COMPREHENSIVE MOCK EXAM 3

Supplemental to: Hypertension Board Review 2026-2027

Rasmus Broberg

AHSCP Certification Examination for Hypertension Specialists (CHS) — 200 questions, four-option single-best-answer format, domain-weighted to the official 2026 content outline

Board-Review Questions

1. A 58-year-old man has had essential hypertension for 15 years. During rapid lowering of mean arterial pressure from 125 to 85 mm Hg, he develops transient lightheadedness despite a pressure that would be well tolerated by a normotensive person. Which physiologic adaptation best explains this response?

A. Rightward resetting of cerebral and arterial baroreflex autoregulation

B. Leftward resetting of renal pressure-natriuresis toward lower pressures

C. Increased sensitivity of carotid baroreceptors to small pressure changes

D. Loss of sympathetic vasoconstrictor tone during chronic hypertension

2. An experimental model of salt-sensitive hypertension shows that a higher renal perfusion pressure is required to excrete the same sodium load as in control animals. Which change best describes the underlying renal-pressure relationship?

A. A leftward shift of the pressure-natriuresis curve

B. A steeper baroreceptor firing curve at low pressures

C. A reduction in proximal sodium reabsorption at every pressure

D. A rightward shift of the pressure-natriuresis curve

3. A healthy volunteer adopts a very low-sodium diet for one week. Which coordinated hormonal response most directly preserves extracellular volume and arterial pressure?

A. Increased nitric oxide with suppression of distal sodium transport

B. Reduced sympathetic tone with increased renal dopamine synthesis

C. Suppressed renin with increased atrial natriuretic peptide release

D. Increased renin, angiotensin II, and aldosterone activity

4. A patient with reduced renal perfusion has activation of the renin-angiotensin system. At usual physiologic concentrations, angiotensin II preferentially constricts which renal vascular segment to help preserve glomerular filtration pressure?

A. The arcuate artery before the glomerulus

B. The efferent arteriole

C. The vasa recta descending from the medulla

D. The afferent arteriole

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

A. The sodium-chloride cotransporter, NCC

B. The sodium-hydrogen exchanger, NHE3

C. The sodium-potassium-2 chloride cotransporter, NKCC2

D. The epithelial sodium channel, ENaC

6. A patient has acute sympathetic activation during severe pain. Stimulation of renal juxtaglomerular cells increases renin secretion predominantly through which receptor?

A. Beta-2 adrenergic receptors

B. Beta-1 adrenergic receptors

C. Alpha-1 adrenergic receptors

D. Muscarinic M2 receptors

7. Endothelial nitric oxide lowers vascular tone after diffusing into adjacent vascular smooth muscle. Which intracellular signaling event most directly mediates this vasodilation?

A. Activation of soluble guanylate cyclase with increased cyclic GMP

B. Inhibition of potassium channels with membrane depolarization

C. Activation of adenylate cyclase with increased cyclic AMP

D. Activation of phospholipase C with increased intracellular calcium

8. Which endogenous endothelial mediator is a potent vasoconstrictor that can promote vascular smooth-muscle hypertrophy and sodium retention when overactive?

A. Endothelin-1

B. Prostacyclin

C. Bradykinin

D. Atrial natriuretic peptide

9. A 78-year-old woman has a blood pressure of 168/66 mm Hg with no secondary cause identified. Which age-related vascular change most directly explains her widened pulse pressure?

A. Decreased velocity of reflected arterial pressure waves

B. Reduced compliance of the large elastic arteries

C. Reduced left ventricular stroke volume

D. Increased compliance of the proximal aorta

10. In a patient with marked aortic stiffness, reflected arterial pressure waves return to the proximal aorta earlier than normal. What is the expected hemodynamic consequence?

A. Prolongation of diastole with increased coronary perfusion time

B. Augmentation of late systolic central aortic pressure

C. Selective lowering of systolic pressure with unchanged diastolic pressure

D. Reduction in left ventricular afterload during ejection

11. A patient with long-standing pressure overload develops concentric left ventricular hypertrophy. Which initial mechanical adaptation most directly normalizes wall stress according to the law of Laplace?

