabolism. All four have proven mortality benefit in HFrEF.

Question 2. While guidelines recommend starting all four simultaneously when possible, practical initiation often involves: Start ACE inhibitor/ARB first → add beta-blocker once euvolemic (start low, titrate slowly) → add MRA → add SGLT2 inhibitor. Diuretics for congestion as needed (symptom control). Titrate each to target doses. Consider switching ACE inhibitor to ARNI once stable.

Question 3. Patients with heart failure have compensatory sympathetic activation maintaining cardiac output. Abrupt beta-blockade can precipitate decompensation. Starting low and titrating slowly allows the heart to adapt, with gradual improvement in function over weeks to months. The negative inotropic effect is offset by long-term benefits of blocking toxic catecholamine effects.

Question 4. Options include: (1) ARB (losartan, valsartan)—no cough, but angioedema still possible (use with caution); (2) Hydralazine + isosorbide dinitrate—alternative vasodilator strategy, particularly effective in Black patients; (3) ARNI should NOT be used if patient had ACE inhibitor angioedema (contains valsartan which can also cause angioedema via similar mechanism).

Diagram/Table Exercise Answers

DT 16.1: Myocardial Oxygen Balance

1. Three determinants of demand: **Heart rate, contractility, wall tension**
2. Beta-blockers reduce demand by decreasing **heart rate** and **contractility**
3. Nitrates reduce demand primarily by reducing **venous return/preload** (wall tension)
4. Longer diastolic time improves supply by allowing more **coronary perfusion**

DT 16.2: Heart Failure Neurohormonal Cascade

1. SNS activation causes increased **heart rate** and vasoconstriction.
 2. RAAS activation causes vasoconstriction and **aldosterone** release.
 3. Drug class that blocks SNS: **Beta-blockers**
 4. Drug class that blocks RAAS: **ACE inhibitors/ARBs/ARNI**
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DT 16.3: Antianginal Drug Comparison Table

Drug Class	Primary Mechanism	Effect on HR	Effect on Preload	Reflex Tachycardia
Beta-blockers	↓ HR, ↓ contractility	Decreased	No change	No
Nitrates	Venodilation (preload reduction)	No direct effect	Decreased	Yes
DHP CCBs	Vasodilation	Increased or no change	Decreased	Yes
Ranolazine	Late INa blockade	No change	No change	No

DT 16.4: HFrEF GDMT Comparison Table

Drug Class	Mechanism	Key Trial(s)	Adverse Effects
ACE inhibitors	Block RAAS	CONSENSUS, SOLVD	Cough, hyperkalemia, AKI
Beta-blockers	Block sympathetic activation	MERIT-HF, CIBIS-II	Bradycardia, hypotension
MRAs	Block aldosterone	RALES, EMPHASIS-HF	Hyperkalemia
SGLT2 inhibitors	Natriuresis, unclear	DAPA-HF, EMPEROR	UTI, DKA, volume depletion
ARNI	Neprilysin inhibition + ARB	PARADIGM-HF	Hypotension, angioedema

Multiple-Choice Question Answers

Question 1. Answer: B

Nitrates primarily relieve angina by venodilation, which reduces preload and wall tension.

Question 2. Answer: B

Nitrate tolerance is prevented by a 10-12 hour nitrate-free interval daily.

Question 3. Answer: B

By slowing heart rate, beta-blockers prolong diastole, allowing more time for coronary perfusion.

Question 4. Answer: C

Digoxin reduces symptoms but is not part of the four pillars (no mortality benefit).

Question 5. Answer: B

Metoprolol succinate (not tartrate), carvedilol, and bisoprolol have proven mortality benefit.

Question 6. Answer: B

Sildenafil + nitrates = severe hypotension due to combined cGMP accumulation.

Question 7. Answer: B

Ranolazine inhibits the late sodium current, reducing calcium overload during ischemia.

Question 8. Answer: A

Neurohormonal activation provides short-term compensation but causes long-term harm.

Question 9. Answer: B

Neprilysin inhibition prevents breakdown of beneficial natriuretic peptides.

Question 10. Answer: B

Beta-blockers in HF: "start low, go slow" to avoid precipitating decompensation.

Question 11. Answer: B

Milrinone is an "inodilator"—increases contractility and causes vasodilation.

Question 12. Answer: B

Spironolactone blocks aldosterone-mediated fibrosis and sodium retention.

Question 13. Answer: C

Cardiogenic shock requires IV inotropes/vasopressors; oral medications are inappropriate.

Question 14. Answer: B

SGLT2 inhibitors cause natriuresis and reduced preload (mechanism not fully understood).

Question 15. Answer: B

Hydralazine + isosorbide dinitrate is the alternative for ACE inhibitor angioedema. ARBs and ARNI carry angioedema risk.

Question 16. Answer: B

Digoxin reduces symptoms and hospitalizations but does not reduce mortality.