COMPREHENSIVE MOCK EXAM 4

Supplemental to: Hypertension Board Review 2026-2027

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American Hypertension Specialist Certification Program (AHSCP) — Certification Examination for Hypertension Specialists (CHS); 200 questions, single-best-answer, four options (A–D), domain-weighted: Basic Science 15%; Diagnostic Evaluation 10%; Secondary Hypertension 20%; Treatment 35%; Treatment: Special Conditions 20%

Board-Review Questions

1. A healthy 28-year-old volunteer stands abruptly after lying supine for 20 minutes. His systolic pressure transiently falls, then returns toward baseline within seconds. Which reflex change is primarily responsible for the rapid recovery of arterial pressure?

A. Increased carotid sinus firing with suppression of sympathetic vasoconstrictor activity

B. Increased atrial natriuretic peptide release with renal sodium excretion over seconds

C. Decreased carotid sinus firing with increased sympathetic outflow and reduced vagal tone

D. Reduced renin release with immediate loss of angiotensin II-mediated vasoconstriction

2. A patient with reduced effective arterial blood volume has activation of the renin-angiotensin-aldosterone system. Which signal directly stimulates renin release from juxtaglomerular cells?

A. Alpha-1 adrenergic stimulation of afferent arteriolar smooth muscle

B. Increased sodium chloride delivery to the macula densa

C. Beta-1 adrenergic stimulation of juxtaglomerular cells

D. Increased stretch of the afferent arteriole from higher renal perfusion

3. A patient with renal hypoperfusion has a modest increase in circulating angiotensin II. At physiologic concentrations, which renal vascular effect helps preserve glomerular filtration pressure?

A. Preferential dilation of the efferent arteriole

B. Preferential constriction of the efferent arteriole

C. Selective constriction of the afferent arteriole without efferent change

D. Complete loss of autoregulatory tone in both renal arterioles

4. A patient with primary aldosteronism has hypertension and potassium wasting. In principal cells of the cortical collecting duct, aldosterone most directly increases sodium reabsorption by increasing the activity and expression of which transporter?

A. Epithelial sodium channels in the apical membrane

B. Na-K-2Cl cotransporters in the thick ascending limb

C. Na-Cl cotransporters in the distal convoluted tubule

D. Sodium-glucose cotransporters in the proximal tubule

5. A patient receives a rapid isotonic saline infusion that markedly increases atrial stretch. Which hormonal response favors natriuresis and opposes the renin-angiotensin-aldosterone system?

A. Increased aldosterone secretion from the adrenal zona glomerulosa

B. Increased vasopressin release caused by reduced atrial stretch

C. Increased renin secretion from juxtaglomerular cells

D. Increased atrial natriuretic peptide release

6. A salt-sensitive patient increases dietary sodium for several days. In a normal pressure-natriuresis relationship, what physiologic response ultimately restores sodium balance?

A. A small rise in arterial pressure increases renal sodium excretion

B. A fall in arterial pressure increases proximal tubular sodium reabsorption

C. A rise in renin secretion causes sustained renal sodium retention

D. A fall in renal perfusion pressure increases sodium delivery to the urine

7. An experimental drug enhances endothelial nitric oxide synthase activity. Which immediate vascular effect is most likely?

A. Increased intracellular calcium in vascular smooth muscle with vasoconstriction

B. Increased cyclic GMP in vascular smooth muscle with vasodilation

C. Increased endothelin receptor signaling with sustained arteriolar constriction

D. Reduced cyclic GMP in vascular smooth muscle with increased peripheral resistance

8. A patient with severe endothelial dysfunction has increased production of endothelin-1. Which effect of endothelin-1 most directly raises systemic blood pressure?

A. Potent vascular smooth-muscle constriction through endothelin receptors

B. Direct opening of vascular potassium channels causing arterial relaxation

C. Suppression of smooth-muscle calcium signaling causing venodilation

D. Stimulation of endothelial nitric oxide release as its dominant systemic action

9. A sympathomimetic drug selectively stimulates alpha-1 adrenergic receptors in systemic resistance vessels. Which hemodynamic change is expected?

A. Decreased arteriolar resistance with a fall in mean arterial pressure

B. Increased arteriolar resistance and increased mean arterial pressure

C. Reduced venous tone with a fall in cardiac preload and arterial pressure

D. Primary slowing of sinoatrial-node firing without a vascular effect

10. A beta-1 selective antagonist lowers blood pressure partly by an effect on the kidney. Which renal effect contributes to this response?

A. Increased renin secretion with greater angiotensin II generation

B. Direct blockade of epithelial sodium channels in collecting ducts

C. Direct inhibition of the Na-Cl cotransporter in the distal tubule

D. Reduced renin secretion from juxtaglomerular cells

11. A 76-year-old patient has a blood pressure of 168/66 mm Hg. Which vascular change most strongly contributes to this widened pulse pressure with aging?

A. Reduced compliance of the large elastic arteries

B. Increased compliance of the aorta with greater systolic energy storage

C. Marked reduction in stroke volume from increased ventricular compliance

D. Loss of arteriolar tone causing uniformly lower systolic pressure

12. Two patients have the same mean arterial pressure, but one has substantially greater carotid-femoral pulse wave velocity. What does the higher pulse wave velocity most directly indicate?

A. Greater compliance of the central elastic arteries

B. Lower sympathetic tone in resistance arterioles

C. Greater stiffness of the central elastic arteries

D. Lower left ventricular ejection velocity during systole

13. A patient with salt-sensitive hypertension has low plasma renin activity while consuming a high-sodium diet. Which mechanism best explains this pattern?

A. Renal sodium wasting contracts volume and stimulates persistent renin release

B. Renal sodium retention expands volume and suppresses renin release

C. High sodium directly activates beta-1 receptors on juxtaglomerular cells

D. Volume expansion increases renin through reduced macula densa chloride delivery

14. A patient with obesity develops hypertension despite normal circulating volume measurements. Which obesity-associated pathway can contribute to sustained sympathetic activation and hypertension?

A. Adiponectin-mediated suppression of sympathetic activity as the dominant pressor mechanism

B. Reduced renal tubular sodium reabsorption caused by visceral adiposity

C. Leptin-mediated activation of central sympathetic pathways

D. Chronic suppression of the renin-angiotensin system by adipose tissue

15. In a patient with metabolic syndrome, hyperinsulinemia may contribute to hypertension. Which effect is most consistent with this relationship?

A. Enhanced sympathetic activity and renal sodium retention

B. Persistent suppression of sympathetic outflow and natriuresis

C. Selective blockade of angiotensin II receptors in resistance vessels

D. Reduced proximal tubular sodium reabsorption with chronic volume contraction

16. A patient with unilateral renal artery narrowing has reduced perfusion pressure distal to the stenosis. Which renal autoregulatory response initially helps maintain glomerular filtration?

A. Afferent arteriolar dilation together with angiotensin II-mediated efferent constriction

B. Afferent arteriolar constriction together with efferent arteriolar dilation

C. Simultaneous maximal constriction of both arterioles independent of hormones

D. Loss of all vascular tone with passive transmission of the reduced pressure

17. An increase in sodium chloride delivery to the macula densa activates tubuloglomerular feedback. What is the expected immediate effect on the same nephron?

A. Afferent arteriolar dilation with stimulation of renin release

B. Efferent arteriolar dilation with maximal aldosterone secretion

C. Afferent arteriolar constriction with suppression of renin release

D. Proximal tubular dilation with immediate activation of vasopressin

18. A thiazide-like diuretic lowers blood pressure by inhibiting sodium reabsorption at which nephron transporter?

A. The Na-K-2Cl cotransporter in the thick ascending limb

B. The epithelial sodium channel in the cortical collecting duct

C. The sodium-glucose cotransporter in the proximal convoluted tubule

D. The Na-Cl cotransporter in the distal convoluted tubule

19. A loop diuretic produces brisk natriuresis in a patient with advanced chronic kidney disease. Which transporter is directly inhibited?

A. The Na-Cl cotransporter in the distal convoluted tubule

B. The epithelial sodium channel in the collecting duct

C. The Na-K-2Cl cotransporter in the thick ascending limb

D. The sodium-hydrogen exchanger in the proximal tubule as its primary target

20. A 19-year-old has severe early-onset hypertension, hypokalemia, metabolic alkalosis, suppressed renin, and suppressed aldosterone. Which molecular abnormality best fits this phenotype?

A. Loss-of-function of epithelial sodium channels causing renal salt wasting

B. Loss-of-function of the thiazide-sensitive Na-Cl cotransporter

C. Failure of aldosterone synthesis causing renal sodium loss and hyperkalemia

D. Gain-of-function activity of epithelial sodium channels in the collecting duct

21. A patient eats large amounts of natural black licorice and develops hypertension, hypokalemia, and suppressed renin and aldosterone. Which mechanism is responsible?

A. Activation of aldosterone synthase causes marked aldosterone excess despite low renin

B. Blockade of epithelial sodium channels causes compensatory sodium retention

C. Inhibition of cortisol synthesis produces secondary hyperaldosteronism

D. Inhibition of 11-beta-hydroxysteroid dehydrogenase type 2 permits cortisol to activate mineralocorticoid receptors

22. A patient with a catecholamine-secreting tumor has episodic headache, palpitations, diaphoresis, and severe hypertension. Which receptor effect most directly accounts for the marked increase in systemic vascular resistance during an episode?

A. Alpha-1 adrenergic receptor-mediated arteriolar vasoconstriction

B. Beta-2 adrenergic receptor-mediated vasodilation as the dominant effect

C. Muscarinic receptor-mediated arteriolar relaxation throughout the systemic circulation

D. Dopamine receptor-mediated renal vasodilation as the dominant systemic response

23. In normal renal principal cells, circulating cortisol concentrations exceed aldosterone concentrations, yet aldosterone selectively regulates mineralocorticoid receptors. What provides this tissue specificity?

A. Preferential renal conversion of aldosterone to an inactive metabolite before receptor binding

B. Local conversion of cortisol to cortisone by 11-beta-hydroxysteroid dehydrogenase type 2

C. Selective degradation of mineralocorticoid receptors whenever cortisol is present

D. Complete exclusion of circulating cortisol from the renal interstitium

24. A family has autosomal dominant early-onset hypertension with suppressed renin, elevated aldosterone, and aldosterone production that is regulated by ACTH. Which mechanism is most characteristic?

A. A chimeric gene places aldosterone synthase under ACTH-responsive regulatory control

B. An activating epithelial sodium channel mutation produces aldosterone-independent sodium retention

C. Loss of 11-beta-hydroxysteroid dehydrogenase allows cortisol to activate mineralocorticoid receptors

D. Bilateral renal artery stenosis causes renin-driven secondary hyperaldosteronism

25. A patient with longstanding hypertension develops left ventricular hypertrophy. Which hemodynamic load most directly drives concentric hypertrophy in uncomplicated systemic hypertension?

A. Chronic reduction of afterload with increased ventricular emptying

B. Chronic elevation of left ventricular afterload

C. Isolated reduction of venous return without arterial pressure elevation

D. Persistent fall in systemic vascular resistance with unchanged wall stress

26. A patient with chronic hypertension develops impaired endothelial function. Which change is most likely to promote further vascular remodeling and increased resistance?

A. Increased nitric oxide bioavailability with sustained smooth-muscle relaxation

B. Reduced nitric oxide bioavailability with increased oxidative stress

C. Reduced endothelin signaling with regression of vascular smooth muscle

D. Reduced sympathetic tone with decreased arteriolar wall stress

27. A patient has a normal daytime blood pressure but persistently elevated nighttime pressure on ambulatory monitoring. Why can this pattern carry additional cardiovascular risk?

A. A higher nighttime pressure proves that daytime office readings are falsely elevated

B. A nocturnal rise indicates normal baroreflex resetting and lower target-organ stress

C. Loss of the normal nocturnal pressure fall reflects abnormal autonomic and volume regulation

D. Normal daytime pressure makes isolated nocturnal hypertension relatively reassuring because the 24-hour average remains lower

28. A patient with long-standing hypertension develops arteriolar wall thickening. Which structural adaptation tends to maintain elevated systemic vascular resistance even when acute vasoconstrictor tone is reduced?

A. Decreased media-to-lumen ratio from chronic outward remodeling

B. Increased media-to-lumen ratio from vascular remodeling

C. Loss of smooth muscle with progressive enlargement of resistance-vessel lumens

D. Replacement of arteriolar muscle by highly compliant elastic tissue

29. A patient with hypertension has increased arterial pulse pressure despite an unchanged stroke volume. Which change best explains the higher systolic pressure?

A. Increased arterial compliance causes a larger pressure rise for the same ejected volume

B. Reduced venous tone directly widens pulse pressure without changing arterial properties

C. Reduced arterial compliance causes a larger pressure rise for the same ejected volume

D. Increased capillary permeability directly increases central systolic pressure

30. A patient starts an angiotensin-converting enzyme inhibitor. Which change in bradykinin metabolism contributes to both vasodilation and a characteristic adverse effect?

A. Accelerated bradykinin degradation lowers bradykinin concentrations

B. Reduced bradykinin degradation increases bradykinin concentrations

C. Complete blockade of bradykinin receptors prevents airway sensory activation

D. Increased aldosterone secretion secondarily raises bradykinin concentrations

31. A 58-year-old man with a large upper-arm circumference repeatedly has office blood pressures near 170/96 mm Hg when measured with a standard adult cuff. His home readings with a properly sized device average 138/82 mm Hg. What is the most important measurement error to exclude first?

A. Use of a cuff bladder that is too large for the patient's arm

B. Measuring after five minutes of quiet seated rest with back supported

C. Use of a cuff bladder that is too small for the patient's arm

D. Positioning the supported arm at approximately heart level

32. A 46-year-old woman has an office blood pressure of 152/92 mm Hg immediately after walking rapidly from the parking garage while talking on her phone. Which step best improves the validity of the repeat measurement?

A. Have her sit quietly for at least several minutes with back and feet supported before repeating the reading

B. Repeat the measurement immediately while she remains standing to avoid delay

C. Ask her to cross her legs and hold the arm unsupported at shoulder height

D. Use the smallest cuff that can encircle the arm to improve signal detection

33. During manual blood pressure measurement, Korotkoff sounds appear at 168 mm Hg, disappear between 150 and 132 mm Hg, and then reappear until 88 mm Hg. If the examiner fails to palpate systolic pressure first, what error can result?

A. The auscultatory gap can cause underestimation of systolic pressure

B. The silent interval can make the first Korotkoff sound appear earlier and therefore overestimate systolic pressure

C. The auscultatory gap proves the patient has severe aortic regurgitation

D. Repeat the measurement with an oscillometric device because an auscultatory gap prevents dependable manual systolic identification once it occurs

34. A 73-year-old man reports lightheadedness when he stands. His blood pressure falls from 148/78 mm Hg supine to 124/66 mm Hg three minutes after standing. How should this finding be classified?

A. Normal orthostatic adaptation because the diastolic pressure remained above 60 mm Hg

B. Orthostatic hypertension because standing increased sympathetic vascular tone

C. White-coat hypertension because the seated office value was above goal

D. Orthostatic hypotension because systolic pressure fell by at least 20 mm Hg

35. A patient is asked to obtain home blood pressure readings to guide treatment. Which technique is most appropriate?

A. Take repeated seated readings with a validated upper-arm cuff after quiet rest and record values over multiple days

B. Use a validated wrist monitor held at heart level and record one reading each morning

C. Take readings mainly during headache or dizziness to capture periods most likely to represent clinically important hypertension

D. Take one seated reading immediately before the morning medication each day without repeating it

36. A 50-year-old untreated man has office blood pressures averaging 146/88 mm Hg, but 24-hour ambulatory monitoring shows an awake average of 124/76 mm Hg and a 24-hour average of 119/72 mm Hg. Which diagnosis best describes this pattern?

A. Masked hypertension

B. Sustained hypertension

C. White-coat hypertension

D. Masked uncontrolled hypertension

37. A 44-year-old untreated woman has repeated office blood pressures of 124/76 mm Hg. Because of left ventricular hypertrophy on ECG, ambulatory monitoring is obtained and shows an awake average of 136/84 mm Hg. Which phenotype is present?

A. Masked hypertension

B. White-coat hypertension

C. Sustained normotension

D. White-coat effect during antihypertensive therapy

38. A 62-year-old woman taking three antihypertensive medications has an office blood pressure of 152/88 mm Hg. Home readings average 124/74 mm Hg. Which interpretation is most appropriate before intensifying therapy?

A. Masked uncontrolled hypertension is present because treated patients are classified primarily by the office pressure

B. Sustained uncontrolled hypertension is likely enough that a fourth medication should be added before out-of-office confirmation

C. A white-coat effect should be confirmed with reliable out-of-office measurements

D. Possible home-device underestimation makes repeat standardized office readings sufficient without further out-of-office confirmation

39. Ambulatory blood pressure monitoring in a patient with chronic kidney disease shows an awake average of 132/78 mm Hg and a sleep average of 130/76 mm Hg. What abnormality is most evident?

A. Exaggerated physiologic nocturnal dipping of blood pressure

B. Markedly blunted nocturnal dipping of blood pressure

C. A normal circadian pattern because systolic pressure fell by at least 20%

D. White-coat hypertension because nighttime pressure remained below daytime pressure

40. An untreated 57-year-old has an average standardized office blood pressure of 134/84 mm Hg. Under the contemporary ACC/AHA classification, how is this blood pressure categorized?

A. Stage 1 hypertension

B. Normal blood pressure

C. Elevated blood pressure without hypertension

D. Stage 2 hypertension

41. A 35-year-old untreated patient has standardized office readings averaging 126/76 mm Hg. Which classification is most accurate?

A. Normal blood pressure

B. Elevated blood pressure

C. Stage 1 hypertension

D. Stage 2 hypertension

42. At three separate follow-up visits, an untreated 41-year-old has standardized clinic blood pressures averaging 142/86 mm Hg. How should this blood pressure be classified?

A. Normal blood pressure

B. Stage 2 hypertension

C. Elevated blood pressure

D. Stage 1 hypertension

43. A newly diagnosed hypertensive patient has no symptoms or known comorbid disease. Which initial laboratory assessment is most useful for identifying kidney disease and treatment-relevant metabolic abnormalities?

A. Plasma free metanephrines to screen broadly for occult catecholamine excess

B. An aldosterone-to-renin ratio as the sole initial laboratory evaluation before kidney or metabolic testing

C. Serum electrolytes and creatinine together with urinalysis and urine albumin assessment

D. Renal artery duplex ultrasonography to screen routinely for renovascular hypertension

44. A 56-year-old patient with hypertension has an ECG showing increased QRS voltage with a lateral strain pattern. Which target-organ effect is most likely?

