

COMPREHENSIVE MOCK EXAM 7

Supplemental to: Hypertension Board Review 2026-2027

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AHSCP Certification Examination for Hypertension Specialists (CHS) — maximum 200 multiple-choice questions in 3.5 hours; A-D single-best-answer format for this MEDREV parent book; domain-weighted to the 2026 AHSCP content outline

Board-Review Questions

1. A patient with new hypertension has central weight gain, proximal muscle weakness, easy bruising, wide violaceous striae, and recently diagnosed diabetes. Which secondary cause best unifies these findings?

- A. Primary aldosteronism can cause hypertension and metabolic abnormalities, but it does not explain the catabolic skin and muscle findings described.
- B. Pheochromocytoma can produce severe hypertension and hyperglycemia, but it more typically causes episodic headache, palpitations, and diaphoresis than violaceous striae.
- C. Hypothyroidism can cause weight gain and predominantly diastolic hypertension, but it does not account for easy bruising, proximal myopathy, and wide purple striae.
- D. Cushing syndrome can cause hypertension, central adiposity, proximal myopathy, easy bruising, violaceous striae, and glucose intolerance from chronic cortisol excess.

2. Which integrated mechanism is a major driver of obesity-associated hypertension?

- A. Increased sympathetic activity, renin-angiotensin-aldosterone signaling, and renal sodium retention raise vascular tone and effective circulating volume.
- B. Obstructive sleep apnea can add intermittent hypoxic sympathetic surges, but that mechanism alone does not explain the broader renal sodium-retentive physiology of obesity.
- C. Insulin resistance and endothelial dysfunction accompany obesity, but a sustained fall in sympathetic tone with renal salt wasting is not the usual hemodynamic pattern.
- D. Adipose tissue alters natriuretic-peptide biology, but obesity does not characteristically produce chronic natriuresis with contraction of extracellular volume.

3. An adult with hypertension recently had a myocardial infarction and has left ventricular systolic dysfunction. Which antihypertensive strategy best incorporates therapies with established post-MI benefit?

- A. A long-acting dihydropyridine calcium-channel blocker can lower BP, but it does not replace therapies that improve post-MI ventricular remodeling and outcomes.
- B. An alpha-1 blocker can reduce peripheral resistance, but it is not a preferred post-MI agent for improving ventricular remodeling or survival.

C. Use evidence-based post-MI therapy that also lowers BP, including a beta blocker and renin-angiotensin system blockade when clinically tolerated.

D. A central alpha-2 agonist can lower BP, but it is not a substitute for guideline-directed post-MI therapy in left ventricular systolic dysfunction.

4. A patient whose home average is 134/84 mm Hg wants a food plan that can lower pressure before the next visit. Which eating pattern should the clinician recommend as the best-studied hypertension-specific approach?

A. A Mediterranean-style diet can improve overall cardiovascular risk, but its blood-pressure effect is less specifically established than the DASH eating pattern.

B. A calorie-restricted low-carbohydrate diet may lower BP when it produces weight loss, but its benefit depends more on weight change than on a hypertension-specific food pattern.

C. A vegetarian diet may lower BP in some patients, but eliminating all animal products is not required to obtain the best-studied dietary antihypertensive effect.

D. A DASH-style diet emphasizing vegetables, fruits, whole grains, low-fat dairy, and reduced saturated fat has strong evidence for lowering blood pressure.

5. An adult with hypertension also has recurrent calcium nephrolithiasis and documented hypercalciuria. Which antihypertensive class may provide an additional stone-prevention benefit?

A. A thiazide or thiazide-like diuretic lowers BP and can reduce urinary calcium excretion, making it useful when hypercalciuria is clinically relevant.

B. A loop diuretic is useful for volume overload, but it increases urinary calcium excretion and therefore does not provide the same hypercalciuria benefit.

C. An ACE inhibitor may be preferred for albuminuric kidney disease, but it does not specifically reduce urinary calcium enough to serve as the stone-directed choice here.

D. A direct vasodilator such as hydralazine can lower BP, but it has no specific therapeutic effect on renal calcium handling in hypercalciuric stone disease.

6. An adult with severe symptomatic aortic stenosis also has persistent hypertension. What is the best general management principle?

- A.** Defer all antihypertensive treatment until valve intervention, an approach that avoids hypotension but leaves potentially harmful excess afterload untreated.
- B.** Treat hypertension cautiously with gradual dose titration and close clinical monitoring while arranging definitive evaluation and treatment of the valve lesion.
- C.** Use a rapidly acting arterial vasodilator to normalize outpatient BP promptly, a strategy that can provoke hypotension when fixed outflow obstruction limits compensation.
- D.** Increase intravascular volume routinely to maintain preload, an approach that may worsen pulmonary congestion and does not address the elevated systemic pressure.

7. A patient with previously controlled hypertension develops higher home and office readings after the dose of a serotonin-norepinephrine reuptake inhibitor is increased. What is the best next consideration?

- A.** Progression of primary hypertension remains possible, but the close temporal relationship to a noradrenergic dose increase should not be ignored.
- B.** Noradrenergic antidepressant effects can raise blood pressure, so the medication, dose, indication, and alternatives should be reviewed as potential contributors.
- C.** Adding another antihypertensive agent may eventually be needed, but doing so before assessing a newly introduced pressor medication can miss a reversible cause.
- D.** Depression-related inactivity can worsen cardiovascular risk, but it does not explain as directly as the medication change does a new dose-related pressure rise.

8. A patient has hypertension, hyperkalemia, normal renal function, and hyperchloremic metabolic acidosis that improves with a thiazide. Which inherited tubular disorder best fits this pattern?

- A.** Liddle syndrome causes increased epithelial sodium-channel activity and hypertension, but it usually produces hypokalemic metabolic alkalosis rather than hyperkalemic acidosis.
- B.** Gitelman syndrome impairs the distal Na-Cl cotransporter and is thiazide-like physiologically, but it typically causes salt wasting, hypokalemia, and metabolic alkalosis.
- C.** Bartter syndrome impairs thick ascending-limb salt transport and can cause hypokalemic alkalosis, but hypertension with hyperkalemic acidosis is not its typical phenotype.
- D.** Gordon syndrome increases thiazide-sensitive Na-Cl cotransport, producing hypertension with hyperkalemia and metabolic acidosis that often responds well to a thiazide.

9. An adult appears to have resistant hypertension on three medications because office pressures remain markedly elevated, yet ambulatory monitoring shows normal daytime and nighttime pressures. What should be concluded before therapy is intensified?

- A.** Masked uncontrolled hypertension is characterized by normal office but elevated out-of-office pressure, the opposite pattern from the ambulatory findings in this case.
- B.** A white-coat effect can produce apparent treatment resistance, so normal ambulatory pressures argue against automatically escalating therapy from office values alone.
- C.** True resistant hypertension remains a concern when both office and out-of-office pressures are elevated despite an appropriate regimen, which is not demonstrated here.
- D.** Pseudohypertension from arterial stiffness can cause spuriously high cuff readings, but the office-ambulatory discrepancy should first be interpreted as a white-coat pattern.

10. Why is therapeutic inertia an important quality-of-care problem in hypertension management?

- A.** Overtreatment refers to intensifying therapy despite low or symptomatic pressures, a different quality problem from failing to act on persistent above-goal readings.
- B.** Therapeutic inertia is failure to initiate or intensify appropriate treatment despite reliable evidence that blood pressure remains above the individualized goal.
- C.** Measurement error can create misleading readings and should be corrected, but it is not the definition of therapeutic inertia once persistent hypertension is established.
- D.** Medication nonadherence can mimic treatment failure and must be assessed, but therapeutic inertia specifically describes inadequate clinician or system treatment intensification.

11. A patient with resistant hypertension has loud snoring, witnessed apneas, and daytime sleepiness. Which test provides the diagnostic standard when a comprehensive sleep evaluation is required?

- A.** Overnight polysomnography is the reference diagnostic study; a validated home sleep-apnea test can also be appropriate in selected uncomplicated patients.
- B.** A STOP-BANG questionnaire is useful for screening risk, but a high score alone does not establish the apnea-hypopnea burden needed for diagnosis.
- C.** Overnight pulse oximetry may show desaturation and support suspicion, but it cannot characterize sleep stages and respiratory events as comprehensively as formal sleep testing.

D. Ambulatory blood-pressure monitoring may reveal nondipping or nocturnal hypertension, but those patterns are not specific enough to diagnose obstructive sleep apnea.

12. A 34-year-old woman develops abrupt hypertension and has an abdominal bruit. Renal-artery imaging shows alternating areas of stenosis and dilation. Which diagnosis is most likely?

A. Atherosclerotic renal-artery stenosis commonly affects older patients and the ostial/proximal artery rather than producing a beaded mid-distal pattern in a young woman.

B. Fibromuscular dysplasia can produce a string-of-beads appearance from alternating stenosis and dilation and is a classic renovascular cause in younger women.

C. Polyarteritis nodosa can involve medium-sized renal arteries and cause hypertension, but systemic vasculitic features and aneurysmal lesions are more typical than this classic beading pattern.

D. Renal-vein thrombosis can impair kidney function and cause flank symptoms, but it does not produce alternating stenosis and dilation of the renal artery.

13. An adult with biochemically confirmed pheochromocytoma is being prepared for surgical resection. Which medication sequence is essential if tachycardia also requires treatment?

A. Starting a beta blocker before alpha blockade may control pulse, but unopposed alpha-adrenergic vasoconstriction can precipitate a dangerous hypertensive crisis.

B. A calcium-channel blocker can be used as adjunctive or alternative pressure control in selected patients, but it does not eliminate the usual need for adequate alpha blockade.

C. Establish alpha-adrenergic blockade first; after adequate alpha blockade, a beta blocker can be added if persistent tachycardia requires treatment.

D. An ACE inhibitor can lower pressure in many hypertensive patients, but catecholamine-mediated vasoconstriction is not adequately addressed by ACE inhibition alone before tumor resection.

14. A patient with edema receives intravenous furosemide and promptly increases urinary sodium excretion. Blockade of which nephron transporter produces this loop-diuretic effect?

A. The Na-K-2Cl cotransporter in the thick ascending limb, the principal molecular target of loop diuretics such as furosemide.

- B.** The Na-Cl cotransporter in the distal convoluted tubule, which is the principal target of thiazide and thiazide-like diuretics rather than furosemide.
- C.** The epithelial sodium channel in the collecting duct, which is targeted by amiloride and triamterene rather than by loop diuretics.
- D.** The mineralocorticoid receptor in the collecting duct, which is antagonized by spironolactone and eplerenone rather than by furosemide.

15. An older adult develops recurrent orthostatic dizziness after recent intensification of antihypertensive therapy. What is the preferred first management strategy?

- A.** Maintaining the intensified regimen may preserve seated BP control, but persistent orthostatic symptoms raise fall risk and require evaluation rather than simple observation.
- B.** Stopping every cardiovascular medication may relieve symptoms, but it can unnecessarily remove therapies with important indications before the responsible contributors are identified.
- C.** Increasing antihypertensive intensity may be appropriate for sustained severe pressure, but dizziness on standing after a dose change suggests possible overtreatment rather than undertreatment.
- D.** Review medications and volume status, identify reversible contributors, measure orthostatic pressures, and add individualized nonpharmacologic measures before making targeted drug adjustments.

16. An adult taking amlodipine develops symmetric ankle edema without dyspnea, orthopnea, elevated jugular venous pressure, or other evidence of heart failure. Which mechanism best explains the edema?

- A.** Generalized sodium and water retention can cause dependent edema in heart or kidney failure, but amlodipine edema often occurs without true intravascular volume expansion.
- B.** Preferential precapillary arteriolar dilation raises capillary hydrostatic pressure, promoting fluid movement into dependent tissues despite otherwise stable volume status.
- C.** Chronic venous insufficiency can cause ankle edema, but its mechanism is venous hypertension rather than the characteristic arteriolar-capillary effect of dihydropyridine therapy.
- D.** Nephrotic syndrome can cause edema through hypoalbuminemia and renal sodium retention, but the vignette provides no proteinuric or systemic features suggesting that process.

17. A patient with hypertension has an eGFR of 28 mL/min/1.73 m², albuminuria, and bilaterally atrophic echogenic kidneys on ultrasonography. Which mechanism most likely contributes to the hypertension?

- A.** Renovascular disease can cause hypertension through renal ischemia and RAAS activation, but bilateral chronic parenchymal changes and albuminuria point more directly to intrinsic kidney disease.
- B.** Primary aldosteronism can coexist with CKD and cause resistant hypertension, but the structural renal abnormalities themselves provide a more immediate explanation in this vignette.
- C.** Obstructive sleep apnea is common in resistant hypertension, but it does not account for the reduced filtration, albuminuria, and chronic echogenic renal atrophy shown here.
- D.** Renal parenchymal disease promotes sodium retention and neurohormonal activation, making chronic kidney disease itself a common secondary contributor to hypertension.

18. An adult taking amlodipine has excellent blood-pressure control but troublesome dependent ankle edema without clinical volume overload. Which adjustment can be reasonable?

- A.** Reduce the amlodipine dose or combine it with an appropriate ACE inhibitor or ARB, strategies that may lessen vasodilatory edema while preserving pressure control.
- B.** Add a loop diuretic solely for the ankle swelling, an approach that may have limited benefit because dihydropyridine edema is not primarily caused by systemic volume excess.
- C.** Increase the amlodipine dose to improve arteriolar dilation, a change that commonly worsens rather than relieves dose-related dependent edema.
- D.** Diagnose heart failure and begin chronic decongestive therapy from ankle edema alone, despite the absence of dyspnea, orthopnea, or other evidence of congestion.

19. An adult with asthma has a compelling cardiovascular indication for beta blockade. Which prescribing principle is most accurate?

- A.** A nonselective beta blocker provides effective cardiovascular therapy, but beta-2 blockade can provoke bronchospasm and is generally less attractive in a patient with reactive airways.
- B.** When beta blockade is necessary, a beta-1 selective agent may be better tolerated, although active bronchospasm still requires caution, low-dose initiation, and individualized monitoring.
- C.** Avoiding all beta blockers is reasonable when there is no compelling indication, but an absolute lifetime prohibition may deny important benefit in selected patients with stable asthma.

D. Using a beta blocker with intrinsic sympathomimetic activity may seem attractive, but that property does not reliably protect against bronchospasm or improve cardiovascular outcome indications.

20. A 55-year-old has repeated standardized office blood pressures near 146/90 mm Hg but no previous hypertension diagnosis. When feasible, what is the best way to confirm persistent hypertension before committing to long-term therapy?

A. Additional standardized office measurements help establish persistence, but relying only on clinic values can miss white-coat and masked blood-pressure patterns.

B. An exercise stress test can evaluate ischemia and exercise pressure response, but it is not the standard method for confirming chronic resting hypertension.

C. Use validated home or ambulatory out-of-office measurements together with repeated standardized office readings to confirm the diagnosis and identify discordant patterns.

D. A cuffless smartwatch may provide convenient trends, but current unvalidated cuffless measurements should not replace validated cuff-based home or ambulatory confirmation.

21. An adult taking verapamil develops constipation and clinically important bradycardia. Which pharmacologic property best explains the slow heart rate?

A. Dihydropyridine calcium-channel blockers mainly dilate arterioles and have much less direct nodal suppression, so their mechanism does not explain verapamil-associated bradycardia.

B. Beta-1 receptor blockade slows sinus and AV nodal activity, but verapamil is not a beta blocker and produces the effect through calcium-channel inhibition.

C. Non-dihydropyridine calcium-channel blockade depresses sinoatrial automaticity and atrioventricular nodal conduction, which can produce bradycardia and conduction delay.

D. Mineralocorticoid-receptor antagonism can lower BP and potassium handling, but it does not directly account for the nodal conduction effects of verapamil.

22. Which broad lesson from ALLHAT remains relevant when selecting initial antihypertensive therapy for many patients?

A. A thiazide-like diuretic is an effective evidence-based first-line option with strong cardiovascular outcome data and remains appropriate for many patients.

B. An ACE inhibitor is an important first-line class in selected patients, but ALLHAT did not establish universal superiority over thiazide-like therapy for every outcome and population.

C. A long-acting dihydropyridine calcium-channel blocker is also an effective first-line option, but the trial did not show that thiazide-like diuretics should be abandoned.

D. An alpha-1 blocker can lower pressure and may help symptomatic benign prostatic hyperplasia, but its ALLHAT arm raised concern about heart-failure outcomes as routine first-line therapy.

23. A patient has confirmed resistant hypertension despite an optimized ACE inhibitor, long-acting dihydropyridine calcium-channel blocker, and chlorthalidone. Potassium is 4.0 mmol/L and eGFR is 68 mL/min/1.73 m². Which fourth-line agent is usually preferred?

A. A beta blocker can be appropriate for ischemic heart disease, tachyarrhythmia, or another compelling indication, but it is not usually the preferred generic fourth-line choice in this profile.

B. A central alpha-2 agonist such as clonidine can lower resistant pressure, but adverse effects and withdrawal hypertension usually place it after a mineralocorticoid-receptor antagonist.

C. An alpha-1 blocker can lower BP and may help coexisting prostatic symptoms, but evidence generally favors a mineralocorticoid-receptor antagonist as the next add-on here.

D. Spironolactone is usually the preferred fourth-line agent for resistant hypertension when potassium and kidney function permit, with subsequent laboratory monitoring.

24. A patient taking cyclosporine, a chronic NSAID, and a prescription stimulant develops newly resistant hypertension. What is the best first interpretation?

A. Primary aldosteronism remains an important cause of resistant hypertension and may merit screening, but several known pressor drugs should first be recognized as potentially reversible contributors.

B. Renovascular disease can present with abrupt or difficult hypertension, but the medication list provides a more immediate explanation that should be addressed before invasive vascular testing.

C. Pheochromocytoma can cause severe hypertension, but the absence of a suggestive syndrome and the presence of multiple pressor medications make it a less direct first explanation.

D. Cyclosporine, NSAIDs, and stimulants can each raise blood pressure, so the regimen should be reviewed systematically before labeling the hypertension biologically refractory.

25. A patient with acute type A aortic dissection has a blood pressure of 198/112 mm Hg and heart rate of 108/min while definitive surgical care is being arranged. Which pharmacologic sequence is preferred?

- A.** Begin an intravenous arterial vasodilator alone to reduce pressure quickly, but doing so before heart-rate control can provoke reflex tachycardia and increase aortic shear stress.
- B.** Use an oral long-acting antihypertensive regimen first, an approach too slow and difficult to titrate for the immediate anti-impulse control required in acute dissection.
- C.** Begin intravenous beta blockade for rapid heart-rate and impulse control, then add a titratable vasodilator if additional blood-pressure reduction is required.
- D.** Give a diuretic as the primary emergency therapy, which may reduce volume but does not rapidly control the tachycardia and contractile shear forces central to dissection management.

26. An adult presents with severe hypertension, agitation, tachycardia, and chest pain shortly after using cocaine. Which mechanism most directly drives the acute cardiovascular toxicity?

- A.** Cocaine has local anesthetic sodium-channel effects that can contribute to arrhythmia, but those effects do not primarily explain the marked hypertension and adrenergic agitation.
- B.** Coronary thrombosis can complicate cocaine use and cause chest pain, but it does not by itself account for the generalized tachycardic hypertensive syndrome.
- C.** Blockade of catecholamine reuptake produces acute sympathetic excess, vasoconstriction, tachycardia, and increased myocardial oxygen demand.
- D.** Panic can produce tachycardia and transient pressure elevation, but the recent stimulant exposure provides a direct pharmacologic cause of the severe sympathetic state.

27. During an episode of reduced effective arterial volume, pressure at the renal afferent arteriole falls. Which change at the juxtaglomerular apparatus directly promotes the compensatory increase in renin?

- A.** Reduced afferent-arteriolar perfusion pressure is sensed by juxtaglomerular cells and directly promotes renin secretion.
- B.** Increased sodium chloride delivery to the macula densa signals adequate tubular flow and generally suppresses, rather than stimulates, renin release.
- C.** Expansion of effective arterial volume raises renal perfusion and provides feedback that tends to reduce renin secretion.

D. Atrial natriuretic peptide rises with volume expansion and promotes natriuresis while generally opposing renin-angiotensin-aldosterone activation.

28. A 76-year-old with diffuse atherosclerotic disease has difficult hypertension, asymmetric kidneys, and two admissions for abrupt pulmonary edema without a new myocardial infarction. Which secondary hypertension mechanism deserves particular attention?

A. Severe atherosclerotic renal-artery stenosis can produce renovascular hypertension and recurrent flash pulmonary edema, especially with bilateral or functionally important disease.

B. Primary aldosteronism is an important cause of resistant hypertension, but recurrent abrupt pulmonary edema in an older patient with diffuse atherosclerosis particularly raises renovascular concern.

C. Obstructive sleep apnea commonly contributes to resistant hypertension, but it does not characteristically produce the recurrent flash-pulmonary-edema pattern described.

D. Fibromuscular dysplasia can cause renovascular hypertension, but the patient's age, smoking history, and diffuse atherosclerosis favor atherosclerotic rather than nonatherosclerotic renal-artery disease.

29. A patient with newly recognized hypertension is being evaluated for early kidney target-organ damage. Which urine test is particularly useful for detecting clinically important albuminuria before a major decline in eGFR?

A. A spot urine protein-to-creatinine ratio detects total protein and can be useful in kidney disease, but it is less specific than albumin measurement for early albuminuric risk assessment.

B. A spot urine albumin-to-creatinine ratio provides a practical quantitative measure of albuminuria and is useful for detecting early hypertensive or diabetic kidney injury.

C. Routine urinalysis may show protein or blood and remains useful, but a negative or semiquantitative dipstick can miss lower levels of clinically relevant albuminuria.

D. A 24-hour urine protein collection can quantify protein excretion, but it is cumbersome and is not usually the preferred first test for screening albuminuria in this setting.

30. Clinicians reviewing SPRINT note that intensive systolic blood-pressure treatment reduced cardiovascular events and mortality in a selected high-risk population. Which major group was excluded from the trial?

- A.** Adults 75 years and older were included and formed a prespecified subgroup, so advanced age alone was not an exclusion from SPRINT.
- B.** Adults at increased cardiovascular risk without diabetes comprised the core study population, making this group central rather than excluded.
- C.** Patients with chronic kidney disease were represented within defined eGFR limits, so CKD itself was not a blanket exclusion.
- D.** Patients with diabetes mellitus were excluded, which is important when applying SPRINT findings to hypertensive adults with diabetes.

31. Automated office readings vary widely from beat to beat in a patient with chronic atrial fibrillation. How should the clinician improve the estimate of the patient's resting blood pressure?

- A.** Auscultatory measurement can remain useful in atrial fibrillation, but a single reading is vulnerable to the same cycle-length variability that affects automated devices.
- B.** Obtain several careful measurements with a validated technique and average them because the irregular rhythm increases beat-to-beat blood-pressure variability.
- C.** An automated device may be acceptable if validated for irregular rhythms, but one isolated reading should not be assumed to represent the patient's average pressure.
- D.** Selecting only the highest systolic value may avoid underestimation, but it systematically biases the estimate upward rather than characterizing the average blood pressure.

32. A patient who consumes large amounts of natural licorice develops hypertension, hypokalemia, suppressed renin, and suppressed aldosterone. Which enzyme is functionally inhibited?