A. Increased end-diastolic volume without wall thickening

B. Increased ventricular chamber radius

C. Increased ventricular wall thickness

D. Reduced myocardial mass with eccentric remodeling

12. A patient with severe chronic hypertension has no neurologic symptoms at a mean arterial pressure of 135 mm Hg. Which statement best describes cerebral blood-flow autoregulation in this setting?

A. The autoregulatory range is shifted toward higher arterial pressures

B. Autoregulation is abolished once chronic hypertension develops

C. The lower autoregulatory limit shifts toward substantially lower pressures

D. Cerebral blood flow becomes pressure independent at all arterial pressures

13. Two patients ingest the same sodium load. One has a large rise in blood pressure while the other has little change. Which phenotype best describes the first patient?

A. Autonomic failure with supine hypertension

B. White-coat hypertension

C. Isolated diastolic hypertension

D. Salt-sensitive blood pressure

14. A patient with severe obesity develops hypertension despite normal renal imaging. Which mechanism is most strongly implicated in obesity-associated hypertension?

A. Loss of insulin signaling causing obligatory renal sodium wasting

B. Persistent suppression of sympathetic outflow and renin release

C. Reduced intravascular volume from chronic natriuresis

D. Sympathetic activation with renal sodium retention

15. A researcher studies vascular remodeling in chronic hypertension. Which structural change most directly increases systemic vascular resistance in small resistance arteries?

A. Loss of vascular smooth muscle with passive dilation

B. An increased media-to-lumen ratio

C. Reduced extracellular matrix within the arteriolar wall

D. A larger lumen with a thinner smooth-muscle media

16. Epidemiologic studies link low birth weight with later hypertension. Which renal developmental mechanism is most often proposed to contribute to this association?

A. Excess nephron formation with chronic sodium wasting

B. Congenital suppression of proximal tubular sodium transport

C. Reduced nephron endowment with greater single-nephron filtration demand

D. Persistent renal vasodilation from excess nitric oxide synthesis

17. Angiotensin-converting enzyme inhibition lowers angiotensin II and also increases the concentration of which vasodilator peptide by reducing its degradation?

A. Endothelin-1

B. Norepinephrine

C. Bradykinin

D. Vasopressin

18. Which component of the renin-angiotensin system generally counterbalances classic angiotensin II signaling by promoting vasodilation and natriuretic effects?

A. ACE generation of angiotensin II with AT1-receptor signaling

B. ACE2 generation of angiotensin-(1-7) with Mas-receptor signaling

C. Aldosterone activation of alpha-1 receptors in resistance vessels

D. Renin activation of mineralocorticoid receptors in the collecting duct

19. Acute atrial stretch increases atrial natriuretic peptide release. Which renal response is expected?

A. Increased sodium reabsorption with stimulation of aldosterone

B. Increased natriuresis with suppression of renin and aldosterone

C. Reduced glomerular filtration with increased sympathetic activity

D. Enhanced distal sodium retention through epithelial sodium channels

20. Within the kidney, locally generated dopamine normally contributes to sodium balance during sodium excess. What is its principal effect on tubular transport?

A. It directly stimulates aldosterone secretion from the adrenal cortex

B. It constricts afferent arterioles to reduce filtered sodium delivery

C. It activates ENaC and increases collecting-duct sodium retention

D. It inhibits several sodium transporters and promotes natriuresis

21. A 19-year-old patient has early-onset hypertension, hypokalemic metabolic alkalosis, suppressed renin, and suppressed aldosterone. A parent had the same phenotype. Which molecular defect is most likely?

A. Loss-of-function of the mineralocorticoid receptor

B. Deficient aldosterone synthase causing impaired mineralocorticoid production

C. Gain-of-function of the epithelial sodium channel

D. Loss-of-function of the thiazide-sensitive sodium-chloride cotransporter

22. A 32-year-old patient has hypertension, persistent hyperkalemia, metabolic acidosis, and low renin despite normal kidney function. Which inherited transport abnormality is most consistent with this phenotype?

A. Loss of NKCC2 function causing salt wasting and hypokalemia

B. Constitutive ENaC activation causing potassium wasting

C. Loss of NCC function causing salt wasting and secondary aldosteronism

D. Increased WNK pathway activity causing excessive NCC-mediated sodium reabsorption

23. A child has severe low-renin, low-aldosterone hypertension with hypokalemic alkalosis. The phenotype is caused by failure to inactivate cortisol within mineralocorticoid-sensitive tissues. Which enzyme is deficient?

