COMPREHENSIVE MOCK EXAM 2

Supplemental to: Hypertension Board Review 2026-2027

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American Hypertension Specialist Certification Program (AHSCP) Certification Examination for Hypertension Specialists - 200-question MEDREV supplemental simulation; A-D single-best-answer format; domain-weighted to the official 2026 AHSCP content outline

Board-Review Questions

1. In an otherwise stable adult, mean arterial pressure is most directly determined by which hemodynamic relationship?

A. The product of cardiac output and systemic vascular resistance, with central venous pressure usually contributing little.

B. The sum of stroke volume and systemic vascular resistance, because flow and resistance add directly in the arterial tree.

C. The product of heart rate and arterial compliance, because elastic recoil alone determines steady perfusion pressure.

D. The difference between systolic and diastolic pressure, because pulse pressure is the principal determinant of mean pressure.

2. A patient with salt-sensitive hypertension requires a higher arterial pressure than before to excrete the same sodium load. Which renal concept best explains this finding?

A. A leftward shift of the pressure-natriuresis relationship, so sodium excretion increases at lower pressures than before.

B. Loss of glomerular autoregulation alone, causing sodium excretion to become independent of renal perfusion pressure.

C. A rightward shift of the pressure-natriuresis relationship, so a higher pressure is required to achieve sodium balance.

D. Suppression of tubular sodium reabsorption at every pressure, which would favor lower rather than higher long-term pressure.

3. Renal perfusion pressure falls abruptly in a patient with otherwise intact kidney function. Which change most directly stimulates renin release from juxtaglomerular cells?

A. Increased afferent arteriolar stretch with increased macula densa sodium chloride delivery, signaling that renal perfusion is adequate.

B. Reduced afferent arteriolar stretch together with increased beta-1 sympathetic signaling and reduced macula densa sodium chloride delivery.

C. Direct stimulation of mineralocorticoid receptors in the collecting duct, which is the principal sensor controlling renin secretion.

D. Increased atrial natriuretic peptide signaling at the juxtaglomerular apparatus, which normally suppresses rather than stimulates renin.

4. Which effect is most characteristic of angiotensin II acting at the AT1 receptor?

A. Arteriolar vasoconstriction with aldosterone stimulation, increased proximal sodium reabsorption, and facilitation of sympathetic activity.

B. Generalized vasodilation with reduced aldosterone secretion and suppression of proximal tubular sodium transport.

C. Selective blockade of sympathetic neurotransmission with increased renal sodium excretion and reduced thirst.

D. Direct opening of epithelial sodium channels in the collecting duct without any vascular or adrenal effects.

5. Aldosterone increases distal nephron sodium retention primarily through which cellular effect?

A. Upregulation and activation of epithelial sodium channels and the basolateral sodium-potassium ATPase in principal cells.

B. Inhibition of the thiazide-sensitive sodium-chloride cotransporter in the distal convoluted tubule, producing natriuresis.

C. Blockade of the sodium-potassium-2 chloride cotransporter in the thick ascending limb, reducing medullary concentration.

D. Inhibition of proximal tubular sodium-hydrogen exchange, causing bicarbonaturia and sodium wasting.

6. A patient with chronic hypertension develops diminished heart-rate responses to acute changes in arterial pressure. What is the best explanation?

A. Carotid baroreceptors become permanently maximally activated, causing persistent vagal stimulation despite the elevated pressure.

B. Aortic baroreceptors stop sensing vessel stretch completely, so all short-term autonomic regulation is abolished.

C. Baroreceptor firing becomes the main long-term controller of extracellular volume, replacing renal pressure-natriuresis mechanisms.

D. Arterial baroreceptors reset to operate around a higher pressure, reducing their ability to provide sustained long-term pressure correction.

7. Which endothelial mediator normally lowers vascular tone by activating soluble guanylate cyclase in vascular smooth muscle?

A. Nitric oxide, which increases cyclic GMP and promotes smooth-muscle relaxation.

B. Endothelin-1, which primarily activates potent vasoconstrictor pathways in vascular smooth muscle.

C. Angiotensin II, which primarily activates AT1-mediated vasoconstriction and sodium-retaining pathways.

D. Norepinephrine, which commonly raises resistance through alpha-1 adrenergic receptor activation.

8. An older adult has a blood pressure of 168/66 mm Hg with no secondary cause identified. Which vascular change most strongly contributes to this pattern?

A. Marked increase in large-artery compliance, which amplifies elastic buffering and selectively raises systolic pressure.

B. Loss of large-artery compliance, which raises systolic pressure and widens pulse pressure as the aorta stiffens.

C. Isolated reduction in venous capacitance, which primarily lowers pulse pressure while leaving systolic pressure unchanged.

D. A fall in stroke volume with preserved arterial stiffness, which typically narrows rather than widens pulse pressure.

9. Why can reflected arterial pressure waves contribute more to late systolic pressure in an older patient with stiff arteries?

A. Higher pulse-wave velocity returns the reflected wave earlier, augmenting central systolic pressure before ventricular ejection has ended.

B. Lower pulse-wave velocity returns reflected waves earlier, causing them to merge with early diastole and raise systolic pressure.

C. Greater venous compliance accelerates reflected waves through the venous system and transfers them directly to the aorta.

D. Reduced arterial stiffness causes the reflected wave to return during systole instead of diastole, increasing central pressure.

10. A patient has volume expansion and increased atrial stretch. Which response is expected from natriuretic peptide signaling?

A. Increased natriuresis and vasodilation with suppression of renin and aldosterone activity.

B. Increased renin release and aldosterone secretion with enhanced distal sodium reabsorption.

C. Systemic vasoconstriction with increased sympathetic outflow and reduced glomerular filtration.

D. Selective potassium retention without meaningful effects on sodium balance or vascular tone.

11. Which nephron transporter is directly inhibited by thiazide and thiazide-like diuretics?

A. The sodium-potassium-2 chloride cotransporter in the thick ascending limb of Henle.

B. The sodium-chloride cotransporter in the distal convoluted tubule.

C. The epithelial sodium channel in the cortical collecting duct and connecting tubule.

D. The sodium-glucose cotransporter in the proximal tubule that reabsorbs filtered glucose.

12. Which nephron transporter is directly inhibited by loop diuretics such as furosemide?

A. The sodium-potassium-2 chloride cotransporter in the thick ascending limb of the loop of Henle.

B. The sodium-chloride cotransporter in the distal convoluted tubule, the principal target of thiazide diuretics.

C. The epithelial sodium channel in principal cells, the target of amiloride and triamterene.

D. The mineralocorticoid receptor in principal cells, the target of spironolactone and eplerenone.

13. Dihydropyridine calcium channel blockers lower blood pressure mainly through which action?

A. Blockade of beta-1 receptors in the sinoatrial node, reducing heart rate as the dominant antihypertensive mechanism.

B. Direct inhibition of renin enzymatic activity, preventing conversion of angiotensinogen to angiotensin I.

C. Blockade of L-type calcium channels in vascular smooth muscle, reducing arteriolar contraction and systemic vascular resistance.

D. Activation of endothelial mineralocorticoid receptors, increasing nitric oxide production and vasodilation.

14. A patient develops a persistent dry cough after starting an ACE inhibitor. Which mechanism best explains this adverse effect?

A. Excessive activation of AT1 receptors by accumulated angiotensin II, increasing bronchial smooth-muscle tone.

B. Direct stimulation of histamine release from mast cells by blockade of the angiotensin II receptor.

C. Inhibition of epithelial sodium channels in airway epithelium, causing local sodium accumulation and cough.

D. Reduced degradation of bradykinin and related peptides after inhibition of angiotensin-converting enzyme.

15. How do angiotensin receptor blockers lower blood pressure?

A. They inhibit angiotensin-converting enzyme and therefore directly increase bradykinin concentrations to the same degree as ACE inhibitors.

B. They block mineralocorticoid receptors in the collecting duct while leaving vascular angiotensin signaling unchanged.

C. They directly inhibit renin secretion from juxtaglomerular cells without affecting downstream angiotensin receptors.

D. They block AT1 receptor signaling, reducing angiotensin II-mediated vasoconstriction and aldosterone-dependent sodium retention.

16. A beta-1 selective blocker lowers blood pressure partly by reducing which renal signal?

A. Renin release from juxtaglomerular cells, thereby reducing downstream angiotensin II and aldosterone generation.

B. Aldosterone binding to mineralocorticoid receptors, an effect produced directly by spironolactone rather than beta blockade.

C. Epithelial sodium channel activity through direct channel blockade, an effect produced by amiloride rather than beta blockade.

D. Sodium-chloride cotransporter activity in the distal convoluted tubule, the direct target of thiazide diuretics.

17. An alpha-1 adrenergic antagonist lowers arterial pressure primarily through which immediate vascular effect?

A. Increased alpha-1 receptor signaling in resistance vessels, producing reflex vasoconstriction and a lower cardiac output.

B. Direct inhibition of aldosterone synthesis in the adrenal cortex, causing natriuresis without a vascular effect.

C. Reduced arteriolar and venous smooth-muscle constriction, lowering systemic vascular resistance and venous tone.

D. Selective slowing of atrioventricular nodal conduction, which is the principal action of non-dihydropyridine calcium channel blockers.

18. Central alpha-2 agonists such as clonidine reduce blood pressure mainly through which mechanism?

A. Increased norepinephrine release from peripheral sympathetic nerve terminals, producing sustained vasoconstriction.

B. Direct blockade of L-type calcium channels in resistance arteries, producing the same primary action as amlodipine.

C. Irreversible inhibition of angiotensin-converting enzyme, reducing angiotensin II while increasing bradykinin.

D. Reduced sympathetic outflow from the central nervous system, lowering peripheral vascular resistance and heart rate.

19. Spironolactone lowers blood pressure by antagonizing which receptor?

A. The glucocorticoid receptor, reducing cortisol-mediated vascular responsiveness while leaving sodium handling unchanged.

B. The angiotensin II AT2 receptor, preventing vasodilator signaling and thereby lowering systemic vascular resistance.

C. The vasopressin V2 receptor, causing selective aquaresis as the primary mechanism of chronic blood pressure control.

D. The mineralocorticoid receptor, reducing aldosterone-mediated sodium retention and potassium secretion in the distal nephron.

20. A young patient has severe hypertension, hypokalemia, metabolic alkalosis, suppressed renin, and suppressed aldosterone. Which inherited mechanism is most likely?

A. An aldosterone-producing adrenal adenoma causing autonomous mineralocorticoid secretion and elevated aldosterone levels.

B. Loss of sodium-chloride cotransporter function causing Gitelman syndrome with renal salt wasting and low blood pressure.

C. A gain-of-function mutation of the epithelial sodium channel causing Liddle syndrome.

D. Loss of NKCC2 function causing Bartter syndrome with renal salt wasting and secondary hyperreninemia.

21. A patient with hypertension and hypokalemic metabolic alkalosis has low renin and low aldosterone after chronic ingestion of large amounts of natural licorice. What mechanism is responsible?

A. Inhibition of 11-beta-hydroxysteroid dehydrogenase type 2 allows cortisol to activate renal mineralocorticoid receptors.

B. Activation of aldosterone synthase causes marked autonomous aldosterone production despite suppression of renin.

C. Inhibition of epithelial sodium channels produces renal sodium wasting followed by secondary hyperaldosteronism.

D. Blockade of glucocorticoid receptors increases ACTH and causes isolated cortisol deficiency without mineralocorticoid effects.

22. A patient has hyperkalemic hypertension and metabolic acidosis caused by a familial increase in distal sodium-chloride cotransporter activity. Which disorder is most consistent?

A. Gordon syndrome, also called pseudohypoaldosteronism type II, which often responds dramatically to a thiazide.

B. Liddle syndrome, which typically produces hypokalemic metabolic alkalosis from constitutive epithelial sodium channel activation.

C. Bartter syndrome, which causes renal salt wasting, secondary hyperreninemia, and usually normal or low blood pressure.

D. Gitelman syndrome, which causes distal salt wasting with hypokalemia, metabolic alkalosis, and low or normal blood pressure.

23. Why are Black adults, older adults, patients with chronic kidney disease, and people with metabolic syndrome often described as having greater salt sensitivity of blood pressure?

A. They uniformly have autonomous aldosterone-producing adenomas, making dietary sodium irrelevant to long-term pressure control.

B. They have complete loss of the renin-angiotensin system, so blood pressure becomes independent of extracellular volume.

C. Their blood pressure may change more substantially with sodium balance because renal sodium handling and neurohormonal responses favor volume dependence.

D. They cannot increase natriuretic peptide secretion, making sodium intake the sole determinant of systolic and diastolic pressure.

24. What is the principal immediate hemodynamic consequence of increased systemic sympathetic nerve activity in hypertension?

A. Selective venous dilation with reduced heart rate and suppressed renin release, lowering both preload and systemic resistance.

B. Direct inhibition of vascular alpha-1 receptors, producing reduced resistance despite increased circulating catecholamines.

C. Increased arteriolar vasoconstriction and cardiac stimulation, often accompanied by enhanced renal renin release and sodium retention.

D. Isolated reduction in proximal tubular sodium reabsorption without meaningful cardiac or vascular effects.

25. Endothelin-1 contributes to elevated vascular tone through which general action?

A. Potent vasoconstriction through endothelin receptor signaling in vascular smooth muscle.

B. Potent systemic vasodilation through cyclic GMP generation identical to the action of nitric oxide.

C. Direct blockade of sympathetic ganglia with reduced norepinephrine release into resistance vessels.

D. Selective inhibition of aldosterone synthesis without any direct effect on vascular smooth muscle.

26. In untreated essential hypertension, increased vascular resistance may persist even after an initiating neurohormonal stimulus wanes. Which structural process contributes to this persistence?

A. Vascular remodeling with increased wall-to-lumen ratio and reduced small-artery caliber.

B. Progressive enlargement of resistance-vessel lumens with thinning of the media, which would lower rather than raise resistance.

C. Loss of all smooth-muscle contractile proteins, making arteriolar tone independent of local and neural stimuli.

D. Selective dilation of precapillary arterioles without changes in vessel structure, which would reduce systemic resistance.

27. Which change in renal microvascular physiology is expected with efferent arteriolar constriction by angiotensin II?

A. A selective fall in glomerular capillary pressure with a lower filtration fraction despite preserved renal perfusion.

B. Complete abolition of proximal sodium reabsorption because angiotensin II has no tubular actions beyond the glomerulus.

C. A marked increase in renal blood flow from preferential efferent vasodilation, lowering glomerular filtration pressure.

D. Maintenance or increase of glomerular capillary pressure despite reduced renal perfusion, at the cost of increased filtration fraction.

28. A patient starts an ACE inhibitor and has a modest fall in intraglomerular pressure. Which arteriolar change is primarily responsible?

A. Efferent arteriolar dilation from reduced angiotensin II signaling.

B. Afferent arteriolar constriction from increased angiotensin II signaling at the AT1 receptor.

C. Efferent arteriolar constriction from increased aldosterone release after ACE inhibition.

D. Afferent arteriolar dilation caused by direct blockade of renal L-type calcium channels.

29. Which epidemiologic statement best describes the relationship between usual blood pressure and cardiovascular risk across adult populations?

A. Cardiovascular risk is flat across all pressures below 160/100 mm Hg and rises only after that threshold is crossed.

B. Cardiovascular risk rises progressively with higher blood pressure, without a sharp biologic threshold at the diagnostic cutoff for hypertension.

C. Diastolic pressure determines essentially all risk after age 60, whereas systolic pressure becomes clinically unimportant.

D. Blood pressure predicts stroke but has little relationship to heart failure, chronic kidney disease, or coronary events.

30. Which mechanism best explains why obesity can promote hypertension?

A. Obesity consistently suppresses sympathetic activity and renal sodium reabsorption, producing a chronic low-volume state.

B. Adiposity prevents aldosterone secretion and therefore lowers distal sodium reabsorption despite increased caloric intake.

C. Expanded sodium retention together with sympathetic and renin-angiotensin-aldosterone activation can increase both volume and vascular tone.

D. Obesity lowers renal venous pressure and improves pressure natriuresis, making hypertension uncommon unless another disease is present.

31. A large patient has an upper-arm circumference that exceeds the range of the standard adult cuff. What is the most important measurement correction?

A. Use a validated cuff whose bladder dimensions are appropriate for the measured arm circumference rather than forcing a cuff that is too small.

B. Use the standard cuff and subtract 10 mm Hg from the systolic reading to compensate for the patient's arm size.

C. Place the standard cuff over clothing so the fabric distributes pressure more evenly around the larger upper arm.

D. Measure only at the wrist with the hand below heart level because wrist pressure is more reliable in large arms.

32. Which office technique best minimizes avoidable error before obtaining a routine seated blood pressure?

A. Measure immediately after the patient walks into the room while standing, because exertion reveals the patient's usual pressure burden.

B. Seat the patient quietly with back supported, feet on the floor, arm supported at heart level, and allow several minutes of rest before measurement.

C. Have the patient sit on the examination table with legs dangling and keep the cuffed arm unsupported below heart level.

D. Permit conversation during the reading and measure through a thin sleeve, because these factors do not materially affect pressure.

33. A patient's first office blood pressure is 148/86 mm Hg after proper preparation. What is the best way to improve the reliability of the visit-level estimate?

A. Accept the first reading as definitive because repeated measurements introduce regression to the mean and reduce diagnostic accuracy.

B. Obtain additional readings using the same standardized technique and use the average rather than relying on a single measurement.

C. Repeat the pressure only after exercise so the clinician can determine the patient's maximum physiologic systolic response.

D. Switch immediately to a wrist device for the second reading because changing measurement sites reduces observer-related bias.

34. A patient has repeated clinic pressures around 146/88 mm Hg but well-performed home readings average 124/76 mm Hg. Which phenotype is most likely?

A. Masked hypertension, in which clinic pressure is normal but home or ambulatory pressure is persistently elevated.

B. Sustained hypertension, because any clinic systolic value above 140 mm Hg proves that out-of-office pressure is also elevated.

C. White-coat hypertension or white-coat effect, with office pressure higher than out-of-office pressure.

D. Orthostatic hypertension, because the discrepancy between clinic and home readings indicates an abnormal standing response.

35. A patient has clinic pressures near 122/74 mm Hg but repeated home and daytime ambulatory pressures are clearly hypertensive. What is the best description?

A. Masked hypertension, which carries cardiovascular risk despite apparently normal office readings.

B. White-coat hypertension, which requires elevated office pressure with lower values outside the clinic setting.

C. Isolated systolic hypertension, which requires an elevated systolic pressure and does not depend on measurement setting.

D. Pseudohypertension, which is caused by noncompressible arteries and cannot be diagnosed from this office-home pattern alone.

36. A patient with treated hypertension has a normal daytime ambulatory average but elevated pressures during sleep and no expected nocturnal fall. Why is this finding clinically important?

A. Nocturnal hypertension and a nondipping pattern can identify residual blood pressure burden associated with higher cardiovascular and kidney risk.