- A.** 11-beta-hydroxysteroid dehydrogenase type 1 interconverts glucocorticoids in metabolic tissues, but inhibition of this isoenzyme does not cause the classic apparent-mineralocorticoid-excess phenotype.
- B.** Aldosterone synthase inhibition would lower aldosterone, but it would not allow cortisol to activate mineralocorticoid receptors and produce the characteristic licorice syndrome.
- C.** Renin suppression is a downstream consequence of volume expansion in this disorder, not the primary enzymatic target of glycyrrhetic acid.
- D.** Inhibition of 11-beta-hydroxysteroid dehydrogenase type 2 prevents renal conversion of cortisol to cortisone, allowing cortisol to activate mineralocorticoid receptors.

33. An adult repeatedly has office blood pressures near 150/88 mm Hg, while a technically adequate 24-hour ambulatory study shows normal daytime and nighttime averages. Which phenotype best fits?

- A.** Sustained hypertension requires elevated pressure both in and outside the clinic, which is not demonstrated by the normal ambulatory study.
- B.** Masked hypertension has normal office pressure but elevated home or ambulatory pressure, the reverse of the pattern described here.
- C.** White-coat hypertension describes elevated office readings with normal out-of-office blood pressure in an untreated patient.
- D.** Pseudohypertension can produce spuriously high cuff values in selected patients with stiff arteries, but it is not the standard phenotype defined by normal ambulatory pressure.

34. Three months after starting a combined estrogen-progestin contraceptive, a previously normotensive patient has a sustained pressure increase. Which hepatic hormonal effect of estrogen can contribute to this change?

- A.** Estrogen can alter vascular and hepatic physiology, but a pure vasodilator effect would not explain the recognized pressure rise seen in some users.
- B.** Progestins differ in mineralocorticoid and androgenic properties, but the classic estrogen-related mechanism involves hepatic renin-angiotensin substrate production rather than universal direct sodium-channel activation.
- C.** Oral estrogen can increase hepatic angiotensinogen synthesis and thereby enhance renin-angiotensin-aldosterone system activity in susceptible patients.
- D.** Sympathetic activation can raise blood pressure in many settings, but estrogen-containing contraceptives are not primarily hypertensive through direct catecholamine release.

35. A patient has confirmed resistant hypertension while taking an ACE inhibitor, amlodipine, and hydrochlorothiazide. Before selecting a fourth drug, which diuretic optimization is evidence based when kidney function permits?

- A.** Increasing hydrochlorothiazide may produce additional natriuresis, but a longer-acting thiazide-like agent generally provides stronger and more sustained pressure control for resistant hypertension.
- B.** A loop diuretic becomes preferable when advanced CKD or significant volume overload limits thiazide effectiveness, but that is not established in this vignette.

C. Spironolactone is a preferred fourth-line add-on when appropriate, but the question first asks how to optimize the existing thiazide component of the three-drug regimen.

D. Replace hydrochlorothiazide with a long-acting thiazide-like diuretic such as chlorthalidone or indapamide when clinically appropriate.

36. An adult with ADHD develops higher blood pressure and pulse after an amphetamine dose increase. Which mechanism best accounts for the change?

A. Anxiety associated with stimulant treatment can transiently raise pressure, but the medication also has a direct catecholaminergic mechanism that better explains a reproducible dose-related rise.

B. Serotonergic effects can influence autonomic tone, but amphetamine-associated hypertension is driven principally by increased catecholamine signaling rather than serotonin-mediated vasodilation.

C. Sympathomimetic catecholaminergic stimulation increases heart rate, cardiac output, and systemic vascular tone, producing a dose-related rise in blood pressure in susceptible patients.

D. Mineralocorticoid excess can cause hypertension through sodium retention, but amphetamine does not act primarily as a mineralocorticoid-receptor agonist.

37. An adult with chronic kidney disease has confirmed average blood pressure of 136/84 mm Hg. Which general management principle is most consistent with current adult hypertension guidance?

A. Use lifestyle measures together with antihypertensive medication because CKD places the patient in a higher cardiovascular and renal-risk group at this blood-pressure level.

B. Lifestyle therapy alone for three to six months is an option for lower-risk stage 1 hypertension, but CKD is a high-risk condition that changes the treatment threshold.

C. Waiting for pressure to reach 140/90 mm Hg reflects an older threshold and does not account for the higher-risk status conferred by chronic kidney disease.

D. Albuminuria should still be assessed because it influences kidney and cardiovascular risk and can guide drug selection; CKD does not make urinary albumin information redundant.

38. A patient with severe bilateral atherosclerotic renal-artery disease has recurrent episodes of rapidly developing pulmonary edema despite otherwise optimized medical therapy. Which feature makes revascularization more reasonable to discuss?

A. Recurrent flash pulmonary edema or otherwise unexplained recurrent heart-failure destabilization suggests a high-risk renovascular syndrome in which revascularization may be considered.

B. Stable unilateral stenosis with controlled blood pressure and preserved kidney function is usually managed medically rather than routinely revascularized.

C. An incidental calcified renal-artery plaque without evidence of hemodynamically important stenosis does not establish a renovascular indication for intervention.

D. A single mildly elevated office reading that normalizes outside the clinic suggests a measurement phenotype rather than a high-risk renal-artery syndrome requiring revascularization.

39. An adult receiving an erythropoiesis-stimulating agent for chronic kidney disease develops a sustained rise in blood pressure after therapy begins. What is the best interpretation?

A. Volume overload from CKD can worsen blood pressure and should be assessed, but the temporal relationship also requires consideration of the erythropoiesis-stimulating agent itself.

B. Renovascular disease is a possible cause of resistant hypertension, but it should not be presumed solely because pressure rises after introduction of a known pressor medication.

C. Erythropoiesis-stimulating agents can increase blood pressure, so dose, hemoglobin trajectory, volume status, and the antihypertensive regimen should be reviewed.

D. Improved anemia can change hemodynamics, but hypertension is a recognized adverse effect of erythropoiesis-stimulating therapy rather than merely a benign consequence of better oxygen delivery.

40. A 12-year-old with exertional leg fatigue and headaches has arm pressure 152/86 mm Hg, ankle systolic pressure 108 mm Hg, and delayed femoral pulses. Which cardiovascular diagnosis should be pursued?

A. Renovascular hypertension can cause severe pediatric hypertension, but it does not characteristically produce a fixed arm-leg pressure gradient with delayed femoral pulses.

B. Obstructive sleep apnea can contribute to pediatric hypertension, but it does not explain the pulse delay and lower-extremity pressure deficit.

C. Coarctation of the aorta produces upper-extremity hypertension with diminished or delayed femoral pulses and lower leg pressure, often with a posterior systolic murmur.

D. Primary aldosteronism can produce hypertension and hypokalemia, but it does not create the characteristic upper-to-lower extremity pulse and pressure findings.

41. Soon after hydralazine is given, systemic resistance falls and the pulse accelerates. Which primary action of hydralazine triggers this baroreflex response?

A. Minoxidil also causes direct arteriolar dilation through potassium-channel effects and can provoke reflex tachycardia, but that is a different drug mechanism.

B. Organic nitrates preferentially dilate veins at lower doses and reduce preload, a vascular profile different from hydralazine's predominant arteriolar action.

C. Central alpha-2 agonists reduce sympathetic outflow and tend to slow heart rate, which is opposite to the reflex response produced by hydralazine.

D. Hydralazine directly dilates arterioles, lowering systemic vascular resistance and triggering baroreflex sympathetic activation with tachycardia.

42. An adult has a persistent bothersome cough from an ACE inhibitor but no history of angioedema. If renin-angiotensin system blockade remains indicated, which substitution is most logical?

A. A different ACE inhibitor usually reproduces the class-related bradykinin cough, so switching within the same class is unlikely to solve the problem.

B. An angiotensin receptor blocker preserves downstream RAAS blockade without inhibiting bradykinin breakdown and therefore usually avoids ACE-inhibitor cough.

C. A direct renin inhibitor also affects the RAAS, but it is not the usual substitute when a well-established ARB option is available for ACE-inhibitor cough.

D. Combining a direct renin inhibitor with an ARB increases RAAS blockade but adds adverse-effect risk and is not a strategy for treating ACE-inhibitor cough.

43. Six months after documented normal readings, a 38-year-old presents repeatedly with pressures near 190/110 mm Hg. Which aspect of this history is the strongest clue that a secondary hypertension workup may be warranted?

A. A strong family history and gradual pressure rise with weight gain would favor common primary hypertension rather than an abrupt secondary process.

- B.** A transient high reading during acute pain or sleep deprivation can be reactive and should be repeated before being interpreted as persistent disease.
- C.** Mild stage 1 hypertension that improves substantially with weight loss and sodium reduction is common in primary hypertension and is less suggestive of a secondary cause.
- D.** Abrupt onset of severe hypertension at a relatively young age is a classic clue that should prompt evaluation for an underlying secondary cause.

44. A patient asks whether one lifestyle change can substitute for broader cardiovascular-risk management once hypertension is diagnosed. What is the best counseling?

- A.** Sodium reduction can lower blood pressure substantially, but it does not replace management of smoking, lipids, diabetes, weight, physical inactivity, sleep, and other risks.
- B.** Regular aerobic exercise improves pressure and cardiovascular health, but exercise alone does not neutralize persistent tobacco exposure, excess alcohol, dyslipidemia, or other major risks.
- C.** Address diet, activity, weight, tobacco, sleep, alcohol, and other modifiable cardiovascular risks together rather than relying on a single lifestyle intervention.
- D.** Weight loss can reduce medication requirements in some patients, but persistent stage 2 hypertension still requires pressure-specific treatment rather than assuming weight change alone is sufficient.

45. A patient with proteinuric CKD asks whether adding an ARB to an existing ACE inhibitor would provide “double kidney protection.” Why is this combination generally avoided?

- A.** Dual RAAS blockade can reduce proteinuria more than either drug alone in some settings, but this surrogate benefit has not justified the added safety risk for routine hypertension treatment.
- B.** An ACE inhibitor and an ARB act at different points in the same pathway, so pharmacologic overlap can intensify adverse renal and potassium effects rather than making either drug inactive.
- C.** Dual blockade increases the risk of hyperkalemia, hypotension, and kidney injury without sufficient added clinical-outcome benefit to support routine combined use.
- D.** Mineralocorticoid-receptor antagonists also affect the RAAS and may be appropriate for resistant hypertension, but that does not make ACE inhibitor-ARB combination therapy preferable.

46. A patient taking three antihypertensive medications begins a successful structured weight-loss and exercise program. What monitoring issue becomes particularly important as fitness and weight improve?

- A.** Maintaining the original doses may be reasonable initially, but lifestyle-related pressure reductions can occur early enough that fixed dosing without reassessment risks overtreatment.
- B.** Reassess home blood pressure and symptoms because medication requirements may fall and orthostatic dizziness can emerge as lifestyle treatment lowers pressure.
- C.** Stopping all antihypertensive therapy as soon as weight begins to fall may avoid hypotension, but it can produce rebound loss of control before the new requirement is known.
- D.** Relying only on occasional office readings may miss low home pressures, so out-of-office monitoring is more useful than assuming exercise-related changes are clinically negligible.

47. A patient with hypertension has fatigue, weight gain, cold intolerance, bradycardia, predominantly diastolic hypertension, and an elevated TSH. Which disorder is the most likely contributor?

- A.** Hypothyroidism can increase systemic vascular resistance and produce diastolic hypertension together with bradycardia, cold intolerance, and weight gain.
- B.** Obstructive sleep apnea can cause fatigue and hypertension, but it does not explain the elevated TSH, cold intolerance, and bradycardic hypothyroid pattern.
- C.** Pheochromocytoma can cause episodic hypertension and adrenergic symptoms, but bradycardia and cold intolerance are not its typical clinical pattern.
- D.** Cushing syndrome can cause hypertension and weight gain, but proximal myopathy, bruising, striae, and cortisol-related features would be expected rather than this thyroid profile.

48. A 17-year-old has an elevated oscillometric blood-pressure reading at a routine visit. What is the preferred next step before diagnosing sustained hypertension?

- A.** Initiating two medications from the first screening value would bypass confirmation and is reserved for selected patients with established or severe hypertension, not one oscillometric reading.
- B.** Repeat the measurement by proper auscultatory technique, confirm persistence over appropriate visits or ambulatory monitoring, and apply the fixed adolescent thresholds used from age 13 onward.
- C.** Percentile-based thresholds remain important for children younger than 13, but a 17-year-old is classified using the fixed adolescent blood-pressure categories.

D. Accepting a single oscillometric value as permanent hypertension ignores cuff fit, rest conditions, device variability, and the need for confirmatory measurements.

49. A 24-year-old with a bicuspid aortic valve is found to have upper-extremity hypertension, delayed femoral pulses, and substantially lower systolic pressure in the legs. Which associated diagnosis should be suspected?

A. Renal-artery fibromuscular dysplasia can cause hypertension in a young adult, but it does not explain delayed femoral pulses and a reproducible arm-leg pressure gradient.

B. Primary aldosteronism can occur in younger adults and cause hypertension, but it does not produce a structural pulse delay between the upper and lower extremities.

C. Takayasu arteritis can create pulse deficits and pressure asymmetry, but the bicuspid-valve association and classic arm-leg gradient particularly suggest congenital aortic coarctation.

D. Coarctation of the aorta is associated with bicuspid aortic valve and causes upper-body hypertension with delayed femoral pulses and lower leg pressures.

50. When renal perfusion varies within the autoregulatory range, afferent arteriolar tone changes in response to wall stretch and distal tubular sodium delivery. Which two local mechanisms produce this response?

A. Angiotensin II helps regulate efferent arteriolar tone during low perfusion, but renal autoregulation across ordinary pressure ranges is not produced by circulating RAAS activity alone.

B. The afferent arteriolar myogenic response and tubuloglomerular feedback adjust resistance locally to stabilize renal blood flow and glomerular filtration.

C. Renal sympathetic nerves alter vascular tone during stress and volume changes, but they are not the principal intrinsic mechanisms responsible for moment-to-moment autoregulation.

D. Atrial natriuretic peptide can influence glomerular hemodynamics and sodium excretion, but it does not provide the primary local feedback system that stabilizes GFR across changing pressures.

51. A pregnant patient requires medication for chronic hypertension. Which oral agents are commonly used because of their established pregnancy safety profile?

A. An ACE inhibitor is effective for many nonpregnant adults, but fetal renin-angiotensin system toxicity makes the class inappropriate during pregnancy.

- B.** An angiotensin receptor blocker is useful for RAAS blockade outside pregnancy, but fetal renal toxicity makes it inappropriate for chronic treatment during pregnancy.
- C.** A direct renin inhibitor also interferes with the fetal renin-angiotensin system and is not an established pregnancy antihypertensive option.
- D.** Labetalol or extended-release nifedipine are commonly used, with methyldopa remaining an established alternative for selected pregnant patients.

52. A patient with advanced heart failure has hypertension and severe bilateral edema from marked volume overload. Why is a loop diuretic often preferred over a thiazide as the main decongestive agent?

- A.** A thiazide-like diuretic can be added for sequential nephron blockade in refractory congestion, but it is not usually the main agent for severe volume overload.
- B.** Mineralocorticoid-receptor antagonists improve outcomes in selected heart-failure patients and provide mild natriuresis, but they are not the primary rapid decongestive therapy for major edema.
- C.** Loop diuretics produce potent natriuresis and remain useful for substantial volume overload, including when kidney function is reduced.
- D.** An SGLT2 inhibitor can provide modest natriuretic and heart-failure benefit, but it does not replace loop-diuretic dosing when immediate treatment of severe congestion is required.

53. A younger adult with renal-artery fibromuscular dysplasia has difficult hypertension and a focal hemodynamically significant lesion. When revascularization is appropriate, which approach is generally preferred?

- A.** Percutaneous transluminal renal angioplasty is generally preferred, with stenting reserved for selected complications or lesions rather than used routinely.
- B.** Primary renal-artery stenting is standard for atherosclerotic ostial disease in selected cases, but routine stenting is usually unnecessary for uncomplicated fibromuscular dysplasia.
- C.** Open surgical revascularization can be considered for complex anatomy or failed endovascular therapy, but it is not the usual first intervention for a focal treatable lesion.
- D.** Nephrectomy can be appropriate for a nonfunctional kidney in exceptional circumstances, but it is far more destructive than angioplasty for a salvageable stenotic renal artery.

54. A dry cough resolves after lisinopril is replaced by losartan while blood-pressure control is maintained. Which pharmacologic difference best explains why the ARB is less likely to reproduce the cough?

- A.** Both drug classes inhibit the renin-angiotensin system, but ARBs do not block ACE and therefore do not produce the same bradykinin accumulation responsible for much ACE-inhibitor cough.
- B.** Histamine can contribute to cough from allergic or pulmonary disease, but ARBs do not prevent ACE-inhibitor cough by functioning as pulmonary antihistamines.
- C.** Angiotensin receptor blockers do not inhibit bradykinin degradation, so they usually avoid the bradykinin-mediated cough associated with ACE inhibition.
- D.** Central cough suppression is a property of antitussive medications, not the mechanism by which ARBs reduce the likelihood of cough after ACE-inhibitor intolerance.

55. Why can ambulatory blood-pressure monitoring add useful information in a hypertensive patient with suspected obstructive sleep apnea?

- A.** A formal sleep study determines the apnea-hypopnea index, whereas ambulatory BP monitoring evaluates pressure behavior and cannot directly diagnose respiratory events.
- B.** Home or ambulatory pressure can help distinguish white-coat effects, but only 24-hour monitoring also characterizes the nocturnal blood-pressure profile during usual sleep.
- C.** Medication adherence remains a separate part of resistant-hypertension evaluation; a normal or abnormal nocturnal pattern does not substitute for assessing whether prescribed drugs are taken.
- D.** Ambulatory monitoring can identify nocturnal hypertension or a nondipping pattern that is missed by daytime office measurements and is common in sleep apnea.

56. Which classes are standard first-line pharmacologic options for uncomplicated hypertension in most adults, with selection individualized to comorbidity and patient factors?

- A.** Beta blockers are appropriate when coronary disease, arrhythmia, or another compelling indication is present, but they are not universally preferred as the sole routine first-line class.
- B.** Loop diuretics are useful for major volume overload or advanced kidney disease, but uncomplicated hypertension is usually treated first with agents that have broader outcome evidence.
- C.** Central alpha-2 agonists and direct vasodilators can lower pressure in resistant cases, but tolerability and adverse effects generally place them later in treatment.

D. ACE inhibitors or ARBs, long-acting dihydropyridine calcium-channel blockers, and thiazide or thiazide-like diuretics are standard first-line choices.

57. A patient with obesity, obstructive sleep apnea, and resistant hypertension uses CPAP every night and asks whether weight reduction still matters. What is the best response?

A. CPAP treats upper-airway obstruction, but it does not eliminate the independent effects of excess adiposity on sympathetic tone, sodium retention, metabolic disease, and blood pressure.

B. Sodium reduction remains helpful, but dietary sodium is only one contributor and does not replace weight management when obesity is driving both apnea severity and hypertension.

C. Weight reduction remains important because obesity contributes independently to obstructive sleep apnea severity and to several mechanisms of hypertension.

D. Bariatric or anti-obesity treatment may not be appropriate for every patient, but persistent obesity continues to influence hypertension physiology even with excellent CPAP adherence.

58. A patient remains hypertensive on six appropriately selected medications. Ambulatory monitoring confirms persistent hypertension, adherence is verified, secondary causes have been addressed, and the regimen and volume status are optimized. What is the preferred next step?

A. Refer to a hypertension specialist or experienced center for further phenotyping and individualized consideration of advanced pharmacologic or device-based strategies.

B. Rechecking adherence and interfering substances remains useful over time, but stopping all medications simultaneously would expose the patient to severe uncontrolled pressure without diagnostic benefit.

C. Additional secondary-cause evaluation can be reconsidered when new clues appear, but the number of medications does not make further expert assessment futile.

D. Simply adding drugs without reassessing doses, interactions, tolerability, volume status, and specialist options increases polypharmacy without ensuring that the next treatment is rational.

59. A patient with stable claudication and hypertension worries that lowering systemic pressure will “starve the legs.” Which long-term blood-pressure strategy is most appropriate in otherwise stable peripheral artery disease?

A. Severe limb ischemia requires individualized hemodynamic assessment, but stable peripheral artery disease is not a reason to accept uncontrolled systemic hypertension as a chronic strategy.

- B.** Antiplatelet and lipid-lowering therapy address other vascular risks, but those therapies complement rather than replace appropriate blood-pressure treatment in peripheral artery disease.
- C.** Short-acting vasodilators can cause pressure variability and are not used intermittently to raise limb perfusion while avoiding sustained cardiovascular risk reduction.
- D.** Treat hypertension to reduce overall cardiovascular risk while monitoring claudication, limb symptoms, orthostatic tolerance, and perfusion in patients with advanced disease.

60. A patient beginning antihypertensive therapy is asked to keep a home blood-pressure log. Which method best improves the clinical usefulness of those readings?

- A.** Use a validated upper-arm cuff, follow standardized seated technique, and record repeated morning and evening readings over several days rather than isolated values.
- B.** Symptom-triggered measurements can help investigate dizziness or palpitations, but measuring only when symptomatic creates a biased record that does not represent usual pressure.
- C.** Postexercise or postcaffeine readings can demonstrate physiologic responses, but they should not replace standardized resting measurements used for treatment decisions.
- D.** Wrist devices can be appropriate in selected circumstances if validated and positioned correctly, but an upper-arm cuff is generally preferred for routine home monitoring.

61. A patient arrives with pressure 226/134 mm Hg, progressive confusion, retinal hemorrhages with disc edema, and a new rise in creatinine. Which finding makes this a hypertensive emergency rather than severe hypertension without emergency features?

- A.** Acute hypertension-mediated brain and kidney injury is present, making this a hypertensive emergency that requires monitored, titratable intravenous treatment.
- B.** A pressure above 180/120 mm Hg is severe and demands prompt assessment, but the diagnosis of emergency depends on acute target-organ injury rather than the number alone.
- C.** Headache or anxiety can accompany severe hypertension without acute organ injury, but confusion, papilledema, and new kidney injury provide objective evidence of emergency in this case.
- D.** Chronic kidney disease can coexist with severe hypertension, but an acute deterioration in renal function together with neurologic findings changes the urgency and treatment setting.

62. An 82-year-old has a bounding pulse and blood pressure 176/64 mm Hg despite a normal cardiac output and no major valvular lesion. Which age-related arterial property best explains the large pulse pressure?

- A.** Baroreflex sensitivity often declines with age and can affect pressure variability, but it does not directly explain the characteristic widened pulse pressure from stiff large arteries.
- B.** Reduced venous compliance can influence preload, but venous mechanics are not the principal determinant of the age-related rise in systolic pressure and pulse pressure.
- C.** Systemic vascular resistance may change with aging, but a uniform fall in resistance would tend to lower rather than explain the characteristic systolic pressure rise.
- D.** Large-artery stiffening increases pulse-wave velocity and returns reflected waves earlier during systole, augmenting systolic pressure and widening pulse pressure.

63. A patient reports dizziness after several normal-to-low home readings but has an elevated clinic pressure. Which approach is preferred before intensifying therapy?