A. Isolated right ventricular hypertrophy from pulmonary hypertension

B. Acute pericarditis without chronic pressure loading

C. A normal physiologic finding that excludes cardiac target-organ damage

D. Hypertensive left ventricular hypertrophy

45. A patient with longstanding poorly controlled hypertension has flame-shaped retinal hemorrhages, cotton-wool spots, and optic disc swelling. What does this examination most strongly indicate?

A. Uncomplicated mild hypertension without evidence of target-organ injury

B. Isolated diabetic retinopathy that cannot be related to blood pressure

C. Severe hypertensive retinopathy with acute target-organ injury

D. Normal age-related retinal vascular changes with no urgency for evaluation

46. A patient with hypertension and diabetes has an estimated GFR of 78 mL/min/1.73 m2 but a urine albumin-to-creatinine ratio of 220 mg/g. What is the most important interpretation?

A. Albuminuria is evidence of kidney target-organ damage despite preserved estimated GFR

B. Kidney disease is excluded because the estimated GFR remains above 60 mL/min/1.73 m2

C. Albuminuria at this level is physiologic and has no cardiovascular significance

D. The result primarily indicates acute liver injury rather than renal vascular damage

47. A 64-year-old smoker with hypertension has exertional calf discomfort and an ankle-brachial index of 0.62. What does this finding indicate?

A. Peripheral artery disease as hypertension-mediated vascular comorbidity

B. Normal lower-extremity arterial perfusion for an older adult

C. Venous insufficiency without evidence of arterial disease

D. Isolated autonomic dysfunction causing a falsely high ankle pressure

48. A 29-year-old woman develops abrupt severe hypertension that was absent one year earlier. She has an abdominal bruit and no family history of hypertension. Which feature most strongly justifies evaluation for a secondary cause?

A. A stable mildly elevated pressure that developed gradually after age 60

B. Abrupt onset of severe hypertension at a young age

C. A normal physical examination with excellent control on one low-dose drug

D. A family history of typical essential hypertension in several older relatives

49. A patient with atrial fibrillation has highly variable beat-to-beat stroke volume. How should office blood pressure be assessed most reliably?

A. Use a single automated reading after quiet rest because the device compensates for beat-to-beat rhythm variation

B. Obtain multiple carefully performed readings and use an average rather than relying on a single measurement

C. Use the highest of three systolic readings to avoid underestimating pressure during low-stroke-volume beats

D. Defer formal blood pressure assessment until ventricular rate control reduces beat-to-beat variability

50. A patient taking four antihypertensive medications has office readings of 158/92 mm Hg. Before labeling the condition true resistant hypertension, which diagnostic step is essential?

A. Order renal artery imaging before confirming adherence or the out-of-office blood pressure phenotype

B. Escalate therapy when refill history suggests adherence without directly assessing how medications are actually taken

C. Confirm out-of-office hypertension and assess adherence and measurement technique

D. Classify the patient as resistant based on repeated office readings while assuming cuff technique and white-coat effect are not important

51. A 52-year-old man has newly confirmed hypertension without hypokalemia or resistant disease. Under the 2025 Endocrine Society primary aldosteronism guideline, which screening strategy is suggested?

A. Limit screening to hypertension accompanied by spontaneous or diuretic-induced hypokalemia

B. Measure aldosterone and renin and calculate an aldosterone-to-renin ratio in adults with hypertension

C. Limit screening to apparent treatment-resistant hypertension despite at least three medications

D. Screen primarily after an adrenal incidentaloma has already been identified on imaging

52. A hypertensive patient has potassium 3.1 mmol/L, plasma renin activity that is suppressed, and an inappropriately high plasma aldosterone concentration. Which diagnosis is most likely?

A. Renin-secreting renal tumor

B. Bilateral renal artery stenosis

C. Diuretic-induced secondary hyperaldosteronism with high renin

D. Primary aldosteronism with renin suppression

53. A patient with resistant hypertension is being evaluated for primary aldosteronism. Potassium is 2.9 mmol/L. What should be done before interpreting aldosterone-renin screening results?

A. Intentionally worsen hypokalemia to amplify the aldosterone response

B. Correct the hypokalemia because potassium depletion can suppress aldosterone secretion

C. Give a loop diuretic immediately before blood sampling to standardize renin

D. Restrict sodium severely for several days to eliminate aldosterone variability

54. A patient taking spironolactone is scheduled for aldosterone-renin screening because primary aldosteronism is strongly suspected. Why can the drug complicate interpretation?

A. Spironolactone lowers aldosterone while leaving renin unchanged, producing a predictably low ratio

B. Mineralocorticoid receptor blockade can raise renin and alter the aldosterone-renin relationship

C. Spironolactone suppresses adrenal aldosterone secretion so completely that renin becomes uninterpretable

D. Spironolactone mainly alters serum potassium, so the aldosterone-renin ratio remains reliable during treatment

55. A patient has a positive aldosterone-renin screen and an intermediate probability of lateralizing primary aldosteronism. He wants surgery if unilateral disease is present. Which next step is most appropriate?

A. Start lifelong beta-blocker therapy without confirming aldosterone autonomy

B. Proceed directly to bilateral adrenalectomy without additional testing

C. Exclude primary aldosteronism because potassium is currently normal

D. Perform an aldosterone suppression test before proceeding to subtype evaluation

56. A 46-year-old woman has confirmed primary aldosteronism and a 1.6-cm left adrenal nodule on CT. She is considering curative surgery. Which test best determines whether aldosterone secretion is truly unilateral?

A. Adrenal venous sampling to determine aldosterone lateralization

B. Repeat office blood pressure measurement alone

C. Renal duplex ultrasonography

D. Twenty-four-hour ambulatory blood pressure monitoring

57. A 43-year-old patient has confirmed unilateral aldosterone production on adrenal venous sampling and is an acceptable operative candidate. Which treatment offers the best chance of disease-specific cure?

A. Laparoscopic unilateral adrenalectomy

B. Bilateral adrenalectomy with lifelong glucocorticoid replacement

C. Long-term loop diuretic monotherapy without mineralocorticoid blockade

D. Renal artery stenting to reduce renin secretion

58. A 59-year-old man has confirmed bilateral primary aldosteronism and does not want surgery. Which disease-specific medical therapy is preferred?

A. A direct arterial vasodilator alone without blocking aldosterone action

B. A loop diuretic alone because aldosterone receptors are not involved

C. A beta blocker alone as definitive treatment of autonomous aldosterone secretion

D. A mineralocorticoid receptor antagonist such as spironolactone

59. A man with bilateral primary aldosteronism develops painful gynecomastia on spironolactone despite good blood pressure control. Which alternative most directly preserves disease-specific therapy with fewer antiandrogen effects?

A. Hydralazine

B. Clonidine

C. Doxazosin

D. Eplerenone

60. A young adult with glucocorticoid-remediable aldosteronism has hypertension, suppressed renin, and elevated aldosterone. Which treatment mechanism is unique to this disorder?

A. Low-dose glucocorticoid suppression of ACTH-driven aldosterone production

B. High-dose mineralocorticoid administration to suppress renal renin

C. Renal artery angioplasty to eliminate renin-driven aldosterone release

D. Beta-2 agonist therapy to suppress adrenal catecholamine secretion

61. A 38-year-old woman has episodic pounding headache, palpitations, diaphoresis, and paroxysmal hypertension. Which initial biochemical test is most appropriate for suspected pheochromocytoma or paraganglioma?

A. Random serum aldosterone without measuring renin

B. Urine albumin-to-creatinine ratio as the definitive endocrine test

C. Plasma free metanephrines or fractionated urinary metanephrines

D. Obtain a 24-hour urinary free cortisol first because episodic hypertension with diaphoresis is more specific for hypercortisolism

62. A patient has borderline-elevated plasma metanephrines after blood was drawn while seated immediately following a stressful commute. Which step is most appropriate before ordering invasive testing?

A. Diagnose pheochromocytoma immediately because any elevation is diagnostic

B. Begin beta blockade alone before confirming the biochemical abnormality

C. Repeat plasma metanephrines under controlled resting conditions, ideally after supine rest

D. Perform adrenal venous sampling because it is the confirmatory test for catecholamine excess

63. A patient with a biochemically confirmed pheochromocytoma is preparing for adrenalectomy. Which medication sequence is safest?

A. Start beta blockade first to control tachycardia, then add alpha blockade after the heart rate is controlled

B. Use combined alpha/beta blockade from the outset without first establishing adequate alpha-adrenergic blockade

C. Control pressure with calcium-channel blockade and reserve alpha blockade for the intraoperative period

D. Establish alpha-adrenergic blockade before adding beta blockade for persistent tachycardia

64. A patient with pheochromocytoma develops severe hypertension after receiving propranolol before alpha blockade. What mechanism best explains the deterioration?

A. Abrupt loss of alpha-adrenergic tone causes profound systemic vasodilation

B. Propranolol directly stimulates adrenal catecholamine synthesis through ACTH

C. Unopposed alpha-adrenergic vasoconstriction after beta receptor blockade

D. Beta blockade activates mineralocorticoid receptors in renal collecting ducts

65. A 31-year-old patient is diagnosed with a paraganglioma. Which additional consideration is especially important because these tumors often have heritable associations?

A. Defer germline testing when the family history is negative because most apparently sporadic paragangliomas lack inherited risk

B. Restrict genetic testing to bilateral adrenal pheochromocytomas because extra-adrenal paragangliomas are usually sporadic

C. Offer genetic evaluation based on the tumor phenotype and clinical context

D. Use the biochemical catecholamine phenotype alone and reserve germline testing for recurrent disease

66. A patient has resistant hypertension, central obesity, proximal muscle weakness, easy bruising, and wide violaceous striae. Which screening test is appropriate for hypercortisolism?

A. Obtain a morning serum cortisol and ACTH as the preferred screening pair for endogenous hypercortisolism

B. Obtain adrenal CT imaging first and perform biochemical testing after a discrete adrenal lesion is identified

C. Obtain pituitary MRI before documenting endogenous cortisol excess because proximal weakness suggests a pituitary source

D. A validated test such as a 1-mg overnight dexamethasone suppression test

67. A patient has weight loss, heat intolerance, tremor, tachycardia, and predominantly systolic hypertension with widened pulse pressure. Which secondary cause is most likely?

A. Hypothyroidism

B. Primary adrenal insufficiency

C. Hyperthyroidism

D. Advanced autonomic failure

68. A patient has fatigue, cold intolerance, constipation, weight gain, bradycardia, and predominantly diastolic hypertension. Which secondary cause is most likely?

A. Hypothyroidism

B. Hyperthyroidism

C. Pheochromocytoma

D. Primary adrenal insufficiency

69. A hypertensive patient has recurrent nephrolithiasis, constipation, and hypercalcemia. Which endocrine disorder should be considered as a contributor to hypertension?

A. Central diabetes insipidus

B. Primary hyperparathyroidism

C. Primary adrenal insufficiency

D. Hypoparathyroidism

70. A patient with coarse facial features, enlarged hands, sleep apnea, insulin resistance, and hypertension is suspected of having acromegaly. Which initial biochemical test is most appropriate?

A. Random growth hormone concentration as a single definitive test

B. Plasma aldosterone-to-renin ratio as the primary acromegaly test

C. Urinary metanephrines as the most specific test for growth hormone excess

D. Serum insulin-like growth factor 1 concentration

71. A 55-year-old patient has hypertension, edema, rising creatinine, hematuria, and proteinuria. Which secondary hypertension category should be evaluated first?

A. Renal parenchymal disease causing hypertension and urinary abnormalities

B. Renovascular hypertension from renal artery stenosis

C. Primary aldosteronism with secondary glomerular hyperfiltration

D. Medication-associated kidney injury with a secondary rise in blood pressure

72. A 72-year-old man with diffuse atherosclerosis develops recurrent episodes of sudden pulmonary edema and increasingly difficult-to-control hypertension. Which secondary cause is most likely?

A. Uncomplicated primary hypertension with no reason to evaluate renal arteries

B. Primary hypothyroidism as the classic cause of flash pulmonary edema

C. Atherosclerotic renovascular hypertension

D. White-coat hypertension caused by office anxiety alone

73. A 32-year-old woman with no atherosclerotic risk factors has abrupt hypertension and a systolic-diastolic abdominal bruit. Renal imaging shows a beaded appearance of the mid-distal renal artery. What is the most likely diagnosis?

A. Atherosclerotic ostial renal artery stenosis

B. Renal vein thrombosis

C. Polycystic kidney disease

D. Fibromuscular dysplasia

74. A 74-year-old smoker with peripheral artery disease has resistant hypertension. CT angiography shows severe ostial stenosis of the right renal artery. Which etiology is most likely?

A. Fibromuscular dysplasia of the mid-distal renal artery

B. Congenital coarctation of the aorta

C. Atherosclerotic renal artery disease

D. Primary aldosteronism causing a false angiographic lesion

75. A patient with bilateral severe renal artery stenosis starts an ACE inhibitor. Serum creatinine rises from 1.2 to 2.0 mg/dL within one week. What mechanism best explains this change?

A. ACE inhibition causes intense efferent arteriolar constriction and raises glomerular pressure

B. ACE inhibition directly obstructs the renal arteries through smooth-muscle spasm

C. Loss of angiotensin II-mediated efferent arteriolar constriction lowers intraglomerular pressure

D. The drug increases aldosterone enough to cause acute renal cortical ischemia

76. A patient with suspected renovascular hypertension has preserved kidney function and an experienced vascular laboratory is available. Which noninvasive test is a reasonable initial study?

A. Renal duplex ultrasonography

B. Adrenal venous sampling

C. Coronary angiography

D. Pituitary magnetic resonance imaging

77. A patient with atherosclerotic renal artery stenosis has stable kidney function and hypertension controlled with medications. Which management approach is generally most appropriate?

A. Perform renal artery stenting in every anatomically significant stenosis

B. Stop all antihypertensive therapy to preserve renal perfusion

C. Optimize medical therapy and cardiovascular risk reduction rather than routine renal artery stenting

D. Use bilateral nephrectomy as first-line treatment of renovascular disease

78. A patient with severe atherosclerotic renal artery stenosis has repeated hospitalizations for flash pulmonary edema despite optimized therapy. Which feature most strongly supports considering renal revascularization?

A. A stable incidental stenosis with normal kidney function and controlled blood pressure

B. A single mildly elevated office pressure with normal home readings

C. Isolated hyperlipidemia without clinical evidence of renal ischemia

D. Recurrent flash pulmonary edema suggesting hemodynamically important renovascular disease

79. A 17-year-old boy has upper-extremity hypertension, weak delayed femoral pulses, and lower leg pressures well below arm pressures. Which cause of secondary hypertension is most likely?

A. Primary aldosteronism

B. Pheochromocytoma

C. Renal parenchymal disease without vascular obstruction

D. Coarctation of the aorta

80. A 50-year-old man with obesity, loud snoring, witnessed apneas, daytime sleepiness, and resistant hypertension is being evaluated for a secondary contributor. Which test best establishes the suspected diagnosis?

A. Formal sleep testing for obstructive sleep apnea

B. Adrenal venous sampling

C. Renal intra-arterial angiography

D. Overnight dexamethasone suppression testing as the first test

81. A patient with hypertension and severe obstructive sleep apnea asks what effect continuous positive airway pressure is likely to have on blood pressure. Which statement is most accurate?

A. Use CPAP as the principal antihypertensive intervention because severe obstructive sleep apnea commonly produces a large enough pressure fall to replace drug therapy

B. CPAP may produce a modest blood pressure reduction and complements weight loss and antihypertensive therapy

C. Avoid CPAP when hypertension is present because positive intrathoracic pressure can raise sympathetic tone and worsen resting pressure

D. Use CPAP for sleepiness only, because treating obstructive events does not meaningfully complement cardiovascular risk reduction

82. A patient with resistant hypertension has severe obstructive sleep apnea and obesity. Which mechanism most directly links OSA to elevated blood pressure?

A. Persistent suppression of sympathetic activity during recurrent apneic episodes

B. Reduced oxidative stress causes chronic systemic vasodilation

C. Intermittent hypoxemia and arousals increase sympathetic nervous system activity

D. Loss of renin-angiotensin signaling produces renal sodium wasting

83. A patient with difficult-to-control hypertension takes ibuprofen 800 mg three times daily for chronic arthritis. Which effect can explain worsening blood pressure?

A. Renal prostaglandin inhibition produces natriuresis that augments the effect of diuretics

B. Renal sodium retention and attenuation of antihypertensive drug effects

C. NSAIDs block mineralocorticoid receptors and therefore lower pressure through renal sodium loss

D. NSAIDs directly stimulate beta-1 receptors, raising renin as their dominant pressor mechanism

84. A patient with controlled hypertension develops higher readings after starting pseudoephedrine for nasal congestion. Which mechanism is most likely?

A. Direct mineralocorticoid receptor blockade causes compensatory hypertension

B. Prostaglandin stimulation produces renal vasodilation and sodium loss

C. Sympathomimetic vasoconstriction increases peripheral vascular resistance

D. Selective vagal activation increases cardiac output and arterial pressure

85. A woman with hypertension has a significant rise in blood pressure after starting an estrogen-containing oral contraceptive. Which approach is most appropriate if the contraceptive is contributing?

A. Continue the combined contraceptive and intensify antihypertensive therapy without reassessing the hormonal exposure

B. Consider a lower-estrogen, progestin-only, or nonhormonal contraceptive strategy as clinically appropriate

C. Attribute the rise to progression of essential hypertension because contemporary combined contraceptives have negligible blood pressure effects

D. Switch to a higher-estrogen formulation to improve cycle control while monitoring the blood pressure more closely

86. A kidney transplant recipient develops new hypertension after starting cyclosporine. Which mechanism is most consistent with calcineurin inhibitor-associated hypertension?

A. Corticosteroid-related sodium retention is the defining direct vascular mechanism of cyclosporine

B. Transplant renal artery stenosis is the specific pharmacologic effect produced by calcineurin inhibition

C. Chronic allograft dysfunction from reduced nephron mass is the immediate mechanism by which cyclosporine raises pressure

D. Renal vasoconstriction and sodium retention with increased vascular tone

87. A patient receiving an erythropoiesis-stimulating agent for chronic kidney disease develops a progressive increase in blood pressure. Which interpretation is most appropriate?

A. The drug reliably lowers blood pressure through vasodilation

B. The blood pressure change proves primary aldosteronism

C. The association excludes a role for chronic kidney disease

D. The erythropoiesis-stimulating agent can itself contribute to hypertension

88. A patient with hypertension reports consuming four to five alcoholic drinks nightly. Which intervention addresses a plausible secondary contributor to poor control?

A. Switch from spirits to wine while maintaining the same ethanol intake because beverage type is the main determinant of blood pressure effect

B. Substantially reduce alcohol intake, with abstinence or guideline-limited consumption

C. Concentrate the same weekly alcohol intake on weekends to reduce its effect on weekday blood pressure

D. Prioritize sodium reduction and medication escalation because this level of alcohol intake is unlikely to affect treatment response

89. A patient with hypertension, hypokalemia, metabolic alkalosis, low renin, and low aldosterone reports eating imported licorice candy daily. Which diagnosis best describes the resulting syndrome?