B. A nondipping pattern is physiologic in all adults and therefore has no relationship to target-organ risk or treatment assessment.

C. Nighttime pressure should be ignored because only awake systolic blood pressure predicts stroke, heart failure, and kidney outcomes.

D. The finding proves pheochromocytoma because catecholamine tumors are the only cause of elevated blood pressure during sleep.

37. How should orthostatic blood pressure be assessed in a patient with dizziness after standing?

A. Measure only after the patient has walked for 10 minutes, because early standing values are too unstable to identify orthostatic hypotension.

B. Compare right-arm and left-arm seated readings, because an inter-arm difference is the diagnostic definition of orthostatic hypotension.

C. Measure blood pressure and heart rate after a period supine and again after standing, including an assessment within the first 3 minutes.

D. Measure one standing systolic pressure without heart rate, because diastolic pressure and pulse response do not add useful information.

38. Repeated simultaneous measurements show a persistent systolic blood pressure difference of 22 mm Hg between arms. What is the best next interpretation?

A. Average the two arms permanently and ignore the difference because inter-arm pressure differences have no vascular significance.

B. Confirm the finding and consider upper-extremity arterial disease or other vascular pathology; subsequent routine measurements generally use the higher-pressure arm.

C. Use only the lower-pressure arm for treatment decisions because it minimizes the risk of overtreating systemic hypertension.

D. Diagnose primary aldosteronism because a large inter-arm pressure difference is a characteristic endocrine screening sign.

39. An untreated adult has an average standardized office blood pressure of 136/84 mm Hg. How is this classified under the current U.S. adult framework?

A. Elevated blood pressure, which is limited to systolic pressure of 120-129 mm Hg with diastolic pressure below 80 mm Hg.

B. Stage 2 hypertension, which begins at systolic pressure of 140 mm Hg or diastolic pressure of 90 mm Hg.

C. Stage 1 hypertension, because either systolic pressure of 130-139 mm Hg or diastolic pressure of 80-89 mm Hg meets that category.

D. Normal blood pressure, because treatment categories begin only when both systolic and diastolic pressures are at least 140/90 mm Hg.

40. Which initial laboratory evaluation is most appropriate for a newly diagnosed adult with hypertension when there are no immediately obvious secondary-cause clues?

A. Order plasma metanephrines, adrenal CT, renal angiography, and cortisol testing in every patient before confirming the blood pressure diagnosis.

B. Assess kidney function and electrolytes, glucose status, lipids, urinalysis or albuminuria, and other baseline tests guided by clinical context.

C. Skip laboratory evaluation unless systolic pressure exceeds 180 mm Hg, because uncomplicated hypertension does not affect kidney or metabolic risk.

D. Obtain only a complete blood count, because electrolytes, kidney function, urine findings, and metabolic risk do not influence treatment selection.

41. A hypertensive patient has an elevated urine albumin-to-creatinine ratio with a preserved estimated GFR. Why is the urine finding important?

A. Albuminuria is expected with uncomplicated hypertension and has little prognostic value until estimated GFR falls below 30 mL/min/1.73 m2.

B. Albuminuria is a marker of kidney target-organ damage and independently increases cardiovascular and kidney risk even when eGFR is preserved.

C. Albuminuria proves renal artery stenosis and should directly trigger revascularization without additional clinical evaluation.

D. Albuminuria primarily reflects urinary tract infection and should not influence antihypertensive drug selection or risk assessment.

42. A long-standing hypertensive patient has high-voltage QRS complexes with a lateral strain pattern on ECG. What target-organ change is most likely?

A. Acute right ventricular infarction, because lateral repolarization abnormalities are specific for right-sided ischemia.

B. Pericardial tamponade, which typically produces high QRS voltage and a chronic lateral strain pattern.

C. Isolated left atrial enlargement without ventricular remodeling, because hypertension does not produce ventricular hypertrophy.

D. Left ventricular hypertrophy from chronic pressure overload, producing high QRS voltage with a lateral repolarization strain pattern.

43. When is echocardiography most useful in the evaluation of a hypertensive adult?

A. In every newly diagnosed adult regardless of findings, because echocardiography is required before any antihypertensive medication can be prescribed.

B. When symptoms, examination, ECG findings, or clinical questions make assessment of ventricular structure, function, or other cardiac disease relevant to management.

C. Only after a hypertensive emergency, because stable hypertension never causes structural cardiac changes detectable by echocardiography.

D. Only when the office blood pressure is normal, because echocardiography is used mainly to diagnose masked hypertension rather than cardiac damage.

44. A patient with severe chronic hypertension has flame hemorrhages, cotton-wool spots, and optic-disc swelling. What does this examination indicate?

A. A benign age-related retinal change that does not correlate with systemic blood pressure severity or acute organ injury.

B. Diabetic retinopathy alone, because optic-disc swelling cannot occur as part of severe hypertensive retinal injury.

C. Advanced hypertensive retinopathy consistent with acute or severe target-organ injury requiring urgent clinical assessment.

D. Isolated glaucoma, because retinal hemorrhages and cotton-wool spots are not seen with accelerated hypertension.

45. A patient with long-standing hypertension has a rising serum creatinine and persistent albuminuria. Which interpretation is most appropriate?

A. These findings may represent hypertensive kidney target-organ damage, but alternative or primary kidney disease must still be considered.

B. The findings prove that hypertension is secondary to pheochromocytoma, because kidney injury is not caused by sustained primary hypertension.

C. The creatinine rise is irrelevant to blood pressure management because renal function does not influence drug choice or cardiovascular risk.

D. Albuminuria excludes hypertensive nephrosclerosis, because hypertension can reduce GFR but never increases urinary albumin excretion.

46. A 34-year-old with newly recognized hypertension has no obesity, no family history, and a blood pressure of 178/112 mm Hg. What is the most appropriate diagnostic approach?

A. Confirm accurate blood pressure measurement and evaluate for secondary hypertension because the severity and young age raise pretest probability.

B. Assume essential hypertension without further evaluation because secondary causes occur only when potassium is low.

C. Delay evaluation for several years because age under 40 makes target-organ damage and secondary hypertension unlikely.

D. Order only a lipid profile because secondary hypertension cannot present with severe pressure unless kidney failure is already advanced.

47. Which out-of-office method provides the most complete assessment of daytime, nighttime, and 24-hour blood pressure patterns?

A. A single pharmacy kiosk measurement obtained after shopping, because it captures both daytime and nighttime pressure burden.

B. Ambulatory blood pressure monitoring with repeated automated measurements across waking and sleeping periods.

C. One home reading each month, because infrequent home measurements provide the same circadian information as ambulatory monitoring.

D. Palpation of the radial pulse during sleep, because pulse amplitude can substitute for nocturnal systolic and diastolic measurements.

48. A patient brings a home blood pressure log showing highly variable readings obtained immediately after coffee, exercise, and work calls. What is the best next step?

A. Average all readings without regard to technique because a large sample size eliminates systematic measurement error automatically.

B. Replace home monitoring with a cuffless smartwatch because consumer wearable estimates are now preferred for treatment decisions.

C. Use only the highest home reading each day because treatment should always target the patient's maximum observed blood pressure.

D. Standardize the home protocol by using a validated upper-arm device, proper positioning, quiet rest, and repeated readings at consistent times.

49. A 68-year-old has a persistent blood pressure of 162/64 mm Hg. Which derived measurement best captures the unusually wide separation between systolic and diastolic pressure?

A. Pulse pressure, calculated as systolic pressure minus diastolic pressure.

B. Mean arterial pressure, calculated clinically as systolic pressure minus diastolic pressure in every patient.

C. Rate-pressure product, calculated as diastolic pressure multiplied by stroke volume to estimate arterial stiffness.

D. Filtration fraction, calculated from systolic and diastolic pressure to estimate renal perfusion and sodium handling.

50. A patient with apparently resistant hypertension has office readings above goal but a 24-hour ambulatory study is normal. What is the most important conclusion?

A. The ambulatory study should be ignored because resistant hypertension is defined exclusively by office blood pressure measurements.

B. True resistant hypertension has not been established because a white-coat effect should be excluded before labeling the condition resistant.

C. The patient has malignant hypertension because large office-out-of-office differences indicate acute microvascular injury.

D. The patient has masked hypertension because normal ambulatory pressure with elevated office pressure defines the masked phenotype.

51. A patient with hypertension has a long history of diabetic kidney disease, reduced estimated GFR, and persistent albuminuria. Which secondary mechanism is most likely contributing to the hypertension?

A. Primary hyperaldosteronism caused by an adrenal adenoma despite no evidence of renin suppression or mineralocorticoid excess.

B. Renal parenchymal disease with sodium retention, volume expansion, and activation of neurohormonal pathways.

C. Pheochromocytoma caused by episodic catecholamine release despite the chronic kidney findings that dominate the presentation.

D. Coarctation of the aorta causing upper-extremity hypertension despite no pulse delay or arm-leg pressure gradient.

52. A 31-year-old woman has abrupt-onset hypertension and a cervical bruit. Renal imaging shows alternating stenosis and dilation in a distal renal artery. What is the most likely diagnosis?

A. Atherosclerotic renal artery stenosis, which usually affects the ostium and proximal vessel in older patients with vascular risk factors.

B. Renal parenchymal hypertension from chronic glomerulonephritis, which does not produce the described arterial beading pattern.

C. Primary aldosteronism, which can cause resistant hypertension but does not create alternating renal-artery stenosis and dilation.

D. Renal-artery fibromuscular dysplasia, a nonatherosclerotic arteriopathy that can produce the characteristic beaded appearance.

53. An older patient with diffuse atherosclerosis has recurrent episodes of sudden pulmonary edema and severe hypertension. Which secondary cause deserves particular consideration?

A. Isolated white-coat hypertension, which would not explain recurrent flash pulmonary edema in this vascular context.

B. Hemodynamically important atherosclerotic renal artery stenosis, especially if bilateral or affecting a solitary functioning kidney.

C. Primary hypothyroidism, which more often produces a different hemodynamic pattern and does not typically cause abrupt pulmonary edema.

D. Liddle syndrome, which usually presents much earlier in life and is not associated with diffuse atherosclerotic disease.

54. A patient with severe bilateral renal artery stenosis starts an ACE inhibitor and the serum creatinine rises sharply over several days. What mechanism best explains the change?

A. ACE inhibition directly constricts the afferent arteriole and increases renal vascular resistance in all patients.

B. The drug causes immune-complex glomerulonephritis that abruptly reduces filtration whenever renal arteries are narrowed.

C. ACE inhibition increases aldosterone secretion, causing acute potassium loss and prerenal azotemia.

D. Loss of angiotensin II-mediated efferent arteriolar constriction reduces intraglomerular pressure when renal perfusion is critically dependent on that mechanism.

55. Under the 2025 Endocrine Society guideline, which general screening strategy for primary aldosteronism is suggested for adults with hypertension?

A. Screen only patients who have spontaneous hypokalemia because normal potassium essentially excludes primary aldosteronism.

B. Screen adults with hypertension using aldosterone and renin measurements with an aldosterone-to-renin ratio, recognizing implementation and assay considerations.

C. Screen only patients younger than 30 years because primary aldosteronism is too uncommon later in life to justify testing.

D. Use adrenal CT as the initial screening test because imaging is more sensitive than biochemical assessment for autonomous aldosterone production.

56. A patient is being screened for primary aldosteronism. Which approach best improves interpretability of the aldosterone-renin testing?

A. Obtain aldosterone and renin under standardized conditions, measure potassium, and avoid interpreting the ratio in isolation from the absolute hormone values and clinical context.

B. Restrict dietary sodium severely for several days because sodium depletion reliably unmasks autonomous aldosterone secretion.

C. Ignore serum potassium because hypokalemia has no meaningful effect on aldosterone secretion or screening sensitivity.

D. Diagnose primary aldosteronism whenever the ratio is elevated, even if aldosterone itself is very low and renin is artifactually suppressed.

57. A patient with resistant hypertension and hypokalemia has a negative primary aldosteronism screen obtained while potassium is 2.9 mEq/L. What is the best next step?

A. Exclude primary aldosteronism permanently because hypokalemia increases aldosterone enough that false-negative screening cannot occur.

B. Correct the hypokalemia and repeat aldosterone-renin testing if clinical suspicion remains high.

C. Proceed directly to adrenalectomy because hypokalemia alone proves unilateral aldosterone production.

D. Start a high-sodium diet indefinitely and avoid repeat testing because potassium status does not alter hormone interpretation.

58. A patient has hypertension, spontaneous hypokalemia, repeatedly suppressed renin, and clearly elevated aldosterone concentrations. Which statement about confirmatory suppression testing is most appropriate?

A. Suppression testing is mandatory in every patient regardless of the biochemical phenotype or the intended treatment approach.

B. A normal adrenal CT eliminates the diagnosis and makes further biochemical evaluation unnecessary.

C. Confirmatory testing should be replaced by a plasma metanephrine level because catecholamines are the principal cause of hypokalemic hypertension.

D. In a high-probability phenotype with overt renin-independent aldosterone excess, confirmatory suppression testing may be unnecessary before moving to subtype-directed evaluation.

59. A 49-year-old with confirmed primary aldosteronism wants curative surgery if disease is unilateral. CT shows a 1.4-cm left adrenal nodule and a normal right adrenal gland. What is the best next step in most surgical candidates?

A. Use adrenal venous sampling to determine whether aldosterone secretion is truly unilateral before adrenalectomy, unless a well-defined exception applies.

B. Remove the left adrenal gland solely because any visible adrenal nodule in primary aldosteronism is necessarily the source of hormone excess.

C. Perform bilateral adrenalectomy because imaging cannot distinguish unilateral from bilateral disease in any circumstance.

D. Repeat office blood pressure for six months because subtype classification is unnecessary once aldosterone excess has been confirmed.

60. Adrenal venous sampling demonstrates clear unilateral aldosterone production in a healthy patient with primary aldosteronism who wants definitive therapy. What is the preferred treatment?

A. Bilateral adrenalectomy because unilateral secretion predicts occult disease in the contralateral gland.

B. Long-term loop diuretic monotherapy because mineralocorticoid receptor blockade is ineffective in aldosterone excess.

C. Unilateral adrenalectomy after appropriate preoperative management.

D. Renal sympathetic denervation because adrenal hormone production is driven primarily by renal sympathetic nerves.

61. A patient with confirmed bilateral primary aldosteronism is not a surgical candidate. Which drug class directly targets the disease mechanism?

A. A loop diuretic alone, because blocking NKCC2 specifically prevents aldosterone secretion from both adrenal glands.

B. A direct vasodilator alone, because hydralazine neutralizes aldosterone action at the distal nephron receptor.

C. A beta-2 agonist, because stimulating beta-2 receptors suppresses adrenal aldosterone synthesis and corrects potassium wasting.

D. A mineralocorticoid receptor antagonist such as spironolactone or eplerenone.

62. A patient has episodic headache, palpitations, diaphoresis, and paroxysmal severe hypertension. Which biochemical test is most appropriate for suspected pheochromocytoma or paraganglioma?

A. Aldosterone-to-renin ratio because catecholamine-secreting tumors characteristically suppress aldosterone while increasing renin.

B. Serum cortisol measured only at 8 AM because a normal morning cortisol excludes catecholamine-secreting tumors.

C. Plasma free metanephrines or urinary fractionated metanephrines obtained with appropriate preanalytic conditions.

D. Urine albumin-to-creatinine ratio because tumor catecholamine production is best detected by glomerular protein excretion.

63. A patient with a biochemically confirmed catecholamine-secreting adrenal tumor is being prepared for surgery. Which sequencing principle is essential?

A. Start beta blockade first to prevent reflex tachycardia, then add alpha blockade only after heart rate is below 60 beats/min.

B. Avoid all adrenergic blockade because preoperative treatment increases the risk of intraoperative catecholamine release.

C. Use a mineralocorticoid receptor antagonist as the sole preoperative therapy because catecholamines act mainly through aldosterone.

D. Establish adequate alpha-adrenergic blockade before adding beta blockade if tachycardia subsequently requires treatment.

64. A patient with obesity, resistant hypertension, loud snoring, witnessed apneas, and daytime somnolence has normal potassium and kidney function. Which secondary cause should be strongly considered?

A. Coarctation of the aorta, because nocturnal upper-airway symptoms are typical of congenital aortic narrowing.

B. Primary hyperparathyroidism, because loud snoring and witnessed apneas are manifestations of hypercalcemia.

C. Obstructive sleep apnea causing recurrent nocturnal obstruction, intermittent hypoxemia, and sympathetic activation.

D. Reninoma, because renin-secreting tumors commonly produce witnessed apneas before hypokalemia develops.

65. A patient has hypertension, proximal muscle weakness, easy bruising, wide violaceous striae, and new diabetes. Which endocrine disorder should be considered?

A. Primary hypothyroidism, which typically causes wide violaceous striae and proximal catabolic myopathy through low thyroid hormone levels.

B. Cushing syndrome from pathologic glucocorticoid excess, which fits the catabolic skin, muscle, and metabolic findings.

C. Isolated primary aldosteronism, which commonly produces cushingoid body habitus and spontaneous bruising from cortisol excess.

D. Pheochromocytoma, which usually causes chronic glucocorticoid skin changes as its defining physical finding.

66. A patient with untreated hyperthyroidism has tachycardia, tremor, and a blood pressure of 166/62 mm Hg. Which hemodynamic pattern is most consistent with the thyroid disorder?

A. Marked reduction in cardiac output with isolated diastolic hypertension and a narrowed pulse pressure.

B. Suppressed sympathetic responsiveness with profound bradycardia and fixed low systolic pressure.

C. Primary renal sodium retention with low cardiac output and no change in pulse pressure.

D. Increased cardiac output with a widened pulse pressure and predominantly systolic hypertension.

67. A patient with fatigue, weight gain, cold intolerance, and hypertension has a high TSH and low free thyroxine. Which blood pressure pattern can occur with hypothyroidism?

A. High-output systolic hypertension caused by persistent beta-adrenergic stimulation despite low thyroid hormone.

B. Paroxysmal hypertension caused by episodic adrenal catecholamine release from thyroid tissue.

C. Low-resistance shock with a very wide pulse pressure as the usual chronic hemodynamic pattern.

D. Increased systemic vascular resistance with a tendency toward diastolic hypertension.

68. A patient with hypertension has recurrent nephrolithiasis, constipation, and a serum calcium concentration of 11.8 mg/dL. Which endocrine cause should be evaluated?

A. Primary adrenal insufficiency, which usually causes hypotension and hyperkalemia rather than hypercalcemic hypertension.

B. Primary hyperparathyroidism with hypercalcemia, consistent with the kidney stones, constipation, and elevated calcium level.

C. Central diabetes insipidus, which directly causes persistent hypertension through aldosterone excess.

D. Growth hormone deficiency, which characteristically causes hypercalcemia, kidney stones, and severe hypertension.

69. A young adult has hypertension in both arms, weak delayed femoral pulses, and a substantially lower leg blood pressure. What is the most likely secondary cause?