- A.** The office value may reflect a white-coat effect, so choosing the highest clinic measurement as the sole titration target risks worsening symptomatic out-of-office hypotension.
- B.** Review home technique, obtain orthostatic measurements, and integrate symptoms with out-of-office pressures rather than automatically intensifying therapy from the clinic value alone.
- C.** Temporarily holding a suspected medication may be appropriate if symptoms are severe, but stopping every antihypertensive without first characterizing pressure and orthostasis can cause rebound loss of control.
- D.** A vestibular cause of dizziness can coexist, but treatment decisions should still incorporate the temporal relation to medication, home pressure, and standing measurements.

64. A patient with established coronary artery disease has a confirmed average blood pressure of 132/82 mm Hg. Which treatment principle applies under current adult hypertension guidance?

- A.** A 140/90 mm Hg drug threshold is too high for this higher-risk patient because established cardiovascular disease supports treatment at stage 1 pressure levels.
- B.** Established cardiovascular disease places the patient in a group for whom pharmacologic treatment is recommended at stage 1 levels together with lifestyle measures.
- C.** PREVENT-CVD risk estimation is valuable for primary prevention, but a risk score is not required to prove high risk once coronary artery disease is already established.

D. Excessively low diastolic pressure may require individualized caution in symptomatic coronary disease, but that concern does not justify a routine systolic target of 140-150 mm Hg for everyone.

65. An older adult has very high cuff pressures but little evidence of target-organ injury, and the radial artery remains palpable even when the cuff is inflated above the measured systolic pressure. What phenomenon may be considered?

A. A white-coat effect can cause high office pressure with normal out-of-office values, but it does not specifically explain persistence of a palpable artery above cuff systolic pressure.

B. Masked hypertension is the opposite office-out-of-office pattern and would not explain markedly elevated clinic readings with an abnormal cuff-compressibility sign.

C. Orthostatic hypertension can produce a rise in pressure on standing, but it does not account for persistence of a palpable artery despite suprasystolic cuff inflation.

D. Pseudohypertension from poorly compressible calcified arteries can produce spuriously high cuff readings and is suggested by an Osler-type maneuver in the appropriate clinical context.

66. A 15-year-old has repeated auscultatory blood pressures averaging 134/84 mm Hg. Which classification applies using the fixed adolescent thresholds used from age 13 onward?

A. Stage 1 hypertension applies because adolescents 13 years and older use fixed thresholds of 130-139 systolic or 80-89 diastolic for stage 1.

B. Stage 2 hypertension begins at 140/90 mm Hg or higher in this age group, so 134/84 mm Hg does not meet stage 2 criteria.

C. Height-based percentiles remain central for children younger than 13, but fixed adolescent categories apply to a 15-year-old.

D. Elevated blood pressure is 120-129 systolic with diastolic below 80 mm Hg, so the diastolic value of 84 mm Hg moves this patient into stage 1 hypertension.

67. Which physiologic effect links insulin resistance and hyperinsulinemia to hypertension in some patients with metabolic dysfunction?

A. Endothelial dysfunction can alter vascular tone in insulin resistance, but metabolic disease is not isolated from renal and autonomic mechanisms of blood-pressure regulation.

- B.** Enhanced sympathetic activity and increased renal sodium retention can accompany hyperinsulinemia and contribute to higher vascular tone and effective volume.
- C.** Insulin has vasodilator signaling under normal conditions, but insulin resistance does not characteristically produce a sustained shutdown of sympathetic activity that lowers pressure.
- D.** Natriuresis can lower pressure in other settings, but insulin resistance is associated more often with sodium retention than with chronic renal salt wasting.

68. A patient has uncontrolled office pressure despite four prescribed antihypertensive drugs, but pharmacy records show that two medications have not been filled for months. What should precede further treatment escalation?

- A.** Address nonadherence, cost, adverse effects, understanding, and regimen complexity before labeling the patient as having true resistant hypertension.
- B.** A fifth drug could lower pressure if all current agents were being taken, but adding therapy before resolving major refill gaps may increase complexity without treating the actual problem.
- C.** Secondary-cause testing can be important in resistant hypertension, but apparent resistance caused by nonadherence should be corrected before escalating to extensive advanced testing.
- D.** Prescribed medication count alone does not prove treatment exposure, so classifying the patient as truly resistant without verifying adherence would be premature.

69. A kidney-transplant recipient develops new difficult hypertension as tacrolimus exposure increases, without a new vascular lesion on imaging. Which calcineurin-inhibitor effect can account for the pressure rise?

- A.** Transplant renal-artery stenosis is an important post-transplant cause of hypertension, but the temporal association with tacrolimus also raises a medication-specific mechanism.
- B.** Corticosteroids can increase pressure through volume and metabolic effects, but the question asks for a recognized mechanism of the calcineurin inhibitor itself.
- C.** Chronic allograft dysfunction can cause sodium retention and hypertension, but tacrolimus can raise pressure even without structural graft failure.
- D.** Calcineurin inhibitors can cause renal vasoconstriction, sympathetic and endothelial effects, and sodium retention that contribute to hypertension.

70. After hydrochlorothiazide is increased, a patient's serum urate rises to 9.6 mg/dL and podagra recurs. Which thiazide-related metabolic effect is the most likely contributor?

- A.** Loop diuretics can also raise uric acid, but switching automatically to a loop would not remove the diuretic-related gout risk unless another indication favored it.
- B.** Thiazide therapy can reduce renal urate excretion and raise serum uric acid, precipitating gout in susceptible patients.
- C.** Metabolic syndrome can independently increase gout risk, but the dose-related temporal association makes the thiazide effect clinically relevant rather than merely coincidental.
- D.** Dietary purine intake affects uric acid and should be reviewed, but thiazides do not cause gout primarily by increasing intestinal purine absorption.

71. A patient with chronic kidney disease and difficult hypertension is started on spironolactone. Which complication requires especially close laboratory surveillance?

- A.** Gynecomastia and breast tenderness are recognized adverse effects of spironolactone, but they do not replace the need for urgent electrolyte monitoring in chronic kidney disease.
- B.** A decline in kidney function can increase drug-related potassium risk, but isolated respiratory acid-base changes are not the principal toxicity being monitored.
- C.** Eplerenone has fewer antiandrogen effects than spironolactone, but both mineralocorticoid-receptor antagonists can impair potassium excretion.
- D.** Hyperkalemia is the major concern, particularly as eGFR falls or when an ACE inhibitor or ARB is used concurrently.

72. Why must SPRINT blood-pressure targets be interpreted in the context of measurement technique when applied in routine practice?

- A.** Different validated techniques can yield clinically meaningful differences, so assuming every office workflow produces identical values can lead to inappropriate target translation.
- B.** Exercise blood pressure has prognostic information in selected settings, but SPRINT targets were based on standardized resting measurements rather than maximal exercise values.
- C.** Accurate standardized measurement is essential when applying SPRINT-derived targets because treatment intensity depends on how the pressure was obtained.

D. Upper-arm cuff measurement remains the clinical standard; SPRINT did not derive its targets from unvalidated cuffless wrist or smartwatch measurements.

73. A patient with mild-to-moderate spontaneous intracerebral hemorrhage presents early with systolic blood pressure 188 mm Hg and no contraindication to treatment. Which acute pressure approach is reasonable?

A. Permissive severe hypertension may be necessary in selected cerebrovascular conditions, but early intracerebral hemorrhage with systolic pressure in this range generally benefits from controlled lowering rather than routine deferral.

B. Rapid reduction below 120 mm Hg can threaten cerebral perfusion and has not shown benefit; overly aggressive lowering should be avoided.

C. Intermittent oral dosing can produce pressure variability and is less controllable than titratable intravenous therapy during the early management of intracerebral hemorrhage.

D. Use smooth titratable treatment toward about 140 mm Hg, generally maintaining systolic pressure in the 130-150 mm Hg range for appropriate mild-to-moderate ICH.

74. A patient arrives hurried and has an initial office blood pressure of 154/92 mm Hg. Which technique best improves the accuracy of the repeat measurement?

A. A smaller cuff can overestimate pressure when the arm is large, so cuff size should be matched to arm circumference rather than intentionally minimized.

B. Letting the arm hang below heart level adds hydrostatic pressure and can falsely elevate the reading; the arm should instead be supported at heart level.

C. Talking during measurement can raise pressure and increase variability, so the patient should rest quietly and remain silent during the repeat reading.

D. After quiet rest, seat the patient with back supported and feet on the floor, support the arm at heart level, and use an appropriately sized validated upper-arm cuff.

75. A patient asks whether a cuffless smartwatch can replace validated home cuff measurements for hypertension diagnosis and medication titration. What is the best advice under current adult guidance?

A. Do not rely on cuffless devices for accurate clinical blood-pressure decisions until their precision, calibration stability, and reliability are adequately validated.

- B.** A cuffless device may be useful for exploratory trends in the future, but a normal office reading does not make an unvalidated wearable clinically accurate.
- C.** Calibration requirements and motion or position effects remain important technical limitations, so a smartwatch should not be treated as the preferred diagnostic standard.
- D.** Treatment decisions should be confirmed with validated cuff-based measurements rather than triggered automatically by any isolated wearable systolic value above 120 mm Hg.

76. During manual blood-pressure measurement, Korotkoff sounds disappear temporarily and then return as the cuff deflates. If this auscultatory gap is not recognized, what error can occur?

- A.** Diastolic pressure can be misread if later Korotkoff phases are interpreted incorrectly, but the classic auscultatory-gap error is missing the true initial systolic sounds.
- B.** Arterial stiffness can widen pulse pressure and make auscultation difficult, but the gap itself does not make the true systolic pressure higher than the first genuine Korotkoff sound.
- C.** An irregular rhythm can increase beat-to-beat variability and complicate measurement, but an auscultatory gap is a recognized phenomenon even with a regular rhythm.
- D.** Systolic pressure may be underestimated if the cuff is not first inflated above the true systolic level and the examiner mistakes the reappearance of sounds for the initial systolic phase.

77. A treated adult has a sustained fall in blood pressure from 138/76 mm Hg supine to 114/64 mm Hg within 3 minutes of standing, accompanied by dizziness. What diagnosis is present?

- A.** White-coat hypertension produces elevated clinic pressure with normal out-of-office pressure, not a symptomatic fall after standing.
- B.** Masked hypertension produces normal clinic pressure with elevated out-of-office pressure, not a posture-related pressure decline.
- C.** Orthostatic hypotension is present because systolic pressure falls by at least 20 mm Hg within 3 minutes of standing and symptoms occur.
- D.** Hypertensive emergency requires severe pressure with acute target-organ injury, the opposite hemodynamic problem from this symptomatic postural fall.

78. A patient with hypertension sleeps about 5 hours nightly on an irregular schedule but has no evidence of obstructive sleep apnea. Which counseling is reasonable?

- A.** Sedative medication may be indicated for a defined sleep disorder, but routine hypnotic treatment is not a primary antihypertensive strategy for insufficient sleep.
- B.** Encourage adequate, regular sleep as one component of a broader lifestyle program that also addresses diet, activity, weight, alcohol, and other cardiovascular risks.
- C.** Sodium reduction remains valuable, but sleep duration and regularity also influence cardiometabolic health and should not be dismissed once dietary counseling begins.
- D.** Chronic sleep restriction can increase sympathetic and metabolic stress, so intentionally maintaining short sleep is not a strategy for lowering blood pressure.

79. An adult has resistant hypertension, spontaneous hypokalemia, suppressed renin, and inappropriately elevated aldosterone. Which diagnosis best explains the biochemical pattern?

- A.** Liddle syndrome can cause low-renin hypertension and hypokalemia, but aldosterone is typically suppressed rather than inappropriately elevated.
- B.** Renovascular hypertension activates renin and secondarily raises aldosterone, so suppressed renin argues against that mechanism.
- C.** Cushing syndrome can cause hypertension and hypokalemia through mineralocorticoid effects, but the specific high-aldosterone, low-renin profile points elsewhere.
- D.** Primary aldosteronism causes autonomous aldosterone production with renin suppression and can produce hypokalemia, especially in more florid disease.

80. Why can primary aldosteronism produce metabolic alkalosis together with hypokalemia?

- A.** Loop-diuretic physiology can increase distal sodium delivery and promote potassium and hydrogen loss, but primary aldosteronism produces the effect through mineralocorticoid receptor activation.
- B.** Mineralocorticoid activity increases distal sodium reabsorption while enhancing potassium and hydrogen-ion secretion, producing hypokalemia and metabolic alkalosis.
- C.** Vomiting also causes hypokalemic metabolic alkalosis, but that mechanism depends on gastric acid loss and secondary renal responses rather than autonomous aldosterone action.
- D.** Renal tubular acidosis can produce hypokalemia, but it causes metabolic acidosis rather than the alkalosis expected with excess mineralocorticoid activity.

81. A patient on four antihypertensive agents has a BMI of 37 kg/m², large neck circumference, morning headaches, witnessed respiratory pauses, and daytime sleepiness. Which secondary contributor should be evaluated first from these clues?

- A.** Primary aldosteronism is common in resistant hypertension and can coexist with sleep apnea, but it does not explain the loud snoring, witnessed respiratory pauses, and daytime sleepiness.
- B.** Hypothyroidism can cause fatigue and weight gain and may worsen sleep-disordered breathing, but the witnessed apneas make obstructive sleep apnea the more direct diagnosis.
- C.** Obstructive sleep apnea causes recurrent upper-airway obstruction, intermittent hypoxia, sympathetic activation, sleepiness, and can contribute to resistant hypertension.
- D.** Renal parenchymal disease can cause resistant hypertension, but the vignette lacks kidney findings and instead provides a classic sleep-disordered-breathing syndrome.

82. Which laboratory pattern in a patient with hypertension most directly indicates chronic kidney disease as hypertension-mediated target-organ damage?

- A.** Persistently reduced estimated GFR and/or persistent albuminuria provide evidence of chronic kidney damage and carry cardiovascular and renal prognostic significance.
- B.** A mildly elevated fasting glucose indicates metabolic risk and possible diabetes, but it does not itself establish chronic kidney target-organ injury.
- C.** Dyslipidemia increases cardiovascular risk and should be treated, but a lipid abnormality is not laboratory evidence of hypertensive kidney damage.
- D.** A transient exercise-related creatine kinase elevation reflects skeletal-muscle injury or exertion, not chronic renal target-organ damage from hypertension.

83. A patient repeatedly obtains high home blood-pressure readings immediately after climbing stairs. What should be done before changing treatment?

- A.** Review technique and repeat measurements after an appropriate period of seated rest because postexertional readings do not represent standardized resting home blood pressure.
- B.** Exercise blood pressure can be informative in selected evaluations, but medication titration for chronic hypertension should not be based solely on immediate postexercise peaks.
- C.** Standing measurements are useful for orthostatic assessment, but vigorous activity should not be deliberately used to establish a baseline home-treatment pressure.

D. A correctable technique error does not invalidate home monitoring as a method; retraining and validated standardized measurements are preferable to abandoning it.

84. A patient with suspected renovascular hypertension has normal kidney function and no iodinated-contrast allergy. Which noninvasive study can directly define renal-artery anatomy?

A. Duplex renal ultrasonography can screen for hemodynamically important stenosis without contrast, but image quality depends heavily on anatomy and operator expertise.

B. MR angiography can also depict renal arteries and may be selected in some patients, but the vignette specifically permits a high-resolution iodinated-contrast study.

C. CT angiography provides noninvasive cross-sectional visualization of the renal arteries and is an appropriate anatomic test when renal function and contrast tolerance permit.

D. Plasma or urine metanephrines evaluate catecholamine excess from pheochromocytoma, not the anatomic lumen of the renal arteries.

85. A patient with diabetes and antihypertensive treatment reports dizziness on standing. Which bedside assessment is preferred for suspected orthostatic hypotension?

A. A seated-to-standing comparison can identify some abnormalities, but a standardized supine baseline improves detection and interpretation of a posture-related pressure fall.

B. Measure blood pressure and pulse after several minutes of supine rest and again after standing, including readings within the first 3 minutes.

C. Exercise testing evaluates exertional hemodynamics, but orthostatic hypotension is defined by the pressure response to upright posture rather than exercise stress.

D. Waiting five minutes before the first standing measurement can miss an early diagnostic fall that occurs during the first three minutes upright.

86. Endothelin-1 can contribute to hypertension primarily through which vascular effect?

A. Activation of endothelin receptors on vascular smooth muscle produces potent vasoconstriction and can promote vascular remodeling.

B. Endothelial nitric oxide is a major vasodilator pathway, but endothelin-1 is not primarily a systemic nitric-oxide agonist.

C. Natriuretic peptides promote sodium excretion and vasodilation, whereas endothelin-1 is better known for vasoconstrictive and proliferative signaling.

D. Suppression of vascular smooth-muscle growth would be protective against remodeling, but endothelin signaling can instead promote hypertrophy and proliferation.

87. A patient asks why smoking cessation is emphasized during hypertension treatment even though nicotine-related pressure increases may be transient. What is the best answer?

A. Nicotine can acutely stimulate sympathetic activity and raise pressure, so smoking should not be viewed as physiologically neutral between office measurements.

B. Blood-pressure medication reduces one major risk pathway, but it does not erase the independent atherothrombotic, pulmonary, and malignant risks from continued smoking.

C. Smoking cessation can be accompanied by weight change and withdrawal symptoms, but those concerns should be managed without using tobacco as a cardiovascular-risk strategy.

D. Smoking substantially increases total cardiovascular risk, so cessation complements pressure control and improves prognosis beyond eliminating nicotine's acute pressor effect.

88. A patient with hypertension and urinary hesitancy feels faint when standing the morning after the first dose of doxazosin. Which class adverse effect best explains the episode?

A. ACE-inhibitor cough is mediated by bradykinin accumulation, but doxazosin is an alpha-1 blocker and does not cause that class-specific adverse effect.

B. Mineralocorticoid-receptor antagonists can cause hyperkalemia, but alpha-1 blockade does not directly impair renal potassium excretion in the same way.

C. Orthostatic hypotension and first-dose dizziness can occur because alpha-1 blockade reduces vascular resistance and impairs compensatory vasoconstriction on standing.

D. Non-dihydropyridine calcium-channel blockers can cause bradycardia through nodal suppression, but doxazosin does not directly block atrioventricular nodal calcium channels.

89. The ACCOMPLISH trial compared two fixed combinations in high-risk hypertensive patients. Which practical lesson is most consistent with its outcome results?

A. Both regimens lowered blood pressure, but outcome differences show that equal pressure reduction does not guarantee identical cardiovascular event rates for every drug combination.

B. Benazepril plus amlodipine reduced major cardiovascular events more than benazepril plus hydrochlorothiazide in the trial population.

C. The comparator used hydrochlorothiazide rather than a thiazide-like agent such as chlorthalidone, so the result should not be interpreted as proving all diuretic combinations inferior in every setting.

D. Hydrochlorothiazide remains a commonly used drug, but ACCOMPLISH did not establish it as the mandatory second agent whenever an ACE inhibitor is prescribed.

90. A patient with hypertension eats restaurant food most days. Which practical strategy is most useful for reducing dietary sodium?

A. Restaurant sauces, dressings, soups, cured meats, and prepared foods can contain substantial sodium even when extra table salt is not added.

B. Fresh fruits and vegetables are generally low in sodium and can support a DASH-style eating pattern rather than being the major sodium source to eliminate.

C. Request lower-sodium preparation, limit sauces and processed foods, choose less-salted items, and compare posted or packaged sodium information when available.

D. Cured and processed meats are typically sodium-dense, so substituting them for minimally processed foods would usually increase rather than reduce sodium intake.

91. A hemodialysis patient gains 4.5 kg between treatments and remains hypertensive before and after dialysis. What management issue should be addressed before simply adding another antihypertensive drug?

A. Additional medication may ultimately be required, but marked interdialytic weight gain raises concern that extracellular volume is a major modifiable driver of the hypertension.

B. Assess sodium and fluid balance, dietary sodium intake, dialysis adequacy, and achievement of an appropriate dry weight before escalating antihypertensive polypharmacy.

C. Longer or more frequent dialysis can improve volume and pressure control in selected patients, but the immediate principle is to evaluate whether current volume targets are appropriate.

D. Dialysis-related thirst can be worsened by sodium intake, so increasing dietary sodium is unlikely to improve either interdialytic weight gain or blood-pressure control.

92. A patient with encephalopathy from severe hypertension has a mean arterial pressure near 170 mm Hg and no aortic dissection, eclampsia, or acute stroke requiring a different target. How much should pressure generally be reduced during the first hour?

- A.** Immediate normalization to below 120/80 mm Hg risks hypoperfusion because chronic severe hypertension shifts autoregulatory relationships and requires controlled rather than precipitous reduction.
- B.** Short-acting oral drugs are difficult to titrate in an emergency and can cause abrupt pressure changes; monitored intravenous therapy is preferred.
- C.** Untreated hypertensive encephalopathy can progress, so deferring all pressure reduction for a day would leave ongoing acute brain injury unaddressed.
- D.** Lower mean arterial pressure by approximately 20%-25% during the first hour, then reduce more gradually while monitoring neurologic and other organ responses.

93. A patient with uncomplicated chronic hypertension takes short-acting nifedipine capsules as needed for isolated high home readings. What is the key concern with this strategy?

- A.** Long-acting dihydropyridine calcium-channel blockers are useful for chronic hypertension, but the short-acting formulation can produce much more abrupt and unpredictable pressure reduction.
- B.** Isolated home spikes should first be interpreted in the context of technique, symptoms, and average pressure rather than routinely treated with rapid-onset rescue dosing.
- C.** Rapid, unpredictable blood-pressure reduction with short-acting nifedipine can provoke ischemic complications; stable long-acting therapy is preferred for chronic control.
- D.** Autoregulation protects organs across a pressure range, but abrupt large reductions can exceed that protection rather than making all time above goal harmless.

94. A newly diagnosed adult with hypertension and normal serum potassium asks whether primary aldosteronism screening is reserved for resistant cases. What approach is suggested by the 2025 Endocrine Society guideline when feasible?

- A.** Hypokalemia increases suspicion for primary aldosteronism, but normal potassium is common enough that potassium alone should not determine who is screened.
- B.** Screen hypertensive adults with aldosterone and renin measurements, interpreting the aldosterone-renin relationship together with potassium, medications, and assay context.

C. An adrenal incidentaloma is one traditional indication for screening, but the updated approach is broader than waiting for a large adrenal mass to be discovered.

D. Cortisol testing evaluates hypercortisolism, a different secondary cause of hypertension, and does not replace aldosterone-renin testing for primary aldosteronism.

95. A health system finds that many patients remain above blood-pressure goal despite frequent visits. Which population-level redesign is most likely to improve control?

A. Use standardized measurement, evidence-based treatment protocols, registries or recall systems, and team-based follow-up to identify uncontrolled patients and close treatment gaps.

B. Specialist referral is valuable for complex or resistant cases, but requiring it before routine primary-care titration would delay treatment for the much larger uncomplicated population.

C. Different measurement methods can produce systematic differences, so allowing each clinic or visit to improvise technique makes longitudinal control harder to assess.

D. Registries can identify patients who are lost to follow-up or above goal; avoiding population tracking removes an important tool for reducing therapeutic inertia.

96. A patient with refractory hypertension is started on minoxidil. Which accompanying regimen is commonly required to counter predictable adverse physiologic effects?

A. A thiazide may be adequate for milder volume effects in some patients, but minoxidil can cause substantial sodium and water retention that often requires a potent diuretic.

B. A loop diuretic plus a beta blocker or other appropriate rate-control strategy is commonly used to counter fluid retention and reflex tachycardia.