A. Primary aldosteronism with autonomous aldosterone overproduction

B. Secondary hyperaldosteronism from renal artery stenosis

C. Apparent mineralocorticoid excess caused by licorice-induced 11-beta-HSD2 inhibition

D. Addison disease with mineralocorticoid deficiency

90. A 28-year-old man presents with chest pain, agitation, tachycardia, diaphoresis, and severe hypertension shortly after recreational drug use. Which drug mechanism most strongly fits this presentation?

A. Mu-opioid receptor agonism producing sedation, miosis, and respiratory depression

B. Cocaine-mediated blockade of catecholamine reuptake with intense sympathetic activation

C. GABA-A receptor potentiation producing anxiolysis, sedation, and reduced sympathetic outflow

D. Cannabinoid CB1 receptor agonism producing altered perception with less intense adrenergic activation

91. A 49-year-old patient with stage 1 hypertension wants to lower blood pressure without medication if possible. Which dietary pattern has the strongest evidence as a structured antihypertensive diet?

A. A diet dominated by processed meats and refined carbohydrates

B. A very-low-fiber diet with unrestricted sodium intake

C. A DASH-style diet rich in fruits, vegetables, whole grains, and low-fat dairy

D. A high-sodium ketogenic diet prescribed solely to increase plasma volume

92. A patient with hypertension consumes approximately 4,000 mg of sodium daily. Which dietary change is most likely to lower blood pressure?

A. Reduce sodium intake substantially, ideally toward a low-sodium dietary pattern

B. Increase sodium intake to improve baroreceptor sensitivity

C. Replace dietary potassium with sodium-containing salt substitutes

D. Maintain the current sodium intake because sodium does not influence blood pressure

93. A 58-year-old patient with hypertension has normal kidney function, potassium 4.1 mmol/L, and no medications that impair potassium excretion. Which dietary strategy can help lower blood pressure?

A. Avoid all dietary potassium because it routinely raises blood pressure

B. Use potassium supplements despite hyperkalemia and advanced CKD

C. Replace fruits and vegetables with sodium-rich processed foods

D. Increase potassium-rich foods as part of a healthy dietary pattern

94. A patient with obesity and hypertension loses 10 kg through a structured program. Which effect on blood pressure is most expected?

A. A clinically meaningful reduction in blood pressure that generally tracks with the amount of weight lost

B. Little blood pressure change is expected unless weight loss exceeds about 20% of baseline body weight

C. Attribute any pressure reduction to concurrent sodium restriction rather than to weight loss itself

D. Antihypertensive doses should be assumed unchanged until the final goal weight is reached, even if pressure falls during treatment

95. A sedentary 61-year-old with hypertension asks about exercise. Which recommendation is most appropriate?

A. Restrict activity to stretching because transient systolic increases during aerobic exercise are unsafe in treated hypertension

B. Emphasize high-intensity isometric exercise as the sole training modality because it provides the most reliable chronic pressure reduction

C. Delay structured exercise until medication has normalized resting blood pressure, then begin activity gradually

D. Regular aerobic activity, supplemented by resistance exercise as tolerated and increased progressively over time

96. A man with hypertension drinks four alcoholic beverages nightly. Which recommendation is most appropriate for blood pressure management?

A. Use alcohol in place of dietary carbohydrate because its acute vasodilator effect provides sustained blood pressure benefit

B. Switch to lower-carbohydrate spirits while keeping four drinks nightly because beverage composition drives the pressor effect

C. Reduce alcohol intake substantially and avoid exceeding recommended daily limits

D. Escalate antihypertensive medication before changing alcohol intake because the current exposure is unlikely to influence treatment response

97. A patient with hypertension asks whether stopping cigarettes is worthwhile even though nicotine cessation may not be the largest direct BP-lowering intervention. What is the best response?

A. Continue smoking because treatment of blood pressure eliminates tobacco-related risk

B. Switch to more frequent nicotine exposure to reduce sympathetic surges

C. Smoking cessation is essential because it markedly reduces overall cardiovascular risk

D. Smoking cessation is unnecessary unless systolic pressure exceeds 180 mm Hg

98. A patient with hypertension reports chronic sleep deprivation, high occupational stress, and no regular relaxation or activity routine. Which intervention is reasonable as part of a comprehensive plan?

A. Improve sleep and use evidence-based stress-reduction strategies alongside diet and exercise

B. Use behavioral stress reduction as the primary intervention and defer reassessment of persistent hypertension until stress symptoms resolve

C. Use over-the-counter calming supplements as the main blood pressure therapy because stress is the dominant presumed mechanism

D. Treat insomnia symptoms without assessing sleep-disordered breathing because apnea contributes mainly to daytime sleepiness rather than hypertension

99. A 45-year-old patient has persistent stage 1 hypertension averaging 134/84 mm Hg, no CVD, diabetes, or CKD, and a PREVENT 10-year CVD risk below 7.5%. What is the preferred initial strategy?

A. Begin antihypertensive medication immediately because any persistent stage 1 hypertension warrants drug therapy independent of predicted risk

B. Lifestyle therapy with planned blood pressure reassessment after about three to six months of intervention

C. Continue lifestyle measures without a defined reassessment interval as long as the patient remains asymptomatic

D. Begin medication immediately because age over 40 substitutes for formal cardiovascular risk assessment in stage 1 hypertension

100. A 58-year-old patient with diabetes has an average blood pressure of 134/82 mm Hg despite lifestyle changes. Which approach is most appropriate?

A. Start antihypertensive medication because diabetes confers higher cardiovascular risk

B. Continue lifestyle therapy alone because pharmacologic treatment is generally deferred until blood pressure reaches 140/90 mm Hg even in diabetes

C. Wait for albuminuria to develop before starting medication because kidney protection is the principal reason to treat hypertension in diabetes

D. Base the medication decision on PREVENT risk alone and do not treat at this pressure until the calculated risk reaches 7.5%

101. A 52-year-old patient without CVD, diabetes, or CKD has an average blood pressure of 136/86 mm Hg and a PREVENT 10-year CVD risk of 11%. What is the best treatment approach?

A. Continue lifestyle therapy alone because systolic pressure below 140 mm Hg outweighs the elevated PREVENT risk

B. Begin antihypertensive medication together with continued lifestyle therapy because cardiovascular risk is elevated

C. Delay medication until stage 2 hypertension develops even though predicted cardiovascular risk is elevated

D. Use aspirin for the elevated cardiovascular risk and defer antihypertensive medication while pressure remains in stage 1

102. A 63-year-old patient has confirmed stage 2 hypertension averaging 154/94 mm Hg and no contraindications. Which initial pharmacologic strategy is preferred?

A. Use lifestyle therapy alone for three to six months before medication despite the confirmed stage 2 pressure

B. Start one first-line agent and titrate it to a maximal dose before considering a second class

C. Start two first-line antihypertensive agents from different classes, preferably as a single-pill combination

D. Start an ACE inhibitor together with an ARB to obtain greater initial renin-angiotensin blockade

103. A 67-year-old patient with treated hypertension asks about the usual blood pressure goal under the 2025 ACC/AHA guideline. Which target is generally appropriate when tolerated?

A. A blood pressure target below 140/90 mm Hg as the default goal for all treated adults

B. A blood pressure target below 130/80 mm Hg when treatment is tolerated

C. A blood pressure target below 120/70 mm Hg as the default goal for every adult with hypertension

D. Use symptom-limited titration without a prespecified numeric goal once drug therapy has begun

104. A patient without a compelling indication needs a first-line antihypertensive drug. Which class is considered appropriate first-line therapy?

A. An alpha-1 blocker as the preferred universal first-line agent

B. A thiazide-type diuretic used as standard first-line therapy

C. A central alpha-2 agonist as routine first-line monotherapy

D. A direct arterial vasodilator as routine first-line monotherapy

105. A patient requires a thiazide-type diuretic and has no contraindications. Which agent has a long duration of action and extensive cardiovascular outcome evidence?

A. Mannitol

B. Acetazolamide

C. Triamterene as a thiazide-type drug

D. Chlorthalidone

106. A patient starts amlodipine and develops bilateral ankle edema without heart failure or venous thrombosis. What is the most likely mechanism?

A. Severe renal sodium retention from mineralocorticoid receptor activation

B. Preferential arteriolar dilation increases capillary hydrostatic pressure

C. Direct myocardial depression causing acute biventricular failure

D. Loss of plasma albumin from calcium-channel blockade of glomerular filtration

107. A patient develops a persistent dry cough several weeks after starting lisinopril. Which mechanism best explains the adverse effect?

A. Direct stimulation of beta-2 receptors in bronchial smooth muscle

B. Excess aldosterone production causing pulmonary sodium retention

C. Accumulation of bradykinin because angiotensin-converting enzyme also degrades kinins

D. Selective blockade of histamine H1 receptors in the respiratory tract

108. A patient taking an ACE inhibitor develops tongue swelling and stridor without urticaria. Which immediate action is most important for long-term medication management?

A. Restart the same ACE inhibitor at a lower dose once swelling resolves

B. Permanently discontinue the ACE inhibitor because this is bradykinin-mediated angioedema

C. Add an NSAID and continue the ACE inhibitor unchanged

D. Switch to combined ACE inhibitor and ARB therapy to prevent recurrence

109. A patient with CKD starts an ARB. Which laboratory abnormality should be monitored because it can worsen after renin-angiotensin system blockade?

A. Hyperkalemia due to reduced aldosterone-mediated potassium excretion

B. Hypophosphatemia from urinary phosphate wasting

C. Severe hypocalcemia from parathyroid suppression

D. Iron deficiency from inhibition of intestinal iron absorption

110. A patient with hypertension is prescribed lisinopril and losartan together to obtain more complete renin-angiotensin system blockade. Why is this combination generally inappropriate?

A. Dual blockade chiefly increases bradykinin accumulation, making cough and angioedema the principal limitation

B. It increases acute kidney injury and hyperkalemia without sufficient added clinical benefit

C. Dual blockade tends to lower potassium through additive aldosterone release, making hypokalemia the major concern

D. The combination is avoided mainly because it adds cost without changing kidney or potassium-related adverse effects

111. A 30-year-old woman with chronic hypertension is planning pregnancy. Which antihypertensive class should be stopped before conception because of fetal toxicity?

A. Angiotensin receptor blocker

B. Long-acting nifedipine therapy

C. Labetalol therapy

D. Methyldopa therapy

112. A patient taking chlorthalidone develops muscle weakness and ventricular ectopy. Serum potassium is 2.8 mmol/L. Which adverse effect is responsible?

A. Thiazide-associated renal potassium wasting causing hypokalemia

B. Thiazide-associated hyperkalemia from mineralocorticoid blockade

C. Calcium-channel blockade causing intracellular potassium sequestration

D. ACE inhibition causing potassium wasting through increased aldosterone

113. An older adult becomes confused and unsteady two weeks after starting a thiazide-type diuretic. Sodium is 122 mmol/L. Which drug complication is most likely?

A. ACE inhibitor-associated hypernatremia

B. Amlodipine-associated diabetes insipidus

C. Thiazide-associated hyponatremia

D. Beta-blocker-associated renal salt wasting

114. A patient with gout has recurrent attacks after a thiazide dose increase. Which metabolic effect of thiazide therapy can contribute?

A. Marked uricosuria with persistent hypouricemia

B. Direct inhibition of xanthine oxidase lowering uric acid

C. Conversion of uric acid to allantoin in the kidney

D. Reduced urate excretion with hyperuricemia

115. A patient with advanced CKD has hypertension and clinically important extracellular volume overload. Which diuretic class is often needed for effective volume removal?

A. A carbonic anhydrase inhibitor as the preferred chronic volume agent

B. An osmotic diuretic as routine outpatient antihypertensive therapy

C. A potassium supplement without a diuretic

D. A loop diuretic for effective natriuresis and decongestion

116. A patient with hypertension and resting heart rate 52/min needs an additional drug. Which agent is least likely to worsen the bradycardia?

A. Amlodipine

B. Verapamil

C. Diltiazem

D. Metoprolol

117. A patient taking verapamil for hypertension develops fatigue, a heart rate of 42/min, and first-degree atrioventricular block. Which pharmacologic property explains these findings?

A. Predominantly peripheral vasodilation triggers reflex vagal slowing of the sinus node and AV conduction

B. Nondihydropyridine calcium-channel blockade slows sinoatrial and atrioventricular nodal conduction

C. Mineralocorticoid receptor antagonism reduces nodal calcium entry and directly produces atrioventricular block

D. Thiazide-like sodium transport inhibition directly slows conduction through the atrioventricular node

118. A 48-year-old patient has uncomplicated primary hypertension with no coronary disease, heart failure, arrhythmia, or other compelling indication. Which statement about beta blockers is most accurate?

A. They are not preferred universal first-line therapy when no compelling indication is present

B. They are preferred first-line therapy in uncomplicated hypertension because suppression of renin provides superior outcome protection

C. They should generally be avoided in uncomplicated hypertension because they worsen cardiovascular outcomes even when another indication exists

D. Their principal antihypertensive action is direct alpha-1 blockade of systemic resistance vessels

119. A 70-year-old man with hypertension and symptomatic benign prostatic hyperplasia asks whether doxazosin could address both conditions. What is the main limitation of using an alpha-1 blocker as primary BP therapy?

A. Orthostatic hypotension and weaker outcome evidence than first-line agents

B. Severe hyperkalemia from mineralocorticoid receptor blockade

C. Persistent cough from bradykinin accumulation

D. Obligatory bradycardia from atrioventricular nodal blockade

120. A patient taking clonidine misses several doses and develops abrupt severe hypertension and tachycardia. Which management principle is most important when discontinuing chronic clonidine?

A. Stop it suddenly because rebound hypertension does not occur

B. Increase a nonselective beta blocker first and stop clonidine immediately

C. Taper the drug rather than stopping it abruptly

D. Replace clonidine with an NSAID to prevent sympathetic activation

121. A patient taking high-dose hydralazine develops fever, arthralgias, myalgias, and a positive antihistone antibody. Which adverse effect is most likely?

A. ACE inhibitor-associated angioedema

B. Thiazide-associated gout without systemic symptoms

C. Drug-induced lupus syndrome

D. Dihydropyridine-associated ankle edema

122. A patient with refractory hypertension is started on minoxidil. Which accompanying adverse-effect pattern is most characteristic?

A. Bradycardia, volume depletion, and hair loss

B. Dry cough, angioedema, and hyperkalemia from bradykinin accumulation

C. Hyponatremia, hypokalemia, and gout from distal Na-Cl blockade

D. Fluid retention, reflex tachycardia, and hypertrichosis

123. A man with resistant hypertension develops gynecomastia and sexual adverse effects on spironolactone. Which alternative mineralocorticoid receptor antagonist has fewer antiandrogen effects?

A. Hydrochlorothiazide

B. Eplerenone

C. Amlodipine

D. Clonidine

124. A patient with genetically confirmed Liddle syndrome has persistent hypertension despite an ACE inhibitor. Which drug directly targets the causal sodium transport abnormality?

A. Spironolactone

B. Amiloride

C. Losartan

D. Aliskiren

125. A patient has true resistant hypertension despite maximally tolerated doses of an ARB, amlodipine, and chlorthalidone. Potassium is 4.2 mmol/L and kidney function is adequate. Which add-on drug has strong evidence?

A. A second ARB

B. A second dihydropyridine calcium channel blocker

C. An ACE inhibitor added to the existing ARB

D. Spironolactone as fourth-line therapy

126. A patient is labeled resistant to treatment while taking lisinopril, amlodipine, and hydrochlorothiazide at modest doses. Which medication optimization is most appropriate before escalating to unusual agents?

A. Add a second renin-angiotensin system blocker before optimizing the current diuretic and calcium channel blocker doses

B. Keep hydrochlorothiazide at a modest dose and add clonidine before addressing possible volume dependence

C. Use maximally tolerated complementary therapy and consider a longer-acting thiazide-like diuretic

D. Add hydralazine before confirming that the complementary three-drug regimen is maximally tolerated

127. A patient has uncontrolled office blood pressure despite four prescribed medications, but pharmacy records show that prescriptions are filled only intermittently. What is the most important next step?

A. Diagnose refractory hypertension and proceed directly to renal denervation

B. Address medication adherence using a nonjudgmental, specific assessment and regimen simplification

C. Increase the doses of the existing regimen before addressing refill gaps because inadequate prescribed intensity is the likelier cause of apparent resistance

D. Confirm the out-of-office pressure phenotype first and postpone a detailed adherence intervention until after ambulatory hypertension is documented

128. A patient with stage 2 hypertension is starting an ACE inhibitor and amlodipine. Which formulation strategy can improve adherence when an appropriate product and doses are available?

A. Keep the drugs as separate pills so each can be titrated independently even when the intended doses are already established

B. Alternate the ACE inhibitor and calcium channel blocker on different days to reduce additive hypotension

C. Use the second agent as needed on days when office or home systolic pressure rises above 160 mm Hg

D. Use an appropriate fixed-dose single-pill combination to reduce pill burden and simplify the daily regimen

129. A patient takes five antihypertensive tablets at four different times each day and frequently misses afternoon doses. Which intervention is most likely to improve adherence?

A. Add additional midday doses so each drug has a separate administration time

B. Avoid discussing adherence because missed doses should be assumed intentional

C. Use short-acting agents exclusively because they require more frequent reminders

D. Simplify to once-daily dosing and combination pills where clinically feasible

130. A patient with previously controlled hypertension develops higher home readings after beginning daily naproxen. Which medication interaction is most likely?

A. NSAID-related sodium retention and reduced effectiveness of antihypertensive therapy

B. Naproxen increases renal prostaglandin activity, enhancing diuretic natriuresis and thereby lowering blood pressure

C. Attribute the higher readings to progression of hypertension because naproxen tends to suppress aldosterone and lower pressure

D. Naproxen accelerates hepatic metabolism of antihypertensive drugs, reducing their serum concentrations as the principal interaction

131. A patient taking spironolactone and an ARB is prescribed trimethoprim-sulfamethoxazole for an infection. Which laboratory complication deserves particular attention?

A. Severe hypokalemia

B. Profound hypocalcemia

C. Drug-associated hyperkalemia

D. Isolated hypernatremia

132. A patient taking metoprolol for coronary disease is started on verapamil for additional blood pressure control. Which adverse interaction is most concerning?

A. Marked reflex tachycardia from opposing nodal effects

B. Excessive bradycardia or atrioventricular block

C. Severe potassium wasting from combined distal sodium blockade

D. Accelerated bradykinin breakdown causing persistent cough

133. A man with hypertension and stable angina uses sublingual nitroglycerin. He asks about sildenafil for erectile dysfunction. Which counseling is most important?

