

ANSWER KEY WITH EXPLANATIONS

FOR CHAPTER 15

Skill Drill Answers

SD 15.1: Drug Class Matching

1. **A** — Lisinopril is an ACE inhibitor
 2. **B** — Losartan is an ARB
 3. **D** — Amlodipine is a DHP CCB
 4. **E** — Verapamil is a non-DHP CCB
 5. **F** — Clonidine is centrally acting
 6. **C** — Aliskiren is a direct renin inhibitor
 7. **A** — Ramipril is an ACE inhibitor
 8. **E** — Diltiazem is a non-DHP CCB
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SD 15.2: True/False with Correction

1. **F** — Cough is due to BRADYKININ accumulation, not angiotensin II.
 2. **T** — ARBs don't affect bradykinin, so cough is rare.
 3. **F** — ACE inhibitors are CONTRAINDICATED in pregnancy (teratogenic).
 4. **F** — Verapamil and diltiazem are NON-DIHYDROPYRIDINES.
 5. **T** — Abrupt clonidine discontinuation causes rebound hypertension.
 6. **F** — Amlodipine is a DHP and does NOT cause bradycardia.
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SD 15.3: Fill in the Blank

1. **angiotensin II; bradykinin**

2. **dry cough**
 3. **AT1**
 4. **methyldopa**
 5. **vascular**
 6. **aliskiren**
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SD 15.4: RAAS Cascade Ordering

Angiotensinogen: **1**

Renin release: **2** (acts on angiotensinogen)

Angiotensin I: **3**

Angiotensin II: **4**

Aldosterone release: **5**

SD 15.5: Drug-Site of Action Matching

1. **B** — Lisinopril blocks ACE
 2. **C** — Losartan blocks AT1 receptor
 3. **A** — Aliskiren blocks renin
 4. **D** — Spironolactone blocks aldosterone receptor
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SD 15.6: Rapid Recall

1. **Cough**
 2. **Amlodipine**
 3. **Methyldopa (none are ideal; methyldopa is preferred)**
 4. **Angioedema**
 5. **Renin**
 6. **Verapamil**
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SD 15.7: Complete the Sentence

1. **increase**
 2. **do not**
 3. **tachycardia**
 4. **decrease**
 5. **stenosis**
 6. **α_2**
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SD 15.8: CCB Classification

1. **DHP** — Amlodipine
 2. **Non-DHP** — Verapamil
 3. **DHP** — Nifedipine
 4. **Non-DHP** — Diltiazem
 5. **DHP** — Felodipine
 6. **DHP** — Clevidipine
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SD 15.9: Compelling Indication Matching

1. **A** — ACE/ARB for diabetic nephropathy
 2. **B or C** — Thiazide or CCB for isolated systolic HTN
 3. **A** — ACE/ARB for HFrEF
 4. **A and D** — ACE/ARB and beta-blocker post-MI
 5. **C** — CCB as monotherapy in Black patients
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SD 15.10: Adverse Effect Identification

1. **A** — ACE inhibitors cause cough
2. **B** — DHP CCBs cause peripheral edema
3. **C** — Non-DHP CCBs cause constipation and bradycardia
4. **D** — Centrally acting agents cause rebound hypertension
5. **E** — Hydralazine causes drug-induced lupus

SD 15.11: Contraindication Identification

1. **T** — ACE inhibitors are teratogenic
 2. **F** — Combination increases adverse events without benefit
 3. **T** — Verapamil's negative inotropy worsens heart failure
 4. **F** — Abrupt discontinuation causes rebound hypertension
 5. **T** — ACE inhibitors cause AKI in bilateral RAS
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SD 15.12: Clinical Scenario Drug Selection

1. **B** — Lisinopril (ACE inhibitor) provides renal protection
2. **B** — Switch to losartan (ARB avoids cough)
3. **C** — Methyldopa is preferred in pregnancy

Case Study Answers

Case 15.1: ACE Inhibitor Initiation

Question 1. ACE inhibitors dilate the efferent arteriole, reducing intraglomerular pressure. In diabetic nephropathy, elevated glomerular pressure causes hyperfiltration and progressive damage. By lowering this pressure, ACE inhibitors reduce proteinuria and slow progression to end-stage renal disease independent of blood pressure effects.

Question 2. ACE inhibitors reduce aldosterone production by decreasing angiotensin II. Aldosterone normally promotes potassium secretion in the collecting duct. Less aldosterone means less potassium excretion, causing hyperkalemia. Risk increases with renal impairment, diabetes, or concurrent K⁺-sparing diuretics.

Question 3. The creatinine increase results from efferent arteriolar dilation reducing GFR. A rise up to 30% is expected and acceptable—it reflects reduced glomerular pressure (the protective mechanism). A rise >30% suggests bilateral renal artery stenosis, where GFR is critically dependent on angiotensin II-mediated efferent constriction.

Question 4. Important history includes: presence of renovascular disease risk factors (atherosclerosis, bruits, peripheral vascular disease), symptoms suggesting renal artery stenosis, use of NSAIDs (which worsen renal effects), potassium supplements or K⁺-sparing diuretics, and whether bilateral renal artery stenosis is possible.