C. Potassium supplementation may be needed for a patient on a kaliuretic diuretic, but it does not prevent the reflex tachycardia produced by marked arteriolar vasodilation.

D. Renin-angiotensin system blockade may remain useful when otherwise indicated; adding minoxidil does not automatically require withdrawal of an ACE inhibitor or ARB.

97. A resistant-hypertension conference reviews PATHWAY-2. Which fourth-line treatment result from that trial is most relevant to practice?

A. Volume and sodium excess often contribute to resistant hypertension, so stopping the diuretic backbone would generally work against the physiology emphasized by the trial.

- B.** Spironolactone was the most effective add-on treatment among the tested fourth-line options for appropriately selected patients with resistant hypertension.
- C.** Bisoprolol and doxazosin lowered pressure in some participants, but spironolactone produced the greatest average home systolic blood-pressure reduction.
- D.** PATHWAY-2 compared chronic oral add-on strategies and did not support short-acting nifedipine as rescue therapy for resistant hypertension.

98. A patient remains hypertensive on an ACE inhibitor, amlodipine, and chlorthalidone, yet potassium is 4.3 mmol/L. Which common endocrine cause should still be actively considered despite the normal potassium?

- A.** Cushing syndrome can cause resistant hypertension, but screening is guided by characteristic clinical features rather than being the most common explanation for normokalemic resistance.
- B.** Obstructive sleep apnea is also common in resistant hypertension and should be assessed when clinically suggested, but normal potassium does not reduce concern for aldosterone excess.
- C.** Primary aldosteronism is common in resistant hypertension and frequently occurs without hypokalemia, so normal potassium does not exclude the diagnosis.
- D.** Renal parenchymal disease is another important contributor, but its evaluation depends on kidney function, urinalysis, albuminuria, and imaging rather than serum potassium alone.

99. A kidney-transplant recipient has hypertension while taking tacrolimus and prednisone. Which management principle is preferred?

- A.** Tacrolimus exposure can contribute to hypertension and may need adjustment with the transplant team, but it should not be changed reflexively before graft function, interactions, and other causes are assessed.
- B.** Transplant renal-artery stenosis, chronic allograft dysfunction, volume excess, and medications can all present with hypertension, so an isolated pressure rise should not automatically be labeled primary.
- C.** Evaluate graft function, volume status, transplant-artery disease, medication effects, and interactions while selecting blood-pressure therapy compatible with the immunosuppressive regimen.
- D.** Dihydropyridine calcium-channel blockers are often useful in calcineurin-inhibitor-associated hypertension; their vasodilatory action does not by itself increase rejection risk.

100. After starting a beta-1 selective blocker, an adult has a lower resting heart rate and lower blood pressure. Which combined physiologic effects explain the antihypertensive action?

- A.** Beta-1 blockade reduces heart rate and contractility and suppresses renin release from juxtaglomerular cells, lowering cardiac output and RAAS activity.
- B.** Epithelial sodium-channel blockade is the mechanism of amiloride and triamterene rather than a defining renal action of beta blockers.
- C.** Some beta blockers have additional vasodilating properties, but direct nitric-oxide donation is not the class-defining mechanism shared by all beta-1 selective agents.
- D.** Beta-1 stimulation would increase renin secretion and cardiac activity, so direct receptor activation would raise rather than explain the observed fall in pulse and pressure.

101. A 79-year-old has blood pressure 172/66 mm Hg and no evidence of significant aortic regurgitation. Which change in the arterial tree most strongly accounts for the disproportionately high systolic pressure?

- A.** Increased peripheral resistance can raise both systolic and diastolic pressure, but it does not specifically explain the age-related combination of high systolic and relatively low diastolic pressure.
- B.** Reduced compliance of the aorta and other large elastic arteries increases systolic pressure and pulse-wave velocity and can contribute to lower diastolic pressure.
- C.** Increased aortic compliance buffers ventricular ejection and tends to narrow rather than widen pulse pressure for a given stroke volume.
- D.** Changes in venous capacitance influence preload, but pulse pressure is determined more directly by stroke volume and large-artery compliance than by venous storage alone.

102. A patient takes antihypertensive medications every other day because the copayments are unaffordable. What is the preferred response?

- A.** Increasing each prescribed dose may appear to compensate for skipped days, but irregular exposure can still produce poor control and adverse effects without solving the access barrier.
- B.** Medication access is a clinically important adherence determinant, so separating financial barriers from hypertension care would miss a reversible cause of treatment failure.
- C.** Identify lower-cost effective alternatives, simplify the regimen, use assistance or coverage resources when available, and preserve evidence-based classes rather than simply escalating doses.

D. Apparent resistant hypertension should not be diagnosed from high readings when the prescribed regimen is not being taken consistently because of a known affordability barrier.

103. Which intervention best uses home blood-pressure monitoring to improve hypertension control rather than merely collecting readings?

A. Validated home measurements are valuable, whereas increasing the number of readings from an unvalidated cuffless wearable does not correct uncertain measurement accuracy.

B. The highest weekly value may reflect stress, technique, or random variability; treatment is better guided by standardized averages and the clinical context.

C. Symptom-triggered readings can investigate episodic complaints, but routine scheduled measurements provide a less biased picture of usual pressure control.

D. Combine validated home readings with structured feedback, adherence support, and timely treatment adjustment by clinicians or a team-based care program.

104. A sedentary adult with stage 1 hypertension wants to become more physically active. Which behavioral strategy is most likely to improve long-term exercise adherence?

A. Self-monitoring through step counts, activity logs, or calendars can reinforce progress, so avoiding feedback removes a useful behavior-change tool.

B. A maximal unsustainable program may produce rapid fatigue or injury; gradual progression is more likely to support long-term adherence and self-efficacy.

C. Set specific achievable activity goals, track progress, anticipate barriers, and integrate regular movement into the patient's daily routine.

D. Most patients with uncomplicated stage 1 hypertension can begin appropriate physical activity while treatment proceeds, rather than waiting for complete medication-based normalization.

105. A clinician plans a short-interval follow-up after initiating lisinopril plus a thiazide-type diuretic. What is the main reason not to postpone reassessment for several months?

A. Early follow-up allows reassessment of blood pressure, adherence, symptoms, electrolytes, and kidney function so treatment can be adjusted safely and effectively.

B. ACE inhibitors and diuretics can alter creatinine and electrolytes relatively soon after initiation, so waiting several months can miss clinically important early changes.

C. Symptoms alone are not a reliable screen for hyperkalemia, hyponatremia, or kidney-function change, making laboratory follow-up important even when the patient feels well.

D. Antihypertensive effects begin before several months have passed, so delaying all reassessment until a presumed final chronic effect is reached can prolong under- or overtreatment.

106. A patient with carotid atherosclerosis has hypertension but no acute stroke or cerebral hypoperfusion syndrome. What is the central long-term treatment principle?

A. Severe bilateral carotid disease may require individualized assessment when symptoms suggest pressure-dependent cerebral perfusion, but routine chronic permissive hypertension increases vascular risk.

B. Control blood pressure as part of comprehensive vascular-risk reduction rather than intentionally maintaining chronic hypertension to augment cerebral perfusion.

C. Permissive hypertension is sometimes used temporarily in selected acute ischemic-stroke settings, but that acute strategy should not be carried forward indefinitely in a stable outpatient.

D. Lipid-lowering, smoking cessation, antiplatelet therapy when indicated, and pressure control address complementary risks and should not be unnecessarily sequenced one at a time.

107. A patient with heart failure with preserved ejection fraction has persistent hypertension. Why is good blood-pressure control an important part of management?

A. Preserved ejection fraction does not eliminate the adverse effects of hypertension on filling pressures, ventricular hypertrophy, arterial stiffness, and future cardiovascular events.

B. Reducing excess afterload and limiting further ventricular and vascular remodeling are important when treating hypertension, a major comorbidity and driver of HFpEF.

C. Heart-rate control may help selected patients with atrial fibrillation or ischemia, but pressure and afterload remain relevant determinants of HFpEF symptoms and risk.

D. Higher afterload increases left-ventricular workload and can worsen diastolic filling, so chronic hypertension is not protective simply because systolic ejection fraction is preserved.

108. Norepinephrine released from renal sympathetic nerves acts on juxtaglomerular cells and increases renin secretion. Which adrenergic receptor mediates this effect most directly?

- A.** Beta-1 adrenergic receptors on juxtaglomerular cells mediate sympathetic stimulation of renin release and thereby augment renin-angiotensin system activity.
- B.** Alpha-1 receptors contribute to renal vasoconstriction, but they are not the principal receptor directly stimulating renin secretion from juxtaglomerular cells.
- C.** Beta-2 receptors have vascular and metabolic effects in other tissues, but juxtaglomerular renin release is linked primarily to beta-1 signaling.
- D.** Muscarinic receptors participate in parasympathetic signaling, but they do not mediate the characteristic sympathetic stimulation of renin release.

109. A patient misses evening doses because six separate antihypertensive pills are difficult to manage. Which regimen change is most likely to improve adherence while preserving necessary drug classes?

- A.** Short-acting formulations increase dosing frequency and can worsen missed-dose problems, so they are generally less attractive when regimen complexity is the barrier.
- B.** Separating each medication into a different dosing time increases cognitive burden and is unlikely to solve adherence problems caused by pill and schedule complexity.
- C.** Use once-daily agents and single-pill combinations when appropriate, while preserving the needed therapeutic classes and confirming affordability and tolerability.
- D.** Nonjudgmental adherence discussion is essential because recognizing the actual barrier allows simplification rather than assuming pharmacologic resistance.

110. A patient taking aliskiren for hypertension becomes pregnant. Which management principle is appropriate?

- A.** Pregnancy alters renin physiology, but increasing a direct renin inhibitor would intensify fetal renin-angiotensin system exposure rather than make treatment safer.
- B.** Adding an ARB would compound renin-angiotensin system blockade and fetal renal risk rather than protect fetal perfusion.
- C.** ACE inhibitors, ARBs, and direct renin inhibitors all interfere with the renin-angiotensin system, so fetal toxicity concern is not limited to ACE inhibitors and ARBs.
- D.** Stop aliskiren and transition to a pregnancy-compatible antihypertensive regimen because direct renin-angiotensin system inhibition is contraindicated during pregnancy.

111. A patient with chronic hypertension taking lisinopril has a positive pregnancy test at 6 weeks' gestation. What medication change should be made now?

- A.** Continuing effective pressure control is important in pregnancy, but it should be achieved with pregnancy-compatible agents rather than by stopping all therapy automatically.
- B.** ACE-inhibitor fetotoxicity is especially established later in pregnancy, but ACE inhibitors are avoided throughout pregnancy rather than intentionally continued after conception.
- C.** Adding an ARB would create dual fetal renin-angiotensin system blockade and increase rather than reduce fetal renal risk.
- D.** Discontinue lisinopril promptly and transition to a pregnancy-compatible antihypertensive regimen while continuing appropriate maternal and fetal monitoring.

112. A patient with hypertension has recurrent nephrolithiasis, constipation, persistent hypercalcemia, and elevated parathyroid hormone. Which endocrine disorder may be contributing?

- A.** Primary hyperparathyroidism causes PTH-dependent hypercalcemia and can present with nephrolithiasis, gastrointestinal symptoms, and an association with hypertension.
- B.** Primary adrenal insufficiency tends to cause hypotension, hyponatremia, and hyperkalemia rather than PTH-dependent hypercalcemia with kidney stones.
- C.** Hyperthyroidism can increase systolic pressure and cause hypercalcemia occasionally, but an elevated PTH in true hypercalcemia points away from a thyroid-driven process.
- D.** Vitamin D intoxication can cause hypercalcemia and stones, but it suppresses PTH rather than producing an inappropriately elevated parathyroid hormone concentration.

113. A patient with disabling acute ischemic stroke is otherwise a candidate for IV tenecteplase, but repeated pressure is 188/112 mm Hg. What should be done before thrombolytic treatment?

- A.** Mechanical thrombectomy may be indicated independently when a treatable large-vessel occlusion is present, but elevated pressure alone does not mean intravenous thrombolysis must be abandoned permanently.
- B.** Carefully lower blood pressure to below 185/110 mm Hg before giving alteplase or tenecteplase, then maintain the recommended post-thrombolysis pressure limits.
- C.** Lowering systolic pressure below 140 mm Hg is not required for thrombolysis eligibility and may create unnecessary delay or excessive reduction in an acutely ischemic brain.

D. Giving thrombolysis before reducing a pressure above the eligibility threshold increases hemorrhagic risk and does not follow standard reperfusion-treatment criteria.

114. A patient with apparent treatment failure takes a chronic NSAID, a prescription stimulant, and high-sodium effervescent supplements. What is the preferred next step before simply adding another antihypertensive drug?

A. Another antihypertensive may eventually be needed, but escalating therapy without reviewing several known pressor exposures can increase polypharmacy while leaving reversible causes in place.

B. Medication count alone does not establish true resistant hypertension when NSAIDs, stimulants, excess sodium, and adherence have not been systematically addressed.

C. A mineralocorticoid-receptor antagonist is effective fourth-line therapy in resistant hypertension, but it does not eliminate the need to remove or reduce avoidable pressor substances.

D. Review the indications and alternatives for pressor medications and supplements, reduce excess sodium exposure, and reassess pressure before labeling the regimen a failure.

115. For an otherwise stable nonpregnant adult receiving treatment for hypertension, which general on-treatment blood-pressure goal is recommended by the 2025 ACC/AHA guideline for most patients?

A. A goal below 140/90 mm Hg may still be individualized in selected frail or special circumstances, but it is not the general target for most treated adults in the updated guideline.

B. A universal target below 110/70 mm Hg would increase adverse-effect risk in many patients and is not recommended regardless of symptoms or comorbidity.

C. A treatment goal below 130/80 mm Hg applies to most adults, with individualized exceptions for pregnancy, institutionalized patients, limited life expectancy, or intolerance.

D. Numeric goals remain useful for guiding treatment; abandoning targets entirely would make it difficult to judge whether antihypertensive therapy is achieving the intended pressure reduction.

116. Several relatives have early-onset hypertension with suppressed renin, elevated aldosterone, and aldosterone production that falls with glucocorticoid administration. What mechanism explains this inherited disorder?

A. Liddle syndrome also causes early low-renin hypertension, but it suppresses aldosterone because the primary defect is gain-of-function epithelial sodium-channel activity.

B. A chimeric gene places aldosterone synthase activity under ACTH-responsive regulation, producing glucocorticoid-remediable familial hyperaldosteronism.

C. Apparent mineralocorticoid excess from 11-beta-HSD2 deficiency causes cortisol-mediated mineralocorticoid receptor activation with low renin and low aldosterone rather than high aldosterone.

D. A catecholamine-secreting tumor can cause familial hypertension in inherited syndromes, but it does not produce glucocorticoid-suppressible aldosterone excess with suppressed renin.

117. At 18 weeks' gestation, a patient with chronic hypertension has repeated pressures around 144/92 mm Hg while off medication. According to the CHAP-informed ACOG approach, at what level should treatment be initiated or titrated?

A. Waiting until severe-range pressure develops exposes the patient to avoidable risk because chronic hypertension treatment is now initiated or titrated before 160/110 mm Hg is reached.

B. Initiate or titrate antihypertensive therapy at approximately 140/90 mm Hg rather than withholding treatment until severe hypertension develops.

C. Proteinuria is used to diagnose superimposed preeclampsia, but chronic hypertension alone still warrants treatment at the recommended pressure threshold.

D. Aggressively normalizing pressure below 100/60 mm Hg is not the goal because excessive maternal hypotension can compromise maternal tolerance and uteroplacental perfusion.

118. A patient with hypertension, eGFR 24 mL/min/1.73 m², and marked edema needs effective immediate volume management. Which diuretic is usually most useful as the primary agent?

A. A mineralocorticoid-receptor antagonist can aid resistant hypertension or heart failure, but advanced CKD increases hyperkalemia risk and its natriuretic effect is insufficient for major edema alone.

B. A thiazide-like diuretic can still add antihypertensive effect in advanced CKD and may be combined with a loop, but severe congestion usually requires a loop as the main decongestive agent.

C. A loop diuretic titrated to produce adequate natriuresis is usually the primary diuretic for substantial volume overload at this level of kidney dysfunction.

D. Acetazolamide has selected adjunctive roles in decongestion and acid-base management, but it is not the universal first-line chronic diuretic for severe CKD-associated edema.

119. A patient begins hydrochlorothiazide and develops increased sodium delivery beyond the distal convoluted tubule. Which transporter has been inhibited to produce the drug's primary natriuretic effect?

- A.** The epithelial sodium channel in the collecting duct is blocked by amiloride and triamterene rather than by thiazide diuretics.
- B.** The Na-K-2Cl cotransporter in the thick ascending limb is the molecular target of loop diuretics such as furosemide.
- C.** The sodium-glucose cotransporter in the proximal tubule is inhibited by SGLT2 inhibitors used for diabetes, kidney disease, and heart failure.
- D.** The Na-Cl cotransporter in the distal convoluted tubule is inhibited by hydrochlorothiazide and other thiazide or thiazide-like diuretics.

120. How can repeated nocturnal hypoxemia from obstructive sleep apnea contribute to persistent daytime hypertension?

- A.** Intermittent hypoxia and arousal activate sympathetic pathways and can sustain vasoconstrictor and neurohumoral activity beyond the sleep period.
- B.** Negative intrathoracic pressure increases cardiac loading stresses during obstructive events, but it does not produce a protective chronic fall in systemic blood pressure.
- C.** Obstructive sleep apnea can promote sodium retention and RAAS activity in some patients, so the mechanism is not isolated renal salt wasting.
- D.** Sleep fragmentation and hypoxia increase rather than permanently suppress catecholamine signaling, helping explain the association with resistant and nocturnal hypertension.

121. A patient with hypertension and symptomatic heart failure with reduced ejection fraction remains on an incomplete regimen. Which principle best integrates heart-failure and blood-pressure treatment?

- A.** Short-acting vasodilators can reduce pressure rapidly, but replacing outcome-improving heart-failure therapies with episodic antihypertensive dosing would sacrifice proven survival benefit.
- B.** Prioritize guideline-directed HFrEF therapies with outcome benefit, using their blood-pressure effects to control hypertension while monitoring renal function, potassium, and tolerance.
- C.** Renin-angiotensin system modulation lowers afterload and improves outcomes in appropriate HFrEF patients, so stable systolic dysfunction is not a reason to avoid it automatically.

D. Treating only the pressure number ignores the prognostic value of heart-failure drug classes that improve survival, hospitalization risk, remodeling, and symptoms.

122. Aldosterone increases distal sodium reabsorption primarily by increasing the activity and expression of which transport pathway in principal cells?

A. Epithelial sodium channels in collecting-duct principal cells are stimulated by mineralocorticoid receptor signaling and couple sodium reabsorption to potassium secretion.

B. The Na-K-2Cl cotransporter is located in the thick ascending limb and is the principal target of loop diuretics rather than aldosterone-regulated principal-cell sodium entry.

C. The Na-Cl cotransporter in the distal convoluted tubule is the thiazide-sensitive pathway and is distinct from aldosterone's major principal-cell target.

D. SGLT2 in the proximal tubule reabsorbs sodium with glucose, but it is not the principal distal sodium channel regulated by aldosterone in collecting-duct principal cells.

123. A stable nonpregnant adult has repeated blood pressure above 180/120 mm Hg but careful evaluation finds no acute neurologic, cardiac, renal, retinal, or aortic target-organ injury. Which general approach is recommended?

A. Intensify oral treatment promptly, address adherence and reversible causes, and arrange close outpatient follow-up rather than automatically using emergency intravenous therapy.

B. Immediate normalization to very low pressure can exceed autoregulatory capacity and is not indicated solely because the numeric reading is severe in the absence of acute organ injury.

C. Severe asymptomatic hypertension still carries long-term cardiovascular risk, so lack of acute injury is not a reason to leave persistent pressure untreated.

D. Intravenous therapy is reserved for hypertensive emergencies with acute target-organ injury or selected disease-specific situations, not every value above 180/120 mm Hg.

124. A patient eats most meals from restaurants and packaged foods and has uncontrolled hypertension. Which nutritional change is most likely to reduce sodium exposure?

A. Increasing dietary potassium can help lower pressure when kidney function and medications permit, but potassium advice does not substitute for reducing a high sodium load.

- B.** Suppressing renin by eating more sodium does not improve cardiovascular risk; greater sodium intake generally increases pressure in salt-sensitive patients.
- C.** Replace highly processed foods with lower-sodium choices, request less salt and sauce when dining out, and compare sodium content on labels or menus.
- D.** Trans fats worsen cardiovascular risk and have no role in improving arterial compliance or offsetting the blood-pressure effects of excess sodium.

125. A patient uses potassium-chloride salt substitute to reduce sodium intake. Which clinical situation warrants the greatest caution because of hyperkalemia risk?

- A.** Advanced chronic kidney disease combined with an ACE inhibitor and spironolactone markedly reduces potassium excretory reserve and increases the risk from potassium-based salt substitutes.
- B.** Normal kidney function without potassium-raising medication provides greater excretory reserve, although excessive supplement use still merits counseling and monitoring when clinically indicated.
- C.** A statin for isolated hyperlipidemia does not usually impair potassium excretion, so it creates far less concern than advanced kidney disease with dual potassium-raising therapy.
- D.** Seasonal allergic rhinitis treated with a standard antihistamine does not materially alter renal potassium handling and is not a major contraindication to potassium-based salt substitution.

126. Adrenal venous sampling in a patient with primary aldosteronism shows bilateral secretion, and the patient will not undergo adrenal surgery. Which drug class provides the most disease-specific chronic therapy?

- A.** A mineralocorticoid-receptor antagonist such as spironolactone or eplerenone blocks aldosterone action and is disease-specific therapy for bilateral primary aldosteronism.
- B.** A thiazide-like diuretic can lower blood pressure and sodium balance, but it does not directly block the inappropriate mineralocorticoid receptor activation driving the disorder.
- C.** A calcium-channel blocker can provide additional pressure control, but it does not directly antagonize aldosterone signaling or correct the disease-specific mechanism.
- D.** A beta blocker may be useful for another cardiovascular indication, but suppressing heart rate alone is not disease-specific treatment for autonomous aldosterone excess.

127. A patient with sudden-onset hypertensive pulmonary edema arrives severely dyspneic with pressure 224/128 mm Hg and diffuse pulmonary congestion. Which rapidly titratable IV approach best addresses the acute afterload problem?

- A.** A large isotonic fluid bolus can help true preload depletion, but pulmonary edema with severe hypertension usually reflects excessive filling pressure and afterload rather than volume deficiency.
- B.** Short-acting oral nifedipine can lower pressure abruptly and unpredictably, making it unsuitable for titratable treatment of a hypertensive emergency.
- C.** Use rapidly titratable intravenous vasodilation, often with high-dose nitroglycerin or another appropriate agent, together with treatment of pulmonary congestion and respiratory distress.
- D.** Beta blockade is essential in aortic dissection, but aggressive isolated beta blockade is not the primary strategy for acute hypertensive pulmonary edema with marked congestion.

128. A patient with hypertension has resting heart rate 48/min and second-degree atrioventricular block. Which antihypertensive drug should generally be avoided because it can worsen nodal conduction?