A. Renal fibromuscular dysplasia, which generally causes an arm-leg systolic gradient with delayed femoral pulses as its defining sign.

B. Primary aldosteronism, which produces differential limb perfusion because aldosterone preferentially constricts leg arteries.

C. Obstructive sleep apnea, which causes radiofemoral delay by increasing nocturnal sympathetic tone.

D. Coarctation of the aorta causing upper-extremity hypertension with diminished delayed femoral pulses and lower leg pressure.

70. A patient with previously controlled hypertension develops higher blood pressure after starting daily ibuprofen for chronic arthritis. What is the most likely explanation?

A. Ibuprofen directly stimulates nitric oxide production, causing reflex hypertension only when the dose is too low.

B. NSAID-related sodium retention and attenuation of renal prostaglandin-mediated vasodilation can raise blood pressure and blunt some antihypertensive effects.

C. NSAIDs cause universal adrenal aldosterone tumors after prolonged exposure, producing irreversible hypertension.

D. Ibuprofen lowers renal sodium reabsorption so dramatically that sympathetic activation is required to preserve blood pressure.

71. A woman with newly worsened hypertension recently started an estrogen-containing oral contraceptive. How should the medication be viewed in the evaluation?

A. As protective against hypertension because estrogen-containing contraceptives consistently suppress hepatic angiotensinogen production.

B. As a potentially contributing medication that can increase blood pressure and should be reassessed in the overall risk-benefit plan.

C. As irrelevant unless she also has hypokalemia because contraceptive-associated hypertension requires mineralocorticoid excess.

D. As diagnostic of pheochromocytoma because oral estrogen provokes catecholamine release from occult adrenal tumors.

72. A patient with hypertension and allergic rhinitis reports taking high-dose pseudoephedrine several times daily. What is the most appropriate interpretation?

A. Pseudoephedrine lowers vascular tone, so continued use is useful when hypertension remains uncontrolled.

B. The drug can raise blood pressure only in patients with pheochromocytoma, so it is otherwise irrelevant.

C. The sympathomimetic decongestant may be contributing to the elevated blood pressure and should be reviewed as a reversible cause.

D. The medication causes hypertension solely through potassium wasting, so a normal potassium level excludes an effect.

73. A kidney transplant recipient develops difficult-to-control hypertension after tacrolimus concentrations increase. Which drug-related mechanism is plausible?

A. Tacrolimus routinely causes systemic vasodilation and renal salt wasting, which should lower rather than raise blood pressure.

B. The drug acts as a mineralocorticoid receptor antagonist and therefore causes compensatory severe hypertension.

C. Calcineurin inhibition produces congenital aortic narrowing that manifests only after transplantation.

D. Calcineurin inhibitor-associated renal vasoconstriction and sodium retention.

74. A patient with chronic kidney disease becomes more hypertensive after the dose of an erythropoiesis-stimulating agent is increased. What is the best interpretation?

A. Erythropoiesis-stimulating agents invariably lower blood pressure by reducing blood viscosity and sympathetic tone.

B. The rise proves new renal artery stenosis because erythropoiesis-stimulating drugs have no effect on vascular pressure.

C. Erythropoiesis-stimulating therapy can contribute to hypertension and should be considered in the medication review.

D. The drug causes hypertension only by producing severe hypokalemia, so a normal potassium concentration rules out causality.

75. A patient has hypertension, hypokalemia, low renin, and low aldosterone and reports consuming large amounts of natural licorice candy. What should be recommended first?

A. Increase licorice intake because additional glycyrrhizin will restore aldosterone secretion and normalize potassium.

B. Start fludrocortisone because low aldosterone proves a true deficiency that is causing the hypertension.

C. Stop the licorice exposure because glycyrrhetinic compounds can inhibit 11-beta-hydroxysteroid dehydrogenase type 2 and create apparent mineralocorticoid excess.

D. Schedule adrenalectomy because suppressed aldosterone with licorice exposure indicates an aldosterone-producing adenoma.

76. A young adult presents with severe hypertension, agitation, chest pain, and marked mydriasis after recreational stimulant use. Which exposure is a likely contributor?

A. Acetaminophen at routine doses, which commonly causes a hyperadrenergic hypertensive syndrome with mydriasis.

B. A proton-pump inhibitor, which raises catecholamines and produces stimulant-type hypertension within minutes.

C. Metformin, which causes acute sympathetic surges and severe hypertension whenever taken without food.

D. Cocaine or an amphetamine-type sympathomimetic producing an acute catecholamine-driven hypertensive syndrome.

77. A patient with resistant hypertension reports drinking six alcoholic beverages every evening. Which intervention is most relevant to secondary contributors?

A. Increase alcohol intake gradually because chronic ethanol exposure causes durable vasodilation and improves resistant hypertension.

B. Ignore alcohol because only distilled spirits, not total ethanol intake, influence blood pressure.

C. Reduce excessive alcohol intake because heavy consumption can raise blood pressure and impair control.

D. Treat the alcohol exposure with mineralocorticoid receptor blockade alone because ethanol raises pressure exclusively through aldosterone.

78. A young patient has severe hypertension, hypokalemia, metabolic alkalosis, markedly elevated renin, and elevated aldosterone. Imaging identifies a small renin-secreting renal tumor. Which diagnosis fits best?

A. Liddle syndrome, which suppresses both renin and aldosterone because ENaC activity is autonomous.

B. Primary aldosteronism, which typically suppresses renin rather than producing a marked renin elevation.

C. A juxtaglomerular cell tumor, or reninoma, causing renin-driven secondary hyperaldosteronism.

D. Apparent mineralocorticoid excess, which causes low renin and low aldosterone rather than renin-driven hyperaldosteronism.

79. A teenager has early severe hypertension, hypokalemic metabolic alkalosis, suppressed renin, and suppressed aldosterone. A parent had a similar disorder. Which treatment most directly targets the likely mechanism?

A. Amiloride to block constitutively active epithelial sodium channels in Liddle syndrome.

B. Spironolactone because Liddle syndrome is caused by autonomous aldosterone secretion from the adrenal cortex.

C. Fludrocortisone because the low aldosterone level indicates mineralocorticoid deficiency.

D. Hydrochlorothiazide because Liddle syndrome is caused by overactivity of the sodium-chloride cotransporter.

80. A patient has familial hypertension with persistent hyperkalemia, mild hyperchloremic metabolic acidosis, and low renin. Which treatment is characteristically effective?

A. Amiloride as the defining therapy for an ENaC gain-of-function disorder with hypokalemic alkalosis.

B. A thiazide or thiazide-like diuretic for Gordon syndrome caused by excessive sodium-chloride cotransporter activity.

C. Fludrocortisone replacement because the disorder is caused by aldosterone deficiency with salt wasting and hypotension.

D. High-dose potassium supplementation because correcting hyperkalemia requires increasing extracellular potassium delivery.

81. A child has low-renin, low-aldosterone hypertension, hypokalemic alkalosis, and no licorice exposure. Genetic testing shows defective 11-beta-hydroxysteroid dehydrogenase type 2. What is the diagnosis?

A. Primary aldosteronism caused by adrenal aldosterone overproduction, which should produce an elevated aldosterone concentration.

B. Gordon syndrome, which usually produces hyperkalemia and metabolic acidosis rather than potassium loss and alkalosis.

C. Apparent mineralocorticoid excess caused by failure to inactivate cortisol in mineralocorticoid-sensitive tissues.

D. Reninoma, which would produce high renin and secondary elevation of aldosterone.

82. An adolescent has hypertension, hypokalemia, sexual precocity, and excess androgen effects. Which congenital adrenal enzyme deficiency can produce this pattern through accumulation of deoxycorticosterone?

A. 11-beta-hydroxylase deficiency, which raises deoxycorticosterone and androgens and can cause hypertension with virilization.

B. 21-hydroxylase deficiency, which classically causes salt wasting and does not generate deoxycorticosterone-mediated hypertension.

C. Aromatase excess, which produces estrogen excess rather than mineralocorticoid hypertension with androgen excess.

D. 5-alpha-reductase deficiency, which alters androgen metabolism without causing deoxycorticosterone accumulation and hypertension.

83. A patient has hypertension, hypokalemia, low renin, sexual infantilism, and reduced sex-steroid production. Which congenital adrenal enzyme deficiency is most consistent?

A. 11-beta-hydroxylase deficiency, which usually produces androgen excess rather than reduced sex-steroid production.

B. 21-hydroxylase deficiency, which most often causes mineralocorticoid deficiency and salt wasting rather than hypertension.

C. 17-alpha-hydroxylase deficiency with excess mineralocorticoid-active precursors such as deoxycorticosterone.

D. Aldosterone synthase deficiency, which causes salt loss, hyperkalemia, and low blood pressure rather than mineralocorticoid hypertension.

84. A patient with enlarged hands, prognathism, sleep apnea, and new hypertension has an elevated insulin-like growth factor 1 concentration. Which diagnosis is most likely?

A. Primary hyperparathyroidism, which explains the acral overgrowth and prognathism through chronic hypercalcemia.

B. Cushing syndrome, which typically causes enlargement of the hands and feet with mandibular overgrowth as its defining feature.

C. Primary aldosteronism, which causes acromegalic facial changes through chronic sodium retention in soft tissues.

D. Acromegaly from growth hormone excess, which fits the acral enlargement, prognathism, sleep apnea, and elevated IGF-1.

85. A patient has a strong clinical suspicion for renovascular hypertension. How should the initial vascular imaging test generally be selected?

A. Use catheter angiography as the mandatory first test for every patient because noninvasive tests cannot identify clinically important renal artery disease.

B. Use a plain abdominal radiograph because calcification reliably determines the hemodynamic severity of renal artery stenosis.

C. Choose a noninvasive modality such as duplex ultrasonography, CT angiography, or MR angiography based on kidney function, local expertise, anatomy, and contraindications.

D. Use adrenal venous sampling because renin gradients from the adrenal veins establish the presence of renal artery stenosis.

86. A young woman has renal artery fibromuscular dysplasia with a hemodynamically significant focal lesion and difficult-to-control hypertension. Which intervention is commonly favored when revascularization is appropriate?

A. Percutaneous transluminal angioplasty, often without routine stent placement.

B. Routine surgical nephrectomy because fibromuscular dysplasia cannot be treated with endovascular techniques.

C. Renal artery stenting in every case because angioplasty alone is ineffective for nonatherosclerotic lesions.

D. Bilateral adrenalectomy because the hypertension is caused by adrenal renin production rather than renal perfusion.

87. An older patient has incidentally discovered atherosclerotic renal artery stenosis, stable kidney function, controlled blood pressure, and no recurrent heart failure. What is the usual management principle?

A. Proceed immediately to stenting because every anatomic renal artery stenosis improves with revascularization regardless of clinical presentation.

B. Stop all renin-angiotensin system blockers permanently because any unilateral renal artery narrowing makes these drugs absolutely contraindicated.

C. Optimize medical therapy and cardiovascular risk reduction rather than routinely performing renal artery revascularization.

D. Perform nephrectomy because medical therapy cannot control the cardiovascular consequences of atherosclerotic renovascular disease.

88. Which patient with atherosclerotic renal artery stenosis is most likely to prompt consideration of revascularization in addition to medical therapy?

A. A patient with mild unilateral stenosis discovered incidentally and blood pressure controlled on one medication.

B. A patient with normal kidney function and no hypertension who has a small calcified renal artery plaque on CT.

C. A patient whose blood pressure normalized after reducing dietary sodium and who has no heart failure or renal deterioration.

D. A patient with recurrent flash pulmonary edema or another high-risk syndrome plausibly driven by severe renovascular disease.

89. A patient is referred for resistant hypertension. Which history element is most likely to uncover a reversible secondary contributor before extensive testing?

A. A history limited to first-degree relatives with hypertension because medication and substance exposures rarely alter blood pressure.

B. A review limited to antihypertensive prescriptions because nonprescription drugs and supplements cannot cause clinically important hypertension.

C. A detailed review of prescription drugs, over-the-counter agents, supplements, substances, alcohol, and adherence.

D. A dietary history that excludes sodium intake because only endocrine and renal diseases qualify as secondary hypertension.

90. A patient develops abrupt hypertension after stopping high-dose clonidine without tapering. What is the likely mechanism?

A. Rebound sympathetic activation after withdrawal of a central alpha-2 agonist.

B. Permanent mineralocorticoid receptor activation caused by chronic clonidine exposure.

C. Acute renal artery thrombosis caused by loss of alpha-2-mediated platelet inhibition.

D. Suppression of catecholamine release after clonidine withdrawal, producing compensatory sodium loss and hypertension.

91. For most nonpregnant adults with hypertension who are treated to reduce cardiovascular risk, what is the overarching blood pressure goal in the 2025 U.S. multisociety guideline?

A. A systolic pressure below 160 mm Hg regardless of age, comorbidity, or baseline cardiovascular risk.

B. A diastolic pressure below 100 mm Hg while leaving systolic pressure untreated unless it exceeds 180 mm Hg.

C. A blood pressure below 130/80 mm Hg, with individualized exceptions when clinical circumstances warrant.

D. A universal target below 110/60 mm Hg because lower pressure is always safer in every treated adult.

92. An untreated 52-year-old has repeated standardized office blood pressures averaging 148/92 mm Hg. What is the recommended treatment principle?

A. Use lifestyle treatment alone indefinitely because medication is reserved for systolic pressure above 180 mm Hg.

B. Delay medication until a cardiovascular event occurs because absolute risk is the only determinant once pressure exceeds 140/90 mm Hg.

C. Start aspirin instead of an antihypertensive because antiplatelet therapy is the preferred first treatment for stage 2 hypertension.

D. Begin antihypertensive drug therapy in addition to lifestyle treatment because pressure is at least 140/90 mm Hg.

93. A 61-year-old with prior myocardial infarction has a repeated blood pressure of 134/82 mm Hg despite lifestyle efforts. Which approach best fits current treatment guidance?

A. Withhold medication until blood pressure reaches 160/100 mm Hg because prior myocardial infarction does not change the treatment threshold.

B. Treat only the diastolic pressure because systolic pressure between 130 and 139 mm Hg is considered normal after myocardial infarction.

C. Use antihypertensive medication because stage 1 hypertension with established cardiovascular disease warrants pharmacologic treatment.

D. Use no further therapy because cardiovascular disease makes blood pressure lowering more hazardous than observation.

94. A healthy 38-year-old has persistent blood pressure around 132/82 mm Hg, no diabetes, CKD, or cardiovascular disease, and a low 10-year cardiovascular risk estimate. What is the preferred initial strategy?

A. Immediate triple-drug therapy because any reading above 120/80 mm Hg requires three agents in a low-risk adult.

B. No follow-up for five years because stage 1 hypertension is clinically irrelevant when short-term risk is low.

C. Intensive lifestyle therapy with reassessment after about 3 to 6 months, adding medication if blood pressure remains at least 130/80 mm Hg.

D. Begin a loop diuretic solely to prevent future sodium sensitivity despite normal kidney function and no volume overload.

95. A patient has confirmed stage 2 hypertension with blood pressure 162/96 mm Hg and no contraindication to standard first-line drugs. Which initial pharmacologic strategy is generally preferred?

A. Start one very low-dose drug and avoid adding another agent for at least one year regardless of the response.

B. Use a short-acting central alpha agonist as sole long-term therapy because rapid pressure fluctuation improves vascular adaptation.

C. Begin a direct vasodilator and alpha blocker as the standard first-line pair before considering thiazides, ACE inhibitors, ARBs, or calcium channel blockers.

D. Start two first-line antihypertensive agents from complementary classes, preferably as a single-pill combination when feasible.

96. Why are fixed-dose single-pill antihypertensive combinations often favored when two drugs are indicated?

A. They eliminate all adverse effects because combining two drugs prevents class-specific toxicity.

B. They allow clinicians to ignore kidney function because fixed doses cannot affect potassium or creatinine.

C. They are effective only when both components belong to the same drug class and act at the same receptor.

D. They reduce pill burden and can improve adherence while combining complementary mechanisms.

97. Which eating pattern has the strongest classic evidence for lowering blood pressure through greater intake of fruits, vegetables, and low-fat dairy with reduced saturated fat?

A. A diet based mainly on processed meats and refined carbohydrates with no emphasis on plant foods.

B. The DASH dietary pattern, emphasizing fruits, vegetables, low-fat dairy, and reduced saturated fat.

C. A high-sodium ketogenic diet designed specifically to increase extracellular volume.

D. An unrestricted fast-food diet because macronutrient composition has no measurable relationship to blood pressure.

98. A patient with hypertension consumes about 5 grams of sodium daily. Which dietary change is most likely to help lower blood pressure?

A. Increase sodium intake to promote pressure natriuresis because more sodium reliably lowers blood pressure in salt-sensitive patients.

B. Avoid all dietary potassium and fiber because these nutrients counteract the antihypertensive effect of sodium reduction.

C. Replace sodium chloride with unlimited potassium salt without checking kidney function or medications.

D. Reduce sodium intake substantially while maintaining an overall heart-healthy dietary pattern.

99. A patient with hypertension asks about increasing dietary potassium. Which statement is most appropriate?

A. Potassium should be increased without limit because hyperkalemia cannot occur from diet when hypertension is present.

B. Higher dietary potassium can help lower blood pressure in appropriate patients, but kidney disease and drugs that raise potassium may make supplementation unsafe.

C. Potassium should be avoided in every hypertensive patient because it uniformly increases systemic vascular resistance.

D. Potassium has no physiologic relationship to sodium balance or blood pressure and therefore should never be discussed.

100. A patient with obesity and hypertension loses 9% of body weight through sustained dietary and behavioral change. What blood pressure effect is expected?

A. Weight loss usually raises blood pressure because reduced adipose tissue activates the renin-angiotensin system.

B. Weight loss is likely to lower blood pressure and can reduce the medication burden in some patients.

C. Weight change has no clinically meaningful relationship to hypertension once a patient is older than 30 years.

D. Weight loss should be avoided during antihypertensive treatment because it makes medications permanently ineffective.

101. Which physical-activity prescription is most appropriate as part of nonpharmacologic hypertension treatment for an otherwise healthy adult?

A. Regular aerobic activity, supplemented by appropriate resistance exercise and reduced sedentary time.

B. Avoid exercise because transient systolic elevation during activity causes progressive resting hypertension.

C. Perform only isometric maximal lifting because aerobic activity has no blood pressure benefit.

D. Exercise once monthly at very high intensity because frequency is less important than a single maximal workload.

102. A patient with hypertension drinks four to five alcoholic beverages nightly. Which recommendation is most appropriate?

A. Increase alcohol intake gradually because chronic vasodilation makes heavy drinking an effective antihypertensive therapy.

B. Continue the same intake because alcohol affects triglycerides but has no meaningful effect on blood pressure.

C. Reduce alcohol intake because excess consumption can raise blood pressure and undermine treatment.

D. Switch from wine to spirits because only fermented beverages influence hypertension.

103. A patient with hypertension has chronic sleep deprivation and high perceived stress. How should these factors be addressed?