Case 15.2: Calcium Channel Blocker Selection

Question 1. A non-dihydropyridine CCB (verapamil or diltiazem) is appropriate because she has both hypertension and atrial fibrillation with rapid ventricular rate. Non-DHP CCBs slow AV conduction, controlling ventricular rate while also lowering blood pressure. Diltiazem is often preferred due to less constipation.

Question 2. Amlodipine (DHP) would reduce blood pressure through vasodilation but would not control her heart rate. In fact, vasodilation may cause reflex tachycardia, potentially worsening her rapid ventricular response. She needs both BP reduction and rate control.

Question 3. Monitor for: bradycardia (excessive rate slowing), AV block (especially if combined with beta-blockers), constipation (more with verapamil), hypotension, and peripheral edema. Check ECG periodically for conduction abnormalities.

Question 4. Black patients often have low-renin hypertension, making the RAAS less activated. RAAS inhibitors (ACE inhibitors, ARBs) are less effective as monotherapy because there is less angiotensin II to block. CCBs work through direct vasodilation independent of the RAAS, making them equally effective regardless of renin status.

Diagram/Table Exercise Answers

DT 15.1: RAAS Cascade and Drug Targets

1. Drug class that blocks renin: **Direct renin inhibitors (aliskiren)**
2. Drug class that blocks ACE: **ACE inhibitors**
3. Drug class that blocks AT1 receptor: **ARBs (angiotensin receptor blockers)**
4. Blocking ACE increases **bradykinin**, causing cough.

DT 15.2: Calcium Channel Blocker Comparison

1. Dihydropyridines are selective for: **Blood vessels (vascular smooth muscle)**
 2. Non-dihydropyridines also affect: **Heart (cardiac muscle, conduction system)**
 3. Example of DHP: **Amlodipine**; Example of non-DHP: **Verapamil or Diltiazem**
 4. Non-DHPs are used for rate control in: **Atrial fibrillation**
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DT 15.3: ACE Inhibitor vs. ARB Comparison Table

Property	ACE Inhibitors	ARBs
Mechanism	Block ACE	Block AT1 receptor
Effect on bradykinin	Increased (not degraded)	No effect
Dry cough	Common (5-20%)	Rare
Angioedema risk	Present	Present (but less common)
Pregnancy	Contraindicated	Contraindicated
Combination together	Not recommended	Not recommended

DT 15.4: Antihypertensive Selection by Comorbidity

Comorbidity	Preferred Class	Rationale
Diabetes with proteinuria	ACE inhibitor/ARB	Reduces glomerular pressure and proteinuria
Heart failure (HFrEF)	ACE inhibitor/ARB + beta-blocker	Mortality benefit
Atrial fibrillation (rate control)	Non-DHP CCB (or beta-blocker)	Slows AV conduction
Elderly/isolated systolic HTN	Thiazide or DHP CCB	Effective, well-tolerated
Pregnancy	Methyldopa	Proven safety

Multiple-Choice Question Answers

Question 1. Answer: C

ACE inhibitors block conversion of angiotensin I to angiotensin II.

Question 2. Answer: B

ACE normally degrades bradykinin; when blocked, bradykinin accumulates and causes cough.

Question 3. Answer: C

Verapamil has the greatest effect on cardiac conduction and contractility among CCBs.

Question 4. Answer: C

Methyldopa has the most safety data in pregnancy; ACE inhibitors and ARBs are teratogenic.

Question 5. Answer: B

ARBs block AT1 receptors without affecting ACE, so bradykinin is not increased.

Question 6. Answer: C

Losartan (ARB) avoids cough because it doesn't affect bradykinin; other ACE inhibitors would cause the same cough.

Question 7. Answer: B

ACE inhibitors dilate the efferent arteriole, reducing glomerular capillary pressure and hyperfiltration.

Question 8. Answer: C

DHPs cause peripheral edema from precapillary dilation; bradycardia and constipation are non-DHP effects.

Question 9. Answer: B

Abrupt clonidine discontinuation causes sympathetic rebound and severe hypertension.

Question 10. Answer: C

ACE inhibitors reduce mortality in HFrEF; verapamil and nifedipine worsen heart failure.

Question 11. Answer: B

The ONTARGET trial showed increased adverse events without cardiovascular benefit from combination.

Question 12. Answer: B

Large creatinine increase suggests bilateral RAS, where GFR depends on angiotensin II-mediated efferent constriction.

Question 13. Answer: C

Angioedema involves multiple mediators including substance P and complement, not just bradykinin.

Question 14. Answer: B

Black patients often have low-renin hypertension, making RAAS inhibitors less effective alone.

Question 15. Answer: B

Hydralazine causes reflex tachycardia and fluid retention, requiring beta-blocker and diuretic.

Question 16. Answer: B

Non-DHP CCBs have negative inotropic effects that worsen cardiac output in systolic heart failure.