- A.** Amlodipine lowers pressure mainly through peripheral arteriolar dilation and usually has little direct effect on atrioventricular nodal conduction.
- B.** Losartan blocks angiotensin II receptors and does not characteristically slow sinoatrial or atrioventricular nodal conduction.
- C.** Chlorthalidone lowers pressure through renal sodium handling and does not directly block cardiac L-type calcium channels in the conduction system.
- D.** Verapamil is a non-dihydropyridine calcium-channel blocker that slows AV nodal conduction and can worsen bradycardia or heart block.

129. Why can an ACE inhibitor cause a persistent dry cough in a susceptible patient?

- A.** ACE inhibitors reduce formation of angiotensin II rather than increasing it, so excessive pulmonary angiotensin II production is not the mechanism of the cough.
- B.** Beta-2 receptor activation causes bronchodilation, but ACE inhibitors do not cause cough by directly stimulating pulmonary beta-adrenergic receptors.
- C.** ACE inhibition decreases degradation of bradykinin and related peptides, allowing accumulation that can trigger cough in susceptible patients.

D. Histamine participates in allergic cough and bronchospasm, but inhibition of histamine metabolism is not the primary pharmacologic action responsible for ACE-inhibitor cough.

130. A patient has a substantially larger blood-pressure increase than peers after a high-sodium diet. Which phenotype does this illustrate?

- A.** White-coat hypertension is defined by an office-out-of-office discrepancy and is not specifically a dietary sodium-response phenotype.
- B.** Pseudohypertension results from noncompressible arteries producing spuriously high cuff readings, a mechanism different from a reproducible sodium-induced pressure rise.
- C.** Isolated systolic hypertension describes the pressure pattern, often related to arterial stiffness, but it does not identify exaggerated physiologic responsiveness to sodium intake.
- D.** Salt-sensitive blood pressure reflects a greater pressure response to sodium loading because renal and vascular mechanisms require a larger pressure change to maintain sodium balance.

131. A patient with uncomplicated hypertension is taking an ACE inhibitor, an ARB, and aliskiren simultaneously. What is the preferred interpretation?

- A.** Greater RAAS suppression can lower proteinuria in selected contexts, but surrogate improvement does not outweigh the renal and potassium risks of routine multiple-pathway blockade.
- B.** When blood pressure remains above goal on one RAAS blocker, adding a different first-line class is generally preferred to stacking multiple renin-angiotensin inhibitors.
- C.** The regimen represents unnecessary multiple renin-angiotensin system blockade and increases the risk of hyperkalemia, hypotension, and acute kidney injury.
- D.** Normal baseline potassium and eGFR reduce immediate risk but do not make triple RAAS blockade an appropriate routine strategy for uncomplicated hypertension.

132. An adult has an average standardized blood pressure of 126/76 mm Hg. How is this classified under current ACC/AHA categories?

- A.** Elevated blood pressure applies because systolic pressure is 120-129 mm Hg while diastolic pressure remains below 80 mm Hg.

B. Normal blood pressure requires systolic pressure below 120 mm Hg and diastolic pressure below 80 mm Hg, so 126/76 is not in the normal category.

C. Stage 1 hypertension begins at systolic pressure 130-139 mm Hg or diastolic pressure 80-89 mm Hg, thresholds not reached by this reading.

D. Isolated systolic hypertension is generally used for a hypertensive systolic value with a nonhypertensive diastolic value, not for systolic pressure of 126 mm Hg.

133. A patient has cardiac output of 5 L/min. If systemic vascular resistance rises substantially while cardiac output remains unchanged, what happens to mean arterial pressure?

A. Central venous pressure contributes only a small downstream pressure term in the systemic circulation and is not the principal determinant of mean arterial pressure in this scenario.

B. Pulse pressure depends strongly on stroke volume and arterial compliance, so it does not have to narrow to zero simply because systemic vascular resistance rises.

C. For a stable cardiac output, greater systemic vascular resistance requires a larger pressure gradient to drive flow and therefore does not make mean arterial pressure fall.

D. Mean arterial pressure increases because, to a useful approximation, it is determined by cardiac output multiplied by systemic vascular resistance plus a small venous-pressure term.

134. A patient with resistant hypertension develops marked hypokalemia while receiving combined loop and thiazide-like diuretics. What is the principal treatment concern?

A. Primary aldosteronism should still be considered when the clinical setting suggests it, but two kaliuretic diuretics provide an immediate medication-based explanation for substantial potassium loss.

B. Sequential nephron blockade can be very effective for sodium and volume control, but intensifying it further before correcting electrolyte depletion can increase arrhythmia and kidney-injury risk.

C. Mineralocorticoid-receptor antagonists tend to raise potassium, whereas loop and thiazide-type diuretics increase distal sodium delivery and commonly lower potassium.

D. Sequential nephron blockade increases the risks of hypokalemia, hyponatremia, volume contraction, and kidney-function change and therefore requires close laboratory and volume monitoring.

135. Ambulatory monitoring shows that a patient's average sleeping systolic blood pressure falls by only 3% compared with the daytime average. Which term best describes this pattern?

- A.** Nondipping describes a nocturnal pressure fall of less than the expected approximately 10% and is associated with sleep apnea, CKD, autonomic dysfunction, and higher cardiovascular risk.
- B.** White-coat hypertension is an office-out-of-office discrepancy and cannot be defined solely from the magnitude of the nocturnal pressure decline.
- C.** Orthostatic hypertension refers to an excessive pressure rise on standing and is not the term for a small fall during sleep while recumbent.
- D.** Normal dipping usually involves a meaningful nocturnal decline; a 3% reduction is substantially less than the expected physiologic decrease.

136. Repeated careful measurements show a persistent 24-mm Hg systolic blood-pressure difference between a patient's arms. What is the best interpretation and follow-up practice?

- A.** A reproducible large inter-arm difference should prompt consideration of upper-extremity or aortic vascular disease, and future measurements are generally taken in the higher-pressure arm.
- B.** Using the lower-pressure arm can underestimate systemic pressure and delay treatment, so it is not the preferred arm merely because it produces a more reassuring value.
- C.** Small inter-arm differences are common, but a persistent difference of this magnitude exceeds ordinary variation and should not be dismissed after repeated standardized measurements.
- D.** Averaging both arms can obscure a clinically important vascular discrepancy; after evaluation, a consistent arm—usually the higher-pressure arm—is used for longitudinal management.

137. A 57-year-old with confirmed primary aldosteronism has a unilateral adrenal nodule on CT and wants potentially curative adrenalectomy. What is generally the preferred next step before surgery?

- A.** CT identifies adrenal anatomy but cannot reliably prove which gland is secreting excess aldosterone because nonfunctioning nodules become common with age.
- B.** Adrenal venous sampling is generally used to establish unilateral aldosterone production before adrenalectomy unless the patient meets a well-defined guideline exception.
- C.** Dexamethasone suppression testing is used in evaluation of cortisol excess and selected familial aldosteronism questions, but it is not the standard method for lateralizing ordinary primary aldosteronism.

D. Renal-vein renin sampling can assess renovascular physiology in uncommon situations, but it does not directly localize autonomous aldosterone secretion to one adrenal gland.

138. Endothelial nitric oxide lowers vascular smooth-muscle tone primarily through which intracellular signaling pathway?

A. Angiotensin II type 1 receptor signaling activates phospholipase C and raises intracellular calcium, producing vasoconstriction rather than nitric-oxide-mediated relaxation.

B. Nitric oxide activates soluble guanylate cyclase, increasing cyclic GMP and promoting smooth-muscle relaxation through downstream protein kinase signaling.

C. Persistent membrane depolarization and calcium entry promote contraction, whereas nitric oxide generally shifts smooth muscle toward reduced intracellular contractile signaling.

D. Endothelin and alpha-1 adrenergic pathways can activate phospholipase C and intracellular calcium, but that is not the primary nitric-oxide second-messenger mechanism.

139. Renal-artery imaging obtained for another reason shows a substantial atherosclerotic stenosis in a patient whose kidney function is stable and pressure is controlled on medication. What management approach is usually favored initially?

A. Anatomic stenosis alone does not guarantee benefit from stenting; randomized data support medical management for many stable patients without high-risk clinical syndromes.

B. ACE inhibitors or ARBs can often be used carefully in unilateral disease with kidney-function and potassium monitoring, so the diagnosis does not require automatic withdrawal of RAAS blockade.

C. Optimize antihypertensive therapy and aggressive cardiovascular risk reduction rather than routinely revascularizing every stable atherosclerotic renal-artery lesion.

D. Surgical revascularization is reserved for selected complex cases and does not replace evidence-based medical therapy in an otherwise stable patient with controlled pressure.

140. A patient with CKD takes an ACE inhibitor and diuretic, develops gastroenteritis with volume depletion, then begins high-dose NSAIDs and develops acute kidney injury. Which interaction is most concerning?

- A.** Diuresis and volume depletion lower renal perfusion, ACE inhibition reduces efferent arteriolar constriction, and NSAIDs reduce prostaglandin-mediated afferent dilation, together markedly lowering glomerular filtration.
- B.** ACE inhibitors dilate rather than constrict the efferent arteriole, so they can impair maintenance of intraglomerular pressure during low-perfusion states.
- C.** NSAIDs inhibit renal prostaglandin synthesis and can constrict the afferent arteriole, particularly when effective circulating volume is low.
- D.** Diuretics can worsen volume depletion during gastroenteritis and therefore do not guarantee renal perfusion or protect against this hemodynamic acute kidney injury.

141. A patient with a stable thoracic aortic aneurysm has persistent hypertension. Which pharmacologic concept is most important for long-term management?

- A.** Higher systemic pressure increases aortic wall stress, so intentionally preserving hypertension is not a strategy for preventing aneurysm enlargement or dissection.
- B.** Surgical thresholds depend on aneurysm size, growth, cause, and anatomy, but medical blood-pressure treatment should continue while surveillance is performed.
- C.** Maintain consistent blood-pressure control with appropriate long-acting agents to reduce aortic wall stress and manage overall cardiovascular risk.
- D.** Stimulants raise heart rate, contractility, and pressure and therefore increase rather than reduce aortic impulse and wall stress.

142. A patient receiving spironolactone for primary aldosteronism develops painful gynecomastia. Which alternative mineralocorticoid-receptor antagonist has greater receptor selectivity and fewer antiandrogen effects?

- A.** Amiloride blocks epithelial sodium channels and can be an alternative in selected patients, but it is not a more selective mineralocorticoid-receptor antagonist.
- B.** Doxazosin blocks alpha-1 receptors and can lower pressure, but it does not directly antagonize aldosterone at the mineralocorticoid receptor.
- C.** Chlorthalidone improves sodium balance and pressure but does not provide disease-specific mineralocorticoid-receptor blockade or avoid aldosterone signaling.
- D.** Eplerenone is a more selective mineralocorticoid-receptor antagonist and generally causes fewer antiandrogen adverse effects than spironolactone.

143. A patient's previously controlled home pressures rise after weeks of frequent pseudoephedrine use for nasal congestion. How should this over-the-counter exposure be handled in the hypertension evaluation?

- A.** Decongestants can provide short-term nasal symptom relief, but chronic sympathomimetic exposure can raise pressure and heart rate rather than reliably producing protective receptor downregulation.
- B.** Nonprescription agents are an important part of medication reconciliation because decongestants, NSAIDs, stimulants, and supplements can materially affect blood pressure.
- C.** Recognize pseudoephedrine as a potentially reversible pressor contributor and reduce, discontinue, or substitute it when clinically feasible before escalating antihypertensive therapy.
- D.** Primary aldosteronism remains a possible secondary cause in resistant hypertension, but a medication history cannot establish that endocrine diagnosis without appropriate biochemical testing.

144. A patient with an adrenal mass also reports episodic pounding headaches, palpitations, sweating, and abrupt pressure surges. Which biochemical study is the preferred initial test for catecholamine excess?

- A.** Plasma free metanephrines or urinary fractionated metanephrines, collected and interpreted under appropriate conditions, provide sensitive initial biochemical testing.
- B.** Random catecholamine concentrations can fluctuate substantially between spells, making metanephrine measurement more reliable for initial biochemical evaluation.
- C.** Aldosterone and renin testing evaluates primary aldosteronism, another endocrine cause of hypertension, but it does not screen for catecholamine-secreting tumors.
- D.** Twenty-four-hour urinary sodium can help assess sodium intake and renal handling, but it is not a biochemical tumor marker for pheochromocytoma or paraganglioma.

145. A patient's pressure and sympathetic symptoms improve after clonidine is started. Which central receptor action accounts for its antihypertensive effect?

- A.** Peripheral alpha-1 blockade lowers vascular resistance with drugs such as doxazosin, but clonidine's dominant antihypertensive action is central rather than peripheral alpha-1 antagonism.
- B.** ACE inhibitors lower angiotensin II and increase bradykinin, but clonidine does not inhibit angiotensin-converting enzyme.

C. Beta blockers can slow heart rate and suppress renin, but clonidine does not act through irreversible beta-1 receptor blockade.

D. Central alpha-2 adrenergic agonism reduces sympathetic outflow from the central nervous system, lowering vascular tone and blood pressure.

146. A patient's LDL cholesterol remains far above goal despite well-controlled blood pressure. Why is lipid treatment still necessary?

A. Severe pressure increases acute vascular risk, but dyslipidemia contributes to atherosclerosis across a wide range of blood-pressure values and should not be treated only during hypertensive crises.

B. Blood pressure and atherogenic lipids are independent, additive cardiovascular risk factors, so each should be treated according to its own indications and overall risk.

C. Achieving a blood-pressure goal reduces risk substantially but does not remove the arterial effects of persistently high LDL cholesterol.

D. Statins lower atherosclerotic risk rather than functioning primarily as antihypertensive drugs, so they complement rather than replace indicated pressure-lowering medication.

147. A patient with albuminuric chronic kidney disease starts an ACE inhibitor for hypertension. Which laboratory monitoring is most important after initiation or dose escalation?

A. Thyroid function testing is appropriate when thyroid disease is suspected, but ACE inhibitors do not routinely cause hypothyroidism requiring post-dose TSH surveillance.

B. Pancreatic enzymes are measured when pancreatitis is suspected, but routine amylase and lipase testing does not monitor the principal renal effects of ACE inhibition.

C. Check serum creatinine and potassium because RAAS blockade can produce an expected creatinine rise and can cause clinically important hyperkalemia, especially in CKD.

D. Creatine kinase monitoring is relevant for selected muscle-toxic therapies such as statins, but it is not the routine safety laboratory for ACE-inhibitor titration.

148. A patient with longstanding hypertension has increased QRS voltage and a lateral repolarization "strain" pattern on ECG. Which target-organ effect should be suspected?

- A.** Right ventricular hypertrophy can alter right-precordial forces, but systemic hypertension more commonly produces increased left-ventricular afterload and left-sided hypertrophy.
- B.** Acute pericarditis causes diffuse ST-segment and PR-segment changes rather than the chronic high-voltage and strain pattern described.
- C.** Chronic pressure overload causes myocardial hypertrophy rather than loss of ventricular muscle mass, making atrophy an implausible explanation for increased voltage.
- D.** Left ventricular hypertrophy from chronic systemic pressure overload can produce increased QRS voltage and secondary ST-T repolarization abnormalities.

149. Standardized clinic and ambulatory readings remain above goal despite verified adherence to maximally tolerated lisinopril, amlodipine, and chlorthalidone. What diagnostic label now applies?

- A.** Masked hypertension refers to normal office readings with elevated out-of-office pressure, whereas this patient is uncontrolled in both settings.
- B.** White-coat hypertension requires elevated office pressure with normal out-of-office pressure, which has been excluded by the home or ambulatory findings.
- C.** True resistant hypertension is present when pressure remains above goal on an appropriate three-drug regimen including a diuretic after adherence and out-of-office elevation are confirmed.
- D.** Refractory hypertension is generally reserved for failure despite a more extensive optimized multidrug regimen and is not synonymous with any three-drug treatment failure.

150. After a year-long obesity treatment program, a patient loses 12% of baseline body weight while sodium intake and antihypertensive adherence remain stable. What change in blood pressure is most likely?

- A.** Blood pressure commonly falls with clinically meaningful weight loss, which can make treatment goals easier to achieve and may reduce medication requirements in some patients.
- B.** Medication doses should be reassessed rather than automatically doubled because successful weight loss can lower pressure and create a risk of overtreatment.
- C.** Weight loss improves glucose metabolism, sleep apnea, and other risk factors and also has a clinically meaningful average effect on blood pressure.
- D.** Obesity promotes sympathetic, renal, and metabolic mechanisms that raise pressure, so sustained weight reduction generally moves physiology in the opposite direction rather than predictably increasing BP.

151. A patient with previously controlled hypertension begins taking high-dose ibuprofen every day for arthritis and the blood pressure rises. Which mechanism best explains the change?

- A.** Pain itself can increase sympathetic tone and raise pressure, but the new daily NSAID also has a direct renal mechanism that can worsen hypertension even when pain improves.
- B.** Some antihypertensive drugs promote natriuresis, whereas NSAID inhibition of renal prostaglandins tends to favor sodium retention rather than epithelial sodium-channel blockade and salt wasting.
- C.** NSAIDs can reduce renal prostaglandin activity, promote sodium and water retention, and blunt the pressure-lowering effects of diuretics and several other antihypertensive agents.
- D.** Reduced sympathetic tone would tend to lower rather than raise pressure, and irreversible autonomic suppression is not the mechanism of NSAID-associated hypertension.

152. A 61-year-old with type 2 diabetes has a confirmed average pressure of 133/82 mm Hg despite lifestyle measures. Which treatment approach best reflects the patient's cardiovascular-risk category?

- A.** Lifestyle treatment remains important, but diabetes confers higher cardiovascular risk and is not a reason to wait until systolic pressure reaches 160 mm Hg before using medication.
- B.** ACE inhibitors and ARBs are useful in many patients with diabetes, especially with albuminuric kidney disease, so diabetes is not an absolute contraindication to RAAS blockade.
- C.** PREVENT-CVD risk estimation is designed mainly for primary prevention when high-risk conditions are absent; diabetes itself already supports treatment at stage 1 pressure levels.
- D.** Use lifestyle measures plus antihypertensive medication rather than waiting for stage 2 hypertension because diabetes places the patient in a higher-risk treatment group.

153. A patient with persistent stage 2 hypertension practices slow breathing and mindfulness and asks whether stress reduction can replace medication. What is the preferred response?

- A.** Use stress-management techniques as adjuncts to a broader lifestyle plan while treating persistent stage 2 hypertension with appropriate pharmacologic therapy.
- B.** Stress reduction can modestly lower pressure in some patients, but stage 2 hypertension should not routinely be left untreated for months while waiting to see whether relaxation alone is sufficient.
- C.** A transient fall during a relaxation session demonstrates short-term autonomic responsiveness but does not prove that a prescribed antihypertensive medication can be safely replaced.

D. Stopping medication to measure the independent effect of mindfulness can expose the patient to uncontrolled pressure and is unnecessary for incorporating behavioral therapy.

154. A patient with resistant hypertension has a high STOP-BANG score, witnessed choking episodes, and excessive daytime sleepiness. What should the clinician arrange next to evaluate this suspected contributor?

A. Primary aldosteronism is also common in resistant hypertension and may warrant biochemical screening, but the symptom cluster specifically points toward sleep-disordered breathing.

B. Sedating antihypertensive drugs can worsen fatigue, but witnessed choking and loud snoring should not be attributed to medication without evaluating obstructive sleep apnea.

C. Antihypertensive therapy does not invalidate a diagnostic sleep study, so medications generally should not be stopped merely to permit objective testing.

D. Perform a formal sleep evaluation with objective testing for obstructive sleep apnea, using polysomnography or an appropriate validated home study when suitable.

155. A 45-year-old without established CVD, diabetes, or CKD has confirmed blood pressure 132/84 mm Hg and a PREVENT-CVD 10-year risk of 3%. Which initial approach is appropriate under the 2025 adult guideline?

A. Low short-term risk does not eliminate lifetime risk, so the patient should receive active lifestyle treatment and planned reassessment rather than no follow-up until older age.

B. Stage 1 hypertension at low PREVENT risk does not require immediate multidrug therapy; drug intensity should be matched to pressure category and overall risk.

C. Begin lifestyle modification and reassess after about 3-6 months; if blood pressure remains at least 130/80 mm Hg, pharmacologic treatment is then recommended.

D. Hypertension causes cardiovascular and organ injury while asymptomatic, so waiting for symptoms before reassessment or treatment would miss the purpose of preventive care.

156. A patient taking hydralazine develops arthralgias, fever, malaise, and positive antihistone antibodies. Which adverse effect best explains the syndrome?

A. Serum sickness can cause fever and arthralgias after selected drugs or biologics, but antihistone antibodies and chronic hydralazine exposure more specifically suggest drug-induced lupus.

- B.** ANCA-associated vasculitis can rarely occur with hydralazine and may cause renal or pulmonary injury, but the classic arthralgia-fever-antihistone pattern points to lupus-like toxicity.
- C.** Primary systemic lupus erythematosus can produce similar symptoms, but the medication exposure and antihistone pattern should raise concern for a drug-induced syndrome first.
- D.** Hydralazine can cause drug-induced lupus, particularly with higher cumulative exposure and susceptible acetylator status, often presenting with arthralgias, fever, and antihistone antibodies.

157. A 68-year-old has confirmed stage 2 hypertension at 154/94 mm Hg and no contraindication to first-line therapy. What initial pharmacologic strategy is preferred under the 2025 adult guideline?

- A.** Starting one agent and waiting for maximal monotherapy titration can eventually lower pressure, but stage 2 hypertension is more likely to require combination therapy from the outset.
- B.** A beta blocker is appropriate when a compelling indication such as coronary disease or arrhythmia exists, but it is not automatically the preferred first drug for uncomplicated stage 2 hypertension.
- C.** Start two first-line agents from different classes, preferably in a single-pill fixed-dose combination when practical, together with lifestyle therapy.
- D.** Lifestyle treatment remains essential, but delaying pharmacologic therapy for three to six months is intended for lower-risk stage 1 hypertension rather than confirmed stage 2 disease.

158. A patient with low health literacy receives a new three-drug antihypertensive regimen. Which communication technique best confirms that the dosing plan is understood?

- A.** Written instructions are helpful when designed clearly, but a dense handout without verbal review does not show whether the patient can correctly carry out the regimen.
- B.** Asking “Do you understand?” often produces a polite yes and does not test whether the patient can recall the actual doses and timing.
- C.** Use teach-back by asking the patient to explain in their own words when and how each medication will be taken, correcting misunderstandings immediately.
- D.** Unexplained abbreviations and medical jargon increase cognitive burden, whereas plain language and concrete schedules improve medication comprehension.

159. After amlodipine is added, a patient's systemic vascular resistance falls without significant slowing of the AV node. Which cellular target best explains this antihypertensive effect?

- A.** Blockade of L-type calcium channels in arteriolar smooth muscle reduces calcium-dependent contraction and lowers systemic vascular resistance.
- B.** Mineralocorticoid-receptor blockade in the collecting duct is the mechanism of spironolactone and eplerenone rather than amlodipine.
- C.** Beta-1 receptor blockade lowers heart rate and renin release with beta blockers, but amlodipine is a dihydropyridine calcium-channel blocker with little direct nodal suppression.
- D.** Central alpha-2 agonism reduces sympathetic outflow with clonidine and related drugs, not with amlodipine.

160. A patient asks whether a beta blocker is the preferred first medication for uncomplicated hypertension when there is no coronary disease, heart failure, arrhythmia, or other compelling indication. What is the best response?