A. Treat sleep deprivation with additional caffeine because sympathetic stimulation lowers nocturnal blood pressure.

B. Include healthy sleep and stress-management strategies as supportive components of a broader lifestyle plan.

C. Ignore sleep and stress because lifestyle counseling should address sodium alone.

D. Use sedative medication as the sole hypertension treatment because adequate sleep removes the need for pressure monitoring.

104. Which drug is a standard first-line option for uncomplicated hypertension and directly inhibits the distal convoluted tubule sodium-chloride cotransporter?

A. A thiazide or thiazide-like diuretic that inhibits the distal sodium-chloride cotransporter and has strong outcome evidence.

B. A loop diuretic, which is the routine preferred first-line agent for every uncomplicated hypertensive patient with normal kidney function.

C. A central alpha-2 agonist, which is preferred before thiazides because it has fewer withdrawal and sedation concerns.

D. An alpha-1 blocker, which is generally first-line because it has superior outcome evidence to thiazide-type therapy.

105. A patient with uncomplicated hypertension needs a thiazide-type agent with a long duration of action and strong outcomes evidence. Which drug is a common choice?

A. Acetazolamide, because carbonic anhydrase inhibition is the standard long-term first-line diuretic strategy for hypertension.

B. Chlorthalidone, with electrolyte and metabolic monitoring as clinically appropriate.

C. Mannitol, because osmotic diuresis provides durable outpatient blood pressure control without electrolyte disturbance.

D. Desmopressin, because increasing renal free-water reabsorption lowers arterial pressure through volume contraction.

106. A patient with isolated systolic hypertension needs a first-line vasodilating drug that has strong evidence in older adults. Which class is appropriate?

A. A long-acting dihydropyridine calcium channel blocker such as amlodipine.

B. A short-acting immediate-release dihydropyridine taken only when systolic pressure feels high.

C. A class IC antiarrhythmic because sodium-channel blockade is a standard method of lowering vascular resistance.

D. A nitrate used as chronic sole antihypertensive therapy because tolerance improves long-term pressure control.

107. A patient with hypertension and albuminuric chronic kidney disease is starting a renin-angiotensin system blocker. Which class is appropriate?

A. A direct vasodilator alone because renin-angiotensin blockade should never be used in chronic kidney disease.

B. An ACE inhibitor or an angiotensin receptor blocker, with monitoring of creatinine and potassium.

C. A nondihydropyridine calcium channel blocker as mandatory monotherapy because albuminuria makes ACE inhibitors contraindicated.

D. A central alpha agonist because it uniquely reduces glomerular albumin leakage by constricting the efferent arteriole.

108. A patient developed cough on lisinopril but has no history of angioedema. Which substitution often preserves renin-angiotensin blockade without the same bradykinin effect?

A. Switch to an angiotensin receptor blocker if otherwise appropriate, preserving RAAS blockade without ACE-mediated bradykinin accumulation.

B. Switch to another ACE inhibitor because all ACE inhibitors completely eliminate bradykinin accumulation after the first drug is stopped.

C. Add a second ACE inhibitor because dual ACE inhibition prevents cough by achieving more complete enzyme blockade.

D. Use aliskiren together with the ACE inhibitor because combining renin and ACE blockade specifically treats bradykinin-mediated cough.

109. A patient develops a dry cough several weeks after starting an ACE inhibitor. What is the most appropriate management if the cough is bothersome and no other cause is found?

A. Continue the ACE inhibitor indefinitely because the cough proves the drug is protecting the lungs.

B. Add another ACE inhibitor at a higher dose because more complete ACE inhibition reduces bradykinin accumulation.

C. Discontinue the ACE inhibitor and use an alternative antihypertensive class, often an ARB when renin-angiotensin blockade is desired.

D. Treat the cough with chronic antibiotics because ACE inhibitor cough represents occult bacterial bronchitis.

110. A patient develops lip and tongue swelling with airway symptoms after starting an ACE inhibitor. Which action is most important for future hypertension treatment?

A. Restart the ACE inhibitor at a lower dose once swelling resolves because angioedema is a harmless dose-dependent side effect.

B. Add an NSAID to prevent recurrent swelling while continuing ACE inhibition.

C. Change only the time of day of the ACE inhibitor because nighttime dosing prevents bradykinin-mediated angioedema.

D. Stop the ACE inhibitor and avoid re-exposure because ACE inhibitor-associated angioedema can recur and be life-threatening.

111. After an ACE inhibitor is started in a patient with CKD, which laboratory tests should generally be reassessed?

A. Serum amylase and lipase only because ACE inhibitors primarily cause pancreatic toxicity.

B. Serum creatinine and potassium to detect clinically important changes in kidney filtration or potassium balance.

C. Platelet count only because thrombocytopenia is the main dose-limiting renal adverse effect.

D. Thyroid-stimulating hormone only because kidney function and potassium are unaffected by renin-angiotensin blockade.

112. A patient with albuminuric CKD has a small stable rise in serum creatinine after starting an ACE inhibitor, with improved blood pressure and no volume depletion or hyperkalemia. What is the best general interpretation?

A. Any creatinine increase proves acute tubular necrosis and requires permanent avoidance of all renin-angiotensin system blockers.

B. A modest creatinine rise can reflect the expected fall in intraglomerular pressure and does not automatically require stopping the drug.

C. The creatinine rise means the ACE inhibitor has increased efferent arteriolar resistance and is damaging glomeruli.

D. Creatinine should never be monitored after starting an ACE inhibitor because changes are unrelated to renal hemodynamics.

113. Why is routine combination therapy with an ACE inhibitor and an ARB generally avoided?

A. The two classes act at exactly the same enzyme and therefore have no additive physiologic effect of any kind.

B. The combination reliably causes severe hypokalemia, which makes it ineffective for lowering blood pressure.

C. ARBs convert into ACE inhibitors in the liver, creating a toxic metabolite unique to combination treatment.

D. Dual renin-angiotensin blockade increases adverse events such as hyperkalemia and kidney injury without sufficient outcome benefit to justify routine use.

114. An older patient becomes confused after a thiazide is added and the serum sodium is 121 mEq/L. What is the most likely medication-related problem?

A. Thiazide-associated hyponatremia, a recognized complication that can be clinically important in susceptible older patients.

B. Thiazide-induced hypernatremia caused by obligatory free-water loss in every treated patient.

C. ACE inhibitor-induced diabetes insipidus because thiazides cannot lower serum sodium.

D. Calcium channel blocker toxicity because dihydropyridines are the principal cause of severe hyponatremia.

115. A patient with recurrent gout develops frequent attacks after a thiazide dose is increased. Which drug effect may be contributing?

A. Thiazide-associated hyperuricemia can increase gout risk in susceptible patients.

B. Thiazides uniformly lower serum urate by blocking proximal uric-acid reabsorption.

C. Gout proves the patient has primary aldosteronism because thiazides have no effect on uric acid.

D. The attacks are caused by thiazide-induced hypocalcemia, which is the primary trigger of urate crystal formation.

116. A patient taking amlodipine has good blood pressure control but develops symmetric ankle edema. What mechanism is most likely?

A. Amlodipine causes severe venous constriction that lowers capillary hydrostatic pressure and traps lymph in the legs.

B. The drug produces nephrotic-range proteinuria in nearly all patients, causing low oncotic pressure and edema.

C. Dihydropyridine calcium channel blockers directly activate aldosterone receptors and create primary mineralocorticoid excess.

D. Preferential arteriolar dilation raises capillary hydrostatic pressure and promotes dependent edema without requiring total-body volume overload.

117. Which statement best describes beta blockers in uncomplicated hypertension?

A. They are mandatory first-line therapy for every adult because no other class reduces cardiovascular events.

B. They are contraindicated in all patients with coronary disease because heart-rate reduction increases myocardial oxygen demand.

C. They are useful when there is a compelling indication, but they are not generally the preferred first-line class for uncomplicated hypertension without such an indication.

D. They should be used only as short-acting rescue medication because chronic beta blockade cannot lower blood pressure.

118. A patient with hypertension also has symptomatic coronary artery disease and exertional angina. Which feature makes a beta blocker particularly useful if there is no contraindication?

A. It increases heart rate and contractility, improving angina by raising coronary oxygen consumption.

B. It can lower blood pressure while reducing heart rate and myocardial oxygen demand.

C. It produces reflex sympathetic activation that improves exercise tolerance in ischemic heart disease.

D. It prevents all coronary events solely by lowering LDL cholesterol independent of hemodynamic effects.

119. A patient with difficult-to-control hypertension develops breast tenderness on spironolactone. Which alternative provides mineralocorticoid receptor blockade with less antiandrogen effect?

A. Hydrochlorothiazide, because it is a selective mineralocorticoid receptor antagonist without endocrine effects.

B. Clonidine, because central alpha-2 activation directly blocks the aldosterone receptor in the collecting duct.

C. Amlodipine, because L-type calcium channel blockade suppresses aldosterone receptor transcription in breast tissue.

D. Eplerenone, a more selective mineralocorticoid receptor antagonist with less antiandrogen activity than spironolactone.

120. A patient has uncontrolled hypertension despite an apparently appropriate regimen. Which step should occur before simply adding multiple new drugs?

A. Assess adherence, measurement quality, interfering substances, and out-of-office blood pressure to identify pseudoresistance.

B. Assume the patient is adherent because prescriptions appear in the electronic record and immediately add three more agents.

C. Ignore home or ambulatory readings because office pressure alone proves true resistance in every patient.

D. Stop all current medication for several weeks because treatment history prevents accurate evaluation of apparent resistance.

121. A patient is learning home blood pressure monitoring. Which practice provides the most useful data for treatment decisions?

A. Use whichever wrist or finger device is cheapest because validation has no effect on treatment accuracy.

B. Measure immediately after exercise and caffeine because peak readings best represent resting cardiovascular risk.

C. Use a validated upper-arm cuff and obtain repeated standardized readings over multiple days rather than reacting to isolated measurements.

D. Record only values that are above goal because normal readings dilute clinically important hypertension.

122. Which care model can improve hypertension control by supporting medication adjustment, monitoring, and adherence?

A. Team-based care involving clinicians, nurses, pharmacists, and other trained professionals using shared protocols and follow-up.

B. Physician-only care with no home monitoring because involvement of other professionals reduces accountability.

C. Emergency-department management for every medication adjustment because outpatient teams cannot safely titrate antihypertensives.

D. Annual visits without interim contact because frequent follow-up increases white-coat hypertension and worsens adherence.

123. A patient wants to replace a validated arm cuff with a smartwatch that estimates blood pressure without an inflatable cuff. What is the best current recommendation?

A. Use the smartwatch exclusively because cuffless devices are now the reference standard for hypertension diagnosis.

B. Calibrate the smartwatch once and assume accuracy for life because vascular aging cannot change device performance.

C. Use only pulse rate from the smartwatch because cuff-based blood pressure devices are no longer recommended.

D. Do not rely on a cuffless wearable as a substitute for validated cuff-based measurements for diagnosis or treatment decisions.

124. What major treatment principle was reinforced by the SPRINT trial in selected high-cardiovascular-risk adults without diabetes?

A. Allowing systolic pressure to remain above 160 mm Hg reduced cardiovascular events compared with any active treatment.

B. Intensive treatment eliminated the need to monitor kidney function, electrolytes, or symptomatic hypotension.

C. The trial showed that systolic blood pressure has no causal relationship to cardiovascular outcomes once diastolic pressure is normal.

D. More intensive systolic blood pressure lowering reduced major cardiovascular events and mortality compared with a less intensive strategy, while requiring attention to adverse effects.

125. Which broad lesson from ALLHAT remains important in hypertension pharmacotherapy?

A. Alpha-1 blocker monotherapy was superior to thiazide therapy for preventing heart failure and therefore became the preferred first-line strategy.

B. Diuretics increased every major cardiovascular outcome and should no longer be used for uncomplicated hypertension.

C. Thiazide-type diuretic-based treatment provides strong cardiovascular outcome protection and remains an effective foundational option for many patients.

D. Only ACE inhibitors lowered blood pressure in older adults, whereas thiazides and calcium channel blockers had no meaningful antihypertensive effect.

126. What treatment lesson is associated with the ACCOMPLISH trial in high-risk hypertensive patients?

A. Combining an ACE inhibitor with an ARB was superior to all other two-drug regimens and became standard first-line therapy.

B. Calcium channel blockers were ineffective when combined with renin-angiotensin blockade and should never be used together.

C. An ACE inhibitor plus a calcium channel blocker produced better cardiovascular outcomes than the tested ACE inhibitor-thiazide combination.

D. The trial established that drug class does not matter at all when achieved office blood pressure is similar.

127. In the ACCORD blood-pressure trial among adults with type 2 diabetes, what is the most appropriate high-level interpretation?

A. Intensive lowering markedly increased stroke and therefore proved that systolic pressure should never be treated below 160 mm Hg in diabetes.

B. Very intensive systolic lowering did not significantly reduce the primary composite cardiovascular outcome overall, although stroke was reduced and treatment burden and adverse effects increased.

C. The trial showed no blood pressure difference between treatment groups, so it provided no information about treatment intensity.

D. The trial proved that diabetes eliminates all benefit from antihypertensive therapy and that glucose control alone should be used.

128. What principle from the AASK trial is most relevant to treatment of hypertension in chronic kidney disease?

A. Renin-angiotensin system blockade, particularly with an ACE inhibitor in the studied population, was beneficial for kidney outcomes especially when proteinuria was present.

B. Alpha-1 blocker therapy was uniquely renoprotective and replaced ACE inhibitors for proteinuric kidney disease.

C. Achieved blood pressure had no relationship to kidney outcomes, so antihypertensive treatment should be avoided in CKD.

D. Loop diuretics were shown to reverse chronic glomerular scarring and eliminate the need for renin-angiotensin blockade.

129. In PATHWAY-2, which add-on drug was especially effective for many patients with resistant hypertension already taking three standard agents?

A. Doxazosin was clearly superior to all other tested add-on therapies and became the preferred universal fourth drug.

B. Spironolactone, which was especially effective as fourth-line therapy in resistant hypertension in PATHWAY-2.

C. Bisoprolol was the only effective add-on therapy and mineralocorticoid receptor blockade had no meaningful blood pressure effect.

D. Placebo lowered blood pressure more than active treatment, demonstrating that resistant hypertension should not be treated pharmacologically.

130. What did HYVET establish in very old adults with hypertension?

A. Antihypertensive therapy should be stopped at age 80 because treatment consistently increases stroke and death in very old adults.

B. Only diastolic hypertension should be treated in adults over 80 because systolic pressure has no prognostic significance in this age group.

C. Very old adults cannot tolerate any thiazide-like therapy, so all diuretics are absolutely contraindicated after age 80.

D. Treating hypertension in selected adults aged 80 years or older can reduce important cardiovascular outcomes and mortality rather than leaving high pressure untreated because of age alone.

131. What major clinical lesson came from the SHEP trial?

A. Isolated systolic hypertension is a benign consequence of aging and should not be treated unless diastolic pressure also exceeds 100 mm Hg.

B. Treating isolated systolic hypertension in older adults reduces stroke and other cardiovascular events.

C. Lowering systolic pressure in older adults increases stroke because cerebral perfusion depends on high pulse pressure.

D. Only beta blockers can improve outcomes in isolated systolic hypertension, whereas diuretics have no benefit.

132. What was an important implication of the Syst-Eur trial?

A. Active treatment of isolated systolic hypertension in older adults, using a calcium channel blocker-based regimen, reduced cardiovascular events including stroke.

B. Calcium channel blockers increased stroke sufficiently that they should never be used in older patients with systolic hypertension.

C. Isolated systolic hypertension could not be lowered pharmacologically without causing severe diastolic hypertension.

D. Only patients younger than 40 years benefited from treatment, making the trial irrelevant to older adults.

133. What safety lesson is associated with ONTARGET regarding combined renin-angiotensin system blockade?

A. Dual ACE inhibitor-ARB therapy eliminated hyperkalemia and was safer for kidney function than either drug alone.

B. ARBs were ineffective as single agents, so they should be prescribed only together with ACE inhibitors.

C. Combining an ACE inhibitor with an ARB increased adverse renal and metabolic effects without enough added cardiovascular benefit to support routine dual therapy.

D. The study showed that renin-angiotensin system blockers cannot lower blood pressure and should be abandoned for hypertension treatment.

134. What was a central finding of the original DASH feeding studies?

A. Diet had no effect on blood pressure unless every participant also lost at least 20% of body weight.

B. A high-sodium processed-food diet lowered pressure more than a plant-rich dietary pattern.

C. Only dietary protein affected blood pressure, whereas fruits, vegetables, minerals, and fat composition had no measurable influence.

D. A dietary pattern rich in fruits, vegetables, and low-fat dairy and lower in saturated fat can lower blood pressure even before considering medication changes.

135. A patient with hypertension, CKD, and substantial albuminuria has no contraindication to renin-angiotensin system blockade. Which regimen principle is most appropriate?

A. Use both an ACE inhibitor and an ARB together to maximize albuminuria reduction regardless of potassium or kidney function.

B. Avoid all renin-angiotensin blockers because albuminuria indicates that efferent arteriolar dilation will always cause irreversible kidney failure.

C. Use one ACE inhibitor or ARB at a tolerated evidence-based dose and monitor kidney function and potassium.

D. Use short-acting nifedipine only when home systolic pressure exceeds 180 mm Hg rather than providing continuous therapy.

136. A patient with type 2 diabetes has hypertension but no albuminuria, CKD, heart failure, or coronary disease. Which statement about initial antihypertensive choice is best?

A. Only an ACE inhibitor can ever be used because diabetes makes all other antihypertensive classes ineffective.

B. All diuretics and calcium channel blockers are contraindicated in diabetes because they uniformly cause severe hyperglycemia.

C. Several first-line classes can be appropriate, including a thiazide-type diuretic, ACE inhibitor or ARB, or long-acting dihydropyridine calcium channel blocker, selected for the whole clinical picture.

D. Blood pressure should not be treated until albuminuria develops because diabetes alone eliminates cardiovascular benefit from pressure lowering.

137. A patient with advanced CKD has hypertension with clinically important extracellular-volume overload despite a thiazide regimen. Which adjustment may be useful?

A. Use or add an appropriately dosed loop diuretic to improve natriuresis and volume control.

B. Stop all diuretics because reduced GFR makes renal sodium retention independent of tubular transport.

C. Use desmopressin to increase water retention because volume expansion lowers blood pressure in advanced CKD.

D. Replace the diuretic with a nitrate alone because venodilation corrects chronic sodium excess without changing kidney sodium handling.

138. Why is an ACE inhibitor or ARB often combined with a long-acting dihydropyridine calcium channel blocker?

A. The combination is used because each drug prevents all adverse effects caused by the other and eliminates the need for monitoring.

B. The classes lower pressure through complementary mechanisms and the renin-angiotensin blocker may reduce calcium channel blocker-associated edema in some patients.