A. Do not combine a phosphodiesterase-5 inhibitor with nitrates because profound hypotension can occur

B. Separate sildenafil and sublingual nitroglycerin by one hour, which is sufficient to prevent the vasodilator interaction

C. Hold nitroglycerin for the calendar day sildenafil is taken, without accounting for the inhibitor's timing or half-life

D. Hold the patient's chronic antihypertensive medications on days sildenafil is used to avoid additive blood pressure lowering

134. A patient taking several antihypertensive medications starts patiromer for chronic hyperkalemia. Which administration strategy helps avoid reduced absorption of certain coadministered drugs?

A. Take antihypertensive medications together with patiromer because the binder is assumed to interact selectively with potassium

B. Crush susceptible oral medications into the patiromer suspension so their absorption occurs at the same time

C. Use the same administration time for cardiovascular drugs and separate patiromer chiefly from antimicrobial agents

D. Separate susceptible oral medications from patiromer by several hours to reduce clinically important binding interactions

135. A patient with albuminuric chronic kidney disease has hypertension and no contraindication to renin-angiotensin system blockade. Which class is particularly appropriate?

A. An ACE inhibitor or angiotensin receptor blocker because renin-angiotensin blockade is favored with albuminuria

B. A dihydropyridine calcium channel blocker because it reduces albuminuria through preferential efferent arteriolar dilation

C. A thiazide-type diuretic because volume reduction is the principal proven strategy for slowing albuminuric kidney disease

D. An alpha-1 blocker because systemic vasodilation preferentially lowers intraglomerular pressure and protein excretion

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

A. Prefer a beta blocker as first-line therapy because diabetes-associated sympathetic activation makes it superior to thiazide-type diuretics or calcium channel blockers

B. Several standard first-line classes can be appropriate, selected according to the overall clinical profile

C. Require an ACE inhibitor as initial therapy in diabetes even without albuminuria because renin-angiotensin blockade has unique outcome benefit in every such patient

D. Continue lifestyle therapy until blood pressure reaches 140/90 mm Hg because diabetes alone does not lower the threshold for pharmacologic treatment

137. An older adult has isolated systolic hypertension without a compelling indication for a specific drug class. Which regimen is reasonable?

A. A long-acting thiazide-type diuretic or dihydropyridine calcium channel blocker

B. Short-acting nifedipine used intermittently when systolic pressure feels high

C. Dual ACE inhibitor and ARB therapy as routine first-line treatment

D. An alpha-1 blocker alone because it best reduces arterial stiffness outcomes

138. A Black adult with stage 2 hypertension has no CKD, heart failure, or other compelling indication. Which initial two-drug combination is a reasonable evidence-based choice?

A. An ACE inhibitor plus an ARB to maximize renin-angiotensin blockade

B. A thiazide-type diuretic plus a long-acting dihydropyridine calcium channel blocker

C. Two beta blockers from different subclasses

D. Two central alpha-2 agonists as routine initial combination therapy

139. A patient with recurrent gout needs an ARB for hypertension. Which ARB has a modest uricosuric effect that may be useful in this setting?

A. Valsartan

B. Losartan

C. Candesartan

D. Olmesartan

140. A patient with hypertension and recurrent calcium nephrolithiasis has hypercalciuria. Which antihypertensive class can reduce urinary calcium excretion?

A. A loop diuretic

B. A direct renin inhibitor

C. A central alpha-2 agonist

D. A thiazide-type diuretic

141. A patient with hypertension and Raynaud phenomenon requires another antihypertensive agent. Which drug can improve both conditions?

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

B. A nonselective beta blocker that promotes peripheral vasoconstriction

C. Clonidine because it is the first-line treatment for Raynaud phenomenon

D. Hydrochlorothiazide because it directly dilates digital arteries

142. A patient with hypertension and frequent migraine headaches has no asthma, bradycardia, or conduction disease. Which class may address both problems?

A. A dihydropyridine calcium channel blocker because vascular smooth-muscle relaxation is an established migraine-prevention mechanism

B. A central alpha-2 agonist because intermittent sympathetic suppression is an established migraine-prevention strategy

C. A beta blocker with established migraine-prevention efficacy, provided no contraindication to beta blockade is present

D. A loop diuretic because mild volume contraction is an established preventive treatment for recurrent migraine

143. A patient with hypertension and permanent atrial fibrillation needs ventricular rate control. Which drug can lower both heart rate and blood pressure?

A. A thiazide-type diuretic

B. A dihydropyridine calcium channel blocker such as amlodipine

C. A mineralocorticoid receptor antagonist as a primary rate-control drug

D. A beta blocker with AV-nodal and antihypertensive effects

144. A patient develops bothersome ankle edema on amlodipine but has otherwise excellent response. Which modification can reduce dihydropyridine-associated edema while maintaining BP control?

A. Switch to a second dihydropyridine calcium channel blocker at an equivalent dose because edema is specific to amlodipine rather than the class

B. Add a loop diuretic while keeping the amlodipine dose unchanged because the edema primarily reflects total-body sodium excess

C. Increase the amlodipine dose to reduce capillary hydrostatic pressure through more complete arteriolar dilation

D. Combine the calcium channel blocker with an ACE inhibitor or ARB when clinically appropriate

145. A patient has office blood pressure 158/90 mm Hg despite four drugs, but ambulatory pressure averages 126/74 mm Hg. What is the best next treatment step?

A. Add two additional medications because office pressure alone defines true resistance

B. Avoid reflex medication escalation and manage the white-coat effect using out-of-office data

C. Proceed directly to renal denervation because ambulatory pressure is irrelevant

D. Stop all antihypertensive medication because any white-coat effect means treatment is unnecessary

146. A patient with apparently resistant hypertension regularly uses pseudoephedrine and high-dose ibuprofen. Which intervention should occur before adding a fifth antihypertensive medication?

A. Continue both agents unchanged because nonprescription drugs do not affect hypertension

B. Add an ACE inhibitor and ARB together to counteract the pressor effect

C. Proceed directly to adrenalectomy without biochemical evaluation

D. Remove or minimize pressor medications when feasible and reassess blood pressure

147. A patient has confirmed resistant hypertension on an optimized regimen. Which endocrine cause deserves particularly strong consideration because it is relatively common in resistant hypertension and has specific screening and targeted therapy?

A. Primary aldosteronism from autonomous aldosterone secretion

B. Pheochromocytoma or paraganglioma

C. Cushing syndrome from endogenous cortisol excess

D. Acromegaly from growth hormone excess

148. A patient with true resistant hypertension has been carefully evaluated, is taking an ACE inhibitor, amlodipine, chlorthalidone, and spironolactone at tolerated doses, and still has stage 2 hypertension. He asks about renal denervation. Which statement is most appropriate?

A. Renal denervation is a guaranteed cure and should replace all antihypertensive medication

B. Renal denervation should be performed before confirming adherence or secondary causes

C. Renal denervation is appropriate in pregnancy and fibromuscular dysplasia without restriction

D. Renal denervation may be considered after multidisciplinary evaluation and shared decision-making in selected patients

149. A patient considering renal denervation asks whether successful treatment would eliminate the need for chronic antihypertensive medication. What is the best counseling point?

A. Plan to discontinue antihypertensive medications immediately after a technically successful procedure if office pressure falls

B. The procedure usually provides modest additional blood pressure lowering and is not considered a curative replacement for medication

C. Use renal denervation mainly to replace lifestyle therapy, because its pressure effect is independent of medication adherence and sodium intake

D. Reserve renal denervation for patients with a proven secondary endocrine cause because that group has the most predictable response

150. An inpatient admitted for pneumonia has repeated blood pressures around 190/104 mm Hg but no chest pain, neurologic symptoms, pulmonary edema, acute kidney injury, or other target-organ damage. What is the best immediate approach?

A. Give intermittent intravenous hydralazine for repeated systolic readings above 180 mm Hg despite the absence of acute organ injury

B. Transfer to an intensive care setting because a pressure threshold above 180/120 mm Hg by itself defines hypertensive emergency

C. Confirm measurement and address underlying factors rather than rapidly lowering pressure with intermittent IV medication

D. Attribute the elevation to acute illness and defer repeat assessment until after discharge if the patient remains asymptomatic

151. A patient with treated hypertension has normal office readings but home blood pressures are consistently 138/84 mm Hg using a validated technique. What is the most appropriate interpretation?

A. Treat the hypertension as controlled because standardized office pressure is normal and the home elevation is modest

B. Interpret the disagreement as a white-coat effect because office and home measurements differ substantially

C. Repeat office measurements before using home data, because home monitoring is primarily useful for assessing adherence rather than control

D. Consider masked uncontrolled hypertension and confirm the pattern before treatment adjustment

152. A patient with newly treated hypertension is willing to measure blood pressure at home. How should the clinician use these readings?

A. Use home averages mainly to screen for symptomatic hypotension rather than to guide upward titration of treatment

B. Use office measurements as the sole titration target and reserve home readings for documenting medication adherence

C. Adjust treatment whenever two consecutive home readings exceed goal without first calculating a multiday average

D. Incorporate validated home measurements into ongoing titration and assessment of white-coat or masked effects

153. A patient repeatedly misses doses because medication costs are high and refills come due on different dates. Which intervention best addresses the barrier?

A. Use lower-cost options when possible and synchronize refills with a simplified regimen

B. Add electronic dose reminders while leaving the medication costs and staggered refill dates unchanged

C. Increase dosing frequency so each missed dose represents a smaller fraction of the prescribed daily regimen

D. Switch to a branded single-pill combination even if the higher copay makes monthly medication costs increase

154. A patient with hypertension and obesity asks whether pharmacologic weight management can influence blood pressure. Which statement is most accurate?

A. Effective weight-loss therapy can contribute to lower blood pressure as part of comprehensive cardiometabolic care

B. Expect blood pressure to remain essentially unchanged because anti-obesity therapy affects weight but not vascular risk

C. Keep antihypertensive doses fixed during substantial weight loss unless the office pressure rises above the pretreatment value

D. Treat hypertension independently and defer obesity therapy because weight reduction is too small a contributor to alter blood pressure management

155. A patient has stage 1 hypertension close to goal and a history of medication-related dizziness. Which initial pharmacologic strategy is reasonable if medication is indicated?

A. Start two first-line agents at standard doses despite the prior history of medication-related dizziness

B. Use low doses of an ACE inhibitor and an ARB together so each individual drug can be kept below its usual dose

C. Use a short-acting oral antihypertensive as needed for episodic office elevations rather than a scheduled first-line agent

D. Begin one first-line antihypertensive agent at a low dose and titrate carefully while monitoring symptoms

156. A nonfrail patient has blood pressure 166/102 mm Hg, more than 20/10 mm Hg above a goal below 130/80 mm Hg. Which strategy is most appropriate?

A. Start one first-line agent and titrate it to the maximum dose before adding a second medication

B. Use initial combination therapy with two complementary first-line agents

C. Use two renin-angiotensin system blockers at low doses to reduce class-specific adverse effects

D. Start a central sympatholytic first because marked hypertension requires a rapidly acting oral agent before standard first-line drugs

157. A patient develops potassium 5.8 mmol/L after spironolactone is added for resistant hypertension. Which response is most appropriate?

A. Continue spironolactone and add another renin-angiotensin blocker to improve pressure control despite the potassium level

B. Switch from spironolactone to an equivalent dose of eplerenone without otherwise addressing the elevated potassium

C. Reassess spironolactone, kidney function, and interacting drugs, and treat the hyperkalemia

D. Continue the current spironolactone dose and recheck potassium in several months if the patient has no symptoms

158. A patient starts an ACE inhibitor and creatinine rises from 1.0 to 1.18 mg/dL while potassium remains normal and volume status is stable. What is the best interpretation?

A. Any rise in creatinine proves bilateral renal artery stenosis and requires emergency angiography

B. A small creatinine increase can be an expected hemodynamic effect and does not by itself require stopping therapy

C. The increase shows ACE inhibition is directly toxic to renal tubular cells

D. The drug should be doubled immediately because creatinine rise predicts better BP control

159. A patient with hypertension is stable on a thiazide-type diuretic. Which routine laboratory issues should be monitored over time?

A. Monitor glucose and uric acid but omit repeat electrolytes when the thiazide dose has been stable

B. Monitor liver enzymes because chronic hepatotoxicity is the principal laboratory concern with thiazide-type diuretics

C. Sodium, potassium, kidney function, and metabolic adverse effects such as uric acid changes

D. No repeat laboratory testing is needed after an initially normal electrolyte and kidney-function panel

160. A patient has severe hypertension but is highly anxious during clinic visits. Repeated home readings are normal. Which approach best prevents overtreatment?

A. Escalate medication until every office reading is below 110/60 mm Hg

B. Disregard all out-of-office information because anxiety cannot affect blood pressure

C. Base decisions on standardized repeated measurements and confirm the out-of-office phenotype

D. Use an IV antihypertensive before every clinic visit to prevent transient elevations

161. A 62-year-old man presents with blood pressure 228/132 mm Hg, confusion, papilledema, and acute kidney injury. Which feature most clearly makes this a hypertensive emergency rather than severe asymptomatic hypertension?

A. A blood pressure above 200/120 mm Hg is sufficient to define a hypertensive emergency even without acute organ injury

B. Abrupt worsening from a previous diagnosis of chronic hypertension establishes a hypertensive emergency

C. A diastolic pressure above 120 mm Hg defines malignant hypertension even when acute target-organ injury is absent

D. Acute hypertension-mediated target-organ injury, demonstrated here by neurologic and renal abnormalities

162. A patient with hypertensive encephalopathy has no aortic dissection, acute ischemic stroke, or other condition requiring a disease-specific blood pressure target. What is a reasonable initial blood pressure-lowering strategy?

A. Normalize blood pressure to below 120/80 mm Hg within 10 minutes

B. Avoid all blood pressure reduction for the first 24 hours despite encephalopathy

C. Lower systolic pressure by at least 100 mm Hg in the first 15 minutes

D. Reduce mean arterial pressure by about 20% to 25% during the first hour, then lower more gradually

163. A 58-year-old man has abrupt tearing chest pain, a proximal aortic dissection on CT, blood pressure 196/112 mm Hg, and heart rate 104/min. Which initial antihypertensive approach best reduces aortic wall stress?

A. Begin a potent arterial vasodilator alone before controlling heart rate

B. Begin intravenous beta blockade promptly, then add a titratable vasodilator if needed

C. Give an oral thiazide and reassess the blood pressure the next morning

D. Use sublingual immediate-release nifedipine to produce the fastest possible pressure fall

164. A patient presents with severe hypertension, acute dyspnea, diffuse pulmonary edema, and preserved perfusion. Which treatment principle is most appropriate?

A. Give a large intravenous fluid bolus to raise filling pressures further

B. Use a nonselective beta blocker as the sole immediate therapy despite acute pulmonary edema

C. Use rapidly titratable intravenous vasodilation while treating congestion and respiratory failure

D. Delay treatment until several outpatient blood pressure readings confirm chronic hypertension

165. A patient with acute coronary syndrome has blood pressure 186/106 mm Hg and ongoing ischemic chest pain without shock, bradycardia, or acute heart failure. Which principle best guides blood pressure treatment?

A. Lower blood pressure while simultaneously treating myocardial ischemia and avoiding an excessive fall in coronary perfusion

B. Lower blood pressure immediately to below 120/70 mm Hg to minimize myocardial oxygen demand as quickly as possible

C. Treat the ischemia first and defer blood pressure therapy because the elevated pressure is primarily compensatory during acute coronary syndrome

D. Use a potent arterial vasodilator without considering reflex tachycardia or the effect of diastolic pressure on coronary perfusion

166. A 70-year-old woman has a mild-to-moderate spontaneous intracerebral hemorrhage and presents with systolic blood pressure 186 mm Hg. Which acute systolic blood pressure strategy is most consistent with contemporary guidance?

A. Use permissive hypertension similar to nonreperfused ischemic stroke and avoid early pressure reduction while neurologic status is stable

B. Lower smoothly toward approximately 140 mm Hg while avoiding sustained values below 130 mm Hg

C. Target systolic pressure between 110 and 120 mm Hg to minimize hematoma expansion as much as possible

D. Use the nonreperfused ischemic-stroke threshold near 220 mm Hg before treating intracerebral hemorrhage

167. A patient with acute ischemic stroke is otherwise eligible for intravenous thrombolysis, but blood pressure is 198/116 mm Hg. What should be done before thrombolytic treatment?

A. Give thrombolysis immediately because blood pressure does not affect hemorrhagic risk

B. Lower blood pressure to below 185/110 mm Hg using a titratable regimen

C. Lower systolic pressure below 120 mm Hg before considering reperfusion

D. Withhold reperfusion permanently because any initial pressure above 180/100 mm Hg is an absolute lifetime contraindication

168. A patient has acute ischemic stroke, is not receiving thrombolysis or thrombectomy, has no aortic dissection or other urgent indication for blood pressure reduction, and has blood pressure 206/112 mm Hg. Which approach is most appropriate during the first 48 to 72 hours?

A. Lower systolic pressure below 140 mm Hg during the first day because the same target used for intracerebral hemorrhage applies to ischemic stroke

B. Reduce pressure promptly by about 25% with intravenous therapy because any acute ischemic stroke above 180 mm Hg represents hypertensive emergency

C. Use the pre-thrombolysis target below 185/110 mm Hg even though no reperfusion therapy is planned

D. Avoid routine active lowering solely for this pressure level and monitor closely

169. A patient with acute ischemic stroke has successful endovascular reperfusion. Which blood pressure strategy should generally be avoided in the first 72 hours when there is no other indication for a lower target?

A. Intensive systolic reduction to below 140 mm Hg

B. Avoiding hypotension and maintaining adequate systemic perfusion

C. Using individualized blood pressure management after reperfusion

D. Monitoring for hemorrhagic transformation and neurologic deterioration

170. A 33-year-old woman with chronic hypertension becomes pregnant. Her preconception regimen includes losartan. Which medication change is most appropriate?

A. Continue losartan through the first trimester and switch later because fetal renal toxicity is primarily a late-pregnancy concern

B. Switch losartan to an ACE inhibitor because ACE inhibition is considered safer than ARB therapy during pregnancy

C. Stop antihypertensive treatment at conception and restart at severe-range pressure to preserve uteroplacental perfusion

D. Discontinue losartan and transition to a pregnancy-compatible antihypertensive such as labetalol or extended-release nifedipine

171. A pregnant patient with chronic hypertension has repeated blood pressures of 146/94 mm Hg at 18 weeks' gestation. Which management approach is most consistent with current evidence?