- A.** Beta blockers are valuable when a specific cardiovascular indication exists, but they are not generally preferred first-line therapy for otherwise uncomplicated hypertension.
- B.** Thiazide-type diuretics, ACE inhibitors or ARBs, and long-acting dihydropyridine calcium-channel blockers have broader routine first-line roles than beta blockers in uncomplicated hypertension.
- C.** Resting tachycardia alone may influence drug selection, but a compelling indication such as coronary disease or HFrEF is more important than an arbitrary requirement for tachycardia.
- D.** Beta blockers need not be withheld until every other class has failed; they can be appropriate earlier when an evidence-based indication makes their cardiac effects useful.

161. Which major blood-pressure-raising effects are mediated predominantly by angiotensin II signaling through the AT1 receptor?

- A.** AT1 signaling stimulates rather than blocks aldosterone release and promotes renal sodium retention through several direct and indirect mechanisms.
- B.** Arteriolar vasoconstriction together with stimulation of aldosterone secretion increases systemic resistance and promotes sodium retention.
- C.** Angiotensin II generally facilitates sympathetic activity rather than producing generalized inhibition of norepinephrine signaling and vasodilation.

D. Angiotensin II preferentially constricts the efferent arteriole at physiologic concentrations, rather than selectively dilating it to reduce intraglomerular pressure.

162. A patient appears to have resistant hypertension but uses an undersized cuff and frequently misses prescribed doses. How should the condition be classified before these issues are corrected?

A. Secondary hypertension remains possible, but neither an incorrect cuff nor missed medication doses proves an endocrine or renal secondary cause.

B. Refractory hypertension implies failure despite a more extensive optimized regimen and cannot be diagnosed while basic measurement and adherence problems remain unresolved.

C. Masked hypertension requires normal office pressure with elevated out-of-office pressure and is not the label for treatment failure caused by measurement error or nonadherence.

D. Apparent treatment-resistant hypertension is present, but pseudoresistance from inaccurate measurement and nonadherence must be excluded before calling it true resistance.

163. A patient with hypertension and bilateral renal-artery stenosis needs medication adjustment. Which class requires particularly careful monitoring of kidney function and potassium because it lowers efferent arteriolar tone?

A. Dihydropyridine calcium-channel blockers lower systemic resistance without the same direct effect on efferent arteriolar angiotensin II signaling and are often useful in renovascular disease.

B. ACE inhibitors and ARBs reduce angiotensin II-mediated efferent arteriolar constriction and can cause a marked fall in GFR when both kidneys are critically perfusion dependent.

C. Central alpha-2 agonists reduce sympathetic outflow but do not characteristically cause a bilateral stenosis-specific fall in glomerular pressure through efferent dilation.

D. Alpha-1 blockers lower peripheral vascular resistance and can cause orthostasis, but they do not directly interrupt the efferent arteriolar RAAS mechanism.

164. A patient with confirmed resistant hypertension takes lisinopril, amlodipine, and hydrochlorothiazide at effective doses. Before moving to later-line agents, which evidence-based optimization of the regimen should be considered?

A. Short-acting clonidine can lower pressure but creates adherence and withdrawal concerns and should not replace an otherwise evidence-based three-drug foundation.

- B.** Optimize the diuretic with a long-acting thiazide-like agent and, if pressure remains resistant and potassium permits, add a mineralocorticoid-receptor antagonist.
- C.** Stopping the diuretic would worsen sodium and volume control in many resistant patients unless a specific adverse effect or volume-depletion problem required it.
- D.** Adding a second ACE inhibitor duplicates the same enzymatic target without adding a complementary mechanism and increases adverse-effect risk.

165. Acute atrial stretch and ventricular wall stress increase release of natriuretic peptides during volume expansion. Which downstream actions help oppose further blood-pressure and volume increase?

- A.** They promote natriuresis and vasodilation while suppressing renin and aldosterone signaling, thereby reducing volume and vascular tone.
- B.** Epithelial sodium-channel activation would increase distal sodium reabsorption and oppose the natriuretic function of these peptides.
- C.** Vasoconstriction and aldosterone stimulation are characteristic pressure-raising pathways, whereas natriuretic peptides generally oppose both responses.
- D.** Sympathetic activation increases cardiac and vascular stress, while natriuretic-peptide signaling tends to counter neurohumoral sodium-retaining systems rather than stimulate them.

166. A patient with long-standing hypertension develops slowly progressive CKD with modest proteinuria, bland urine sediment, and bilaterally small kidneys after other renal diseases are excluded. Which process can produce this pattern?

- A.** Chronic glomerulonephritis can produce small kidneys late in disease, but it often has a history of more active urinary sediment or a defined primary renal disorder.
- B.** Renal-artery stenosis can cause ischemic nephropathy and asymmetric kidney size, but the vignette describes diffuse chronic small-vessel parenchymal injury after other causes are excluded.
- C.** Chronic hypertensive nephrosclerosis causes arteriolar and glomerular injury with progressive GFR loss, modest proteinuria, and often small echogenic kidneys.
- D.** Acute postinfectious glomerulonephritis typically presents with hematuria, active sediment, edema, and an acute course rather than decades of bland slowly progressive disease.

167. Clinic readings repeatedly average 118/74 mm Hg, but a validated home protocol and daytime ambulatory monitoring average about 142/88 mm Hg. Which blood-pressure phenotype is present?

- A.** Masked hypertension describes normal office blood pressure with persistently elevated out-of-office readings and carries cardiovascular risk that can be missed in clinic.
- B.** White-coat hypertension is the reverse pattern, with elevated office pressure but normal home or ambulatory readings.
- C.** Sustained hypertension requires elevated pressure in both office and out-of-office settings rather than a normal clinic value.
- D.** Pseudoresistant hypertension refers to apparent treatment failure from measurement, adherence, or related factors and is not defined by this untreated office-home discrepancy.

168. A patient with hypertension and symptomatic heart failure with reduced ejection fraction has a clear indication for beta blockade. Which principle is preferred?

- A.** Heart-rate lowering alone does not guarantee the same outcome benefit across the entire beta-blocker class; HFrEF evidence is drug specific.
- B.** A systolic pressure below 130 mm Hg may limit titration in some patients, but it does not automatically eliminate the mortality benefit of indicated HFrEF beta blockade when tolerated.
- C.** Initiating or rapidly up-titrating a beta blocker during unstable acute decompensation can worsen hemodynamics; therapy is started or advanced when the patient is clinically stable.
- D.** Use a beta blocker with proven HFrEF outcome benefit and titrate gradually in a clinically stable patient while monitoring heart rate, pressure, congestion, and perfusion.

169. A pregnant patient at 32 weeks has persistent severe-range blood pressure of 168/112 mm Hg. What is the immediate management priority?

- A.** Oral immediate-release nifedipine can be one accepted agent in monitored obstetric protocols, but unsupervised sublingual dosing and uncontrolled hypotension should be avoided.
- B.** Treat acute severe hypertension promptly with a pregnancy-appropriate agent while urgently evaluating for preeclampsia, maternal organ injury, and fetal complications.
- C.** Severe hypertension is not an expected benign feature of the third trimester; delaying treatment increases maternal stroke and other complication risk.

D. ACE inhibitors remain contraindicated in pregnancy because fetal renal toxicity persists after 20 weeks and may be particularly severe later in gestation.

170. A patient runs out of clonidine and, 36 hours later, presents with marked tachycardia, diaphoresis, and severe hypertension. Which physiologic change explains this withdrawal syndrome?

A. Peripheral alpha-1 blockade would lower vascular tone, whereas clonidine withdrawal removes central alpha-2 receptor stimulation rather than causing irreversible alpha-1 antagonism.

B. Sudden loss of central sympathetic suppression produces a surge in adrenergic outflow, causing rebound tachycardia and severe hypertension.

C. Mineralocorticoid deficiency causes hypotension and salt loss, not the hyperadrenergic rebound syndrome seen after abrupt clonidine discontinuation.

D. Clonidine concentrations fall after discontinuation; the problem is loss of central sympatholysis rather than accumulation or direct beta-1 receptor activation.

171. A 37-year-old with hypertension has markedly enlarged kidneys containing numerous bilateral cysts and a family history of kidney failure. Which diagnosis best explains the presentation?

A. Autosomal dominant polycystic kidney disease causes bilateral renal cystic enlargement, can present with early hypertension, and often follows an autosomal dominant family pattern.

B. Renal-artery fibromuscular dysplasia can cause hypertension in younger adults, but it does not produce massively enlarged kidneys filled with bilateral cysts.

C. Acquired cystic kidney disease occurs mainly after advanced chronic kidney failure and usually involves small or normal-sized kidneys rather than inherited enlarged cystic kidneys.

D. Simple renal cysts are common and can be incidental, but numerous bilateral cysts with enlarged kidneys and a strong family history indicate a systemic inherited cystic disorder.

172. An older adult develops a 35% rise in serum creatinine soon after starting an ACE inhibitor and has bilateral renal bruits. Which mechanism should be considered?

A. Severe bilateral renal-artery stenosis or stenosis to a solitary functioning kidney can make GFR dependent on angiotensin II-mediated efferent constriction, so ACE inhibition produces a marked creatinine rise.

B. ACE inhibitors dilate the efferent arteriole; they do not raise filtration by preferentially constricting it, so the large creatinine increase suggests perfusion-dependent renal physiology.

C. A small creatinine rise can occur after ACE inhibition, but an increase greater than about 30% is not expected in every patient and should prompt evaluation of volume, medications, and renovascular disease.

D. Primary aldosteronism does not characteristically make GFR dependent on efferent angiotensin II tone, so it does not explain the bruit-associated large creatinine rise after ACE inhibition.

173. A patient with years of severe hypertension develops neurologic symptoms after systemic pressure is lowered precipitously from 230/130 mm Hg to near-normal levels. Which adaptation explains the risk of cerebral hypoperfusion?

A. In chronic hypertension the cerebral autoregulatory curve shifts toward higher, not lower, pressures, so sudden normalization can move perfusion below the adapted lower limit.

B. Reflex cerebrovascular responses contribute to flow regulation, but the key vulnerability is the chronically shifted autoregulatory range rather than a unique reflex vasoconstriction caused by treatment.

C. Chronic hypertension shifts the cerebral autoregulatory range to higher pressures, making the brain vulnerable when systemic pressure is reduced abruptly below its adapted perfusion range.

D. Autoregulation is impaired in severe disease but is not universally abolished, so cerebral blood flow does not simply become linearly proportional to systemic pressure at all levels.

174. A patient with chronic kidney disease has progressively increasing albuminuria. Why is this finding clinically important?

A. Albumin loss is not protective volume therapy; greater albuminuria generally reflects glomerular injury and is associated with worse renal and cardiovascular outcomes.

B. Albuminuria is both a marker of kidney damage and an independent marker of elevated cardiovascular and kidney-progression risk.

C. Estimated GFR and albuminuria provide complementary prognostic information, so knowing eGFR does not make urinary albumin excretion redundant.

D. Pheochromocytoma can cause hypertension but is not diagnosed by albuminuria, which is a nonspecific marker of renal vascular and glomerular injury.

175. A renal-physiology model shows that a patient's kidneys require a higher arterial pressure than normal to excrete a given sodium load. Which concept best describes this abnormality?

- A.** A rightward shift of the renal pressure-natriuresis relationship means a higher perfusion pressure is required to achieve sodium balance and is a central model of chronic hypertension.
- B.** Suppression of tubular sodium reabsorption would increase sodium excretion at a lower pressure and shift the relationship in the opposite direction.
- C.** Baroreflex resetting can contribute to chronic pressure regulation, but the described defect specifically concerns the kidney's pressure-dependent sodium-excretion curve.
- D.** Complete loss of GFR would cause renal failure rather than the stable higher-pressure equilibrium represented by a shifted pressure-natriuresis relationship.

176. A younger adult has severe hypertension, hypokalemic metabolic alkalosis, suppressed renin, suppressed aldosterone, and a strong family history of early hypertension. Which inherited mechanism best explains the phenotype?

- A.** Gitelman syndrome results from loss of the thiazide-sensitive Na-Cl cotransporter and causes hypokalemic alkalosis, but the renal salt wasting usually lowers rather than raises blood pressure.
- B.** Loss-of-function epithelial sodium-channel disease causes salt wasting and hyperkalemia in pseudohypoaldosteronism, the opposite volume and potassium phenotype from this patient.
- C.** Gain-of-function epithelial sodium-channel activity in Liddle syndrome produces sodium retention, low-renin low-aldosterone hypertension, hypokalemia, and metabolic alkalosis.
- D.** Mineralocorticoid-receptor loss causes renal salt wasting, hypotension, hyperkalemia, and elevated renin-aldosterone signaling rather than this low-renin hypertensive syndrome.

177. A patient with an undiagnosed pheochromocytoma becomes profoundly hypertensive during a catecholamine surge. If tachycardia later requires beta blockade, what sequencing principle prevents worsening vasoconstriction?

- A.** Control alpha-mediated vasoconstriction with appropriate alpha blockade or titratable vasodilation before adding beta blockade for persistent tachycardia, avoiding unopposed alpha stimulation.
- B.** An ACE inhibitor may reduce pressure as adjunctive therapy in some contexts, but catecholamine crisis is not driven exclusively by angiotensin II and requires direct control of adrenergic vasoconstriction.

C. Diuresis alone does not address the intense catecholamine-mediated vascular tone, and affected patients may actually be relatively volume contracted before adequate alpha blockade.

D. Beta blockade before alpha control can leave alpha-mediated vasoconstriction unopposed and may precipitate or worsen severe hypertension rather than safely lowering systemic resistance.

178. A patient with hypertension, chronic kidney disease, and substantial albuminuria needs antihypertensive drug therapy. Which class is particularly useful for kidney protection when tolerated?

A. A direct vasodilator can lower pressure when additional therapy is needed, but it does not provide the same albuminuria-lowering intraglomerular effect as RAAS blockade.

B. A central alpha-2 agonist can lower resistant pressure but is not generally chosen for disease-specific kidney protection in albuminuric chronic kidney disease.

C. Combining an ACE inhibitor with an ARB can reduce albuminuria further, but excess hyperkalemia and kidney-injury risk makes routine dual blockade inappropriate.

D. An ACE inhibitor or ARB reduces systemic and intraglomerular pressure, can lower albuminuria, and is preferred when safely tolerated and monitored.

179. An adult has an average standardized blood pressure of 148/76 mm Hg. Which classification is correct under current ACC/AHA categories?

A. Stage 1 hypertension applies to systolic pressure 130-139 mm Hg or diastolic pressure 80-89 mm Hg, so the systolic value here exceeds that range.

B. Both systolic and diastolic values do not need to be elevated; classification is based on whichever component falls into the higher category.

C. Elevated blood pressure is limited to systolic pressure 120-129 mm Hg with diastolic pressure below 80 mm Hg, not a systolic value of 148 mm Hg.

D. Stage 2 hypertension applies because systolic pressure is at least 140 mm Hg even though the diastolic pressure remains below 80 mm Hg.

180. A patient with proliferative glomerulonephritis develops falling GFR, weight gain, edema, and rising pressure over several days. Which renal mechanism most directly drives the new hypertension?

- A.** Reduced glomerular filtration can activate intrarenal and systemic neurohumoral pathways, but the most immediate hemodynamic driver of edema-associated hypertension is impaired sodium and water excretion.
- B.** Reduced renal sodium excretion expands extracellular and effective circulating volume, increasing cardiac filling and systemic pressure.
- C.** Venodilation would reduce effective preload rather than produce the edema and pressure rise seen when inflamed kidneys retain sodium.
- D.** Renal salt wasting causes volume contraction and generally lowers pressure, making it the opposite mechanism from edema-forming glomerular disease.

181. A patient with type 2 diabetes, albuminuria, and hypertension needs an antihypertensive regimen. Which class has a compelling kidney indication when tolerated?

- A.** A thiazide-like diuretic is an effective pressure-lowering class and may be needed in combination, but it does not provide the same direct albuminuria-focused renal indication.
- B.** An ACE inhibitor or ARB is preferred because RAAS blockade lowers pressure and albuminuria, with monitoring of serum creatinine and potassium.
- C.** Hydralazine can lower pressure through arteriolar dilation, but diabetes does not contraindicate RAAS blockade and hydralazine alone is not the kidney-protective first choice here.
- D.** Dual ACE inhibitor-ARB therapy increases kidney and hyperkalemia adverse events and is not required for nephroprotection despite the presence of diabetes and albuminuria.

182. At a preconception hypertension visit, a woman is taking losartan. What is the best medication principle?

- A.** Discontinue the ARB and transition to a pregnancy-compatible antihypertensive before conception when possible, or promptly once pregnancy is recognized.
- B.** Fetal renin-angiotensin system toxicity can occur during pregnancy, particularly later gestation, so continuing losartan until delivery is unsafe.
- C.** Combining an ARB with an ACE inhibitor would intensify fetal RAAS blockade and maternal renal or potassium risk rather than improve pregnancy safety.
- D.** Pregnancy changes RAAS physiology, but increasing an ARB dose is not an appropriate way to compensate because the drug class itself is contraindicated.

183. A patient taking chlorthalidone develops weakness; sodium is 126 mmol/L and potassium is 3.1 mmol/L. What adverse drug effect is most likely?

- A.** ACE inhibitors can raise potassium and creatinine, but chlorthalidone is a thiazide-like diuretic and does not inhibit angiotensin-converting enzyme.
- B.** Mineralocorticoid-receptor antagonists tend to raise potassium, whereas thiazide-type diuretics commonly lower potassium and can cause hyponatremia.
- C.** Calcium-channel blockers alter vascular smooth-muscle calcium entry, but they do not explain the coupled sodium and potassium depletion produced by chlorthalidone.
- D.** Thiazide-type diuretics can cause hyponatremia and hypokalemia through renal electrolyte loss and impaired urinary dilution, especially in susceptible patients.

184. A patient with obesity and hypertension asks whether effective anti-obesity pharmacotherapy or metabolic surgery can influence blood pressure. What is the best answer?

- A.** Substantial sustained weight loss can meaningfully lower blood pressure, improve obesity-related contributors such as sleep apnea, and reduce medication requirements in some patients.
- B.** Weight-management interventions can alter sodium handling and sympathetic activity favorably; adipose tissue is not required for normal renal sodium excretion.
- C.** Body weight remains mechanistically related to pressure through renal, neurohumoral, metabolic, and sleep-related pathways even after hypertension is established.
- D.** Antihypertensive medication use does not by itself contraindicate metabolic surgery; candidacy depends on obesity severity, comorbidity, operative risk, and informed preference.

185. Potassium rises from 4.5 to 5.9 mmol/L after spironolactone is added to lisinopril in a patient with hypertension. Which interaction explains the new laboratory hazard?

- A.** ACE inhibitors and spironolactone both reduce renal potassium excretion, so the combination does not counterbalance potassium effects even when baseline kidney function is normal.
- B.** Combined RAAS and mineralocorticoid-receptor blockade can substantially increase hyperkalemia risk and requires careful potassium and kidney-function monitoring.
- C.** An eGFR above 20 mL/min/1.73 m² does not eliminate hyperkalemia risk; declining kidney function further reduces potassium excretory reserve with this drug combination.

D. ARBs and ACE inhibitors share potassium-raising RAAS effects, so clinically important hyperkalemia is not unique to ARB combinations with spironolactone.

186. A patient with a pheochromocytoma is found to carry an SDHB pathogenic variant. What is the principal clinical value of identifying this hereditary result beyond management of the current tumor?

- A.** Genotype can influence tumor location and biochemical phenotype, but it does not make future biochemical assessment unnecessary when recurrence or new disease is suspected.
- B.** Some hereditary syndromes predispose to extra-adrenal or multifocal disease, so surveillance should not automatically be restricted to the adrenal glands.
- C.** The result can guide lifelong surveillance for multifocal, recurrent, or associated tumors and can prompt genetic counseling and risk assessment of biologic relatives.
- D.** Predictive testing of at-risk relatives can identify carriers before hypertension or catecholamine symptoms appear, allowing syndrome-appropriate surveillance rather than waiting for clinical disease.

187. A patient with pressure 242/136 mm Hg and visual blurring has retinal flame hemorrhages, cotton-wool spots, and swollen optic discs. How should these ocular findings affect the urgency assessment?

- A.** Mild chronic hypertensive retinopathy may show arteriolar narrowing or AV crossing changes, but hemorrhages, cotton-wool spots, and disc edema indicate much more severe injury.
- B.** Severe hypertensive retinopathy with optic-disc swelling represents acute hypertension-mediated target-organ injury and warrants urgent evaluation and controlled treatment.
- C.** Preserved visual acuity does not make papilledema benign; optic-disc swelling in severe hypertension is a marker of dangerous microvascular injury even before major vision loss.
- D.** Diabetic retinopathy can cause hemorrhages and cotton-wool spots, but the severe hypertension and disc swelling require urgent consideration of hypertensive emergency regardless of diabetes status.

188. A previously sedentary patient with uncomplicated hypertension asks for a safe long-term exercise plan. Which prescription best reflects evidence-based aerobic and resistance activity?

- A.** Aerobic exercise has strong blood-pressure benefits, and adding appropriately performed resistance training can provide further cardiovascular and functional benefit rather than needing to be avoided entirely.

- B.** Isometric exercise may lower resting pressure when prescribed appropriately, but repeated maximal straining and Valsalva maneuvers are not the primary or safest universal program.
- C.** Patients with uncomplicated controlled or mildly elevated pressure can generally begin graded physical activity without waiting for a blood pressure below 120/80 mm Hg.
- D.** Regular aerobic activity supplemented by resistance exercise, with gradual progression according to baseline fitness, symptoms, comorbidity, and treatment response.

189. A 67-year-old with treated hypertension and chronic exertional angina has a resting pulse of 98/min. There is no asthma, heart block, or other contraindication. Which antihypertensive class is especially useful because it also reduces myocardial oxygen demand?

- A.** A thiazide-type diuretic lowers blood pressure and improves cardiovascular outcomes, but it does not directly slow heart rate or provide primary antianginal rate control.
- B.** An angiotensin receptor blocker can be valuable for vascular and renal indications, but it does not directly reduce sinus rate or contractility in a tachycardic patient with angina.
- C.** A mineralocorticoid-receptor antagonist can help resistant hypertension or selected heart-failure patients, but it is not used primarily to lower anginal heart rate and contractile demand.
- D.** A beta blocker lowers blood pressure, heart rate, and myocardial contractility, making it useful for chronic angina when there is no contraindication.

190. A hypertensive patient with normal renal function asks whether increasing dietary potassium can help blood-pressure control. Which advice is preferred?

- A.** Potassium intake affects renal sodium handling and vascular biology and therefore contributes meaningfully to blood-pressure regulation.
- B.** Potassium-rich foods are generally encouraged when safe; routine elimination would remove a potentially beneficial dietary component without evidence of universal pressure elevation.
- C.** A potassium-rich diet can help lower blood pressure, but potassium supplements and potassium-based salt substitutes require caution in CKD or with potassium-raising medications.
- D.** Advanced CKD and mineralocorticoid-receptor blockade substantially increase hyperkalemia risk, so unsupervised potassium supplementation is not safe in those settings.

191. A strength athlete with a very large upper-arm circumference has blood pressure measured in clinic. What error is most likely if the cuff bladder is too small for the arm?