C. Both drugs act only by blocking the same L-type calcium channel, making the pair pharmacologically identical to doubling one dose.

D. The combination is reserved exclusively for hypertensive emergencies because chronic use causes obligatory renal artery thrombosis.

139. A patient remains above goal on an ACE inhibitor. Why can adding a thiazide-type diuretic be effective?

A. The two drugs must be combined because neither class lowers blood pressure when used alone.

B. The diuretic reduces sodium-volume burden while the ACE inhibitor blunts counter-regulatory renin-angiotensin activation, producing complementary pressure lowering.

C. Thiazides increase sodium retention, which is required for ACE inhibitors to reach the vascular wall.

D. ACE inhibitors prevent every thiazide adverse effect, so electrolyte and kidney monitoring is unnecessary when the drugs are combined.

140. A patient planning pregnancy is taking lisinopril for hypertension. What is the key medication principle?

A. ACE inhibitors should be discontinued and replaced with a pregnancy-compatible antihypertensive regimen before or promptly when pregnancy is recognized.

B. Continue lisinopril throughout pregnancy because fetal renal development is protected by ACE inhibition.

C. Double the ACE inhibitor dose during the second trimester because pregnancy-induced vasodilation makes fetal toxicity less likely.

D. Combine lisinopril with an ARB because dual renin-angiotensin blockade prevents pregnancy-related hypertension without fetal risk.

141. A patient on an ACE inhibitor and thiazide develops acute kidney injury after adding high-dose ibuprofen during a dehydrating illness. What treatment interaction is most relevant?

A. Ibuprofen directly dilates the afferent arteriole, so kidney injury occurs because glomerular pressure becomes excessively high.

B. The combination of renin-angiotensin blockade, diuresis, NSAID-related afferent vasoconstriction, and volume depletion can markedly reduce glomerular filtration.

C. The thiazide blocks ACE metabolism, causing an allergic interstitial nephritis unique to the drug pair.

D. NSAIDs prevent sodium retention and therefore create isolated postrenal obstruction when combined with an ACE inhibitor.

142. A patient with controlled hypertension asks whether an over-the-counter decongestant is harmless. What is the best advice?

A. All decongestants lower blood pressure because alpha-adrenergic stimulation dilates systemic resistance vessels.

B. Blood pressure effects occur only in pheochromocytoma, making routine counseling unnecessary.

C. Decongestants are preferred antihypertensive adjuncts because nasal vasoconstriction reduces systemic vascular resistance.

D. Sympathomimetic decongestants can raise blood pressure in susceptible patients, so the specific product and alternatives should be reviewed.

143. A patient asks whether every antihypertensive medication should be moved to bedtime because nighttime dosing is always superior. What is the best response?

A. Every antihypertensive must be taken at midnight because morning dosing consistently increases cardiovascular mortality.

B. All antihypertensives should be taken only in the morning because nighttime blood pressure has no relationship to cardiovascular risk.

C. Dosing time is irrelevant even when a short-acting medication causes end-of-dose loss of control or symptomatic nocturnal hypotension.

D. Routine universal bedtime dosing is not required; dosing time should prioritize a durable 24-hour effect, adherence, symptoms, and the pharmacology of the regimen.

144. A patient frequently misses the evening dose of a twice-daily antihypertensive but reliably takes morning medications. What is the best regimen principle?

A. Add more evening doses because a more complicated regimen reliably improves adherence through greater patient engagement.

B. Simplify to once-daily long-acting therapy or a single-pill combination when clinically appropriate to improve adherence.

C. Stop all long-acting medication because short-acting agents are easier to remember even when several daily doses are required.

D. Ignore the missed doses and classify the patient as pharmacologically resistant before changing the regimen.

145. A patient cannot afford a new brand-name antihypertensive and has begun taking it every other day. What is the most useful treatment response?

A. Document nonadherence without changing the plan because cost is not a legitimate determinant of treatment effectiveness.

B. Increase the prescribed dose so that every-other-day administration produces the same pharmacokinetics as daily treatment.

C. Add a second expensive drug because more prescriptions reduce the financial impact of each individual medication.

D. Address cost directly and select an effective affordable regimen the patient can take consistently.

146. A patient appears to have resistant hypertension but pharmacy refill history shows long gaps in all three prescribed medications. What is the best next step?

A. Proceed directly to renal denervation because refill gaps prove that medications are biologically ineffective.

B. Add three additional agents while leaving the adherence barrier unexplored.

C. Treat adherence as a central diagnostic and therapeutic issue before labeling the blood pressure pharmacologically resistant.

D. Diagnose malignant hypertension because nonadherence is itself evidence of acute target-organ injury.

147. A patient has stage 1 hypertension and an estimated 10-year cardiovascular risk above the current PREVENT-based treatment threshold. Which strategy is appropriate?

A. Add antihypertensive medication to lifestyle treatment because elevated predicted cardiovascular risk supports pharmacologic therapy at the stage 1 level.

B. Ignore the risk estimate because treatment thresholds are based only on diastolic pressure above 100 mm Hg.

C. Use statin therapy instead of blood pressure treatment because predicted risk cannot influence antihypertensive decisions.

D. Delay all therapy until systolic pressure exceeds 180 mm Hg because stage 1 hypertension is never treated pharmacologically.

148. What 10-year cardiovascular risk threshold in the 2025 U.S. hypertension guideline is used with the PREVENT equations to help guide medication treatment for stage 1 hypertension?

A. About 50% or greater, because lower predicted risk is never used in blood pressure treatment decisions.

B. A PREVENT 10-year cardiovascular risk of about 7.5% or greater supports medication treatment at the stage 1 level.

C. Exactly 1% or greater, because every adult above that level requires immediate multidrug therapy.

D. Predicted risk is not used at all; the guideline abandoned risk-based treatment thresholds for stage 1 hypertension.

149. A hypertensive patient smokes cigarettes and asks whether smoking cessation will replace antihypertensive therapy. What is the best response?

A. Continue smoking because nicotine lowers cardiovascular risk once blood pressure is treated pharmacologically.

B. Stop antihypertensive medication after smoking cessation because tobacco exposure is the only cause of cardiovascular events in hypertension.

C. Smoking cessation is essential for cardiovascular risk reduction but should complement, not replace, indicated blood pressure treatment.

D. Smoking has no interaction with overall cardiovascular risk, so counseling is unrelated to hypertension care.

150. Which public-health intervention can lower population blood pressure even among people who do not yet carry a hypertension diagnosis?

A. Increasing sodium in processed foods so the kidney adapts to chronic pressure natriuresis.

B. Reducing sodium in the food supply and improving access to heart-healthy foods and physical activity.

C. Discouraging physical activity because transient exercise-related systolic elevation causes chronic hypertension.

D. Eliminating blood pressure screening because awareness increases anxiety and therefore raises population pressure.

151. Before escalating medication for a clinic blood pressure of 154/88 mm Hg, what should be confirmed?

A. That the patient has eaten a high-sodium meal immediately before the measurement because treatment should be based on peak dietary response.

B. That only one reading was obtained, because averaging repeated measurements reduces the accuracy of treatment decisions.

C. That measurement technique is standardized and the elevated pressure is persistent, with out-of-office data when useful.

D. That the cuff is intentionally smaller than the arm so systolic pressure is not underestimated.

152. An 88-year-old nursing-home resident has recurrent falls, symptomatic orthostatic hypotension, and a seated blood pressure of 126/68 mm Hg while taking four antihypertensives. What treatment principle is most appropriate?

A. Intensify therapy to a universal systolic target below 100 mm Hg because age and falls do not alter treatment goals.

B. Individualize therapy and consider de-intensification when treatment harms such as symptomatic hypotension outweigh incremental benefit.

C. Add clonidine because orthostatic symptoms indicate undertreated sympathetic hypertension.

D. Ignore standing symptoms because only seated office pressure should influence antihypertensive decisions.

153. A patient with persistent uncontrolled hypertension despite optimized medications is interested in catheter-based renal denervation. Which statement best reflects current evidence?

A. Renal denervation reliably normalizes blood pressure in every patient and eliminates the need for lifestyle change or medication.

B. Renal denervation can provide an additional modest blood pressure reduction in selected patients but is an adjunct rather than a cure or replacement for comprehensive care.

C. The procedure is contraindicated whenever a patient is taking antihypertensive drugs because it works only in untreated hypertension.

D. Renal denervation lowers pressure by removing the adrenal glands and therefore treats only primary aldosteronism.

154. Before referring a patient with apparently uncontrolled hypertension for renal denervation, what should be established?

A. A single elevated office reading because procedural therapy should precede confirmation or medication review.

B. Normal ambulatory blood pressure because white-coat hypertension predicts the largest procedural benefit.

C. Complete withdrawal of all antihypertensives for several months because denervation cannot be evaluated in treated patients.

D. Sustained uncontrolled blood pressure with reliable out-of-office confirmation, attention to adherence, and evaluation for important secondary causes.

155. How should a patient be counseled about the expected response to renal denervation?

A. The procedure guarantees a systolic pressure below 120 mm Hg and permanently removes the need for monitoring.

B. The average pressure reduction is modest and individual response is variable, so ongoing follow-up and medication adjustment may still be required.

C. A lack of immediate hypotension means the procedure failed because the full effect must occur during the catheterization.

D. Blood pressure always rises after successful denervation, so antihypertensive drugs must be stopped before discharge.

156. What is the role of implantable baroreflex activation therapy in routine uncomplicated hypertension management?

A. It should replace all oral antihypertensive medication at the time hypertension is first diagnosed.

B. It is preferred for white-coat hypertension because device stimulation corrects office anxiety without affecting ambulatory pressure.

C. It is not a standard first-line treatment and is generally reserved for specialized or investigational contexts rather than routine care.

D. It is a dietary intervention that activates carotid baroreceptors through increased potassium intake.

157. A patient with diabetes is taking an ACE inhibitor. Why should aliskiren not routinely be added for more complete renin-angiotensin blockade?

A. Aliskiren causes irreversible beta-receptor blockade and severe bradycardia only when diabetes is present.

B. Combining direct renin inhibition with an ACE inhibitor or ARB in diabetes increases renal and electrolyte risk without an established outcome advantage.

C. Direct renin inhibitors cannot lower blood pressure, so they are never pharmacologically active in humans.

D. ACE inhibitors convert aliskiren into an aldosterone agonist that causes universal hypokalemia.

158. A patient on hydralazine develops tachycardia and fluid retention as the dose is increased. What pharmacologic principle explains these effects?

A. Hydralazine directly blocks beta receptors and aldosterone receptors, so tachycardia and sodium retention indicate another unrelated disease.

B. The drug primarily constricts arterioles, and the tachycardia results from increased afterload rather than reflex activation.

C. Hydralazine causes obligatory renal sodium wasting, so edema implies that the dose is too low rather than a known compensatory effect.

D. Direct arteriolar vasodilation can trigger reflex sympathetic activation and sodium retention, so additional agents may be needed to counter those responses.

159. A patient with refractory hypertension receives minoxidil and soon develops marked edema and tachycardia. What co-therapy is commonly required?

A. A potent diuretic and usually a beta blocker or other rate-controlling strategy to counter fluid retention and reflex tachycardia.

B. Potassium supplementation alone because minoxidil causes hypertension primarily through potassium wasting.

C. An ACE inhibitor and ARB together because dual renin-angiotensin blockade is mandatory whenever minoxidil is prescribed.

D. No additional therapy because edema and tachycardia prove that minoxidil is producing its desired antihypertensive effect.

160. A patient has persistent hypertension and a high-sodium diet despite taking three medications. Why is dietary sodium still clinically relevant?

A. Once three drugs are prescribed, sodium intake no longer influences blood pressure because medications completely override renal sodium handling.

B. High sodium intake improves the effect of ACE inhibitors by increasing renin release and therefore should be encouraged.

C. High sodium intake can blunt the response to multiple antihypertensive classes and contribute to volume-dependent treatment resistance.

D. Dietary sodium affects only diastolic pressure in children and has no relationship to adult resistant hypertension.

161. A patient has blood pressure 224/132 mm Hg, confusion, papilledema, and acute kidney injury. What distinguishes this presentation from severe asymptomatic hypertension?

A. The presence of new acute hypertension-mediated target-organ injury makes this a hypertensive emergency.

B. Any systolic pressure above 180 mm Hg is automatically an emergency even when there is no acute target-organ injury.

C. Papilledema and kidney injury indicate chronic uncomplicated hypertension rather than an acute emergency.

D. Hypertensive emergency is defined only by headache intensity and does not depend on organ injury or blood pressure context.

162. A nonpregnant patient has repeated blood pressure 188/122 mm Hg but no chest pain, neurologic deficit, pulmonary edema, kidney injury, or other acute target-organ damage. What is the best general approach?

A. Admit every such patient to intensive care for immediate intravenous reduction to below 120/80 mm Hg within minutes.

B. Give no treatment or follow-up because severe blood pressure without symptoms has no cardiovascular significance.

C. Use sublingual short-acting nifedipine to produce the fastest possible pressure drop before discharge.

D. Confirm accurate measurement and absence of acute target-organ damage, then intensify oral therapy with timely outpatient follow-up.

163. A patient has a hypertensive emergency without a condition requiring a disease-specific target. What is the usual initial blood pressure-lowering principle?

A. Normalize blood pressure to below 120/80 mm Hg within five minutes in every patient regardless of the affected organ.

B. Avoid any blood pressure reduction for the first 24 hours because autoregulation makes treatment more dangerous than the emergency itself.

C. Use only a single long-acting oral dose because intravenous titration increases pressure variability and cannot be monitored closely.

D. Use titratable intravenous therapy to achieve a controlled partial reduction, commonly about 20% to 25% in mean arterial pressure during the first hour, then lower more gradually.

164. A patient with tearing chest pain has acute aortic dissection and blood pressure 210/118 mm Hg. Which treatment sequence is preferred?

A. Start a pure arterial vasodilator before heart-rate control because reflex tachycardia reduces aortic wall stress.

B. Begin rapid intravenous beta blockade to reduce heart rate and aortic shear, then add a titratable vasodilator if blood pressure remains above target.

C. Delay treatment until systolic pressure falls spontaneously because lowering pressure increases dissection propagation.

D. Give an oral thiazide alone because gradual volume loss is the fastest way to reduce impulse on the aortic wall.

165. A patient presents with severe hypertension, acute pulmonary edema, diffuse crackles, and respiratory distress. Which acute treatment strategy is appropriate?

A. Use titratable intravenous nitroglycerin with ventilatory support as needed, plus diuresis when volume overload is present.

B. Give a large rapid crystalloid bolus because increasing preload is the primary treatment for hypertensive pulmonary edema.

C. Use a short-acting oral alpha agonist alone and delay respiratory support until blood pressure normalizes.

D. Avoid vasodilators because reducing afterload invariably worsens pulmonary edema in severe hypertension.

166. A patient with severe hypertension develops headache, confusion, seizures, and MRI findings consistent with posterior reversible encephalopathy syndrome. What is the treatment principle?

A. Allow pressure to remain above 220/120 mm Hg because cerebral symptoms indicate that autoregulation requires severe hypertension.

B. Lower blood pressure immediately to a normal level with an unmonitored oral medication because rapid normalization prevents cerebral ischemia.

C. Treat seizures only because blood pressure has no role in hypertensive encephalopathy once neurologic symptoms appear.

D. Treat as a hypertensive neurologic emergency with controlled intravenous blood pressure reduction while addressing seizures and other precipitants.

167. A patient with spontaneous intracerebral hemorrhage presents early with systolic blood pressure 188 mm Hg and otherwise mild-to-moderate ICH. Which acute pressure strategy best reflects modern guidance?

A. Use smooth, sustained lowering toward about 140 mm Hg, generally maintaining systolic pressure in the 130 to 150 mm Hg range rather than driving it below 130 mm Hg.

B. Maintain systolic pressure above 200 mm Hg because lowering blood pressure always increases hematoma expansion.

C. Reduce systolic pressure below 100 mm Hg as quickly as possible because the lowest attainable pressure improves cerebral perfusion.

D. Ignore blood pressure variability because only the single lowest systolic reading influences ICH outcome.

168. A patient with acute ischemic stroke is otherwise eligible for intravenous thrombolysis but the blood pressure is 198/116 mm Hg. What is required before thrombolytic treatment?

A. Proceed with thrombolysis at any pressure because blood pressure has no relationship to intracranial hemorrhage risk.

B. Lower blood pressure to below 100/60 mm Hg because deep hypotension improves penumbral perfusion before thrombolysis.

C. Withhold all reperfusion therapy permanently because any presenting systolic pressure above 180 mm Hg is an absolute uncorrectable contraindication.

D. Lower blood pressure to below 185/110 mm Hg before thrombolysis and maintain it below the post-treatment threshold afterward.

169. A patient with acute ischemic stroke is not receiving thrombolysis or thrombectomy and has blood pressure 206/112 mm Hg with no aortic dissection, acute coronary syndrome, pulmonary edema, or other pressure-dependent emergency. What is the usual early approach?

A. Immediately reduce systolic pressure below 110 mm Hg in every ischemic stroke because cerebral blood flow rises as systemic pressure falls.

B. Give no antihypertensive treatment even if systolic pressure exceeds 260 mm Hg because ischemic stroke permanently abolishes pressure-related complications.

C. Use sublingual nifedipine to cause a rapid fall because pressure variability improves perfusion of the ischemic penumbra.

D. Avoid routine aggressive lowering in the first day because modestly elevated pressure may support cerebral perfusion; treatment is generally reserved for substantially higher levels or another compelling indication.

170. A patient with acute coronary syndrome has severe hypertension and ongoing ischemic chest pain. Which treatment principle is most appropriate?

A. Raise blood pressure further because myocardial oxygen demand falls as afterload increases.

B. Use hydralazine alone to provoke reflex tachycardia because a faster heart rate improves coronary perfusion time.

C. Use titratable anti-ischemic therapy such as nitrates and appropriate beta blockade while avoiding an excessive fall in diastolic pressure.

D. Ignore the hypertension until troponin normalizes because blood pressure treatment has no role during acute myocardial ischemia.

171. A patient develops severe hypertension, tachycardia, agitation, and chest pain after cocaine use. Which acute strategy is appropriate?

A. Use a pure beta blocker as the only initial therapy and ignore agitation because sympathetic activation is unrelated to cocaine toxicity.

B. Give an oral mineralocorticoid receptor antagonist because cocaine hypertension is driven primarily by aldosterone excess.

C. Avoid sedation or vasodilation because spontaneous resolution is required before blood pressure can be treated safely.

D. Treat the hyperadrenergic state with benzodiazepines and use titratable vasodilator therapy when needed for persistent hypertension or ischemia.

172. A pregnant patient with chronic hypertension has repeated blood pressure readings of 144/92 mm Hg early in pregnancy. What treatment threshold is supported by the CHAP-informed ACOG approach?

A. Withhold all medication until blood pressure reaches 180/120 mm Hg because treatment below that level impairs fetal growth.