A. Withhold medication until persistent severe-range pressure of at least 160/110 mm Hg develops

B. Defer medication until preeclampsia or proteinuria develops because treating mild chronic hypertension is thought to restrict fetal growth

C. Initiate or titrate antihypertensive therapy rather than waiting for severe-range hypertension

D. Use renin-angiotensin system blockade after the first trimester because fetal organogenesis is then complete

172. A woman at 32 weeks' gestation has persistent blood pressure 168/112 mm Hg confirmed within minutes. What is the most appropriate next step?

A. Observe with repeated measurements for several hours before treatment because acute therapy is reserved for pressures that remain severe over a prolonged interval

B. Give magnesium sulfate as the sole immediate therapy because seizure prophylaxis also provides sufficient blood pressure control

C. Start oral methyldopa for gradual blood pressure reduction and defer rapidly acting therapy while she remains neurologically asymptomatic

D. Treat severe-range hypertension promptly with a pregnancy-appropriate rapidly acting agent

173. A woman with chronic hypertension asks about reducing the risk of preeclampsia during pregnancy. Which preventive measure is commonly recommended when no contraindication exists?

A. Routine warfarin throughout pregnancy to prevent placental thrombosis

B. High-dose ibuprofen from the first trimester until delivery

C. Low-dose aspirin started during pregnancy according to obstetric guidance

D. Stopping all antihypertensive drugs to increase uteroplacental perfusion

174. A woman with gestational hypertension is discharged after delivery. Which follow-up strategy is most appropriate?

A. Use the routine six-week postpartum visit as the first blood pressure reassessment if the patient has no symptoms

B. Stop home monitoring after delivery because removal of the placenta reliably eliminates the hypertensive physiology

C. Hold antihypertensive medication at discharge unless severe-range blood pressure persists in the hospital

D. Arrange early postpartum blood pressure assessment and encourage validated home monitoring

175. A 67-year-old patient has hypertension, eGFR 48 mL/min/1.73 m², and urine albumin-to-creatinine ratio 520 mg/g. Which drug class has a particularly strong kidney-protective rationale if tolerated?

A. An ACE inhibitor or angiotensin receptor blocker, but not both together

B. A combined ACE inhibitor plus angiotensin receptor blocker regimen

C. Use clonidine as needed for episodic home blood pressure elevations because CKD limits use of renin-angiotensin blockers

D. A direct renin inhibitor combined routinely with an ACE inhibitor

176. A kidney transplant recipient has persistent hypertension while taking tacrolimus. Which contributor should be considered specifically in this setting?

A. Loss of renal sympathetic innervation after transplantation makes persistent hypertension primarily independent of renal vascular tone

B. Tacrolimus lowers vascular resistance but activates the adrenal cortex, making secondary hyperaldosteronism its principal pressor mechanism

C. Calcineurin-inhibitor–related renal vasoconstriction and sodium retention associated with tacrolimus therapy

D. Tacrolimus causes renal sodium wasting that activates the renin-angiotensin system and raises pressure secondarily

177. A patient with HFrEF has hypertension despite guideline-directed therapy. Which calcium channel blocker should generally be avoided because of negative inotropic effects?

A. Amlodipine

B. Felodipine

C. Long-acting nifedipine

D. Verapamil

178. A patient with HFrEF, hypertension, and volume overload remains congested. Which blood pressure strategy best fits the comorbidity?

A. Optimize heart-failure guideline-directed therapy and use diuretics as needed for congestion

B. Prioritize amlodipine escalation before optimizing disease-modifying heart-failure therapy because pressure reduction is the main mortality benefit

C. Avoid loop diuretics despite clinical congestion because lower filling pressure tends to worsen hypertension in HFrEF

D. Use verapamil for additional blood pressure and rate control despite reduced ejection fraction

179. A patient with chronic coronary disease and hypertension has blood pressure 142/84 mm Hg. Which long-term target is generally appropriate when treatment is tolerated?

A. A blood pressure target below 140/90 mm Hg as the routine goal for chronic coronary disease

B. A blood pressure target below 130/80 mm Hg when tolerated as part of secondary cardiovascular prevention

C. A systolic range of 130 to 139 mm Hg with diastolic pressure 80 to 89 mm Hg to avoid a coronary perfusion J-curve in all patients

D. Prioritize antianginal therapy and accept systolic pressure up to 150 mm Hg when angina is controlled

180. An 84-year-old nursing-home resident has advanced dementia, recurrent falls, orthostatic symptoms, and limited life expectancy. How should the intensity of antihypertensive therapy be determined?

A. Use an intensive systolic target below 120 mm Hg because trial results in older adults apply without modification for frailty or symptoms

B. Deprescribe all antihypertensive therapy because age above 80 years reverses the cardiovascular benefit of treatment

C. Use a routine systolic target of 150 to 160 mm Hg for nursing-home residents to reduce fall risk

D. Individualize treatment using goals of care, symptoms, frailty, comorbidity, and likely benefits versus harms

181. A 72-year-old treated patient becomes lightheaded on standing. Supine blood pressure is 148/78 mm Hg and, after standing, it falls to 122/66 mm Hg with symptoms. What is the best next step?

A. Base treatment primarily on seated pressure because standing measurements are too variable for medication decisions

B. Review symptoms, volume status, medications, and the timing of the orthostatic fall before reflexively abandoning hypertension treatment

C. Intensify treatment of the supine systolic pressure first and reassess dizziness after control improves

D. Add a bedtime antihypertensive for the supine pressure before reviewing volume status and the existing regimen

182. A patient with hypertension develops orthostatic dizziness after doxazosin was added for blood pressure control. Which medication-related explanation is most plausible?

A. Alpha-1 blockade augments baroreflex-mediated peripheral vasoconstriction when the patient stands

B. Doxazosin acts mainly in the kidney, producing natriuresis without materially changing systemic venous tone

C. Alpha-1 blockade can impair compensatory vasoconstriction on standing

D. Doxazosin lowers supine pressure while largely preserving the venous constrictor response needed for standing

183. A patient with Parkinson disease has neurogenic orthostatic hypotension plus marked supine hypertension at night. Which management principle is most appropriate?

A. Individualize therapy to balance supine hypertension against symptomatic standing hypotension

B. Use a long-acting antihypertensive throughout the daytime so the supine pressure is controlled continuously

C. Treat the orthostatic symptoms and regard nocturnal supine hypertension as a relatively benign consequence of autonomic failure

D. Schedule most vasodilator therapy in the morning so nocturnal pressure falls without worsening upright symptoms

184. A patient with bipolar disorder takes lithium and develops hypertension requiring therapy. Which medication issue is especially important?

A. Thiazide diuretics increase renal lithium clearance, making subtherapeutic lithium concentrations the main concern

B. Thiazide diuretics can raise lithium concentrations and increase toxicity risk

C. Lithium clearance is independent of renal sodium handling, so antihypertensive choice does not affect lithium exposure

D. ACE inhibitors increase lithium clearance and are therefore preferred when a patient receiving lithium develops hypertension

185. A patient taking venlafaxine for major depressive disorder develops a sustained blood pressure increase after a dose escalation. Which explanation is most appropriate?

A. Venlafaxine produces irreversible adrenal insufficiency that lowers blood pressure

B. The serotonin-norepinephrine reuptake inhibitor can contribute to dose-related blood pressure elevation

C. The antidepressant cannot affect sympathetic tone or blood pressure

D. The pressure increase establishes pheochromocytoma without need for medication review

186. A 14-year-old adolescent has repeated auscultatory blood pressures of 136/86 mm Hg at separate visits. Which classification framework should generally be used for adolescents age 13 years and older?

A. Continue percentile-based pediatric thresholds through age 17 rather than using fixed adult-aligned thresholds at age 13

B. Classify 136/86 mm Hg as elevated blood pressure rather than hypertension because systolic pressure remains below 140 mm Hg

C. Classify the reading as stage 2 hypertension because any diastolic pressure at or above 80 mm Hg is stage 2 in adolescents

D. Use fixed thresholds aligned with the adult-style pediatric classification, making this stage 1 hypertension

187. An 8-year-old child has elevated oscillometric blood pressure at a well visit. What is the most appropriate immediate measurement step?

A. Repeat the measurement and confirm an abnormal reading by auscultation using an appropriate cuff

B. Repeat and average oscillometric readings and accept them as diagnostic if the device is validated, without auscultatory confirmation

C. Use a validated wrist device if an upper-arm cuff is difficult to fit, because wrist and arm measurements are diagnostically interchangeable

D. Repeat the pressure at the next annual visit because routine hypertension screening begins in adolescence

188. A 10-year-old child has auscultatory blood pressure at or above the 95th percentile on three separate visits. Which next diagnostic tool is particularly useful for confirming sustained hypertension and detecting white-coat hypertension?

A. Use structured home upper-arm blood pressure readings as the preferred test to confirm the diagnosis and white-coat phenotype

B. Obtain echocardiography first and infer sustained hypertension if left ventricular hypertrophy is present

C. Perform a broad secondary-hypertension laboratory workup before confirming whether the office phenotype is sustained

D. Ambulatory blood pressure monitoring with a validated pediatric device

189. A 15-year-old with obesity and a strong family history of hypertension has persistent stage 1 hypertension but no history, examination, or laboratory clues to a secondary cause. Which evaluation strategy is most appropriate?

A. Order renal vascular imaging and endocrine testing now because adolescence alone makes secondary hypertension sufficiently likely to justify a broad evaluation

B. Perform the recommended basic evaluation without automatically ordering an extensive secondary-hypertension workup

C. Obtain a comprehensive secondary-hypertension workup despite the obesity and family history because primary hypertension should remain a diagnosis of exclusion in every adolescent

D. Begin lifestyle therapy and postpone even the recommended basic evaluation for six months because the absence of symptoms makes secondary disease unlikely

190. A 12-year-old with stage 2 hypertension has repeat measurements still in the stage 2 range but no acute symptoms. What is the most appropriate next step?

A. Arrange prompt diagnostic evaluation and treatment or subspecialty referral, generally within about one week

B. Arrange ambulatory monitoring and lifestyle counseling, but defer diagnostic evaluation for several months unless symptoms or still higher pressures develop

C. Start antihypertensive medication immediately without further diagnostic evaluation because persistent stage 2 readings already establish the cause as primary hypertension

D. Repeat office measurements in three to six months because asymptomatic stage 2 hypertension in children can be followed like persistent stage 1 hypertension

191. A 13-year-old with stage 2 hypertension develops severe headache and vomiting, and blood pressure is more than 30 mm Hg above the 95th percentile. What is the most appropriate disposition?

A. Refer for immediate emergency evaluation for acute severe hypertension and target-organ injury

B. Schedule routine follow-up in six months because symptoms do not change pediatric management

C. Advise strenuous exercise to lower blood pressure before remeasurement

D. Start an over-the-counter decongestant and repeat the pressure the next day

192. A hypertensive child is about to start long-term pharmacologic therapy. Which test is commonly used to assess cardiac target-organ effects before medication initiation?

A. Routine invasive coronary angiography

B. Annual electrophysiology study in all children

C. Pulmonary artery catheterization before the first antihypertensive dose

D. Echocardiography to evaluate left ventricular hypertrophy

193. A 16-year-old with confirmed primary hypertension, obesity, and no acute target-organ injury is starting treatment. Which intervention should be part of management regardless of whether medication is also needed?

A. Eliminate all physical activity to prevent exercise-related systolic pressure increases

B. Use medication alone because lifestyle treatment is ineffective in adolescents

C. Family-centered lifestyle therapy addressing diet, physical activity, weight, and sodium exposure

D. Encourage high-sodium sports drinks throughout the day to prevent pressure variability

194. A child has hypertension due to chronic kidney disease and persistent proteinuria. Which medication class is often favored when no contraindication exists?

A. An ACE inhibitor or angiotensin receptor blocker, particularly when persistent proteinuria accompanies pediatric CKD

B. Use a thiazide-type diuretic as the sole kidney-protective therapy because reducing volume is more important than reducing proteinuria

C. A routine combination of ACE inhibitor plus angiotensin receptor blocker

D. Use a calcium channel blocker preferentially because renin-angiotensin blockade tends to worsen proteinuria in pediatric CKD

195. A 17-year-old with repaired coarctation of the aorta has normal clinic blood pressure but exertional headaches. Which blood pressure assessment is particularly useful?

A. No further assessment because repaired coarctation eliminates future hypertension risk

B. A single ankle blood pressure taken immediately after sprinting

C. Home blood pressure alone as the preferred diagnostic test for masked pediatric hypertension

D. Ambulatory blood pressure monitoring to look for masked hypertension

196. A patient with severe anxiety has episodic clinic blood pressure elevations but normal validated home and ambulatory readings and no target-organ injury. Which approach is most appropriate?

A. Treat each transient office systolic pressure above 180 mm Hg as a same-day medication-escalation indication even without organ injury

B. Use a short-acting anxiolytic or antihypertensive before clinic visits so the office measurement is normalized

C. Address anxiety while avoiding unnecessary antihypertensive escalation based only on situational office elevations

D. Focus exclusively on antihypertensive pharmacotherapy because treating anxiety does not influence the office blood pressure phenotype

197. A hospitalized patient has repeated blood pressures near 190/100 mm Hg during severe postoperative pain but no acute target-organ injury. Which response is most appropriate?

A. Give a PRN intravenous antihypertensive for postoperative systolic readings above 180 mm Hg because the number alone confers immediate stroke risk

B. Transfer to intensive care because repeated pressure near 190/100 mm Hg meets a numeric definition of hypertensive emergency

C. Attribute the elevation to pain and defer repeat measurement until the pain has completely resolved

D. Treat reversible contributors such as pain and remeasure carefully rather than reflexively giving intravenous antihypertensive medication

198. A patient with pheochromocytoma is preparing for adrenalectomy and remains hypertensive. Which sequencing principle is essential?

A. Start beta blockade first to control tachycardia, then add alpha blockade after the heart rate is controlled

B. Use labetalol alone preoperatively because combined alpha- and beta-receptor blockade provides adequate protection without establishing alpha blockade separately

C. Establish adequate alpha-adrenergic blockade before adding beta blockade for persistent tachycardia

D. Maintain sodium restriction and avoid preoperative volume expansion because catecholamine-associated hypertension implies volume excess

199. A patient with known baroreflex failure develops extreme blood pressure lability with hypertensive surges triggered by emotional stress. Which statement best distinguishes this syndrome from ordinary essential hypertension?

A. Baroreflex failure produces a fixed low blood pressure with no sympathetic surges

B. The disorder is caused by excess aldosterone in every case

C. Loss of afferent baroreflex buffering can produce dramatic stress-triggered pressure and heart-rate swings

D. Emotional stress cannot alter pressure when the carotid baroreflex is impaired

200. A patient with recurrent syncope has autonomic failure and neurogenic orthostatic hypotension. Why is renal sympathetic denervation generally a poor choice for coexisting hypertension in this patient?

A. Neurogenic orthostatic hypotension is a contraindication because further sympathetic reduction may worsen upright hypotension

B. Use renal denervation because reducing supine hypertension is expected to improve neurogenic orthostatic symptoms as well

C. Avoid renal denervation because the procedure increases renal sympathetic tone and can worsen hypertensive surges

D. Consider autonomic failure irrelevant to candidacy as long as renal artery anatomy is technically suitable

Answers

1. C. The arterial baroreflex is the fastest major homeostatic mechanism for short-term blood pressure stabilization. A sudden fall in arterial pressure reduces stretch and firing of carotid sinus and aortic arch baroreceptors, which increases sympathetic tone and decreases parasympathetic tone. The resulting tachycardia, increased contractility, and arteriolar and venous constriction restore pressure within seconds. Renal and natriuretic mechanisms act much more slowly.

2. C. Juxtaglomerular cells release renin in response to beta-1 adrenergic stimulation, reduced afferent arteriolar pressure, and reduced sodium chloride delivery to the macula densa. Increased renal perfusion and increased macula densa sodium chloride suppress renin. Alpha-1 signaling contributes to vasoconstriction but is not the principal direct adrenergic trigger for renin release.

3. B. Angiotensin II preferentially constricts the efferent arteriole at physiologic levels, helping maintain glomerular capillary pressure when renal perfusion falls. Excessive angiotensin II can eventually reduce renal blood flow substantially, but the classic compensatory effect is efferent arteriolar constriction. Efferent dilation would lower intraglomerular pressure.

4. A. Aldosterone acts on mineralocorticoid receptors in principal cells to increase epithelial sodium channel activity at the apical membrane and Na/K-ATPase activity basolaterally. This promotes sodium retention and enhances the electrochemical gradient for potassium secretion. NKCC2 and NCC are targets of loop and thiazide-type diuretics, respectively, rather than the principal aldosterone-regulated apical channel.

5. D. Atrial stretch stimulates atrial natriuretic peptide release. ANP promotes natriuresis, increases glomerular filtration through renal hemodynamic effects, and suppresses renin and aldosterone. Volume expansion ordinarily suppresses renin, aldosterone, and vasopressin rather than increasing them.

6. A. Pressure natriuresis describes the increase in renal sodium excretion that accompanies a rise in arterial pressure. In salt-sensitive hypertension, the pressure-natriuresis curve is shifted so that a higher pressure is required to excrete a given sodium load. Renin is generally suppressed during sodium loading, and lower renal perfusion tends to conserve rather than waste sodium.

7. B. Endothelial nitric oxide diffuses into vascular smooth muscle and activates soluble guanylate cyclase, increasing cyclic GMP and promoting relaxation. Impaired nitric oxide bioavailability contributes to endothelial dysfunction and increased vascular tone. Endothelin and increased intracellular calcium have the opposite effect.

8. A. Endothelin-1 is one of the most potent endogenous vasoconstrictors and increases vascular smooth-muscle tone through endothelin receptor signaling. Endothelial signaling can be complex, but the dominant pressor effect relevant to hypertension is vasoconstriction and vascular remodeling. Nitric oxide and potassium-channel opening generally favor vasodilation.

9. B. Alpha-1 receptor activation causes vascular smooth-muscle contraction, raising systemic vascular resistance and arterial pressure. Venous constriction can also increase venous return. Direct slowing of the sinus node is not an alpha-1 effect; reflex bradycardia may occur secondarily when pressure rises.

10. D. Beta-1 receptors on juxtaglomerular cells promote renin release. Beta-1 blockade therefore reduces renin secretion and downstream angiotensin II and aldosterone signaling, in addition to its cardiac effects. ENaC blockade is produced by amiloride-like drugs, whereas NCC inhibition is the mechanism of thiazide-type diuretics.

11. A. With aging, fragmentation of elastin, collagen deposition, calcification, and other structural changes reduce large-artery compliance. The stiff aorta accommodates stroke volume poorly, increasing systolic pressure, while faster wave reflection can lower or fail to preserve diastolic pressure. The result is widened pulse pressure and isolated systolic hypertension.

12. C. Pulse wave velocity rises as arterial walls become stiffer, making carotid-femoral pulse wave velocity a widely used measure of central arterial stiffness. Increased stiffness causes the pressure wave to travel more rapidly and contributes to earlier wave reflection and systolic pressure augmentation. It is not simply a measure of sympathetic tone or ejection velocity.