- A.** An oversized cuff can sometimes underestimate pressure, whereas an undersized cuff requires excessive inflation pressure and tends to bias the reading upward.
- B.** An undersized cuff on a large arm can falsely overestimate blood pressure, potentially creating an erroneous diagnosis of uncontrolled hypertension.
- C.** A validated automated device still requires an appropriate cuff range; algorithm validation does not compensate for using a cuff outside its intended arm circumference.
- D.** Cuff mismatch can affect both systolic and diastolic measurements, so it should not be assumed that systolic pressure remains reliable in a very muscular arm.

192. A patient with eGFR 20 mL/min/1.73 m² develops potassium 6.0 mmol/L while taking spironolactone for difficult hypertension. What drug-safety issue requires immediate attention?

- A.** A potassium level near 6 mmol/L in advanced CKD is clinically significant and should not be accepted simply because the patient is receiving a mineralocorticoid-receptor antagonist.
- B.** Mineralocorticoid-receptor blockade may be unsafe because advanced kidney dysfunction greatly increases the risk of persistent or worsening hyperkalemia.
- C.** Increasing spironolactone would further reduce potassium excretion; hyperkalemia is a toxicity signal rather than evidence of inadequate receptor blockade.
- D.** Potassium replacement is contraindicated in this setting because CKD and spironolactone already impair renal potassium elimination.

193. A patient with obstructive sleep apnea begins effective CPAP therapy. What average effect on hypertension is most realistic?

- A.** CPAP reduces obstructive events and nocturnal sympathetic stress, so it has physiologic effects even though the blood-pressure response varies substantially among patients.
- B.** Some patients achieve meaningful pressure reduction, but CPAP does not reliably normalize blood pressure enough to eliminate antihypertensive medication in everyone.
- C.** Blood pressure often falls modestly on average, with larger responses in some patients who have resistant hypertension, severe sleep apnea, or high treatment adherence.

D. Eliminating obstructive respiratory events generally reduces rather than increases nocturnal sympathetic activation, so a systematic CPAP-induced pressure rise is not expected.

194. A patient with untreated thyrotoxicosis has pulse 116/min and blood pressure 164/58 mm Hg. Which hemodynamic change best explains the widened pulse pressure?

A. Hyperthyroidism increases beta-adrenergic sensitivity and chronotropy rather than causing complete autonomic failure with loss of heart-rate response.

B. Renal and metabolic changes can influence volume, but hyperthyroid systolic hypertension is not explained by obligatory salt wasting that prevents any arterial pressure rise.

C. Cardiac output is usually increased rather than markedly reduced, while diastolic pressure may remain normal or fall because systemic vascular resistance decreases.

D. Increased heart rate and stroke volume raise cardiac output while systemic vascular resistance falls, producing a hyperdynamic circulation with widened pulse pressure.

195. A patient with hypertension and atrial fibrillation has rapid ventricular response. If ventricular systolic function and conduction status permit, which drug can address both rate control and blood pressure?

A. Hydralazine lowers afterload but can provoke reflex tachycardia, making it a poor choice when the immediate need includes slowing the ventricular response.

B. A beta blocker or a non-dihydropyridine calcium-channel blocker can slow AV nodal conduction and lower pressure, with selection based on ventricular function and other contraindications.

C. A thiazide diuretic can improve blood pressure and volume status but does not directly slow atrioventricular nodal conduction during rapid atrial fibrillation.

D. An ARB is useful for blood-pressure and selected renal or cardiac indications but does not acutely terminate atrial fibrillation or provide ventricular rate control.

196. A 54-year-old has a properly measured average blood pressure of 134/86 mm Hg on repeated visits. How is this categorized under current ACC/AHA adult thresholds?

A. Stage 2 hypertension begins at systolic pressure 140 mm Hg or diastolic pressure 90 mm Hg, thresholds not reached by this patient's average.

- B.** Stage 1 hypertension applies because systolic pressure is 130-139 mm Hg and diastolic pressure is 80-89 mm Hg.
- C.** Elevated blood pressure is limited to systolic pressure 120-129 mm Hg with diastolic pressure below 80 mm Hg, which does not fit 136/84 mm Hg.
- D.** Normal blood pressure requires systolic pressure below 120 mm Hg and diastolic pressure below 80 mm Hg, so this average is above the normal category.

197. A patient with CKD, eGFR 22 mL/min/1.73 m², prominent edema, and uncontrolled hypertension remains on an ACE inhibitor and calcium-channel blocker. Which strategy most directly addresses the volume component?

- A.** Use an adequately dosed diuretic regimen, usually centered on a loop diuretic at this level of kidney dysfunction, with sodium restriction and response monitoring.
- B.** A beta blocker may be useful for a compelling cardiac indication, but replacing RAAS blockade solely because edema is present does not directly treat extracellular volume excess.
- C.** Nitrates can reduce preload in selected cardiac conditions, but they do not substitute for renal sodium removal when edema and CKD indicate significant volume expansion.
- D.** Fluid restriction may help selected patients, but sodium restriction and effective diuretic therapy remain central because reduced GFR still permits clinically useful renal sodium removal.

198. A patient with hypertension drinks about four alcoholic beverages every night. Which counseling is preferred?

- A.** Alcohol can cause transient vasodilation, but sustained heavy intake is associated with higher blood pressure and should not be used as chronic antihypertensive therapy.
- B.** Reducing heavy alcohol consumption can lower blood pressure and should be incorporated into the broader lifestyle and cardiovascular-risk treatment plan.
- C.** High-sodium mixers would add another pressure-raising dietary exposure rather than protect against orthostatic symptoms during alcohol reduction.
- D.** Alcohol can affect triglycerides, sleep, arrhythmia risk, and blood pressure, so its cardiovascular relevance is not limited to lipid metabolism.

199. A healthy volunteer moves rapidly from supine to standing. Which immediate baroreflex response helps preserve arterial pressure?

- A.** Venous return and atrial stretch initially fall rather than rise, so atrial natriuretic-peptide release is not the first compensatory mechanism preserving pressure.
- B.** Renal perfusion can transiently decrease and renin may eventually rise, but the immediate seconds-scale response is neural rather than cessation of renin secretion.
- C.** Reduced stretch of the carotid sinus and aortic arch decreases baroreceptor firing, increasing sympathetic outflow and reducing vagal activity.
- D.** Increased carotid-sinus stretch occurs with rising arterial pressure and would suppress sympathetic tone, the opposite signal from the pressure fall after standing.

200. Repeated standardized office and home measurements in a 58-year-old average 132/83 mm Hg despite lifestyle efforts. The patient has no established cardiovascular disease, diabetes, or chronic kidney disease; PREVENT-CVD estimates 10-year CVD risk at 8.4%. What is the most appropriate next step under the 2025 adult hypertension guideline?

- A.** Add a first-line antihypertensive medication while continuing lifestyle therapy because stage 1 hypertension is present and 10-year PREVENT-CVD risk exceeds the 7.5% treatment threshold.
- B.** A three-to-six-month lifestyle-only trial is used for lower-risk stage 1 hypertension, but the 8.4% PREVENT-CVD risk exceeds the updated risk threshold for starting medication.
- C.** Two-drug initial therapy is preferred for stage 2 hypertension, whereas 132/83 mm Hg is stage 1 and does not by itself require an immediate two-drug regimen.
- D.** Established CVD, diabetes, and CKD are high-risk indications, but their absence does not negate pharmacologic treatment when PREVENT-CVD risk is at or above the recommended threshold.

Answers

1. D. Clinical reasoning: The constellation of specific catabolic features—proximal weakness, easy bruising, wide violaceous striae—plus metabolic disease raises concern for endogenous hypercortisolism.

2. A. Why this is correct: Obesity is associated with sympathetic activation, RAAS and mineralocorticoid activity, renal sodium retention, sleep apnea, and altered renal pressure relationships. These effects increase volume and vascular tone.

- 3. C.** Interpretation: After MI, beta blockade and ACE inhibitor/ARB therapy are often indicated based on ventricular function and clinical status. They reduce myocardial workload and adverse remodeling while contributing to BP control.
- 4. D.** Management principle: The DASH dietary pattern lowers BP and improves overall cardiovascular diet quality. Its benefits are enhanced when sodium intake is reduced and caloric intake supports a healthy weight.
- 5. A.** Key point: Thiazides enhance distal calcium reabsorption and can reduce urinary calcium, making them useful in selected patients with hypertension and recurrent calcium stones.
- 6. B.** Clinical reasoning: Hypertension increases LV afterload even in aortic stenosis and should be treated, but severe fixed obstruction makes abrupt hypotension poorly tolerated. Careful titration and valve-directed management are appropriate.
- 7. B.** Why this is correct: Some SNRIs and other psychiatric drugs can increase BP through noradrenergic or metabolic effects. Hypertension and psychiatric illness should both be treated, with medication choice and monitoring individualized.
- 8. D.** Interpretation: Gordon syndrome (pseudohypoaldosteronism type II) increases NCC activity, causing sodium retention, hypertension, hyperkalemia, and metabolic acidosis. Thiazides directly target the implicated transporter.
- 9. B.** Management principle: True resistant hypertension requires confirmation that BP is uncontrolled outside the office when appropriate, along with adherence assessment and proper measurement. White-coat effect can create pseudoresistance.
- 10. B.** Key point: Therapeutic inertia is failure to initiate or intensify appropriate therapy despite uncontrolled BP. Standardized protocols, home monitoring, and timely follow-up can reduce this gap.
- 11. A.** Clinical reasoning: Sleep apnea diagnosis requires objective sleep testing. Full polysomnography is standard, while validated home testing can be appropriate in selected uncomplicated adults with high pretest probability.
- 12. B.** Why this is correct: Renal fibromuscular dysplasia often affects younger women and can cause the classic beaded appearance from alternating stenosis and aneurysmal dilation. It is a potentially treatable cause of renovascular hypertension.
- 13. C.** Interpretation: Beta blockade before adequate alpha blockade can leave alpha-mediated vasoconstriction unopposed and precipitate severe hypertension. Volume expansion and appropriate alpha blockade precede surgery.
- 14. A.** Management principle: Loop diuretics inhibit NKCC2 in the thick ascending limb, producing potent natriuresis. They are particularly useful for volume control when renal function is substantially reduced or edema is prominent.

- 15. D.** Key point: Management begins with identifying causes such as volume depletion, vasodilators, alpha blockers, autonomic disease, or deconditioning. Slow position changes, hydration when appropriate, compression, and medication adjustment can reduce symptoms.
- 16. B.** Clinical reasoning: Dihydropyridine CCB edema reflects precapillary arteriolar dilation more than total-body volume expansion. Lowering the dose or combining with a RAAS blocker can reduce edema in some patients.
- 17. D.** Why this is correct: Renal parenchymal disease is one of the most common secondary contributors to hypertension. Impaired sodium excretion, volume expansion, RAAS activation, and sympathetic effects can all participate.
- 18. A.** Interpretation: Dihydropyridine edema is largely a capillary hemodynamic effect, not simply total-body volume overload. Dose reduction, switching, or adding a RAAS blocker can be more effective than reflexive diuresis.
- 19. B.** Management principle: Nonselective beta blockade can provoke bronchospasm. Cardioselective agents may be used cautiously when there is a strong cardiovascular indication, considering asthma severity and response.
- 20. C.** Key point: Out-of-office BP measurement helps confirm persistent hypertension and identify white-coat or masked patterns. Proper office technique and repeated measurements are also essential.
- 21. C.** Clinical reasoning: Verapamil and diltiazem have important cardiac electrophysiologic effects in addition to vasodilation. Bradycardia, AV block, and constipation are notable adverse effects of verapamil.
- 22. A.** Why this is correct: ALLHAT reinforced the effectiveness of thiazide-like diuretic therapy for initial treatment and highlighted that BP lowering and outcome evidence, not novelty of a drug class, should drive selection.
- 23. D.** Interpretation: Mineralocorticoid receptor antagonism is highly effective as fourth-line treatment in many patients with true resistant hypertension, provided potassium and renal function allow safe use.
- 24. D.** Management principle: Medication-induced pseudoresistance is common and potentially reversible. NSAIDs, stimulants, calcineurin inhibitors, hormones, and other agents can independently or collectively worsen BP.
- 25. C.** Key point: Acute aortic syndrome requires rapid anti-impulse therapy. IV beta blockade reduces heart rate and dP/dt before vasodilation; typical goals are HR 60-80/min and SBP below 120 mm Hg or the lowest pressure maintaining organ perfusion.
- 26. C.** Clinical reasoning: Cocaine blocks catecholamine reuptake and causes intense sympathetic stimulation, vasoconstriction, tachycardia, and increased myocardial oxygen demand. It can trigger hypertensive emergencies, ischemia, and aortic complications.

- 27. A.** Why this is correct: Renin release is stimulated by reduced renal perfusion, reduced macula-densa sodium chloride delivery, and renal sympathetic beta-1 activation. Volume expansion and increased distal sodium delivery suppress renin.
- 28. A.** Interpretation: Atherosclerotic renovascular disease is suggested by resistant hypertension, diffuse vascular disease, kidney asymmetry, worsening renal function with RAAS blockade, or recurrent flash pulmonary edema.
- 29. B.** Management principle: Albuminuria is an important marker of kidney damage and cardiovascular risk. A spot urine albumin-to-creatinine ratio is convenient and clinically informative.
- 30. D.** Key point: SPRINT compared more intensive with standard systolic targets in high-risk nondiabetic adults. Diabetes was excluded, which is important when applying the results to other populations.
- 31. B.** Clinical reasoning: Irregular rhythms can reduce precision of some oscillometric measurements. Repeated standardized readings, and manual confirmation when needed, improve reliability.
- 32. D.** Why this is correct: Glycyrrhetic acid from licorice inhibits 11 β -HSD2, which normally converts cortisol to cortisone in mineralocorticoid target tissues. Cortisol then activates the mineralocorticoid receptor, mimicking apparent mineralocorticoid excess.
- 33. C.** Interpretation: White-coat hypertension refers to elevated office BP with normal out-of-office BP in an untreated patient. Masked hypertension is the opposite pattern.
- 34. C.** Management principle: Estrogen-containing contraceptives can increase angiotensinogen and raise BP in susceptible patients. The magnitude varies, and alternative contraception may be appropriate when hypertension is clinically significant.
- 35. D.** Key point: Resistant hypertension often has an important volume component. Long-acting thiazide-like diuretics have stronger and more persistent BP effects than low-dose hydrochlorothiazide in many patients.
- 36. C.** Clinical reasoning: Amphetamine-type stimulants increase catecholaminergic activity and may raise BP and heart rate. Medication review is an essential part of evaluating apparent secondary or resistant hypertension.
- 37. A.** Why this is correct: CKD increases cardiovascular risk and is a condition for which stage 1 hypertension merits treatment. Albuminuria and renal function influence drug choice and monitoring.
- 38. A.** Interpretation: Selected high-risk presentations—especially recurrent flash pulmonary edema, progressive ischemic nephropathy, or truly refractory hypertension in appropriate anatomy—can justify revascularization consideration.

- 39. C.** Management principle: ESAs can worsen hypertension through mechanisms that include increased vascular resistance and other hemodynamic effects. BP should be monitored during treatment.
- 40. C.** Key point: Coarctation is an important secondary cause of pediatric hypertension. Arm-leg BP discrepancy and diminished femoral pulses are classic findings and warrant cardiovascular imaging.
- 41. D.** Clinical reasoning: Hydralazine is a direct arteriolar vasodilator. The fall in resistance can activate the baroreflex, causing tachycardia and increased renin; fluid retention is another reason it is often combined with other agents.
- 42. B.** Why this is correct: ARBs provide AT1 blockade without inhibiting bradykinin degradation and are a common alternative for ACE-inhibitor cough. Prior ACE-inhibitor angioedema requires more individualized risk assessment.
- 43. D.** Interpretation: Secondary hypertension should be considered with early or abrupt onset, resistant hypertension, severe or episodic presentations, hypokalemia, renal dysfunction, suggestive symptoms, or characteristic examination findings.
- 44. C.** Management principle: Hypertension clusters with other modifiable risks. Comprehensive prevention causes larger overall benefit than treating BP as an isolated number.
- 45. C.** Key point: ACE inhibitor plus ARB dual RAAS blockade increases adverse renal and potassium effects without a favorable routine risk-benefit balance. One RAAS blocker is usually selected instead.
- 46. B.** Clinical reasoning: Successful lifestyle change can lower BP enough to require medication adjustment. Home readings, symptoms, electrolytes, and renal function guide safe de-intensification when appropriate.
- 47. A.** Why this is correct: Hypothyroidism can increase systemic vascular resistance and is associated with diastolic hypertension. Treating thyroid disease can improve the BP pattern in affected patients.
- 48. B.** At age 13 years and older, pediatric hypertension guidance uses fixed adolescent blood-pressure thresholds rather than height-based percentiles. An elevated oscillometric screening value should be confirmed with proper auscultatory technique and persistent hypertension established over appropriate repeated measurements or ambulatory monitoring.
- 49. D.** Management principle: Coarctation causes a pressure gradient proximal versus distal to the narrowing, classically with arm hypertension, delayed or weak femoral pulses, and lower leg pressures.
- 50. B.** Key point: Renal autoregulation depends mainly on the afferent arteriolar myogenic response and tubuloglomerular feedback. These mechanisms stabilize renal blood flow and filtration over a physiologic pressure range.

- 51. D.** Clinical reasoning: Labetalol and extended-release nifedipine are commonly used during pregnancy, and methyldopa remains an option. Drugs that directly inhibit the renin-angiotensin system are avoided because of fetal toxicity.
- 52. C.** Why this is correct: Loop diuretics are powerful natriuretics and are central to decongestion in HF. BP benefit reflects volume reduction; renal function and electrolytes still require monitoring.
- 53. A.** Interpretation: For renal fibromuscular dysplasia, balloon angioplasty is often the preferred intervention when revascularization is indicated; stents are usually reserved for complications or failed angioplasty.
- 54. C.** Management principle: ARBs block AT1 receptors without inhibiting ACE, so bradykinin breakdown is not directly impaired. They preserve many RAAS-blocking benefits with much less ACE-inhibitor cough.
- 55. D.** Key point: OSA commonly causes nocturnal BP elevation and blunted dipping. ABPM characterizes these patterns and distinguishes them from office-only effects.
- 56. D.** Clinical reasoning: Current adult guidance identifies thiazide-type diuretics, long-acting dihydropyridine CCBs, ACE inhibitors, and ARBs as first-line classes. Other drugs are chosen for compelling indications or resistant hypertension.
- 57. C.** Why this is correct: Obesity adds to OSA and hypertension through multiple pathways. CPAP treats airway obstruction but does not reverse all effects of excess adiposity; weight management remains part of comprehensive care.
- 58. A.** Interpretation: Persistent uncontrolled BP after rigorous confirmation and optimization warrants specialist evaluation. Advanced testing and newer device-based options may be considered selectively, but only after pseudoresistance and standard therapy are addressed.
- 59. D.** Management principle: PAD identifies high systemic atherosclerotic risk. BP control remains important, alongside smoking cessation, lipid therapy, antiplatelet therapy when indicated, exercise, and limb-specific assessment.
- 60. A.** Key point: Structured home BP monitoring is most informative when technique is standardized and multiple readings are averaged. Selective or symptom-only measurement can bias interpretation.
- 61. A.** Clinical reasoning: Hypertensive emergency is severe BP elevation with acute target-organ injury, such as encephalopathy, acute renal injury, pulmonary edema, aortic syndrome, or retinal injury. It requires rapid but controlled IV treatment in a monitored setting.
- 62. D.** Why this is correct: Arterial stiffening increases pulse-wave velocity and brings reflected waves back earlier, augmenting central systolic pressure. This helps explain isolated systolic hypertension in older adults.

- 63. B.** Interpretation: Discordant office and home readings may reflect white-coat effect, technique problems, or intermittent hypotension. Treatment should integrate validated out-of-office data and symptoms.
- 64. B.** Management principle: Patients with established CVD are already high risk; stage 1 hypertension warrants pharmacologic therapy as part of secondary prevention, individualized for symptoms and comorbidities.
- 65. D.** Key point: Marked arterial stiffness and calcification can occasionally make cuff pressure overestimate intra-arterial pressure. This is uncommon and should not be diagnosed solely from a bedside sign.
- 66. A.** Clinical reasoning: For adolescents age 13 years and older, pediatric guidance uses adult-like thresholds: stage 1 is 130-139/80-89 mm Hg and stage 2 begins at 140/90 mm Hg.
- 67. B.** Why this is correct: Insulin resistance clusters with obesity and other mechanisms that favor sympathetic activation, sodium retention, endothelial dysfunction, and vascular remodeling. Hypertension in metabolic syndrome is therefore multifactorial.
- 68. A.** Interpretation: True resistance requires that an effective regimen is actually being taken. Nonadherence is common and should be evaluated nonjudgmentally using history, refill data, pill counts, or biochemical testing when appropriate.
- 69. D.** Management principle: Calcineurin inhibitors commonly add to post-transplant hypertension through renal vasoconstriction, sodium retention, and nephrotoxicity. Management must balance BP control with graft immunosuppression needs.
- 70. B.** Key point: Thiazides can reduce renal urate excretion and raise serum uric acid. This does not prohibit their use in every patient with gout, but recurrent attacks may influence drug selection.
- 71. D.** Clinical reasoning: MRAs can be effective for resistant hypertension but reduce potassium excretion. CKD and concurrent RAAS blockade increase hyperkalemia risk, so laboratory monitoring is essential.
- 72. C.** Why this is correct: Treatment targets depend on how BP is measured. Standardization—rest, positioning, validated equipment, repeated readings—reduces the risk of overtreatment or undertreatment when translating trial targets to practice.
- 73. D.** Interpretation: For mild-to-moderate spontaneous ICH with presenting SBP 150-220 mm Hg, AHA/ASA guidance supports early smooth lowering toward 140, maintaining approximately 130-150. Excessively low or highly variable pressure may be harmful.
- 74. D.** Management principle: Standardized positioning and an appropriate validated cuff reduce common systematic errors. Talking, unsupported limbs, incorrect cuff size, and failure to rest can all distort office BP readings.

- 75. A.** Key point: The 2025 adult guideline emphasizes validated home BP monitoring and advises against relying on cuffless devices, including smartwatches, for accurate BP measurement until performance is sufficiently established.
- 76. D.** Clinical reasoning: An auscultatory gap can cause the examiner to mistake reappearance of sounds for systolic pressure if the cuff was not inflated high enough. Palpating systolic pressure first helps avoid this error.
- 77. C.** Why this is correct: Orthostatic hypotension is usually defined as a persistent fall of at least 20 mm Hg systolic or 10 mm Hg diastolic within 3 minutes of standing. Symptoms strengthen the clinical significance.
- 78. B.** Interpretation: Sleep health is increasingly recognized as part of cardiovascular prevention. Insufficient or irregular sleep can worsen sympathetic and metabolic factors, although it does not replace standard antihypertensive therapy.
- 79. D.** Management principle: Primary aldosteronism classically causes autonomous aldosterone secretion with suppressed renin; hypokalemia strengthens suspicion but is absent in many cases.
- 80. B.** Key point: Aldosterone-driven ENaC activity creates a lumen-negative potential that favors potassium and hydrogen secretion. Volume expansion also enhances renal handling that can sustain alkalosis.
- 81. C.** Clinical reasoning: OSA is common in resistant hypertension. Symptoms include loud snoring, witnessed apneas, nocturnal choking, and daytime sleepiness, although some patients are less symptomatic.
- 82. A.** Why this is correct: CKD is defined by persistent abnormalities of kidney structure or function, including reduced GFR or albuminuria. Both are important hypertension-related target-organ findings and risk markers.
- 83. A.** Interpretation: Home BP is valuable only when measured under standardized resting conditions. Technique should be corrected before medication is intensified based on potentially biased values.
- 84. C.** Management principle: CTA provides high-resolution noninvasive assessment of renal arterial anatomy. Duplex ultrasonography and MR angiography are alternatives depending on local expertise, renal function, body habitus, and contrast considerations.
- 85. B.** Key point: Orthostatic assessment compares recumbent or seated values with standing values, especially within 3 minutes. Symptoms and heart-rate response help clarify volume depletion, medications, or autonomic dysfunction.
- 86. A.** Clinical reasoning: Endothelin-1 is a powerful vasoconstrictor and can promote vascular growth and remodeling. Its net effect is usually pressor, although receptor-specific endothelial effects also exist.