B. Treat only if diastolic pressure exceeds 120 mm Hg because systolic hypertension has no pregnancy-related risk.

C. Initiate or titrate antihypertensive therapy at persistent blood pressure of 140/90 mm Hg or higher rather than waiting for severe-range hypertension.

D. Stop any existing antihypertensive therapy until the third trimester because chronic hypertension should remain untreated early in pregnancy.

173. Which oral antihypertensive is commonly used for chronic hypertension during pregnancy?

A. Lisinopril, because fetal renal toxicity occurs only with ARBs and not with ACE inhibitors.

B. Losartan, because angiotensin receptor blockers are preferred throughout pregnancy for placental perfusion.

C. Extended-release nifedipine, with labetalol and methyldopa also established options depending on the patient.

D. Aliskiren, because direct renin inhibition has the longest safety experience in pregnancy.

174. Why should ACE inhibitors and ARBs generally be avoided during pregnancy?

A. They can disrupt fetal renal development and cause serious fetopathy, particularly with exposure later in pregnancy.

B. They cause maternal hyperthyroidism but have no direct fetal effects.

C. They increase uterine blood flow so profoundly that fetal macrosomia is inevitable.

D. They are avoided only because they are ineffective for lowering maternal blood pressure.

175. A pregnant patient has persistent blood pressure 168/112 mm Hg. What is the best acute management principle?

A. Observe without treatment until seizures occur because severe-range pressure is protective for uteroplacental perfusion.

B. Use an ACE inhibitor intravenously because renin-angiotensin blockade is the fastest and safest pregnancy treatment.

C. Lower systolic pressure below 90 mm Hg immediately because profound hypotension prevents preeclampsia complications.

D. Treat persistent severe-range hypertension promptly with protocol-based labetalol, hydralazine, or immediate-release oral nifedipine.

176. A patient with preeclampsia with severe features is being treated for severe hypertension. Which additional therapy reduces the risk of eclamptic seizures?

A. Mannitol because osmotic diuresis prevents eclampsia by lowering intracranial pressure in every patient.

B. Magnesium sulfate for seizure prophylaxis when indicated in preeclampsia with severe features, in addition to blood pressure treatment.

C. Warfarin because placental microthrombi are the primary mechanism of eclamptic seizures.

D. ACE inhibitor infusion because renin-angiotensin blockade is the preferred anticonvulsant in pregnancy.

177. A patient is discharged after a pregnancy complicated by hypertension and preeclampsia. Which postpartum principle is most important?

A. Stop all monitoring after placental delivery because preeclampsia and severe hypertension cannot occur postpartum.

B. Discontinue every antihypertensive immediately because lactation prevents treatment of postpartum blood pressure.

C. Continue close blood pressure surveillance because hypertension can persist or worsen after delivery and postpartum preeclampsia can present after hospital discharge.

D. Reassess blood pressure only at the next annual visit because early postpartum values do not predict complications.

178. Which patient meets a standard definition of resistant hypertension?

A. A patient with one high office reading while taking no medication and with normal home blood pressure.

B. Blood pressure above goal despite three appropriate drugs, including a diuretic, at tolerated doses after pseudoresistance is excluded.

C. A patient whose blood pressure is controlled on one low-dose thiazide but who reports a family history of hypertension.

D. A patient with untreated stage 1 hypertension who has not yet attempted lifestyle change.

179. A patient takes four antihypertensives and has office blood pressure 156/88 mm Hg, but 24-hour ambulatory blood pressure is normal. What is the best next conclusion?

A. A white-coat effect is contributing, so true uncontrolled resistant hypertension has not been demonstrated.

B. The ambulatory study proves masked hypertension because normal out-of-office pressure defines the masked phenotype.

C. The patient has a hypertensive emergency because discordance between office and ambulatory values is acute target-organ injury.

D. The ambulatory readings should be ignored because resistant hypertension can be diagnosed only from office measurements.

180. A patient appears to have resistant hypertension, but serum drug testing and refill history show inconsistent use of the prescribed regimen. What is the most important next step?

A. Address adherence barriers directly and reassess blood pressure on a regimen the patient is actually able and willing to take.

B. Diagnose refractory hypertension and proceed directly to an invasive device procedure.

C. Add several additional medications because adherence is irrelevant once three drugs have been prescribed.

D. Stop discussing adherence because objective evidence of missed medication cannot explain persistent hypertension.

181. A patient with resistant hypertension is taking lisinopril, amlodipine, and low-dose hydrochlorothiazide but remains volume expanded. What medication optimization is reasonable before moving to unusual therapies?

A. Stop the diuretic because sodium-volume excess is rarely relevant in resistant hypertension.

B. Optimize diuretic therapy, often by using an effective long-acting thiazide-like agent such as chlorthalidone when kidney function permits.

C. Replace all three drugs with short-acting clonidine taken only when systolic pressure exceeds 180 mm Hg.

D. Add an ACE inhibitor on top of lisinopril because two ACE inhibitors improve natriuresis without increasing risk.

182. A patient has true resistant hypertension despite an optimized ACE inhibitor or ARB, long-acting calcium channel blocker, and appropriate diuretic. Which fourth-line drug has particularly strong evidence when potassium and kidney function permit?

A. Spironolactone, provided kidney function and potassium permit mineralocorticoid receptor blockade as fourth-line therapy.

B. Immediate-release nifedipine used intermittently because rapid pressure fluctuations improve long-term control.

C. A second ACE inhibitor because using two agents from the same class is safer than mineralocorticoid receptor blockade.

D. Fludrocortisone because expanding volume is the best strategy in resistant hypertension.

183. A patient with stage 4 CKD and resistant hypertension has edema and high dietary sodium intake. What management issue is especially important?

A. Increase sodium intake because pressure natriuresis requires greater dietary sodium when GFR is reduced.

B. Stop all diuretics because advanced CKD makes tubular sodium transport irrelevant to blood pressure.

C. Control extracellular-volume excess with sodium restriction and an appropriately potent diuretic strategy, often including a loop diuretic at advanced CKD stages.

D. Treat only with a beta blocker because volume status has little influence on resistant hypertension in kidney disease.

184. A patient has hypertension and heart failure with reduced ejection fraction. Which treatment strategy best integrates the two conditions?

A. Treat blood pressure with a dihydropyridine alone and avoid all heart-failure disease-modifying drugs because they lower pressure.

B. Use verapamil as mandatory first-line therapy because negative inotropy improves systolic heart failure outcomes.

C. Stop renin-angiotensin system therapy because reducing afterload worsens forward flow in HFrEF.

D. Prioritize HFrEF guideline-directed therapies that also lower blood pressure, including RAAS-pathway therapy, evidence-based beta blockade, an MRA, and an SGLT2 inhibitor.

185. A patient with heart failure with preserved ejection fraction also has persistent hypertension. What is the key blood pressure principle?

A. Maintain systolic pressure above 180 mm Hg because HFpEF ventricles require severe hypertension for filling.

B. Control hypertension to the usual evidence-based target while avoiding symptomatic hypotension and treating congestion and other HFpEF comorbidities.

C. Avoid all antihypertensive treatment because reducing afterload always worsens diastolic relaxation.

D. Use only short-acting vasodilators as needed because sustained pressure control has no role in HFpEF.

186. A patient with chronic coronary artery disease and hypertension remains above goal. Which general strategy is appropriate?

A. Allow systolic pressure above 160 mm Hg because coronary perfusion requires persistent severe hypertension.

B. Use a pure arterial vasodilator alone even if it causes reflex tachycardia and worsens angina.

C. Avoid all renin-angiotensin blockers and beta blockers because they increase myocardial oxygen demand.

D. Treat toward below 130/80 mm Hg if tolerated, choosing agents that also fit coronary indications such as beta blockade or RAAS inhibition.

187. A patient with an enlarging thoracic aortic aneurysm has hypertension. What chronic treatment principle is most important?

A. Achieve consistent blood pressure control and reduce aortic wall stress, often using beta-blocking or RAAS-directed therapy when appropriate.

B. Encourage large blood pressure swings because intermittent high wall stress promotes adaptive thickening of the aneurysm.

C. Avoid treating systolic hypertension because lower pressure accelerates aneurysm expansion.

D. Use short-acting sympathomimetics to maintain tachycardia and thereby reduce time spent in systole.

188. A patient with a prior ischemic stroke has persistent hypertension after the acute recovery phase. What long-term target is appropriate for most patients?

A. Maintain systolic pressure above 160 mm Hg indefinitely because permissive hypertension remains necessary after the acute stroke period.

B. A blood pressure below 130/80 mm Hg if tolerated as part of secondary stroke prevention.

C. Treat only diastolic pressure because recurrent stroke risk is unrelated to systolic pressure.

D. Stop antihypertensive therapy after neurologic recovery because blood pressure reduction increases all recurrent stroke risk.

189. A patient with peripheral artery disease has hypertension and a high cardiovascular risk burden. Which statement is most appropriate?

A. Maintain severe hypertension because lowering systemic pressure invariably worsens claudication and limb outcomes.

B. Use no ACE inhibitor or ARB in peripheral artery disease because renin-angiotensin blockade causes acute limb ischemia.

C. Treat hypertension according to standard cardiovascular risk-reduction goals rather than withholding therapy because of concern about limb perfusion.

D. Treat only if diastolic pressure exceeds 120 mm Hg because systolic hypertension does not affect vascular-event risk in PAD.

190. A patient has hypertension and type 2 diabetes without symptomatic orthostasis. What blood pressure goal is appropriate for most such adults under current U.S. guidance?

A. A goal below 130/80 mm Hg is appropriate for most adults with diabetes when it can be achieved safely and without symptomatic hypotension.

B. Below 160/100 mm Hg only, because diabetes makes lower pressure targets harmful in all patients.

C. Below 110/60 mm Hg in every patient, regardless of age, autonomic neuropathy, or treatment tolerance.

D. No numerical goal is used in diabetes because glucose control eliminates the cardiovascular effects of blood pressure.

191. A patient with obesity and hypertension asks how intentional weight loss fits into the blood pressure plan. What is the best answer?

A. Weight loss should be avoided because reducing adipose tissue increases sympathetic tone and raises blood pressure.

B. Anti-obesity treatment has no relevance to hypertension because weight and blood pressure are physiologically independent.

C. Sustained weight loss can lower blood pressure and cardiovascular risk, and evidence-based obesity treatment including pharmacotherapy may be appropriate when indicated.

D. Antihypertensive drugs should be stopped whenever weight-loss therapy is started because hypotension is inevitable.

192. A patient with hypertension also has markedly elevated LDL cholesterol and high atherosclerotic cardiovascular risk. What is the best risk-reduction principle?

A. Treat blood pressure only because LDL cholesterol ceases to predict events once systolic pressure is below 140 mm Hg.

B. Treat lipids only because statin therapy directly normalizes blood pressure in most hypertensive patients.

C. Avoid combining lipid-lowering and antihypertensive therapy because the treatments cancel each other's cardiovascular benefit.

D. Treat both blood pressure and lipid risk with evidence-based therapies because controlling one risk factor does not eliminate the harm from the other.

193. Which finding meets the standard definition of orthostatic hypotension?

A. Any rise in heart rate of 10 beats/minute after standing, regardless of blood pressure change.

B. A systolic pressure increase of 20 mm Hg after standing because orthostatic hypotension is defined by pressure variability in either direction.

C. A diastolic fall of 2 mm Hg after ten minutes of standing because any measurable decline meets diagnostic criteria.

D. A sustained fall of at least 20 mm Hg systolic or 10 mm Hg diastolic within 3 minutes of standing.

194. A hypertensive older adult has symptomatic orthostatic hypotension. What should be done before adding a pressor medication?

A. Intensify every antihypertensive because standing symptoms prove that seated blood pressure remains undertreated.

B. Begin high-dose midodrine immediately without reviewing medications or supine blood pressure.

C. Identify reversible causes, review medications and volume status, and use individualized nonpharmacologic measures before adding a pressor.

D. Restrict fluids aggressively because volume contraction improves orthostatic cerebral perfusion.

195. A patient with neurogenic orthostatic hypotension has disabling symptoms and also severe supine hypertension at night. Which treatment issue is especially important?

A. Pressor therapy such as midodrine or droxidopa can worsen supine hypertension, so dose timing and nighttime positioning require careful planning.

B. Supine hypertension proves the orthostatic diagnosis is false because the two conditions cannot coexist.

C. Pressor drugs should be taken immediately before lying flat at bedtime because supine administration prevents nocturnal hypertension.

D. Treat supine hypertension with continuous daytime bed rest because standing increases nocturnal blood pressure.

196. A patient with well-controlled hypertension develops higher pressures after venlafaxine is increased for depression. What is the best management principle?

A. Assume the rise is unrelated because psychiatric medications cannot affect sympathetic tone or blood pressure.

B. Recognize venlafaxine as a possible contributor and coordinate psychiatric and antihypertensive management rather than escalating blindly.

C. Stop all psychiatric treatment abruptly because withdrawal cannot influence blood pressure or mental health.

D. Diagnose primary aldosteronism solely from the venlafaxine dose change because antidepressants do not alter cardiovascular physiology.

197. A 15-year-old has repeated auscultatory blood pressure readings of 132/82 mm Hg. How is this classified under the pediatric framework for adolescents age 13 years or older?

A. Normal blood pressure because pediatric percentiles must be used exclusively until age 18 years.

B. Elevated blood pressure but not hypertension because stage 1 begins only at 140/90 mm Hg for all adolescents.

C. Stage 1 hypertension using the adult-like threshold of 130 to 139 systolic or 80 to 89 diastolic mm Hg.

D. Stage 2 hypertension because any diastolic pressure above 80 mm Hg is stage 2 after age 13.

198. An 8-year-old has auscultatory blood pressure at or above the 95th percentile on three separate visits. What conclusion is appropriate?

A. Hypertension cannot be diagnosed before age 13 because only adolescents develop sustained primary hypertension.

B. A single percentile chart is irrelevant because pediatric hypertension is defined only by an absolute pressure above 160/100 mm Hg.

C. The child has normal blood pressure because percentile-based thresholds are used only for height assessment, not blood pressure.

D. The repeated auscultatory readings meet the office criterion for pediatric hypertension and warrant evaluation appropriate to age and severity.

199. A 10-year-old has stage 1-range office blood pressure at three separate visits without symptoms. Which test is especially useful for confirming sustained hypertension and identifying white-coat hypertension?

A. Ambulatory blood pressure monitoring to confirm sustained pediatric hypertension and help identify a white-coat pattern.

B. A single emergency-department blood pressure because acute-care readings are the preferred pediatric reference standard.

C. A serum troponin level because myocardial injury is required before pediatric hypertension can be confirmed.

D. A renal biopsy in every child because tissue diagnosis must precede confirmation of elevated blood pressure.

200. A child has confirmed hypertension that persists despite lifestyle treatment and has left ventricular hypertrophy. Which pharmacologic approach is appropriate?

A. Avoid medication until adulthood because pediatric target-organ damage does not justify antihypertensive treatment.

B. Use short-acting nifedipine as needed for individual high readings because sustained daily treatment impairs normal growth.

C. Treat only with an alpha-1 blocker because ACE inhibitors, ARBs, calcium channel blockers, and thiazides are contraindicated in children.

D. Begin an age-appropriate first-line drug such as an ACE inhibitor, ARB, long-acting calcium channel blocker, or thiazide and titrate to the pediatric target.

Answers

1. A. Mean arterial pressure is approximately cardiac output multiplied by systemic vascular resistance when right atrial pressure is small relative to arterial pressure. Cardiac output itself equals heart rate times stroke volume, while pulse pressure is influenced strongly by stroke volume and arterial compliance rather than serving as the primary determinant of mean pressure.

2. C. Long-term arterial pressure is tightly linked to renal sodium handling. In salt-sensitive hypertension, the pressure-natriuresis curve is shifted such that higher arterial pressure is needed to excrete a given sodium intake and restore sodium balance.

3. B. Renin secretion rises when renal perfusion pressure falls, when beta-1 adrenergic signaling increases, and when macula densa sodium chloride delivery decreases. These inputs activate the renin-angiotensin-aldosterone system to restore effective circulating volume and pressure.

4. A. AT1 receptor signaling mediates most familiar pressor actions of angiotensin II, including vasoconstriction, aldosterone release, sodium retention, thirst, and sympathetic facilitation. These combined effects increase effective circulating volume and systemic vascular resistance.

5. A. Mineralocorticoid receptor activation in principal cells increases epithelial sodium channel activity and sodium-potassium ATPase expression, promoting sodium retention and potassium secretion. This is why mineralocorticoid excess can produce hypertension, hypokalemia, and metabolic alkalosis.

6. D. Baroreflexes buffer short-term blood pressure fluctuations, but with sustained hypertension they reset toward the prevailing higher pressure. This adaptation limits their ability to normalize chronic hypertension and leaves long-term pressure regulation largely dependent on renal and hormonal mechanisms.

7. A. Endothelial nitric oxide diffuses into vascular smooth muscle and stimulates soluble guanylate cyclase, increasing cyclic GMP and causing relaxation. Reduced nitric oxide bioavailability is one mechanism contributing to endothelial dysfunction and increased vascular resistance in hypertension.

8. B. Age-related stiffening of the aorta and other conduit arteries reduces their ability to buffer ventricular ejection. Systolic pressure rises, diastolic pressure may fall, and pulse pressure widens, producing the common phenotype of isolated systolic hypertension.

9. A. Arterial stiffening increases pulse-wave velocity. The reflected wave therefore returns to the central aorta earlier, often during late systole rather than diastole, increasing central systolic load and pulse pressure.

10. A. Atrial and B-type natriuretic peptides oppose volume and pressure expansion by promoting natriuresis, vasodilation, and inhibition of the renin-angiotensin-aldosterone system. Their actions counter sodium-retaining and vasoconstrictor pathways.

11. B. Thiazide and thiazide-like diuretics inhibit the sodium-chloride cotransporter in the distal convoluted tubule. Loop diuretics inhibit NKCC2, while amiloride and triamterene inhibit epithelial sodium channels in the distal nephron.

12. A. Loop diuretics inhibit NKCC2 in the thick ascending limb, producing potent natriuresis and reducing medullary concentrating ability. Their site and mechanism distinguish them from thiazides, ENaC blockers, and mineralocorticoid receptor antagonists.

13. C. Dihydropyridine calcium channel blockers such as amlodipine act predominantly on vascular L-type calcium channels, producing arteriolar vasodilation. Non-dihydropyridines have more prominent cardiac conduction and rate effects.

14. D. Angiotensin-converting enzyme also degrades bradykinin. ACE inhibition therefore increases bradykinin and related peptides, contributing to cough and, rarely, angioedema; ARBs do not inhibit bradykinin degradation in the same way.

15. D. ARBs selectively block AT1 receptor effects of angiotensin II, reducing vasoconstriction and aldosterone-mediated sodium retention. Unlike ACE inhibitors, they do not inhibit ACE and therefore do not characteristically increase bradykinin to the same extent.