13. B. Salt-sensitive hypertension often reflects impaired renal ability to excrete sodium at normal pressure, producing volume expansion and suppression of renin. High sodium intake increases macula densa chloride delivery and renal perfusion, both of which also suppress renin. Thus a low-renin phenotype can accompany volume-dependent hypertension.

14. C. Obesity can increase sympathetic nervous system activity through several mechanisms, including leptin signaling, sleep-disordered breathing, insulin resistance, and altered central pathways. Obesity also promotes renal sodium retention and activation of the renin-angiotensin-aldosterone system. Adiponectin generally has metabolically favorable effects and is not the dominant pressor mediator described here.

15. A. Insulin resistance and compensatory hyperinsulinemia are associated with sympathetic activation, altered vascular function, and increased renal sodium reabsorption. These effects can contribute to higher blood pressure in metabolic syndrome. They do not act by blocking angiotensin receptors or producing chronic natriuresis.

16. A. Renal autoregulation uses afferent vasodilation when perfusion falls, while angiotensin II supports filtration pressure by preferential efferent constriction. This combination helps preserve glomerular capillary pressure. Blocking the renin-angiotensin system in severe bilateral renovascular disease can therefore cause a substantial fall in GFR.

17. C. The macula densa senses distal tubular sodium chloride delivery. Increased delivery signals that filtration is relatively high and leads to afferent arteriolar constriction while suppressing renin. Reduced sodium chloride delivery has the opposite effect, promoting afferent dilation and renin release.

18. D. Thiazide and thiazide-like diuretics inhibit the sodium-chloride cotransporter, NCC, in the distal convoluted tubule. Loop diuretics inhibit NKCC2, while amiloride and triamterene inhibit ENaC. SGLT2 inhibitors act in the proximal tubule and are not thiazide-type antihypertensive agents.

19. C. Loop diuretics inhibit NKCC2 in the thick ascending limb, a nephron segment responsible for substantial sodium chloride reabsorption and generation of the medullary concentration gradient. Their strong natriuretic effect makes them useful when volume overload accompanies reduced kidney function. Thiazides target NCC and potassium-sparing epithelial channel blockers target ENaC.

20. D. Liddle syndrome results from gain-of-function of ENaC, producing avid distal sodium reabsorption, volume expansion, potassium wasting, and metabolic alkalosis. Because the sodium retention is autonomous, both renin and aldosterone are suppressed. ENaC loss of function causes the opposite salt-wasting, hyperkalemic phenotype.

21. D. Glycyrrhetinic acid in natural licorice inhibits renal 11-beta-hydroxysteroid dehydrogenase type 2, which normally converts cortisol to cortisone in mineralocorticoid-sensitive tissues. Cortisol can then activate mineralocorticoid receptors, producing an apparent mineralocorticoid excess phenotype with hypertension, hypokalemia, low renin, and low aldosterone.

22. A. Catecholamine excess from pheochromocytoma or paraganglioma can strongly stimulate alpha-1 receptors, producing arteriolar vasoconstriction and abrupt increases in systemic vascular resistance. Beta-1 stimulation contributes tachycardia and increased contractility. Beta-2 vasodilation can occur in some vascular beds but does not explain the classic pressor episodes.

23. B. Mineralocorticoid receptors bind cortisol and aldosterone with similar affinity. In aldosterone-sensitive epithelial tissues, 11-beta-hydroxysteroid dehydrogenase type 2 converts cortisol to cortisone, which has little mineralocorticoid activity, allowing aldosterone to signal selectively. Inhibition or deficiency of this enzyme produces apparent mineralocorticoid excess.

24. A. Familial hyperaldosteronism type I, also called glucocorticoid-remediable aldosteronism, is caused by a chimeric CYP11B1/CYP11B2 gene that places aldosterone synthase activity under ACTH regulation. Aldosterone is elevated and renin is suppressed. Liddle syndrome and apparent mineralocorticoid excess instead feature low aldosterone.

25. B. Systemic hypertension increases the pressure the left ventricle must generate to eject blood, increasing wall stress and afterload. The ventricle adapts by developing concentric hypertrophy. This initially normalizes wall stress but eventually contributes to diastolic dysfunction, ischemia, arrhythmia risk, and heart failure.

26. B. Hypertension is associated with oxidative stress, inflammation, endothelial dysfunction, and reduced nitric oxide bioavailability. These changes favor vasoconstriction, smooth-muscle growth, collagen deposition, and vascular remodeling, which can perpetuate elevated resistance. Enhanced nitric oxide and reduced endothelin would generally oppose this process.

27. C. Normal blood pressure generally falls during sleep. Nondipping or reverse dipping is associated with conditions such as sleep apnea, CKD, diabetes, autonomic dysfunction, and volume excess and is linked to greater target-organ risk. Ambulatory monitoring uniquely characterizes this circadian pattern.

28. B. Chronic hypertension causes remodeling of small arteries and arterioles, including increased wall-to-lumen or media-to-lumen ratio. A smaller effective lumen for a given wall mass increases resistance and amplifies responses to vasoconstrictor stimuli. Structural remodeling therefore helps hypertension become self-sustaining.

29. C. Pulse pressure depends importantly on stroke volume and arterial compliance. When the arterial system is stiff, a given stroke volume produces a greater systolic pressure rise because less energy is buffered by elastic expansion of the aorta. This is a key mechanism of isolated systolic hypertension in older adults.

30. B. ACE, also known as kininase II, degrades bradykinin. ACE inhibition therefore increases bradykinin, contributing to vasodilation but also to cough and, rarely, angioedema. Angiotensin receptor blockers do not inhibit bradykinin breakdown, explaining their much lower incidence of cough.

31. C. An undersized cuff can substantially overestimate blood pressure in a patient with a large arm. Accurate office measurement requires a validated device, correct cuff size, quiet rest, supported back and feet, and the arm supported near heart level. A cuff that is too large tends to underestimate rather than exaggerate pressure, while the other choices describe proper technique.

32. A. Recent exertion, talking, unsupported posture, crossed legs, and improper cuff size can all distort office blood pressure. A standardized measurement begins after quiet seated rest, with back and feet supported and the arm supported at heart level. Repeating immediately after exertion or using a small cuff can produce falsely high values.

33. A. An auscultatory gap is a transient disappearance of Korotkoff sounds during cuff deflation, classically in older patients with arterial stiffness. If the cuff is not first inflated above a palpated systolic pressure, the examiner may mistake the reappearance of sounds for the true systolic pressure and underestimate systolic BP. Palpating the systolic level before auscultation helps avoid this error.

34. D. Orthostatic hypotension is conventionally defined by a sustained fall in systolic BP of at least 20 mm Hg or diastolic BP of at least 10 mm Hg within three minutes of standing. This patient has a 24-mm Hg systolic decline with compatible symptoms. The finding is distinct from white-coat hypertension and should prompt review of volume status, medications, and autonomic function.

35. A. Home BP monitoring is most useful when measurements are standardized and repeated over several days. A validated upper-arm cuff is preferred, and readings should be taken while seated after rest, commonly in duplicate in the morning and evening. Selective measurement only during symptoms or after pressor stimuli can bias the estimate of usual BP.

36. C. White-coat hypertension refers to elevated office BP with normal out-of-office BP in an untreated patient. Masked hypertension is the opposite pattern. The terms white-coat effect and masked uncontrolled hypertension are generally used in patients already receiving antihypertensive therapy.

37. A. Masked hypertension is present when office BP is not high but out-of-office BP is elevated in a person not taking antihypertensive medication. It can be associated with target-organ damage and increased cardiovascular risk. ABPM or structured home monitoring is therefore useful when office readings appear normal despite clinical clues suggesting excess BP exposure.

38. C. In treated patients, elevated office BP with normal out-of-office BP is termed a white-coat effect. Confirming this pattern is particularly important in apparent resistant hypertension because unnecessary intensification can cause hypotension outside the office. Home BP monitoring is practical for repeated assessment; ABPM can be used when confirmation remains uncertain.

39. B. Blood pressure normally falls during sleep. Here the sleep systolic pressure is only minimally lower than the awake value, indicating a nondipping pattern. Nondipping is common in CKD, diabetes, sleep apnea, and autonomic disorders and is associated with higher cardiovascular and renal risk.

40. A. Stage 1 hypertension is defined by an average systolic BP of 130 to 139 mm Hg or diastolic BP of 80 to 89 mm Hg. Elevated BP is systolic 120 to 129 mm Hg with diastolic below 80 mm Hg. Stage 2 hypertension begins at 140 mm Hg systolic or 90 mm Hg diastolic.

41. B. Elevated BP is defined by systolic pressure of 120 to 129 mm Hg with diastolic pressure below 80 mm Hg. Normal BP is below 120/80 mm Hg, whereas stage 1 begins at 130 systolic or 80 diastolic. This category identifies patients for lifestyle intervention and longitudinal monitoring rather than automatic drug therapy.

42. B. Stage 2 hypertension is present when average systolic BP is at least 140 mm Hg or diastolic BP is at least 90 mm Hg. Classification is based on the higher category when systolic and diastolic values fall into different ranges. This patient qualifies by the systolic value despite a diastolic value in the stage 1 range.

43. C. Routine evaluation of hypertension includes assessment for kidney disease, diabetes, dyslipidemia, electrolyte abnormalities, and target-organ effects. Serum electrolytes and creatinine plus urinalysis and urine albumin measurement help identify CKD and clues such as hypokalemia. Specialized testing for secondary causes should be targeted to clinical suspicion rather than performed universally.

44. D. Chronic systemic hypertension increases left ventricular afterload and can produce concentric left ventricular hypertrophy. ECG may show increased voltage and secondary repolarization abnormalities, although echocardiography is more sensitive. LVH identifies hypertension-mediated target-organ damage and higher cardiovascular risk.

45. C. Retinal hemorrhages, cotton-wool spots, exudates, and especially papilledema or optic disc swelling indicate severe hypertensive retinal injury. In the setting of markedly elevated BP, these findings support a hypertensive emergency rather than uncomplicated asymptomatic hypertension. They warrant prompt evaluation for injury in other organs as well.

46. A. Albuminuria is an important marker of kidney damage and cardiovascular risk even when estimated GFR is preserved. Hypertension and diabetes can both cause glomerular injury that appears first as increased urinary albumin excretion. The finding also influences antihypertensive drug selection, particularly favoring renin-angiotensin system blockade when clinically appropriate.

47. A. An ankle-brachial index below approximately 0.90 supports peripheral artery disease, and a value of 0.62 is clearly abnormal. Hypertension is a major risk factor for atherosclerotic vascular disease. Identifying PAD changes overall cardiovascular risk assessment and reinforces the importance of comprehensive risk-factor management.

48. B. Secondary hypertension should be considered when hypertension has an unusual presentation: abrupt onset, severe or resistant hypertension, onset at a young age, unexplained hypokalemia, renal dysfunction, or suggestive symptoms and examination findings. In a young woman with an abdominal bruit, renovascular disease from fibromuscular dysplasia is an important possibility.

49. B. Irregular rhythms can increase variability in automated and auscultatory BP measurements. Repeated standardized measurements and averaging improve the estimate of usual pressure. A single extreme reading is especially vulnerable to error and should not be used alone to diagnose or titrate chronic hypertension.

50. C. Apparent resistant hypertension can reflect pseudoresistance from inaccurate measurement, a white-coat effect, medication nonadherence, or interfering substances. True resistant hypertension should therefore be confirmed with reliable out-of-office BP and a careful review of technique, adherence, and the regimen. Only after pseudoresistance is excluded should evaluation focus on secondary causes and advanced therapies.

51. B. The 2025 Endocrine Society guideline broadened case detection by suggesting screening for primary aldosteronism in all individuals with hypertension using aldosterone and renin with an aldosterone-to-renin ratio. Hypokalemia, resistant hypertension, sleep apnea, and adrenal incidentaloma remain important clues, but their absence does not exclude PA. The goal is earlier recognition of a common and treatable form of secondary hypertension.

52. D. Primary aldosteronism is characterized by autonomous aldosterone production with suppression of renin. Hypokalemia may occur but is not required. Renovascular disease, renin-secreting tumors, and many diuretic effects activate renin and therefore produce a secondary rather than primary hyperaldosteronism pattern.

53. B. Hypokalemia can suppress aldosterone secretion and potentially produce a falsely reassuring screening result. Potassium should be corrected when feasible before testing, and medication effects and sodium intake should be considered in interpretation. The screening goal is to identify inappropriately autonomous aldosterone relative to renin under clinically interpretable conditions.

54. B. Mineralocorticoid receptor antagonists can substantially raise renin and alter aldosterone dynamics, complicating ARR interpretation. When clinically safe and diagnostically necessary, they may need to be withdrawn for an appropriate interval before testing. Medication adjustment must be individualized because uncontrolled hypertension and hypokalemia also carry risk.

55. D. When screening indicates an intermediate probability of lateralizing primary aldosteronism and the patient would pursue surgery, an aldosterone suppression test can help confirm autonomous secretion. Contemporary guidance allows some high-probability cases to bypass suppression testing, but it remains useful in intermediate cases. Normal potassium does not exclude PA.

56. A. Adrenal CT defines anatomy but cannot reliably establish functional laterality because nonfunctioning adrenal nodules are common and bilateral or microscopic disease may be missed. Adrenal venous sampling is the reference method for lateralizing aldosterone secretion in most surgical candidates. This distinction determines whether unilateral adrenalectomy is appropriate.

57. A. Lateralized primary aldosteronism is treated with unilateral adrenalectomy when surgery is desired and appropriate. This can normalize potassium and substantially improve or cure hypertension while reducing aldosterone-mediated cardiovascular risk. Bilateral adrenalectomy is not standard treatment for unilateral PA and would create adrenal insufficiency.

58. D. Bilateral primary aldosteronism is generally treated medically with mineralocorticoid receptor antagonism. Spironolactone is commonly preferred because of availability and cost, while eplerenone may be chosen when antiandrogenic adverse effects are problematic. Disease-specific blockade treats both blood pressure and the pathophysiologic consequences of aldosterone excess.

59. D. Eplerenone is a more selective mineralocorticoid receptor antagonist and produces fewer androgen- and progesterone-receptor adverse effects than spironolactone. It may require different dosing and can be more expensive, but it preserves disease-specific aldosterone blockade. Hydralazine, clonidine, and doxazosin lower BP without directly antagonizing aldosterone.

60. A. Familial hyperaldosteronism type I is caused by ACTH-regulated aldosterone synthase activity. Low-dose glucocorticoid therapy can suppress ACTH and reduce aldosterone production; mineralocorticoid receptor antagonists may also be used when needed. This mechanism distinguishes the disorder from Liddle syndrome and renovascular hypertension.

61. C. Catecholamine-secreting tumors continuously metabolize catecholamines to metanephrines, making plasma free or fractionated urinary metanephrines sensitive screening tests. Testing conditions

and pretest probability matter because stress and medications can produce false positives. Aldosterone, albuminuria, and a single random cortisol do not diagnose pheochromocytoma.

62. C. Mild metanephrine elevations can result from sympathetic activation, posture, stress, medications, and collection conditions. Repeating plasma testing after appropriate rest, often supine, can improve specificity when the initial result is borderline. Marked elevations in a compatible clinical setting are more concerning, but adrenal venous sampling is not the diagnostic test for pheochromocytoma.

63. D. Preoperative treatment traditionally begins with alpha blockade to control catecholamine-mediated vasoconstriction and permit volume re-expansion. Beta blockade may be added afterward if tachycardia persists. Giving a beta blocker before adequate alpha blockade can leave alpha-mediated vasoconstriction unopposed and precipitate severe hypertension.

64. C. In catecholamine excess, beta blockade before alpha blockade can remove beta-2 vasodilatory effects while alpha-mediated vasoconstriction continues, producing an abrupt rise in systemic vascular resistance. This is why alpha blockade must be established first when both classes are needed.

65. C. Pheochromocytomas and paragangliomas can occur in hereditary syndromes involving genes such as RET, VHL, NF1, and SDHx. Genetic evaluation is often appropriate, particularly with paraganglioma, young age, multifocal disease, or family history. Results can influence surveillance and testing of relatives.

66. D. Cushing syndrome should be screened with a validated test such as the overnight dexamethasone suppression test, late-night salivary cortisol, or 24-hour urinary free cortisol, selected for the clinical setting. Random cortisol values are not reliable screening tests because cortisol varies diurnally and with stress.

67. C. Thyroid hormone excess increases cardiac output through increased heart rate, contractility, and metabolic demand, often producing systolic hypertension and widened pulse pressure. Hypothyroidism is more often associated with increased systemic vascular resistance and diastolic hypertension. The clinical symptoms strongly support thyrotoxicosis.

68. A. Hypothyroidism can increase systemic vascular resistance and is classically associated with diastolic hypertension. The accompanying cold intolerance, constipation, weight gain, and bradycardia support the diagnosis. Hyperthyroidism more often produces tachycardia and systolic hypertension.

69. B. Primary hyperparathyroidism is associated with hypercalcemia and may coexist with hypertension, while nephrolithiasis and constipation are classic clues. The causal relation between parathyroid disease and BP is less direct than in primary aldosteronism, but the biochemical phenotype warrants evaluation. Hypoparathyroidism produces hypocalcemia rather than hypercalcemia.

70. D. IGF-1 is the preferred initial biochemical test for suspected acromegaly because it integrates growth hormone secretion over time. Random growth hormone is unreliable because secretion is

pulsatile. Acromegaly can contribute to hypertension through sodium retention, vascular effects, sleep apnea, and cardiometabolic disease.

71. A. Renal parenchymal disease is among the most common causes of secondary hypertension. Hematuria, proteinuria, reduced GFR, and edema point directly to intrinsic kidney disease, which can raise BP through sodium retention, volume expansion, and renin-angiotensin activation. Kidney evaluation should therefore precede exotic endocrine testing.

72. C. Recurrent flash pulmonary edema, abrupt worsening of hypertension, and diffuse atherosclerotic disease are classic clues to hemodynamically important renal artery stenosis. Atherosclerotic renovascular disease is especially common in older patients with vascular disease. The finding warrants targeted renal artery evaluation and assessment of kidney function.

73. D. Fibromuscular dysplasia is a nonatherosclerotic, noninflammatory arteriopathy that often affects younger women and classically involves the mid-to-distal renal artery with a string-of-beads appearance. Atherosclerotic stenosis more often involves the ostium or proximal renal artery in older patients with vascular risk factors.

74. C. Atherosclerotic renal artery stenosis typically affects older patients with diffuse vascular disease and commonly involves the ostium or proximal renal artery. Fibromuscular dysplasia occurs in a different demographic and distribution. The angiographic pattern and clinical context strongly favor atherosclerosis.