- 87. D.** Why this is correct: Hypertension management aims to reduce total cardiovascular risk. Tobacco exposure independently increases vascular disease risk and acutely raises sympathetic tone, so cessation remains a major preventive intervention.
- 88. C.** Interpretation: Alpha-1 blockers can improve urinary symptoms but are not preferred first-line hypertension drugs because of outcome data and adverse effects such as first-dose orthostatic hypotension.
- 89. B.** Management principle: ACCOMPLISH demonstrated that combination choice can matter beyond office BP differences in high-risk patients. It does not mean one pair is universally best, but it supports rational complementary combination therapy.
- 90. C.** Key point: Practical sodium reduction focuses on processed foods, sauces, restaurant portions, and label reading. Behavioral specificity makes lifestyle counseling more actionable.
- 91. B.** Clinical reasoning: Volume excess is a major driver of hypertension in dialysis. Sodium restriction, adequate ultrafiltration, and assessment of dry weight are foundational before escalating medications indefinitely.
- 92. D.** Why this is correct: For many hypertensive emergencies, a controlled initial reduction of MAP by about 20-25% limits ongoing injury while avoiding hypoperfusion in vascular beds adapted to chronic hypertension. Specific emergencies have different targets.
- 93. C.** Interpretation: Short-acting immediate-release nifedipine is not appropriate for routine rapid BP lowering because the response can be excessive and unpredictable. Chronic therapy should use long-acting regimens.
- 94. B.** Management principle: The 2025 Endocrine Society guideline broadened case detection by suggesting screening of individuals with hypertension. Screening relies on aldosterone and renin with an aldosterone-to-renin relationship, while potassium and interfering medications affect interpretation.
- 95. A.** Key point: Hypertension control improves with reliable measurement, protocolized treatment, systematic follow-up, and multidisciplinary team care. Population management helps identify patients lost to follow-up or persistently uncontrolled.
- 96. B.** Clinical reasoning: Minoxidil is a potent arteriolar vasodilator that can cause marked sodium/water retention and reflex tachycardia. It is usually reserved for severe refractory hypertension with appropriate diuretic and rate-control coverage.
- 97. B.** Why this is correct: PATHWAY-2 supported the concept that sodium retention and aldosterone biology are important in resistant hypertension and showed strong efficacy of spironolactone as add-on therapy when safe.
- 98. C.** Interpretation: Primary aldosteronism is common among patients with resistant hypertension and often occurs without hypokalemia. Normal potassium therefore does not exclude it.

99. C. Management principle: Post-transplant hypertension is multifactorial. Calcineurin inhibitors, steroids, graft dysfunction, renal artery stenosis, and native renal disease can add, so management requires transplant-specific evaluation.

100. A. Key point: Beta-1 blockade lowers cardiac output and inhibits juxtaglomerular renin release. Some agents have additional vasodilating properties, but these are not universal class effects.

101. B. Clinical reasoning: With aging, aortic and large-artery stiffening reduces Windkessel buffering. The same stroke volume causes a larger systolic rise and faster diastolic runoff, widening pulse pressure.

102. C. Why this is correct: Cost is a modifiable adherence barrier. Generic substitution, formulary-aligned choices, combination pills when affordable, assistance programs, and team support can improve persistent treatment.

103. D. Interpretation: Home monitoring is most effective when linked to a treatment system—standardized technique, communication, protocol-based titration, and multidisciplinary follow-up—not simply data collection.

104. C. Management principle: Specific, measurable, achievable goals and self-monitoring support behavior change. Gradual progression is safer and more sustainable than an abrupt maximal regimen.

105. A. Key point: Hypertension management is iterative. Early follow-up detects adverse effects and inadequate response and supports timely titration rather than therapeutic inertia.

106. B. Clinical reasoning: Outside selected acute neurologic settings, chronic uncontrolled hypertension promotes stroke and vascular events. Long-term management integrates BP control with lipid treatment, antithrombotic therapy when indicated, and smoking cessation.

107. B. Why this is correct: Hypertension is common in HFpEF and adds to LV hypertrophy, stiffness, and congestion. Guideline-directed BP control and treatment of associated conditions are important.

108. A. Interpretation: Juxtaglomerular cells express beta-1 adrenergic receptors. Sympathetic stimulation of these receptors promotes renin release and thereby activates the renin-angiotensin-aldosterone system.

109. C. Management principle: Regimen complexity is a major adherence barrier. Once-daily dosing and fixed-dose combinations can reduce pill burden and are specifically favored for stage 2 treatment when practical.

110. D. Key point: Direct renin inhibition, like ACE inhibition and ARB therapy, can impair fetal renal development and should not be used during pregnancy.

111. D. Clinical reasoning: ACE inhibitors can cause fetal renal and other toxicity, especially later in gestation, and are not used during pregnancy. Chronic hypertension itself should still be treated with pregnancy-compatible medications.

- 112. A.** Why this is correct: Primary hyperparathyroidism can be associated with hypertension and is suggested by hypercalcemia plus an inappropriately elevated PTH. The kidney stones and constipation support hypercalcemia.
- 113. B.** For an otherwise eligible patient receiving intravenous thrombolysis for acute ischemic stroke, blood pressure should be reduced to below 185/110 mm Hg before alteplase or tenecteplase and kept within recommended post-treatment limits to reduce hemorrhagic risk.
- 114. D.** Management principle: Medication reconciliation is part of resistant-hypertension evaluation. Removing or substituting pressor agents may restore control and reduce unnecessary polypharmacy.
- 115. C.** Key point: The 2025 guideline emphasizes a goal below 130/80 mm Hg for most adults, while recognizing the need for individualized decisions in settings such as institutional care, limited life expectancy, or symptomatic orthostasis.
- 116. B.** Clinical reasoning: Familial hyperaldosteronism type I, or glucocorticoid-remediable aldosteronism, reflects a CYP11B1/CYP11B2 chimeric gene so aldosterone synthesis becomes ACTH regulated.
- 117. B.** Why this is correct: Following CHAP, ACOG supports 140/90 mm Hg as the threshold for initiating or titrating treatment of chronic hypertension in pregnancy. The trial established a treatment threshold rather than a single precise target BP.
- 118. C.** Interpretation: Loop diuretics retain substantial natriuretic utility at low GFR and are commonly used for edema and volume-dependent hypertension. Thiazide-like agents may still have adjunctive efficacy in advanced CKD but require close monitoring.
- 119. D.** Management principle: Thiazide and thiazide-like diuretics inhibit the Na-Cl cotransporter in the distal convoluted tubule. Their chronic antihypertensive effect also involves reduction in peripheral vascular resistance.
- 120. A.** Key point: Intermittent hypoxia and arousals trigger sympathetic surges, oxidative stress, and endothelial dysfunction. Repeated activation can persist into wakefulness and add to persistent and resistant hypertension.
- 121. B.** Clinical reasoning: ARNI/ACEi/ARB therapy, evidence-based beta blockade, MRAs, and SGLT2 inhibitors are core HFrEF therapies as clinically appropriate. BP management should be integrated with these outcome-improving drugs rather than competing with them.
- 122. A.** Why this is correct: Aldosterone induces ENaC and Na/K-ATPase activity in collecting-duct principal cells, increasing sodium reabsorption while promoting potassium and hydrogen secretion.
- 123. A.** Interpretation: Severe hypertension without acute target-organ damage is not a hypertensive emergency. The 2025 guideline emphasizes timely oral treatment and follow-up rather than reflexive rapid IV lowering.

- 124. C.** Management principle: Most dietary sodium comes from processed and restaurant foods. Sodium reduction lowers BP, particularly in salt-sensitive patients, and complements antihypertensive drug therapy.
- 125. A.** Key point: Potassium-containing salt substitutes can cause dangerous hyperkalemia when kidney excretion is impaired or when RAAS blockers and MRAs reduce potassium excretion.
- 126. A.** Clinical reasoning: Medical therapy for bilateral or nonsurgical primary aldosteronism centers on mineralocorticoid receptor blockade. Potassium, renal function, BP, and treatment response require follow-up.
- 127. C.** Why this is correct: Hypertensive acute pulmonary edema benefits from rapid afterload/preload reduction with titratable IV vasodilator therapy, oxygen/ventilatory support as needed, and decongestion when volume overloaded. Pure beta blockade can worsen acute decompensation.
- 128. D.** Interpretation: Non-dihydropyridine CCBs such as verapamil and diltiazem slow AV nodal conduction and can worsen bradycardia or conduction block. Dihydropyridines have much less nodal effect.
- 129. C.** Management principle: ACE, also called kininase II, degrades bradykinin. ACE inhibition can therefore increase bradykinin and substance P, contributing to cough and, rarely, angioedema.
- 130. D.** Key point: Salt sensitivity means BP changes substantially with sodium intake. It often reflects renal and neurohormonal differences in sodium handling and is more common with aging, CKD, and several metabolic conditions.
- 131. C.** Clinical reasoning: Stacking multiple RAAS blockers does not provide routine outcome benefit commensurate with risks such as hyperkalemia, hypotension, and renal injury. Combination therapy should use complementary mechanisms instead.
- 132. A.** Why this is correct: Elevated BP is systolic 120-129 with diastolic below 80. Normal is below 120 and below 80; stage 1 begins at 130 systolic or 80 diastolic.
- 133. D.** Interpretation: At a simplified physiologic level, MAP is approximately cardiac output times systemic vascular resistance, with central venous pressure contributing little. If output is fixed and resistance rises, MAP rises.
- 134. D.** Management principle: Combining loop and thiazide-type diuretics can cause powerful natriuresis but also hypokalemia, hyponatremia, magnesium loss, and kidney dysfunction. Laboratory surveillance is essential.
- 135. A.** Key point: Normal nocturnal BP usually falls relative to daytime values. A blunted nocturnal decline is termed nondipping and is associated with conditions such as sleep apnea, CKD, and autonomic dysfunction.

- 136. A.** Clinical reasoning: A reproducible inter-arm difference can reflect subclavian or other vascular disease. Using the higher-reading arm helps avoid undertreatment; the cause should be evaluated in context.
- 137. B.** Why this is correct: Cross-sectional imaging cannot reliably distinguish a functioning aldosterone-producing lesion from an incidental nodule. AVS is usually recommended to determine unilateral versus bilateral secretion before surgery, with limited exceptions.
- 138. B.** Interpretation: Nitric oxide diffuses into vascular smooth muscle and activates soluble guanylate cyclase, increasing cGMP and promoting relaxation. Reduced endothelial NO bioavailability adds to vascular dysfunction in hypertension.
- 139. C.** Management principle: Most stable atherosclerotic renal artery stenosis is managed initially with optimized medical therapy. Revascularization is reserved for selected clinical syndromes rather than anatomy alone.
- 140. A.** Key point: The classic hemodynamic combination of diuretic/volume depletion, ACE inhibitor or ARB, and NSAID can compromise both afferent and efferent support of filtration and precipitate AKI.
- 141. C.** Clinical reasoning: Chronic BP control reduces hemodynamic stress on the aortic wall. Beta blockers and RAAS-directed agents are commonly used depending on aneurysm etiology and patient factors.
- 142. D.** Why this is correct: Eplerenone is a more selective MRA and causes fewer sex-hormone-related adverse effects, although it is often more expensive and may require more frequent dosing.
- 143. C.** Interpretation: Decongestants with sympathomimetic activity can raise BP. A careful medication and substance inventory—including nonprescription products—is a core step in evaluating uncontrolled hypertension.
- 144. A.** Management principle: Catecholamines are metabolized continuously within these tumors, making plasma free or urinary fractionated metanephrines sensitive screening tests. Sampling conditions and medications can influence false-positive results.
- 145. D.** Key point: Clonidine stimulates central alpha-2 receptors, decreasing sympathetic outflow. Abrupt withdrawal can cause rebound sympathetic activation and severe hypertension.
- 146. B.** Clinical reasoning: Hypertension care is risk based. Lipid lowering, diabetes treatment, tobacco cessation, and BP control provide complementary cardiovascular protection.
- 147. C.** Why this is correct: RAAS blockade can raise potassium and cause an expected modest creatinine change; a large rise suggests volume depletion, renal artery disease, NSAID interaction, or other problems. Monitoring supports safe titration.

148. D. Interpretation: Chronic pressure overload can cause LV hypertrophy, which may be suggested by ECG and assessed more sensitively by echocardiography when indicated.

149. C. Management principle: Resistant hypertension is above-goal BP despite three complementary agents, commonly a RAAS blocker, long-acting CCB, and diuretic at maximally tolerated doses, or controlled BP requiring at least four drugs. Adherence and out-of-office control should be confirmed.

150. A. Key point: Weight reduction is an effective nonpharmacologic antihypertensive intervention and also improves metabolic risk, sleep apnea, and medication requirements in many patients.

151. C. Clinical reasoning: NSAIDs can reduce renal prostaglandin-mediated vasodilation, promote sodium retention, and blunt the effect of diuretics and RAAS blockers in susceptible patients.

152. D. Why this is correct: Diabetes is a high-risk condition in which stage 1 hypertension generally merits pharmacologic treatment plus lifestyle therapy; drug choice is influenced by kidney and cardiovascular status.

153. A. Interpretation: Behavioral stress-reduction approaches may provide modest BP benefit and support adherence, but they are adjuncts. Stage 2 hypertension generally warrants drug therapy in addition to lifestyle intervention.

154. D. Management principle: OSA is highly prevalent in resistant hypertension. Recognition and treatment improve sleep-related risk and can modestly improve BP, while drug therapy and lifestyle management continue.

155. C. Key point: For lower-risk stage 1 hypertension, the 2025 guideline recommends an initial lifestyle trial, with medication if average BP remains at least 130/80 after 3-6 months.

156. D. Clinical reasoning: Hydralazine can cause a dose- and susceptibility-related drug-induced lupus syndrome, classically associated with arthralgias and antihistone antibodies.

157. C. Why this is correct: Current guidance favors two first-line drugs of different classes for stage 2 hypertension, preferably in a single-pill combination to improve adherence and shorten time to control.

158. C. Interpretation: Teach-back reveals misunderstandings and supports safer medication use. Clear language, written schedules, pill organization, and family or pharmacist support can further improve adherence.

159. A. Management principle: Dihydropyridine calcium-channel blockers such as amlodipine act predominantly on vascular L-type calcium channels, lowering arteriolar resistance. Peripheral edema is a common dose-related adverse effect.

160. A. Key point: First-line therapy generally centers on thiazide-type diuretics, ACE inhibitors/ARBs, and long-acting dihydropyridine CCBs. Beta blockers remain important when comorbid conditions provide a compelling reason.

161. B. Clinical reasoning: AT1-receptor activation causes vasoconstriction, stimulates aldosterone, enhances sodium retention and sympathetic activity, and constricts the efferent arteriole. These actions support arterial pressure and extracellular volume.

162. D. Why this is correct: Improper measurement and nonadherence can mimic resistance. True resistant hypertension requires accurate BP measurement, appropriate regimen intensity, adherence assessment, and exclusion of a white-coat effect.

163. B. Interpretation: RAAS blockers dilate the efferent arteriole and can reduce GFR when filtration depends on angiotensin II, especially in bilateral severe renal artery stenosis or stenosis to a solitary functioning kidney.

164. B. Management principle: Resistant hypertension management emphasizes an effective long-acting thiazide-like diuretic and, when safe, an MRA such as spironolactone. Pseudoresistance and secondary causes should be addressed first.

165. A. Key point: Natriuretic peptides promote sodium excretion and vasodilation and oppose the renin-angiotensin-aldosterone system. They are physiologic counter-regulators of pressure and volume expansion.

166. C. Clinical reasoning: Chronic hypertension can cause arteriolar and glomerular injury leading to progressive CKD, usually with a relatively bland sediment. The diagnosis is clinical and requires exclusion of competing renal disease when features are atypical.

167. A. Why this is correct: Masked hypertension is normal office BP with elevated out-of-office BP. It can be missed if assessment relies only on clinic readings and carries cardiovascular risk.

168. D. Interpretation: Certain beta blockers—such as carvedilol, metoprolol succinate, and bisoprolol—have established HFrEF outcome benefit. Initiation and titration should occur in a stable, compensated patient.

169. B. Management principle: Persistent severe hypertension in pregnancy is an obstetric emergency because maternal stroke risk rises sharply. IV labetalol, IV hydralazine, or immediate-release oral nifedipine are commonly used according to protocol while the cause and need for delivery are assessed.

170. B. Key point: Chronic clonidine suppresses central sympathetic outflow. Abrupt discontinuation can cause marked rebound sympathetic activity; the drug should generally be tapered rather than stopped suddenly.

171. A. Clinical reasoning: ADPKD causes bilateral renal cystic disease, often with early hypertension from intrarenal ischemia and RAAS activation. Family history and enlarged multicystic kidneys are characteristic.

172. A. Why this is correct: When renal perfusion is critically reduced, angiotensin II-mediated efferent constriction helps maintain filtration. ACE inhibition can therefore cause a substantial creatinine rise in bilateral severe RAS or stenosis of a solitary functioning kidney.

173. C. Interpretation: Chronic hypertension causes adaptive vascular remodeling and a rightward shift of cerebral autoregulation. A pressure that is normal for another patient may fall below this patient's lower autoregulatory limit if BP is reduced too abruptly.

174. B. Management principle: Albuminuria independently reflects renal injury and cardiovascular risk. It also influences antihypertensive drug selection, particularly the use of renin-angiotensin system blockade when appropriate.

175. A. Key point: Long-term pressure regulation depends heavily on renal sodium balance. A rightward-shifted pressure-natriuresis curve means higher pressure is needed to achieve sodium balance, favoring persistent hypertension.

176. C. Clinical reasoning: Liddle syndrome is caused by increased ENaC activity, leading to sodium retention, low renin, low aldosterone, hypokalemia, and alkalosis. It responds to ENaC blockade with amiloride or triamterene rather than spironolactone.

177. A. Why this is correct: In catecholamine excess, isolated beta blockade can worsen vasoconstriction by leaving alpha effects unopposed. Alpha blockade or titratable vasodilation is established before beta blockade if needed.

178. D. Interpretation: ACE inhibitors or ARBs reduce intraglomerular pressure and albuminuria and are favored in albuminuric CKD when tolerated. Potassium and renal function require monitoring.

179. D. Management principle: Blood-pressure category is determined by the higher category of systolic or diastolic pressure. Systolic pressure at least 140 mm Hg is stage 2 hypertension even when diastolic pressure is normal.

180. B. Key point: Many renal parenchymal disorders promote sodium and water retention. Volume expansion, together with RAAS and sympathetic activation in some conditions, can elevate BP.

181. B. Clinical reasoning: In diabetes with albuminuric CKD, ACE inhibitor or ARB therapy reduces albuminuria and is a preferred foundational option when tolerated. Dual ACEi/ARB therapy should not be used routinely.

182. A. Why this is correct: ACE inhibitors, ARBs, and direct renin inhibitors can injure the fetal kidneys and are contraindicated in pregnancy. Preconception planning allows transition to a safer regimen.

183. D. Interpretation: Thiazide and thiazide-like diuretics can cause hyponatremia, hypokalemia, hypomagnesemia, and hyperuricemia. Electrolytes should be monitored after initiation and dose changes in susceptible patients.

- 184. A.** Management principle: Obesity adds to hypertension through renal, sympathetic, hormonal, and sleep-related pathways. Effective weight loss—behavioral, pharmacologic, or surgical when indicated—often improves BP.
- 185. B.** Key point: ACE inhibitors reduce aldosterone signaling, and MRAs block the mineralocorticoid receptor. Their combination can be useful but requires careful potassium and kidney-function monitoring.
- 186. C.** Clinical reasoning: A substantial fraction of pheochromocytomas/paragangliomas are hereditary. Genotype can influence tumor distribution, metastatic risk, surveillance, and family counseling.
- 187. B.** Why this is correct: Hemorrhages, exudates/cotton-wool spots, and especially papilledema indicate advanced hypertensive retinal injury and raise concern for a hypertensive emergency when accompanied by severe BP elevation.
- 188. D.** Interpretation: Regular aerobic activity lowers resting BP and cardiovascular risk; resistance exercise can add benefit. Exercise plans should reflect baseline fitness, symptoms, and CVD.
- 189. D.** Beta blockers are useful when hypertension coexists with chronic ischemic heart disease and tachycardia because they lower blood pressure and heart rate, reducing myocardial oxygen demand when no contraindication is present.
- 190. C.** Key point: Higher dietary potassium can favor natriuresis and lower BP, but CKD, ACE inhibitors, ARBs, MRAs, and other factors can increase hyperkalemia risk. Advice must therefore be individualized.
- 191. B.** Clinical reasoning: A cuff that is too small for the arm tends to overestimate BP. Proper cuff selection remains important even with automated devices.
- 192. B.** Why this is correct: MRAs reduce potassium excretion and can cause clinically important hyperkalemia, especially in CKD or with ACE inhibitors/ARBs. Safety depends on renal function, potassium, dose, and monitoring.
- 193. C.** Interpretation: Effective CPAP reduces apneas, hypoxemia, and sympathetic surges. Average BP reductions are usually modest, so antihypertensive therapy and lifestyle measures often remain necessary.
- 194. D.** Management principle: Thyroid hormone increases heart rate, contractility, and cardiac output. Peripheral vasodilation can lower diastolic pressure, producing a widened pulse pressure and predominantly systolic hypertension.
- 195. B.** Key point: Beta blockers and non-dihydropyridine CCBs can slow AV nodal conduction and lower BP. Choice depends on LV systolic function, conduction disease, pulmonary disease, and other comorbidities.

196. B. Clinical reasoning: Stage 1 hypertension is systolic 130-139 mm Hg or diastolic 80-89 mm Hg. Elevated BP is systolic 120-129 with diastolic below 80.

197. A. Why this is correct: Volume excess is a frequent cause of difficult BP control in advanced CKD. Loop diuretics are often needed for effective natriuresis when GFR is substantially reduced or edema is present.

198. B. Interpretation: Heavy alcohol use can elevate BP and impair adherence. Reduction toward low intake or abstinence when appropriate can improve BP and overall health.

199. C. Management principle: Standing transiently reduces venous return and arterial pressure. Less baroreceptor stretch increases sympathetic tone and reduces parasympathetic activity, producing tachycardia and vasoconstriction within seconds.

200. A. For primary prevention without established CVD, diabetes, or CKD, the 2025 guideline uses PREVENT-CVD risk to guide therapy in stage 1 hypertension. A 10-year risk of at least 7.5% supports adding medication to lifestyle treatment.