16. A. Juxtaglomerular cells express beta-1 adrenergic receptors, and sympathetic activation increases renin release. Beta blockade can therefore reduce renin in addition to lowering heart rate and myocardial contractility.

17. C. Alpha-1 receptor blockade reduces catecholamine-mediated vascular smooth-muscle contraction. The resulting arteriolar and venous dilation can lower pressure but may also produce orthostatic symptoms, especially after the first dose.

18. D. Central alpha-2 agonists decrease sympathetic outflow, lowering vascular resistance and often heart rate. Abrupt withdrawal, especially of clonidine, can produce rebound sympathetic activation and marked hypertension.

19. D. Spironolactone and eplerenone are mineralocorticoid receptor antagonists. They reduce aldosterone-driven sodium retention and are particularly effective in primary aldosteronism and many patients with resistant hypertension.

20. C. Liddle syndrome causes constitutive ENaC activation, sodium retention, hypertension, hypokalemia, and metabolic alkalosis with both renin and aldosterone suppressed. Treatment targets ENaC with amiloride or triamterene rather than mineralocorticoid receptor blockade.

21. A. Glycyrrhetinic acid in licorice can inhibit 11-beta-HSD2, the enzyme that normally protects mineralocorticoid receptors from cortisol. Cortisol then acts as a mineralocorticoid, producing sodium retention, hypokalemia, hypertension, and suppression of renin and aldosterone.

22. A. Gordon syndrome results from abnormalities in WNK-related signaling that increase sodium-chloride cotransporter activity. The phenotype includes hypertension, hyperkalemia, and metabolic acidosis, and low-dose thiazide therapy is characteristically effective.

23. C. Salt sensitivity is a phenotype in which changes in sodium intake produce larger changes in arterial pressure. It reflects interacting renal, vascular, genetic, and neurohormonal factors and is more common in several high-risk clinical groups, but it is not synonymous with one specific endocrine disorder.

24. C. Sympathetic activation raises pressure through alpha-mediated vasoconstriction, beta-1 cardiac stimulation, and renal effects that include renin release and enhanced sodium retention. Chronic sympathetic overactivity can therefore reinforce both resistance and volume components of hypertension.

25. A. Endothelin-1 is a powerful endothelial-derived vasoconstrictor. Excess endothelin activity can contribute to increased vascular resistance and vascular remodeling, counterbalancing vasodilator pathways such as nitric oxide.

26. A. Chronic pressure and neurohormonal stimuli promote hypertrophy and remodeling of resistance vessels. A higher wall-to-lumen ratio narrows the effective lumen and helps maintain elevated systemic vascular resistance even when the original trigger is less prominent.

27. D. Angiotensin II preferentially constricts the efferent arteriole at physiologic concentrations, helping preserve glomerular capillary pressure when renal perfusion falls. This can maintain filtration while reducing renal blood flow and increasing filtration fraction.

28. A. ACE inhibition reduces angiotensin II-mediated efferent arteriolar constriction, so the efferent arteriole dilates and intraglomerular pressure falls. This is therapeutically useful in albuminuric kidney

disease but can produce a larger creatinine rise when glomerular filtration is highly angiotensin dependent.

29. B. Population studies show a graded relationship between blood pressure and vascular risk. Diagnostic thresholds are useful for clinical decisions, but the underlying risk relationship is continuous, especially for stroke, heart failure, coronary disease, kidney disease, and mortality.

30. C. Obesity-related hypertension is multifactorial. Renal sodium retention, sympathetic activation, RAAS and mineralocorticoid activity, sleep apnea, insulin resistance, and altered adipokines can all contribute to increased effective volume and vascular tone.

31. A. A cuff that is too small tends to overestimate blood pressure, while a cuff that is too large can underestimate it. Accurate diagnosis begins with a validated device and a cuff sized to the patient's arm circumference.

32. B. Standardized technique reduces systematic error. Rest, supported posture, uncrossed feet, an arm supported at heart level, an appropriate cuff, and avoidance of talking are core elements of reliable office blood pressure measurement.

33. B. Blood pressure varies beat to beat and from moment to moment. Repeated standardized measurements, often averaged, provide a more reliable estimate than a single office reading.

34. C. White-coat hypertension describes elevated office blood pressure with normal out-of-office pressure in an untreated patient; a similar pattern during treatment is often called a white-coat effect. Out-of-office monitoring helps prevent unnecessary intensification based only on clinic values.

35. A. Masked hypertension is the inverse of the white-coat pattern: office values appear normal while home or ambulatory pressure is elevated. Because the true pressure burden is missed in clinic, target-organ and cardiovascular risk can be underestimated.

36. A. Ambulatory monitoring provides information that office measurements cannot, including nocturnal pressure and dipping status. Elevated sleep pressure and absent or reversed dipping are associated with increased target-organ and cardiovascular risk and can occur with conditions such as CKD and sleep apnea.

37. C. Classic orthostatic hypotension is defined by a sustained fall in systolic pressure of at least 20 mm Hg or diastolic pressure of at least 10 mm Hg within 3 minutes of standing. Heart-rate behavior helps distinguish volume depletion from impaired autonomic compensation.

38. B. A reproducible inter-arm difference can reflect subclavian or other arterial disease and is associated with vascular risk. The finding should be confirmed, and the higher-pressure arm is typically used for ongoing blood pressure assessment unless a specific vascular issue dictates otherwise.

39. C. The current U.S. classification defines normal as less than 120/80 mm Hg, elevated as systolic 120-129 with diastolic below 80, stage 1 as 130-139 systolic or 80-89 diastolic, and stage 2 as at least 140 systolic or at least 90 diastolic.

40. B. Baseline evaluation aims to identify cardiovascular risk factors, kidney disease, treatment-relevant electrolyte abnormalities, and evidence suggesting secondary hypertension or target-organ injury. Extensive endocrine or vascular testing is reserved for patients whose history, examination, or initial results raise suspicion.

41. B. Albuminuria can precede a decline in eGFR and signals glomerular injury. It refines kidney and cardiovascular risk and may support use of renin-angiotensin system blockade when clinically appropriate.

42. D. Chronic pressure overload promotes concentric left ventricular hypertrophy. ECG criteria have limited sensitivity but high voltage with a repolarization strain pattern can be a useful marker of hypertensive cardiac target-organ damage.

43. B. Echocardiography can define left ventricular hypertrophy, systolic or diastolic dysfunction, valvular disease, and other structural abnormalities when the result would affect care. It is not mandatory for every uncomplicated case of newly diagnosed hypertension.

44. C. Retinal hemorrhages, exudates, and especially optic-disc swelling can accompany severe or accelerated hypertension and signal significant target-organ injury. The clinical setting determines whether the patient has a hypertensive emergency requiring immediate treatment.

45. A. Chronic hypertension can cause and accelerate kidney injury, including declining GFR and albuminuria. The relationship is bidirectional, however, so clinicians should also assess whether primary kidney disease is driving the hypertension.

46. A. Severe, abrupt, unusually early, or resistant hypertension increases suspicion for a secondary cause. Accurate measurement and confirmation remain essential, followed by a focused evaluation based on history, examination, laboratory findings, and likely etiologies.

47. B. Ambulatory blood pressure monitoring samples blood pressure repeatedly over a full day and night. It can identify white-coat and masked hypertension, quantify nocturnal pressure and dipping, and provide a more complete estimate of pressure burden than isolated office readings.

48. D. Home monitoring is valuable only when technique is reliable. A validated upper-arm cuff, appropriate sizing, rest, proper posture, and repeated readings at consistent times reduce noise and make the average more useful for clinical decisions.

49. A. Pulse pressure is the difference between systolic and diastolic pressure. A widened pulse pressure in older adults often reflects increased large-artery stiffness and is common in isolated systolic hypertension.

50. B. Resistant hypertension requires confirmation that blood pressure remains uncontrolled outside the office, along with assessment of adherence and adequate treatment. A normal ambulatory profile despite elevated clinic readings suggests a white-coat effect rather than true resistance.

51. B. Chronic kidney disease is a common secondary contributor to hypertension. Impaired sodium excretion, extracellular-volume expansion, sympathetic activation, and renin-angiotensin system effects can all raise blood pressure as kidney disease progresses.

52. D. Fibromuscular dysplasia is a nonatherosclerotic, noninflammatory arteriopathy that can produce the classic beaded appearance and renovascular hypertension, often in younger women. The location and imaging pattern differ from typical atherosclerotic renal artery disease.

53. B. Recurrent flash pulmonary edema is a classic high-risk presentation that should raise concern for clinically significant renovascular disease. Atherosclerotic renal artery stenosis is especially plausible in an older patient with widespread vascular disease.

54. D. In severe bilateral renovascular disease, filtration pressure may depend heavily on angiotensin II-mediated efferent vasoconstriction. Blocking that response can cause a large fall in GFR and a marked creatinine rise.

55. B. The 2025 Endocrine Society guideline broadens screening by suggesting biochemical screening for primary aldosteronism in adults with hypertension. Aldosterone, renin, and their ratio are interpreted together, with attention to potassium, medications, sodium status, and the assay used.

56. A. Primary aldosteronism screening depends on both aldosterone and renin, not on the ratio alone. Potassium, medication effects, sodium intake, posture, timing, and assay-specific thresholds can materially affect interpretation.

57. B. Hypokalemia can suppress aldosterone secretion and create a falsely reassuring result. When suspicion remains high, potassium should be corrected and screening repeated under more interpretable conditions.

58. D. Modern primary aldosteronism guidance allows selective use of suppression testing. When biochemical evidence is unequivocal and pretest probability is high, additional suppression testing may add little before subtype evaluation or treatment.

59. A. Incidental nonfunctioning adrenal nodules are common, especially with increasing age. Adrenal venous sampling is generally the preferred method for lateralization in patients considering surgery because anatomy alone can be misleading.

60. C. Unilateral, surgically correctable primary aldosteronism is treated with adrenalectomy in an appropriate operative candidate. Surgery can correct hypokalemia and may cure or substantially improve hypertension.

61. D. Bilateral or nonsurgical primary aldosteronism is treated medically, most directly with mineralocorticoid receptor antagonism. Treatment reduces sodium retention and potassium wasting and targets the cardiovascular consequences of aldosterone excess.

62. C. Metanephrines are continuously produced from catecholamines within these tumors and are sensitive biochemical markers. Testing technique matters because stress, posture, medications, and other factors can increase false-positive results.

63. D. Blocking beta receptors before alpha receptors can leave unopposed alpha-adrenergic vasoconstriction and precipitate a severe hypertensive crisis. Alpha blockade, with volume preparation as appropriate, precedes beta blockade when beta blockade is needed.

64. C. Obstructive sleep apnea is common in patients with resistant hypertension and is suggested by snoring, witnessed apneas, sleepiness, and obesity. Evaluation and treatment of sleep-disordered breathing should accompany optimization of antihypertensive therapy.

65. B. The combination of hypertension, catabolic skin and muscle findings, and new metabolic abnormalities is characteristic of hypercortisolism. Cushing syndrome should be evaluated with an appropriate validated screening strategy.

66. D. Excess thyroid hormone increases heart rate, contractility, and cardiac output and can lower systemic vascular resistance. The resulting pattern may feature systolic hypertension and a widened pulse pressure.

67. D. Hypothyroidism can increase systemic vascular resistance and is associated with diastolic hypertension in some patients. Treating the underlying thyroid disorder may improve the pressure abnormality.

68. B. Hyperparathyroidism and hypercalcemia can be associated with hypertension. The accompanying nephrolithiasis and hypercalcemia make parathyroid disease a relevant secondary diagnostic consideration.

69. D. Coarctation classically causes upper-extremity hypertension with diminished or delayed femoral pulses and lower pressure in the legs. A murmur over the back and collateral vessels may provide additional clues.

70. B. NSAIDs can increase blood pressure by promoting sodium retention and altering renal hemodynamics. They may also reduce the effectiveness of several antihypertensive regimens, so medication review is important when control worsens.

71. B. Estrogen-containing contraceptives can raise blood pressure in susceptible individuals. A careful medication history is therefore part of evaluating new or worsening hypertension.

72. C. Sympathomimetic drugs can increase vascular tone and heart rate and may worsen blood pressure control. Decongestants and stimulants should be included in a secondary-hypertension medication review.

73. D. Calcineurin inhibitors such as tacrolimus and cyclosporine can cause or worsen hypertension through renal vasoconstriction, sodium retention, and other vascular effects. Drug exposure and kidney function should be considered in transplant-associated hypertension.

74. C. Hypertension is a recognized adverse effect of erythropoiesis-stimulating agents. In a patient with CKD and worsening control, timing of the pressure change relative to therapy can provide an important clue.

75. C. Licorice can permit cortisol to activate mineralocorticoid receptors in the kidney by inhibiting 11-beta-HSD2. The resulting sodium retention, potassium loss, hypertension, and renin-aldosterone suppression can improve after the exposure is removed.

76. D. Cocaine and amphetamine-type stimulants can produce abrupt catecholamine-mediated hypertension, tachycardia, vasoconstriction, and ischemic complications. Exposure history is essential in sudden severe hypertension.

77. C. Heavy alcohol consumption can contribute to hypertension and treatment resistance. Reducing excessive intake is a reversible lifestyle target and should be addressed as part of the hypertension evaluation.

78. C. A reninoma causes renin-driven secondary hyperaldosteronism, often producing hypertension, hypokalemia, and metabolic alkalosis in a young patient. The high-renin pattern distinguishes it from primary mineralocorticoid states.

79. A. Liddle syndrome is caused by gain-of-function ENaC activity, producing sodium retention with suppression of renin and aldosterone. Direct ENaC blockers such as amiloride or triamterene target the abnormal pathway; spironolactone does not.

80. B. Gordon syndrome, also called pseudohypoaldosteronism type II, produces hyperkalemic hypertension and metabolic acidosis through increased distal sodium-chloride transport. Thiazide therapy is particularly effective because it targets that transporter.

81. C. 11-beta-HSD2 normally converts cortisol to cortisone in mineralocorticoid target tissues. Loss of that protection permits cortisol to activate mineralocorticoid receptors, causing sodium retention, hypokalemia, and suppression of renin and aldosterone.

82. A. 11-beta-hydroxylase deficiency increases deoxycorticosterone, a mineralocorticoid-active steroid, while also increasing androgen production. The combination can produce hypertension, hypokalemia, and androgen excess.

83. C. 17-alpha-hydroxylase deficiency impairs cortisol and sex-steroid synthesis while shunting precursors toward mineralocorticoid-active pathways. Hypertension and hypokalemia therefore coexist with sexual underdevelopment.

84. D. Growth hormone excess in acromegaly is associated with hypertension, sleep apnea, insulin resistance, and characteristic acral and facial changes. The phenotype plus elevated IGF-1 makes acromegaly the unifying diagnosis.

85. C. Several noninvasive approaches can evaluate renal arteries, and the best first test depends on the patient and available expertise. Invasive angiography is generally reserved for selected cases, especially when an intervention is contemplated or noninvasive studies are inconclusive.

86. A. Renal fibromuscular dysplasia may respond well to angioplasty when revascularization is indicated. Stenting is not routinely required unless there is a specific procedural reason such as a complication or inadequate angioplasty result.

87. C. Randomized evidence has not supported routine revascularization for stable atherosclerotic renal artery stenosis. Intervention is generally reserved for selected high-risk clinical syndromes rather than the anatomic lesion alone.

88. D. Selected high-risk syndromes, including recurrent flash pulmonary edema and some cases of rapidly worsening kidney function or refractory hypertension, may justify revascularization consideration. Stable incidental disease is usually managed medically.

89. C. Medication and substance review is a high-yield part of evaluating apparent resistant or newly worsened hypertension. NSAIDs, sympathomimetics, stimulants, hormones, calcineurin inhibitors, alcohol, licorice, and other exposures can contribute and may be reversible.

90. A. Clonidine suppresses central sympathetic outflow. Abrupt withdrawal can produce a marked sympathetic rebound with tachycardia and severe hypertension, so tapering is generally preferred when the drug is discontinued.

91. C. The 2025 U.S. guideline retains an overarching treatment target below 130/80 mm Hg for most adults. Clinical judgment is important in settings such as limited life expectancy, institutional care, symptomatic hypotension, or pregnancy.

92. D. Current U.S. guidance recommends pharmacologic treatment when average blood pressure is at least 140/90 mm Hg, regardless of the lower-risk threshold framework. Lifestyle modification remains part of treatment rather than an alternative to indicated medication.

93. C. For stage 1 hypertension, established cardiovascular disease is a high-risk feature that favors medication in addition to lifestyle therapy. The overall goal for most such patients is below 130/80 mm Hg if tolerated.

94. C. In lower-risk adults with stage 1 hypertension, current guidance allows a structured trial of lifestyle treatment first. Persistent pressure at or above 130/80 mm Hg after 3 to 6 months supports adding medication.

95. D. Stage 2 hypertension is commonly treated initially with two first-line agents because a single drug is unlikely to achieve the required reduction. Single-pill combinations can simplify treatment and improve adherence.

96. D. Single-pill combinations can make a multidrug regimen easier to take consistently. They do not remove the need for dose adjustment, monitoring, or attention to class-specific adverse effects.

97. B. The DASH eating pattern is a foundational nonpharmacologic treatment for hypertension. Its blood pressure effect can be enhanced further by reducing dietary sodium.

98. D. Lower sodium intake reduces blood pressure, particularly in salt-sensitive individuals. Dietary counseling should be practical and should also consider kidney function and the safety of potassium-containing substitutes.

99. B. Dietary potassium can support blood pressure reduction, but the recommendation is not universal. CKD, hyperkalemia, and medications such as ACE inhibitors, ARBs, and mineralocorticoid antagonists require caution.

100. B. Excess adiposity contributes to hypertension through sympathetic, renal, hormonal, and sleep-related pathways. Sustained weight loss is therefore an important treatment strategy and may improve several cardiovascular risk factors simultaneously.

101. A. Regular physical activity lowers resting blood pressure and improves overall cardiovascular health. Aerobic exercise has strong evidence, and resistance or isometric training can also contribute when appropriately prescribed.

102. C. Excess alcohol use can elevate blood pressure and increase cardiovascular risk. Reducing heavy intake is a modifiable component of comprehensive hypertension treatment.

103. B. Sleep quality and chronic stress can influence sympathetic activation, health behaviors, and blood pressure. They should be addressed alongside diet, physical activity, weight, alcohol, and medication adherence rather than as stand-alone substitutes for indicated therapy.

104. A. Thiazide-type and thiazide-like diuretics are established first-line antihypertensive drugs. They reduce sodium reabsorption in the distal convoluted tubule and have extensive cardiovascular outcome evidence.

105. B. Chlorthalidone is a long-acting thiazide-like diuretic with extensive trial experience. Choice among thiazide-type agents should also account for dose, kidney function, electrolytes, gout risk, and patient tolerance.