75. C. When renal perfusion is critically reduced, angiotensin II-mediated efferent arteriolar constriction helps maintain intraglomerular pressure. ACE inhibition removes this support and can sharply reduce GFR in bilateral severe renal artery stenosis or stenosis to a solitary functioning kidney. A modest creatinine rise can occur with routine therapy, but a large abrupt rise warrants evaluation.

76. A. Renal duplex Doppler ultrasonography is a noninvasive option for evaluating renal artery stenosis when local expertise and patient anatomy permit. CT angiography and MR angiography are alternatives depending on kidney function and other factors. Adrenal venous sampling is used for primary aldosteronism, not renovascular disease.

77. C. Most patients with atherosclerotic renal artery stenosis are managed initially with intensive medical therapy, including BP and atherosclerotic risk-factor control. Routine stenting has not shown broad benefit over medical therapy in stable patients. Revascularization is considered selectively, such as with recurrent flash pulmonary edema, refractory hypertension, or progressive renal dysfunction in an appropriate anatomy.

78. D. Recurrent flash pulmonary edema is a high-risk clinical syndrome associated with hemodynamically significant renal artery disease and is one of the situations in which revascularization may be considered. Stable incidental stenosis without resistant hypertension, pulmonary edema, or progressive renal dysfunction usually favors medical management.

79. D. Coarctation of the aorta produces higher pressure proximal to the obstruction and lower pressure in the legs, with diminished or delayed femoral pulses. A systolic murmur over the back may also be present. Measuring blood pressure in both arms and at least one leg is important when coarctation is suspected.

80. A. Obstructive sleep apnea is common in patients with resistant hypertension and should be suspected with obesity, loud snoring, witnessed apneas, and daytime sleepiness. Sleep testing establishes the diagnosis and severity. OSA can raise BP through sympathetic activation, intermittent hypoxemia, oxidative stress, and altered fluid distribution.

81. B. Treating OSA with CPAP improves apnea-related symptoms and can lower BP modestly, with greater effects in some patients with severe disease or good adherence. It should not be presented as a guaranteed cure for hypertension. Weight management, lifestyle measures, and appropriate antihypertensive medications remain important.

82. C. Repeated upper-airway obstruction causes intermittent hypoxemia, hypercapnia, arousals, and large intrathoracic pressure swings. These activate sympathetic and neurohumoral pathways and impair vascular function, contributing to sustained hypertension and nondipping. OSA often coexists with obesity and resistant hypertension.

83. B. Systemic NSAIDs can raise BP by reducing renal prostaglandin-mediated vasodilation, promoting sodium retention, and blunting the effects of several antihypertensive classes. They are a common reversible contributor to apparent resistant hypertension. Alternatives or the lowest feasible exposure should be considered when clinically appropriate.

84. C. Pseudoephedrine and related decongestants are sympathomimetics that can increase vascular tone and BP, particularly in patients with severe or uncontrolled hypertension. Medication reconciliation should include nonprescription products. Intranasal saline, topical corticosteroids, or other alternatives may avoid the systemic pressor effect when appropriate.

85. B. Estrogen-containing oral contraceptives can raise BP in susceptible patients. When hypertension is uncontrolled or the temporal relation is strong, an alternative contraceptive strategy can be considered while balancing reproductive goals and other risks. ACE inhibitors are contraindicated in pregnancy and do not solve the underlying medication contribution.

86. D. Calcineurin inhibitors such as cyclosporine and tacrolimus can cause hypertension through renal vasoconstriction, sodium retention, sympathetic effects, and endothelial pathways. The problem is common after transplantation and may require adjustment of immunosuppression when feasible plus antihypertensive therapy.

87. D. Erythropoiesis-stimulating agents are recognized medication-related contributors to hypertension, particularly in CKD. Mechanisms may include increased vascular resistance and changes associated

with correction of anemia. The medication history should be reviewed alongside the underlying kidney disease and volume status.

88. B. Excess alcohol intake can raise BP and contribute to resistant hypertension. Reducing intake can lower BP and also improves other health risks. Contemporary guidance generally advises no more than one drink daily for women and two for men if alcohol is consumed, while some patients benefit from abstinence.

89. C. Natural licorice contains glycyrrhetinic compounds that inhibit 11-beta-HSD2, allowing cortisol to activate renal mineralocorticoid receptors. The resulting sodium retention and potassium wasting suppress both renin and aldosterone. This biochemical pattern distinguishes the syndrome from primary and secondary hyperaldosteronism.

90. B. Cocaine and other potent sympathomimetics can cause abrupt severe hypertension, tachycardia, coronary vasoconstriction, ischemia, and neurologic complications. A careful history and, when appropriate, toxicology testing are important in unexplained hypertensive crises. The other agents are not classic acute sympathomimetic causes of this presentation.

91. C. The DASH dietary pattern lowers blood pressure and emphasizes fruits, vegetables, whole grains, legumes, nuts, and low-fat dairy while limiting saturated fat and highly processed foods. Its BP effect is enhanced by reducing sodium. Lifestyle treatment is recommended across the spectrum of elevated BP and hypertension, including when medication is also indicated.

92. A. Reducing dietary sodium lowers BP, with larger effects in many older, Black, CKD, and salt-sensitive patients. A practical strategy is to reduce processed and restaurant foods, read labels, and use non-sodium flavoring. The benefit often complements a DASH-style diet and medication therapy.

93. D. Higher dietary potassium intake can lower BP and partially counter sodium's pressor effects. The safest approach is usually potassium-rich foods, but increased potassium is not appropriate for everyone. CKD, hyperkalemia, or drugs that markedly reduce potassium excretion require individualized counseling.

94. A. Weight loss is one of the most effective lifestyle interventions for patients with overweight or obesity and hypertension. BP often falls progressively as weight decreases because of improvements in sympathetic activity, insulin resistance, sodium handling, sleep apnea, and vascular function. Medication doses may need reassessment as control improves.

95. D. Regular aerobic exercise lowers resting BP and reduces cardiovascular risk; dynamic resistance exercise can provide additional benefit. Programs should be tailored to fitness, comorbidity, and symptom status. Severe uncontrolled hypertension or unstable cardiovascular disease may require stabilization before vigorous exercise, but routine activity is a core treatment strategy.

96. C. Excess alcohol raises blood pressure and can contribute to resistant hypertension, arrhythmias, and medication nonadherence. Reducing intake can lower BP. If alcohol is consumed, contemporary

hypertension guidance recommends limiting intake, generally to no more than two drinks daily for men and one for women, while abstinence is appropriate for many patients.

97. C. Tobacco use acutely activates sympathetic pathways and is a major independent driver of cardiovascular morbidity and mortality. Even if the sustained clinic BP reduction from cessation is not the dominant effect, stopping tobacco substantially reduces total cardiovascular risk and should be part of comprehensive hypertension care.

98. A. Psychosocial stress and poor sleep can worsen BP through sympathetic and behavioral pathways, and sleep apnea is a common secondary contributor. Stress-management and healthy sleep practices can complement dietary, activity, and pharmacologic treatment. They should not replace indicated BP measurement, risk assessment, or medication.

99. B. For lower-risk stage 1 hypertension, the 2025 guideline supports a period of guideline-directed lifestyle therapy, with medication added if BP remains at or above 130/80 mm Hg after roughly three to six months. This approach balances absolute cardiovascular benefit with treatment burden while still addressing lifetime risk.

100. A. The 2025 hypertension guideline recommends drug treatment at a lower threshold for stage 1 hypertension when cardiovascular risk is increased, including diabetes, CKD, established CVD, or PREVENT 10-year CVD risk at least 7.5%. Lifestyle therapy remains important but is combined with medication rather than used alone in these higher-risk groups.

101. B. The 2025 guideline uses PREVENT total CVD risk to guide therapy in stage 1 hypertension. A 10-year risk of at least 7.5% identifies a group for whom medication plus lifestyle therapy is recommended at an average BP of 130/80 mm Hg or higher. The risk threshold replaces the prior reliance on the older pooled cohort equation approach.

102. C. Stage 2 hypertension generally requires two complementary first-line agents, and the 2025 guideline prefers a fixed-dose single-pill combination when feasible to improve adherence and speed control. Combining an ACE inhibitor with an ARB is specifically avoided because dual renin-angiotensin blockade increases kidney injury and hyperkalemia without added outcome benefit.

103. B. The 2025 ACC/AHA multisociety guideline generally recommends a treatment goal below 130/80 mm Hg for adults with hypertension, while clinical judgment remains important in patients with symptomatic hypotension, frailty, limited life expectancy, or other special circumstances. Accurate measurement and treatment tolerance are essential when interpreting any target.

104. B. First-line classes for uncomplicated primary hypertension include thiazide-type diuretics, ACE inhibitors or ARBs, and long-acting dihydropyridine calcium channel blockers. Alpha blockers, central sympatholytics, and direct vasodilators have important roles in selected patients but are not preferred universal first-line agents because of tolerability and outcome considerations.

105. D. Chlorthalidone is a long-acting thiazide-like diuretic with extensive outcome data and potent 24-hour BP lowering. Indapamide is another thiazide-like option. Mannitol and acetazolamide act at different nephron sites, while triamterene is a potassium-sparing ENaC blocker rather than a thiazide-type drug.

106. B. Dihydropyridine calcium channel blockers dilate precapillary arterioles more than postcapillary venules, increasing capillary hydrostatic pressure and causing dependent edema. The edema is not primarily due to total-body sodium overload. Combining a dihydropyridine CCB with an ACE inhibitor or ARB can reduce this adverse effect in some patients.

107. C. ACE inhibition reduces bradykinin degradation, increasing bradykinin and related peptides that can trigger cough. The same pathway contributes to ACE inhibitor-associated angioedema. An ARB is often used when ACE inhibitor cough is troublesome because ARBs do not inhibit bradykinin breakdown.

108. B. ACE inhibitor-associated angioedema can threaten the airway and is mediated by bradykinin rather than histamine. The ACE inhibitor should be permanently discontinued. Airway management is the immediate clinical priority during an acute episode; future renin-angiotensin therapy requires individualized consideration, with ARBs carrying a much lower but not zero angioedema risk.

109. A. ACE inhibitors and ARBs reduce aldosterone signaling and can increase serum potassium, particularly in CKD, diabetes, or when combined with potassium supplements or potassium-sparing drugs. Kidney function should also be monitored after initiation or dose escalation. A modest creatinine increase can be expected, but a large rise warrants evaluation for volume depletion, NSAID exposure, or renovascular disease.

110. B. Dual blockade with an ACE inhibitor and ARB increases renal adverse events and hyperkalemia and does not provide enough outcome benefit to justify routine combination. The same principle applies to combining these drugs with direct renin inhibition in many settings. Complementary classes with different mechanisms are preferred when more than one drug is needed.

111. A. ACE inhibitors, ARBs, direct renin inhibitors, mineralocorticoid receptor antagonists, and certain other agents are avoided in pregnancy because of fetal risk. Labetalol and long-acting nifedipine are commonly used, with methyldopa an additional option. Medication review should occur before conception whenever possible.

112. A. Thiazide-type diuretics increase distal sodium delivery and can promote potassium and hydrogen ion loss, causing hypokalemia and metabolic alkalosis. Severe hypokalemia can produce weakness and arrhythmias. Monitoring is particularly important after initiation, dose changes, or when other potassium-lowering factors are present.

113. C. Thiazide-associated hyponatremia can be severe, especially in older adults, patients with low solute intake, and those taking other drugs that impair water excretion. Neurologic symptoms require

prompt assessment and careful correction. Electrolyte monitoring after starting or intensifying a diuretic helps identify this complication early.

114. D. Thiazide-type diuretics can reduce renal urate excretion and increase serum uric acid, occasionally precipitating gout. This does not absolutely prohibit their use, but the history should influence drug selection and urate management. Losartan can modestly lower uric acid and may be useful when otherwise appropriate.

115. D. Loop diuretics retain substantial natriuretic efficacy as kidney function declines and are especially useful when hypertension is accompanied by edema or volume overload. Thiazide-like drugs can still contribute to BP control in CKD, sometimes in combination, but loop therapy is often central for significant volume excess.

116. A. Amlodipine is a dihydropyridine calcium channel blocker that primarily acts on vascular smooth muscle and usually does not produce clinically important bradycardia. Verapamil and diltiazem slow atrioventricular nodal conduction, as do beta blockers such as metoprolol. In a patient with baseline bradycardia, the latter agents require caution.

117. B. Verapamil and diltiazem are nondihydropyridine calcium channel blockers with substantial cardiac electrophysiologic effects. They slow sinus rate and AV nodal conduction and can worsen bradycardia or heart block, especially when combined with beta blockers. Dihydropyridines such as amlodipine have much less direct nodal effect.

118. A. Beta blockers are valuable when specific indications exist, such as ischemic heart disease, certain arrhythmias, and heart failure with reduced ejection fraction. For uncomplicated primary hypertension, first-line therapy more commonly uses thiazide-type diuretics, ACE inhibitors or ARBs, and long-acting dihydropyridine CCBs. Treatment should be individualized rather than class-based dogma.

119. A. Alpha-1 blockers can improve urinary outflow in benign prostatic hyperplasia and lower BP, but they commonly cause orthostatic symptoms and are not preferred as sole first-line hypertension therapy. They may be useful as add-on agents when another indication is present. Their adverse-effect profile differs from ACE inhibitors, MRAs, and nodal blockers.

120. C. Chronic central alpha-2 agonist therapy suppresses sympathetic outflow. Abrupt withdrawal can produce a rebound catecholamine surge with severe hypertension and tachycardia. Gradual tapering reduces this risk. Special caution is needed when clonidine is combined with beta blockers because withdrawal sequencing can affect adrenergic balance.

121. C. Hydralazine can cause a dose- and exposure-related drug-induced lupus syndrome, particularly in susceptible slow acetylators. Symptoms often include arthralgias, myalgias, fever, and serologic abnormalities. Hydralazine is a direct vasodilator generally reserved for selected settings rather than routine first-line monotherapy.

122. D. Minoxidil is a potent direct arteriolar vasodilator that can cause reflex sympathetic activation and marked sodium and water retention, so it is often paired with a beta blocker and a potent diuretic. Hypertrichosis is a distinctive adverse effect. Because of these limitations, it is reserved for difficult cases.

123. B. Eplerenone is more selective for the mineralocorticoid receptor than spironolactone and therefore causes fewer androgen- and progesterone-receptor adverse effects. Spironolactone is often more potent and less expensive, but eplerenone is an important alternative when endocrine side effects limit adherence.

124. B. Liddle syndrome is caused by gain-of-function epithelial sodium channels in the collecting duct, producing sodium retention with low renin and low aldosterone. Amiloride directly blocks ENaC and is the targeted therapy. Drugs that suppress the renin-angiotensin-aldosterone system are less mechanistically effective because that system is already suppressed.

125. D. After confirming true resistant hypertension and optimizing a complementary three-drug regimen that includes a diuretic, a mineralocorticoid receptor antagonist such as spironolactone is a highly effective next agent for many patients. Potassium and kidney function must be monitored. Duplicating a class or combining ACE inhibitor with ARB adds risk without appropriate benefit.

126. C. Resistant hypertension management begins by ensuring that the foundational regimen is appropriately dosed and mechanistically complementary, commonly an ACE inhibitor or ARB, a long-acting dihydropyridine CCB, and an effective diuretic. Switching from hydrochlorothiazide to a longer-acting thiazide-like agent such as chlorthalidone can improve control in some patients.

127. B. Medication nonadherence is a common cause of apparent resistant hypertension. A respectful discussion should identify cost, adverse effects, complexity, beliefs, and practical barriers. Simplifying the regimen, using once-daily or single-pill combinations, refill synchronization, and team-based support can improve adherence before adding unnecessary therapy.

128. D. Single-pill combinations reduce pill burden, simplify routines, and generally improve adherence and persistence compared with equivalent free-pill combinations. The 2025 guideline prefers a single-pill fixed-dose combination for most patients starting two agents for stage 2 hypertension when clinically appropriate.

129. D. Regimen complexity is a major barrier to adherence. Once-daily drugs and fixed-dose combinations can reduce pill burden and missed doses, while refill synchronization, home BP monitoring, and team-based care can reinforce persistence. The goal is to make the effective regimen easier, not harder, to follow.

130. A. Systemic NSAIDs can increase BP through renal sodium retention and attenuation of the effects of several antihypertensive agents. They are common contributors to loss of control and apparent

resistant hypertension. The medication list should include nonprescription analgesics, and alternatives should be considered when clinically feasible.

131. C. Spironolactone, ARBs, and trimethoprim can each impair potassium excretion, and the combination can produce clinically important hyperkalemia, particularly in older adults or patients with CKD. Potassium and renal function should be monitored and alternative antibiotics considered when appropriate.

132. B. Beta blockers and nondihydropyridine calcium channel blockers both slow sinus and AV nodal conduction and reduce contractility. Combining them can produce symptomatic bradycardia, heart block, or heart failure in susceptible patients. A dihydropyridine such as amlodipine is often a safer CCB choice when additional BP lowering is needed without nodal suppression.

133. A. PDE-5 inhibitors enhance cyclic GMP signaling, and nitrates increase nitric oxide-mediated cyclic GMP production. Their combination can cause severe, potentially life-threatening hypotension. PDE-5 inhibitors can often be used with many other antihypertensives, but nitrates are a major contraindicated combination.

134. D. Patiromer can bind other oral medications in the gastrointestinal tract and reduce their absorption. Separating administration by the recommended interval helps minimize this interaction. Potassium binders can sometimes facilitate continued renin-angiotensin system therapy in patients prone to hyperkalemia.

135. A. ACE inhibitors and ARBs reduce systemic and intraglomerular pressure and are particularly valuable in albuminuric CKD. They can slow progression of kidney disease and reduce proteinuria, although potassium and creatinine require monitoring. Dual ACE inhibitor plus ARB therapy should not be used routinely.

136. B. Diabetes increases cardiovascular risk and lowers the threshold for treating stage 1 hypertension, but it does not mandate a single universal first-line class in the absence of specific comorbidity. ACE inhibitors or ARBs are favored with albuminuric CKD, while thiazide-type diuretics and long-acting dihydropyridine CCBs are also effective options depending on the patient.

137. A. Isolated systolic hypertension reflects large-artery stiffness and responds well to standard first-line agents, particularly thiazide-type diuretics and long-acting dihydropyridine CCBs. Treatment should begin carefully in older adults with attention to orthostatic symptoms and diastolic pressure. Short-acting nifedipine is not appropriate for routine chronic BP control.

138. B. For many Black adults without a compelling indication for renin-angiotensin blockade, thiazide-type diuretics and calcium channel blockers provide strong BP lowering and outcome evidence. Stage 2 hypertension generally warrants two complementary first-line agents. ACE inhibitor or ARB therapy can still be appropriate when CKD with albuminuria, heart failure, or another specific indication is present.

139. B. Losartan has a unique uricosuric effect among commonly used ARBs and can modestly lower serum uric acid. It should not replace standard gout therapy when that is indicated, but the property can influence drug selection in a hypertensive patient with hyperuricemia or gout.

140. D. Thiazide-type diuretics increase distal tubular calcium reabsorption and reduce urinary calcium excretion, which can be useful in selected patients with recurrent calcium stones and hypertension. Loop diuretics increase urinary calcium excretion and are not chosen for this purpose.

141. A. Dihydropyridine calcium channel blockers produce peripheral arterial vasodilation and are commonly used for Raynaud symptoms while also lowering BP. Nonselective beta blockers may worsen peripheral vasospasm in susceptible patients. Comorbid conditions can therefore guide selection among otherwise acceptable antihypertensive classes.

142. C. Certain beta blockers, such as propranolol and metoprolol, are effective migraine-prevention agents and also lower BP. Although beta blockers are not preferred universal first-line therapy for uncomplicated hypertension, a compelling coexisting indication can make them an efficient choice.

143. D. Beta blockers slow AV nodal conduction and can provide both rate control and BP lowering. Nondihydropyridine CCBs can do the same in selected patients, but dihydropyridines such as amlodipine do not provide meaningful rate control. Comorbidity-guided drug selection can reduce the number of medications needed.

144. D. Renin-angiotensin system blockers can reduce the precapillary-postcapillary mismatch that contributes to dihydropyridine CCB edema, decreasing its frequency or severity in some patients. Lowering the CCB dose or switching classes is another option. Simply adding more dihydropyridine therapy often worsens the edema.

145. B. A high office BP with controlled out-of-office BP in a treated patient represents a white-coat effect. Escalating medication solely on the office value can cause hypotension outside the clinic. Resistant hypertension requires confirmation that BP is truly elevated with reliable out-of-office measurement.

146. D. Medication and substance review is central to resistant hypertension management. Sympathomimetic decongestants and systemic NSAIDs can raise BP or blunt antihypertensive response. Removing reversible pressor exposures may restore control and prevents unnecessary escalation to more complex therapies.

147. A. Primary aldosteronism is common among patients with resistant hypertension and carries cardiovascular risk beyond the BP level itself. Screening with aldosterone and renin is therefore high yield, along with evaluation for sleep apnea, renal parenchymal disease, renovascular disease, and medication-related causes. Contemporary endocrine guidance has broadened screening beyond only resistant cases.

148. D. The 2025 guideline incorporates renal denervation as a possible option for selected patients with resistant or otherwise difficult-to-treat stage 2 hypertension after careful evaluation. Candidates should be assessed by a multidisciplinary team, secondary causes and pseudoresistance should be addressed, and expected modest BP lowering and procedural uncertainties should be discussed. It is not a cure or automatic substitute for medication.

149. B. Renal denervation lowers BP modestly on average, with variable individual response. Current guidance emphasizes that it is neither curative nor a complete replacement for pharmacologic and lifestyle therapy. Long-term cardiovascular outcome data are less extensive than for standard antihypertensive medication.

150. C. Severe inpatient BP elevation without acute target-organ damage is not a hypertensive emergency. The 2025 guideline advises against reflex intermittent IV or oral treatment aimed at rapidly lowering BP in such noncardiac hospitalized patients. Measurement error, pain, anxiety, hypoxia, medications, missed chronic therapy, and follow-up needs should be addressed.

151. D. Normal office BP with elevated out-of-office BP in a treated patient is masked uncontrolled hypertension. It is associated with higher risk than truly controlled hypertension. Reliable home monitoring or ABPM can confirm the phenotype and guide safe intensification when appropriate.

152. D. Structured home BP monitoring provides repeated measurements in the patient's usual environment and can improve engagement and control. Clinicians can use averages to assess response and identify white-coat or masked patterns. Single extreme readings should be interpreted in context rather than triggering automatic dose changes.

153. A. Adherence is influenced by cost, pill burden, refill logistics, adverse effects, health literacy, and trust. Matching the regimen to the patient's circumstances, reducing cost, synchronizing refills, and using combination pills can be more effective than simply prescribing additional medication.

154. A. Substantial weight loss can lower BP, and contemporary hypertension guidance recognizes obesity treatment as part of comprehensive risk reduction. Some anti-obesity therapies, including GLP-1-based agents in appropriate patients, can improve cardiometabolic risk and reduce BP. Antihypertensive doses may need reassessment as weight and pressure fall.

155. D. Although stage 2 hypertension usually favors initial combination therapy, selected patients with stage 1 hypertension near goal can begin with monotherapy. This is particularly reasonable in older adults or people prone to hypotension or adverse effects. Follow-up and titration should be based on standardized office and out-of-office readings.

156. B. When BP is substantially above goal, a single drug is unlikely to achieve control. Two complementary first-line agents improve the magnitude and speed of BP reduction, and a fixed-dose single-pill combination can improve adherence. The regimen should avoid redundant or harmful combinations such as ACE inhibitor plus ARB.

157. C. Mineralocorticoid receptor antagonists can cause clinically important hyperkalemia, especially in CKD or with ACE inhibitors, ARBs, potassium supplements, or trimethoprim. Hyperkalemia requires active assessment and management; the treatment plan may include dose reduction, discontinuation, addressing contributors, or use of a potassium binder in selected patients.

158. B. ACE inhibitors and ARBs dilate the efferent arteriole and can cause a modest early increase in creatinine. A small stable change may be acceptable, whereas a larger rise, progressive dysfunction, hypotension, or hyperkalemia should prompt evaluation for volume depletion, NSAID use, renal artery stenosis, or other causes.

159. C. Thiazide-type diuretics can cause hyponatremia, hypokalemia, changes in kidney function, hyperuricemia, and modest metabolic effects. Monitoring is especially important after initiation or dose escalation and in older adults or patients taking interacting medications. Clinical follow-up should also assess volume status and symptoms.

160. C. Blood pressure diagnosis and titration should not rely on a single context. Standardized office technique plus home or ambulatory monitoring can identify a white-coat effect and prevent unnecessary intensification. The objective is sustained control while avoiding treatment-related hypotension.

161. D. Hypertensive emergency is defined by severe blood pressure elevation accompanied by acute target-organ injury, such as encephalopathy, acute kidney injury, myocardial ischemia, pulmonary edema, aortic dissection, or certain cerebrovascular syndromes. The absolute blood pressure is important but does not, by itself, distinguish emergency from severe asymptomatic elevation. Emergencies generally require monitored intravenous treatment tailored to the affected organ.

162. D. In most hypertensive emergencies without a special disease-specific target, overly rapid normalization risks organ hypoperfusion because autoregulation is shifted in chronic hypertension. A commonly used approach is to lower mean arterial pressure by no more than roughly 20% to 25% initially, then proceed more gradually. Aortic syndromes, stroke, pregnancy, and several other conditions require tailored targets.

163. B. Acute aortic syndrome requires immediate anti-impulse therapy. Intravenous beta blockade reduces heart rate and contractility and is generally established before an arterial vasodilator is added, because vasodilation alone can provoke reflex tachycardia and increase shear stress. Guideline targets emphasize heart rate about 60 to 80/min and systolic pressure below 120 mm Hg, or the lowest pressure that preserves organ perfusion.

164. C. Hypertensive acute pulmonary edema is a hypertensive emergency in which rapid afterload and often preload reduction can improve ventricular loading conditions and pulmonary congestion. Titrated intravenous vasodilators such as nitroglycerin or clevidipine may be useful depending on the clinical setting, along with oxygenatory/ventilatory support and diuresis when volume overloaded. Treatment should not be delayed for outpatient confirmation.

165. A. In acute coronary syndrome, hypertension increases myocardial oxygen demand, but excessive reduction—particularly of diastolic pressure—can compromise coronary perfusion. Therapy is individualized and often uses agents that also address ischemia, such as nitrates and selected beta blockers when appropriate. Abrupt, uncontrolled pressure reduction should be avoided.

166. B. For mild-to-moderate spontaneous intracerebral hemorrhage with presenting systolic pressure 150 to 220 mm Hg, smooth sustained reduction toward about 140 mm Hg, with a maintenance range around 130 to 150 mm Hg, is reasonable. Excessive lowering below 130 mm Hg may be harmful. Minimizing blood pressure variability is also important.

167. B. Current acute ischemic stroke guidance uses a blood pressure below 185/110 mm Hg before intravenous thrombolysis, reflecting the thresholds used in reperfusion trials and concern for hemorrhagic complications. Treatment should be controlled rather than excessively aggressive because cerebral perfusion can be pressure dependent in acute ischemia.

168. D. In acute ischemic stroke without reperfusion therapy or another compelling indication, routine early blood pressure lowering has not improved outcomes when pressure is below about 220/120 mm Hg. Cerebral autoregulation is impaired, so aggressive reduction can worsen ischemia. If pressure is extremely high or another emergency indication exists, modest individualized reduction is used.

169. A. The 2026 AHA/ASA acute ischemic stroke guideline identifies intensive systolic reduction below 140 mm Hg after successful endovascular reperfusion as harmful when there is no other indication for that target. Trials of very intensive post-thrombectomy lowering have shown worse functional outcomes. The goal is controlled pressure without inducing hypoperfusion.

170. D. ACE inhibitors, ARBs, and direct renin inhibitors are avoided in pregnancy because of fetal toxicity, particularly renal and developmental injury. Labetalol and extended-release nifedipine are commonly preferred agents for chronic hypertension in pregnancy, with methyldopa another option in selected patients.

171. C. Modern pregnancy guidance supports treatment of chronic hypertension beginning at a threshold around 140/90 mm Hg rather than waiting for severe hypertension. The CHAP trial showed better pregnancy outcomes with active treatment at this threshold without a significant increase in fetal growth restriction. Pregnancy-compatible medications should be used.

172. D. Acute severe hypertension in pregnancy, generally systolic at least 160 or diastolic at least 110 mm Hg when persistent, warrants prompt treatment to reduce maternal risks such as stroke. Common acute options include intravenous labetalol, intravenous hydralazine, or immediate-release oral nifedipine under appropriate monitoring. Evaluation for preeclampsia and fetal status proceeds simultaneously.

173. C. Chronic hypertension is a high-risk condition for preeclampsia. Low-dose aspirin prophylaxis is recommended by major obstetric and cardiovascular guidance for high-risk pregnant patients when not

contraindicated, typically started in the late first or early second trimester according to local obstetric protocols. Warfarin or routine NSAID therapy is not a substitute.

174. D. Blood pressure can rise or remain elevated after delivery, and postpartum hypertensive complications can occur after hospital discharge. Early postpartum assessment—often within days—and home blood pressure monitoring can identify worsening hypertension promptly. Antihypertensive therapy is adjusted to pressure, symptoms, breastfeeding considerations, and clinical context rather than stopped automatically.

175. A. In hypertensive CKD with clinically important albuminuria, renin-angiotensin system inhibition with either an ACE inhibitor or an ARB is recommended to reduce cardiovascular risk and slow kidney disease progression. Dual blockade increases risks such as hyperkalemia and acute kidney injury without sufficient added benefit.

176. C. Hypertension is common after kidney transplantation and can reflect preexisting disease, allograft dysfunction, renal artery stenosis, corticosteroids, and calcineurin inhibitors. Tacrolimus and cyclosporine can raise pressure through renal vasoconstriction, sodium retention, and other mechanisms. Medication review and assessment of the graft are important parts of management.

177. D. Nondihydropyridine calcium channel blockers such as verapamil and diltiazem can worsen systolic heart failure because of negative inotropic effects and are generally avoided in HFrEF. Dihydropyridine agents such as amlodipine may be used for additional blood pressure control when needed after optimization of heart-failure guideline-directed therapy.

178. A. In HFrEF, therapies that improve heart-failure outcomes—renin-angiotensin system/ARNI therapy, evidence-based beta blockade, MRA, and SGLT2 inhibition as appropriate—form the foundation of treatment. Loop diuretics are used to manage congestion. Additional antihypertensive therapy can be added if pressure remains above goal, while avoiding agents that worsen systolic function.

179. B. Contemporary hypertension guidance recommends a target below 130/80 mm Hg for most adults with chronic coronary disease when tolerated. Blood pressure control reduces recurrent cardiovascular risk. Therapy should also reflect coronary indications and avoid excessive hypotension or symptoms in an individual patient.

180. D. Hypertension treatment benefits many older adults, but patients with substantial frailty, limited life expectancy, recurrent symptomatic hypotension, or high treatment burden require individualized decisions. Contemporary guidance supports clinician-patient or caregiver assessment of benefits and harms rather than rigid targets based on age alone.

181. B. Orthostatic hypotension requires a structured assessment of symptoms, reversible contributors, medication timing, volume status, and possible autonomic disease. It does not automatically mean all blood pressure treatment should be stopped; randomized hypertension trials do not show that appropriate

first-line intensive treatment universally causes orthostatic hypotension. Therapy should be individualized to standing symptoms and overall risk.

182. C. Peripheral alpha-1 blockers reduce arteriolar and venous tone and can worsen orthostatic hypotension, particularly in older adults or those with autonomic impairment. Medication review is therefore central when orthostatic symptoms appear after a regimen change. The same class can still have selected indications, such as concomitant benign prostatic hyperplasia, when risks are acceptable.

183. A. Neurogenic orthostatic hypotension with supine hypertension is a difficult autonomic syndrome in which treating one component can worsen the other. Management emphasizes medication review, nonpharmacologic measures, timing of therapies, avoidance of prolonged supine posture when appropriate, and individualized treatment of severe nocturnal hypertension while preserving upright function. A rigid conventional target can be unsafe.

184. B. Thiazide diuretics reduce lithium clearance and can substantially increase serum lithium concentrations. ACE inhibitors and some other agents can also increase lithium levels in susceptible patients. If these combinations are used, clinicians should coordinate monitoring of lithium concentration, kidney function, hydration status, and toxicity symptoms.

185. B. Serotonin-norepinephrine reuptake inhibitors can increase blood pressure in some patients, particularly at higher doses, through enhanced noradrenergic activity. A medication history is an essential part of evaluating new or worsening hypertension, and treatment decisions should balance psychiatric benefit with cardiovascular effects.

186. D. The AAP pediatric guideline uses fixed adult-aligned thresholds beginning at age 13 years: stage 1 hypertension is 130–139/80–89 mm Hg and stage 2 is at least 140/90 mm Hg. In younger children, percentile-based thresholds remain central. Diagnosis still requires appropriate repeat measurements rather than a single reading.

187. A. Oscillometric devices can be used for pediatric screening, but an elevated value should be repeated and confirmed by auscultation. Proper cuff size and technique are essential. Hypertension is generally diagnosed from persistently elevated auscultatory readings across separate visits unless the clinical situation requires urgent evaluation.

188. D. The AAP guideline gives ambulatory blood pressure monitoring an important role in confirming pediatric hypertension after persistent elevated or stage 1 office readings and in evaluating suspected white-coat hypertension. Pediatric-valid equipment and pediatric normative interpretation are required. Home monitoring may be useful after diagnosis but is not the preferred diagnostic confirmation tool.

189. B. Primary hypertension is increasingly common in older children and adolescents with obesity and a positive family history. In children age 6 years or older who fit this pattern and lack findings suggesting a secondary cause, the AAP guideline does not require an extensive secondary evaluation. A careful history, examination, basic studies, and assessment for comorbidities remain necessary.

190. A. Persistent pediatric stage 2 hypertension warrants timely evaluation and treatment or referral rather than prolonged observation. The AAP guideline recommends repeat confirmation and, if stage 2 values persist, diagnostic evaluation including ABPM when appropriate and treatment or specialty assessment within about one week. Symptomatic or extreme hypertension requires immediate care.

191. A. Symptomatic stage 2 hypertension or an extreme elevation—more than about 30 mm Hg above the 95th percentile, or above 180/120 mm Hg in an adolescent—requires immediate evaluation. The clinician should assess for hypertensive emergency and secondary causes and treat according to the affected organ system.

192. D. The AAP pediatric guideline recommends echocardiographic assessment for left ventricular hypertrophy when pharmacologic treatment is being considered, rather than routine echocardiography for every child at the moment hypertension is first suspected. LVH can influence treatment urgency and provides evidence of target-organ effects.

193. C. Lifestyle treatment is foundational in pediatric primary hypertension, particularly when obesity, diet, and inactivity contribute. Family-based dietary changes, healthy weight management, regular physical activity, and sodium reduction are appropriate. Medication is added for selected children, including persistent hypertension despite lifestyle therapy, symptomatic hypertension, stage 2 disease, CKD, diabetes, or target-organ damage.

194. A. Renin-angiotensin system blockers are commonly preferred in hypertensive children with CKD and proteinuria because they reduce proteinuria and provide kidney-protective effects. Kidney function and potassium require monitoring. Dual ACE inhibitor plus ARB therapy is not routine because it increases adverse-event risk.

195. D. Hypertension, including masked hypertension, can persist or develop after coarctation repair. The AAP guideline specifically supports ABPM in children and adolescents after repair to detect abnormal blood pressure patterns that clinic measurements may miss. Ongoing cardiovascular surveillance remains important even after anatomically successful repair.

196. C. Anxiety can transiently raise office blood pressure and contribute to a white-coat phenotype. Standardized measurement and out-of-office confirmation help distinguish sustained hypertension from situational elevation. The anxiety disorder should be treated on its own merits while avoiding overtreatment that could produce hypotension outside the clinic.

197. D. Asymptomatic elevated inpatient blood pressure is common and may be driven by pain, anxiety, sleep disruption, missed medications, or measurement problems. In the absence of acute target-organ damage, observational data raise concern about harm from reflexive acute treatment. Clinicians should address reversible factors, confirm measurement, and plan appropriate chronic follow-up.

198. C. In pheochromocytoma, beta blockade before adequate alpha blockade can produce unopposed alpha-mediated vasoconstriction and severe hypertension. Preoperative preparation therefore establishes

alpha blockade first, followed by beta blockade only if needed for tachyarrhythmia after alpha blockade is adequate. Volume expansion is often necessary because chronic catecholamine excess causes volume contraction.

199. C. Afferent baroreflex failure, often after neck surgery, radiation, or carotid injury, can cause marked blood pressure lability with hypertensive crises, tachycardia, and hypotensive episodes. Because normal buffering of sympathetic surges is lost, emotional or physical stress may provoke dramatic increases. Management differs from routine essential hypertension and focuses on reducing extremes while avoiding hypotension.

200. A. Contemporary renal-denervation guidance excludes or cautions against the procedure in several settings, including neurogenic orthostatic hypotension. Reducing renal sympathetic signaling in a patient already unable to maintain upright pressure may worsen symptoms. Candidate selection requires confirmation of hypertension, assessment of secondary causes, and multidisciplinary shared decision-making.