106. A. Long-acting dihydropyridine calcium channel blockers are effective first-line agents and are particularly useful for systolic hypertension. Short-acting formulations that cause rapid pressure swings are not preferred for routine chronic treatment.

107. B. ACE inhibitors and ARBs lower systemic pressure and intraglomerular pressure and are particularly useful when CKD is accompanied by albuminuria. Kidney function and potassium should be reassessed after initiation or dose escalation.

108. A. ACE inhibitor cough is related largely to bradykinin and other peptide accumulation. ARBs block AT1 receptors without inhibiting ACE and therefore cause cough much less often.

109. C. ACE inhibitor-associated cough can be persistent and is not dose-corrected by adding more ACE inhibition. Substitution with an ARB is often reasonable when the clinical indication for renin-angiotensin system blockade remains.

110. D. ACE inhibitor-associated angioedema is potentially dangerous and requires permanent avoidance of the offending class. Acute airway management takes priority when tongue or laryngeal involvement is present.

111. B. ACE inhibitors and ARBs can reduce GFR in susceptible settings and increase serum potassium. Follow-up creatinine and potassium help identify clinically important renal hypoperfusion, hyperkalemia, or excessive hemodynamic change.

112. B. Renin-angiotensin blockade dilates the efferent arteriole and can cause a predictable hemodynamic decrease in GFR. A large or progressive rise should prompt evaluation for volume depletion, NSAID use, renovascular disease, or other contributors.

113. D. Combining ACE inhibitors and ARBs can produce greater laboratory changes but has not shown a favorable benefit-risk profile for routine hypertension management. One renin-angiotensin blocker is generally selected rather than using both together.

114. A. Thiazide and thiazide-like diuretics can cause clinically important hyponatremia, especially in older adults and other susceptible patients. Electrolytes should be checked when symptoms or risk factors suggest this complication.

115. A. Thiazides can raise serum uric acid and may precipitate gout. The overall regimen should balance blood pressure benefit against adverse effects and alternative drug choices.

116. D. Dihydropyridine-associated edema results mainly from an imbalance between precapillary arteriolar and postcapillary venous dilation. It is not simply sodium retention, which is why escalating a diuretic may not fully address it.

117. C. Beta blockers remain important for conditions such as ischemic heart disease, certain arrhythmias, and selected heart-failure settings. For otherwise uncomplicated hypertension, thiazide-type

diuretics, ACE inhibitors or ARBs, and long-acting calcium channel blockers are generally favored as initial classes.

118. B. In coronary disease, beta blockade can serve both antihypertensive and antianginal goals by reducing heart rate, contractility, and myocardial oxygen demand. The specific drug and duration depend on the patient's cardiac condition.

119. D. Spironolactone can cause gynecomastia, breast tenderness, and sexual adverse effects because of off-target steroid receptor activity. Eplerenone is more selective, though dosing, cost, potassium, and kidney function still matter.

120. A. Apparent treatment resistance is frequently explained or amplified by measurement error, nonadherence, white-coat effect, excess sodium, or interfering medications. These factors should be addressed before labeling hypertension truly resistant.

121. C. Structured home monitoring can improve diagnosis and treatment adjustment when technique and device accuracy are reliable. Multiple readings over time are more informative than isolated high or low values.

122. A. Team-based hypertension care can improve treatment intensity, follow-up, education, adherence, and achievement of blood pressure goals. It is particularly useful when paired with reliable home measurements and clear titration protocols.

123. D. Current hypertension guidance emphasizes validated blood pressure measurement. Cuffless devices are promising but should not replace validated cuff-based measurements for clinical diagnosis and management without adequate validation.

124. D. SPRINT provided pivotal evidence that intensive blood pressure treatment can improve cardiovascular outcomes in selected high-risk adults. Its findings must be interpreted with the trial's standardized measurement methods, population, and adverse-event monitoring in mind.

125. C. ALLHAT reinforced the effectiveness of thiazide-type diuretic therapy for major cardiovascular outcomes. It also showed why antihypertensive choice should be guided by outcome evidence rather than novelty alone.

126. C. ACCOMPLISH demonstrated that combination composition can matter beyond the office blood pressure number. The result supported use of complementary first-line classes, including renin-angiotensin blockade plus a long-acting dihydropyridine calcium channel blocker.

127. B. ACCORD BP did not show a significant reduction in its primary composite outcome with the more intensive strategy, although stroke reduction was observed. The trial remains important when weighing intensity, patient selection, and adverse effects in diabetes.

128. A. AASK helped establish the importance of renin-angiotensin system blockade in hypertensive CKD, particularly with proteinuria. Modern care integrates this principle with current CKD and cardiovascular guidelines and individualized blood pressure targets.

129. B. PATHWAY-2 provided strong evidence for spironolactone as an effective fourth-line option in resistant hypertension. The result is consistent with the importance of sodium retention and relative aldosterone excess in many resistant cases.

130. D. HYVET showed that selected very old adults benefit from antihypertensive treatment. Age alone is not a reason to ignore hypertension, although frailty, orthostasis, comorbidity, and treatment tolerance remain important.

131. B. SHEP was a landmark trial showing that isolated systolic hypertension is clinically important and treatable. Its findings helped overturn the older view that elevated systolic pressure in later life should be accepted as normal aging.

132. A. Syst-Eur supported treatment of isolated systolic hypertension and demonstrated effective outcome reduction with a calcium channel blocker-based strategy. Together with other trials, it established systolic hypertension as a major therapeutic target.

133. C. ONTARGET reinforced that more complete blockade of the same hormonal pathway does not necessarily improve outcomes. Routine ACE inhibitor plus ARB therapy is therefore avoided because the adverse-effect burden outweighs incremental benefit.

134. D. DASH established that dietary composition itself can meaningfully lower blood pressure. Subsequent work showed additional benefit when the DASH pattern is paired with sodium reduction.

135. C. Albuminuric CKD is a strong setting for ACE inhibitor or ARB therapy because these drugs lower systemic and intraglomerular pressure and reduce proteinuria. Dual ACE inhibitor-ARB therapy is not routinely recommended.

136. C. In diabetes without albuminuria or another compelling indication, more than one standard first-line class can be reasonable. Comorbidities, kidney function, adverse-effect risk, cost, and the amount of pressure reduction needed guide selection.

137. A. Volume management is central to hypertension treatment in advanced CKD. Loop diuretics often become important when GFR is low or edema is prominent, although selected thiazide-like agents can still have activity in advanced disease.

138. B. Combining complementary first-line mechanisms often produces greater blood pressure reduction than maximizing one drug alone. Renin-angiotensin blockade can also mitigate some precapillary-postcapillary imbalance that contributes to dihydropyridine edema.

139. B. A diuretic and a renin-angiotensin blocker target different contributors to hypertension and often have additive effects. The combination still requires monitoring for volume depletion, kidney-function change, and electrolyte abnormalities.

140. A. ACE inhibitors and ARBs can cause serious fetal toxicity and are not used for chronic hypertension during pregnancy. Preconception planning should transition patients to agents with established pregnancy use.

141. B. Renal filtration can fall when effective circulating volume is reduced, efferent support is blocked, and prostaglandin-mediated afferent dilation is inhibited. This interaction is especially important during dehydration or acute illness.

142. D. Pseudoephedrine and related sympathomimetics can raise heart rate and vascular tone. Nonprescription products are therefore relevant when blood pressure control worsens or cardiovascular risk is high.

143. D. The key objective is sustained blood pressure control with a regimen the patient can take consistently. Dosing can be individualized rather than applying a universal bedtime rule to every medication and patient.

144. B. Medication complexity is a common barrier to blood pressure control. Long-acting agents and single-pill combinations can reduce dosing burden and make adherence more feasible.

145. D. A regimen cannot work reliably if the patient cannot obtain it. Cost, insurance coverage, pill burden, adverse effects, and health literacy are practical treatment variables and should be addressed explicitly.

146. C. True resistant hypertension requires that the prescribed regimen is actually being taken. Nonadherence can mimic resistance and should be approached nonjudgmentally by identifying cost, adverse effects, complexity, beliefs, or other barriers.

147. A. Current U.S. guidance incorporates predicted cardiovascular risk when deciding whether to start medication for stage 1 hypertension. This complements, rather than replaces, consideration of established CVD, diabetes, and CKD.

148. B. The 2025 guideline uses a PREVENT 10-year cardiovascular risk threshold of 7.5% to identify additional stage 1 patients for pharmacologic treatment. Other high-risk conditions can support treatment even without relying on that calculation.

149. C. Hypertension management should reduce total cardiovascular risk, not only the cuff measurement. Smoking cessation is a major preventive intervention, while blood pressure should still be treated to the appropriate goal.

150. B. Population-level prevention complements clinical treatment. Food-environment changes, physical activity, healthy weight, reduced excess alcohol, and reliable screening can shift blood pressure distribution and cardiovascular risk across communities.

151. C. Treatment decisions depend on accurate pressure measurement. Proper technique, repeat measurements, and home or ambulatory data can prevent unnecessary escalation for artifact or white-coat effect.

152. B. Guideline targets are not a substitute for clinical judgment. Frailty, recurrent falls, limited life expectancy, institutional care, and symptomatic orthostasis may justify a less intensive or simplified regimen.

153. B. Modern sham-controlled trials and regulatory approvals support renal denervation as an option for selected patients. Average effects are comparable to adding roughly one effective antihypertensive drug, with substantial individual variability.

154. D. Device-based treatment should not be used to bypass basic diagnostic work. Confirming the phenotype, excluding pseudoresistance, reviewing secondary causes, and using shared decision-making are core parts of patient selection.

155. B. Renal denervation is not uniformly effective and does not abolish all mechanisms of hypertension. Patients should expect continued blood pressure surveillance and possible ongoing pharmacologic treatment.

156. C. Most hypertension is managed with lifestyle measures and well-established pharmacologic classes. Device therapies occupy selected niches and require specialist assessment rather than routine first-line use.

157. B. More complete renin-angiotensin system blockade is not necessarily better. In diabetes, combining aliskiren with an ACE inhibitor or ARB is avoided because of adverse renal and potassium outcomes.

158. D. Hydralazine reduces arteriolar resistance but can evoke baroreflex tachycardia and renin-mediated sodium retention. It is therefore commonly used with other agents rather than as isolated therapy in difficult hypertension.

159. A. Minoxidil is a powerful arteriolar vasodilator reserved for difficult cases. Significant sodium-water retention and reflex tachycardia are expected concerns, so it is generally paired with strong diuretic and sympathetic control.

160. C. Excess sodium can sustain extracellular-volume expansion and reduce the effectiveness of antihypertensive therapy. Sodium reduction remains important even when multidrug pharmacologic treatment is necessary.

161. A. Hypertensive emergency is severe blood pressure elevation accompanied by new or worsening acute target-organ damage. The clinical syndrome, not the pressure number alone, determines the need for emergency intravenous treatment.

162. D. Severe hypertension without acute target-organ damage is not managed like a hypertensive emergency. Current acute-care guidance emphasizes reliable measurement, evaluation for organ injury, correction of contributors, and timely adjustment of oral therapy rather than abrupt pressure reduction.

163. D. Most hypertensive emergencies require prompt but controlled reduction to limit further organ injury while preserving perfusion. Important exceptions, such as acute aortic syndromes, eclampsia, and some neurologic conditions, have more specific targets and timelines.

164. B. Acute aortic dissection is a special hypertensive emergency in which both pressure and the force of ventricular ejection must be reduced quickly. Beta blockade is established before or with vasodilation to avoid reflex tachycardia and increased shear stress.

165. A. Hypertensive acute pulmonary edema is driven by excessive afterload and often volume redistribution or overload. Rapid afterload and preload reduction, respiratory support, and diuresis when volume overloaded address the acute physiology.

166. D. Hypertensive encephalopathy and PRES reflect failure of cerebral autoregulation and vasogenic edema. Controlled, titratable pressure reduction is required, avoiding both ongoing severe hypertension and overly abrupt hypoperfusion.

167. A. In selected patients with mild-to-moderate spontaneous ICH and presenting systolic pressure between 150 and 220 mm Hg, early smooth lowering toward 140 mm Hg is considered safe and reasonable. Excessive lowering below 130 mm Hg can be harmful, and variability should be minimized.

168. D. Blood pressure must be reduced below 185/110 mm Hg before intravenous thrombolysis and then maintained below 180/105 mm Hg after treatment. Controlled titration is used so eligibility can be preserved without causing hypotension.

169. D. In acute ischemic stroke without reperfusion therapy, permissive hypertension is often accepted unless blood pressure is very high, commonly above about 220/120 mm Hg, or another condition requires treatment. Overly rapid lowering can reduce perfusion to ischemic tissue.

170. C. Severe hypertension increases myocardial oxygen demand and afterload in acute coronary syndromes. Treatment should reduce ischemic stress while preserving coronary perfusion and accounting for heart rate, ventricular function, and other contraindications.

171. D. Cocaine toxicity produces a catecholamine-driven syndrome with agitation, vasoconstriction, tachycardia, and ischemia. Benzodiazepines reduce central sympathetic activation, and agents such as nitrates or other titratable vasodilators can be used when pressure or ischemia remains severe.

172. C. After the CHAP trial, ACOG recommended using 140/90 mm Hg as the threshold for initiating or titrating chronic hypertension therapy in pregnancy. The trial established a treatment threshold rather than a single universal achieved blood pressure goal.

173. C. Labetalol and extended-release nifedipine are commonly used first-line agents in pregnancy, and methyldopa has long experience as well. Drug selection considers maternal comorbidities, adverse effects, and contraindications.

174. A. Renin-angiotensin system blockade can cause fetal renal dysfunction, oligohydramnios, skull and lung complications, and fetal death. Patients who may become pregnant should receive anticipatory medication counseling.

175. D. Persistent severe hypertension in pregnancy is an obstetric emergency because of maternal stroke and other complications. Treatment should be prompt and protocolized using agents with established pregnancy safety and experience.

176. B. Magnesium sulfate is used for seizure prophylaxis and treatment in preeclampsia with severe features and eclampsia. It is not itself the primary antihypertensive, so severe blood pressure still requires appropriate antihypertensive therapy.

177. C. Delivery does not immediately eliminate hypertensive risk. Blood pressure can rise postpartum, and patients need timely follow-up, medication adjustment, symptom education, and evaluation for postpartum preeclampsia when warning signs occur.

178. B. Resistant hypertension is defined by uncontrolled pressure despite an appropriate three-drug regimen or by pressure controlled only with four or more drugs. Adherence, measurement quality, and out-of-office pressure must be assessed before confirming true resistance.

179. A. Out-of-office blood pressure is central to distinguishing true resistance from white-coat pseudoresistance. Escalating therapy solely on office readings could produce unnecessary hypotension outside the clinic.

180. A. Nonadherence is a common cause of apparent resistant hypertension. Effective management requires understanding barriers such as adverse effects, cost, complexity, health beliefs, and communication rather than simply increasing the prescription count.

181. B. Volume excess is common in resistant hypertension, and the diuretic component should be adequate for kidney function and sodium intake. Long-acting thiazide-like therapy is often favored before more complex add-on strategies.

182. A. Mineralocorticoid receptor antagonism is highly effective in many patients with resistant hypertension, as reinforced by PATHWAY-2 and other evidence. Hyperkalemia, kidney function, and endocrine adverse effects must be monitored.

183. C. Advanced CKD often produces sodium retention and volume-dependent hypertension. Diuretic selection and dose must match kidney function and volume status, while renin-angiotensin blockade and other agents are used as tolerated and indicated.

184. D. In HFrEF, the antihypertensive plan should prioritize therapies proven to improve heart-failure outcomes. Blood pressure, kidney function, potassium, congestion, and symptoms determine how rapidly and how far the regimen can be titrated.

185. B. Hypertension is a major driver of HFpEF and should be treated effectively. Therapy is integrated with diuresis for congestion and other guideline-directed HFpEF interventions, with attention to symptoms and renal function.

186. D. Hypertension treatment in coronary disease should reduce overall cardiovascular risk while supporting anti-ischemic goals. Excessively low diastolic pressure may be poorly tolerated in some patients with advanced coronary disease, so treatment remains individualized.

187. A. Chronic hypertension increases aortic wall stress and is an important modifiable risk in thoracic aortic disease. The regimen is selected with the specific aortopathy, heart rate, kidney function, and comorbidities in mind.

188. B. After the acute stroke period, blood pressure control is one of the most important components of secondary prevention. For most patients, a target below 130/80 mm Hg is appropriate when tolerated.

189. C. Peripheral artery disease marks high systemic atherosclerotic risk. Effective blood pressure treatment, smoking cessation, lipid management, antiplatelet therapy when indicated, exercise, and diabetes care are integrated rather than treating limb symptoms in isolation.

190. A. Diabetes increases cardiovascular and kidney risk and supports active treatment of hypertension. A target below 130/80 mm Hg is appropriate for most adults, while autonomic neuropathy, orthostasis, CKD, and frailty may require individualization.

191. C. Obesity is mechanistically linked to hypertension through renal sodium retention, sympathetic activation, sleep apnea, and metabolic pathways. Effective obesity treatment can improve pressure, although antihypertensive doses still require individualized monitoring.

192. D. Hypertension frequently clusters with dyslipidemia and other cardiometabolic risks. Comprehensive prevention addresses each major modifiable risk rather than assuming control of one factor makes the others unimportant.

193. D. Orthostatic hypotension is defined by a sustained fall in systolic pressure of at least 20 mm Hg or diastolic pressure of at least 10 mm Hg within 3 minutes of standing. Symptoms are clinically important but are not required for the numerical definition.

194. C. Initial management of orthostatic hypotension focuses on the cause and on removing aggravating factors. Antihypertensive therapy may need adjustment, but simply abandoning blood pressure control can worsen cardiovascular risk and supine hypertension.

195. A. Neurogenic orthostatic hypotension and supine hypertension frequently coexist. Treatment must balance upright symptoms against nighttime pressure, often avoiding late pressor doses and using nonpharmacologic strategies such as head-of-bed elevation.

196. B. Some psychiatric medications, including norepinephrine-active antidepressants and MAO inhibitor interactions, can raise blood pressure. Management should preserve psychiatric stability while addressing the offending exposure or modifying the antihypertensive regimen when needed.

197. C. For adolescents age 13 years and older, the AAP framework uses fixed adult-like categories: stage 1 is 130 to 139 systolic or 80 to 89 diastolic mm Hg. This simplifies transition toward adult hypertension definitions.

198. D. In children younger than 13 years, blood pressure classification depends on age, sex, and height percentiles. Repeated auscultatory values at or above the 95th percentile across appropriate visits support the diagnosis.

199. A. Ambulatory monitoring is recommended in several pediatric scenarios to confirm hypertension and characterize white-coat or masked patterns. It is particularly useful when office elevation persists over repeated visits.

200. D. Persistent pediatric hypertension with target-organ damage is an indication for pharmacologic therapy in addition to lifestyle measures. Drug choice depends on comorbidities and secondary causes, and pressure is titrated to an age-appropriate target.