A. Deficiency of 11-beta-hydroxylase in adrenal steroid synthesis

B. Deficiency of 11-beta-hydroxysteroid dehydrogenase type 2

C. Deficiency of 21-hydroxylase in adrenal steroid synthesis

D. Deficiency of 17-alpha-hydroxylase in adrenal steroid synthesis

24. A family has autosomal dominant early-onset hypertension with primary aldosteronism that improves with low-dose glucocorticoid therapy. Which mechanism is most likely?

A. ACTH-regulated aldosterone synthesis from a chimeric CYP11B1/CYP11B2 gene

B. Activating mutation of NCC causing hyperkalemic hypertension

C. Loss of 11-beta-HSD2 with cortisol activation of mineralocorticoid receptors

D. Constitutive activation of ENaC independent of adrenal steroid synthesis

25. During a catecholamine surge from a pheochromocytoma, marked peripheral vasoconstriction is mediated predominantly by which receptor?

A. Dopamine D1 receptors in the renal circulation

B. Alpha-1 adrenergic receptors on vascular smooth muscle

C. Beta-2 adrenergic receptors on vascular smooth muscle

D. Muscarinic receptors in resistance arterioles

26. A patient receiving chronic high-dose glucocorticoid therapy develops new hypertension. Which mechanism can contribute to the rise in blood pressure?

A. Persistent suppression of hepatic angiotensinogen production

B. Selective blockade of vascular alpha-adrenergic receptors

C. Obligate renal sodium wasting with chronic volume depletion

D. Enhanced sodium retention and increased vascular responsiveness to pressor hormones

27. A patient with untreated severe obstructive sleep apnea has nocturnal hypertension and a nondipping ambulatory pattern. Which mechanism most directly links repetitive apneas to hypertension?

A. Chronic reduction in renin release from the juxtaglomerular apparatus

B. Intermittent hypoxemia with recurrent sympathetic activation

C. Persistent nocturnal vasodilation from excess nitric oxide

D. Sustained suppression of carotid chemoreceptor signaling

28. Why does aging commonly increase systolic blood pressure even when cardiac output is unchanged?

A. Baroreceptor firing permanently eliminates sympathetic vascular tone

B. Systemic vascular resistance invariably falls to near zero with age

C. Large-artery compliance increases and stores more systolic energy

D. Large-artery stiffening reduces buffering of pulsatile ventricular ejection

29. A 24-hour ambulatory blood-pressure tracing shows an average daytime pressure of 142/86 mm Hg and an average sleep pressure of 137/82 mm Hg. Which pattern is present?

A. Blunted nocturnal dipping

B. Exaggerated nocturnal dipping

C. Isolated white-coat hypertension

D. Normal nocturnal dipping of more than 20 percent

30. A patient has normal clinic blood pressures but repeatedly elevated daytime ambulatory and home readings. Which mechanism most plausibly contributes to the prognostic risk of this phenotype?

A. Systematic overestimation of ambulatory pressure caused by larger cuff bladders

B. A transient clinic-only sympathetic surge with normal pressure at all other times

C. An obligatory fall in nighttime pressure that eliminates target-organ stress

D. Sustained out-of-office pressure load despite apparently normal clinic measurements

31. A muscular man with a 42-cm arm circumference has repeated office readings near 168/96 mm Hg using a standard adult cuff that is too small. What is the most appropriate next step?

A. Repeat after five minutes using the same standard adult cuff

B. Apply a fixed mathematical correction for the 42-cm arm

C. Repeat measurement with a validated device and a cuff sized to the arm circumference

D. Move to a wrist device without first confirming its validation and positioning

32. A hypertension clinic wants to reduce systematic error in office blood-pressure measurement. Which protocol is most appropriate?

A. Rest five minutes but leave the unsupported arm below heart level

B. Seat the patient quietly, support the back and arm, use an appropriate cuff, and average repeated readings

C. Take two readings over a thin shirt with an appropriately sized cuff

D. Take one seated reading after rest and use it as the visit average

33. A 52-year-old woman has office blood pressures of 150-158/88-94 mm Hg at four visits, but a technically adequate 24-hour ambulatory study shows normal awake and asleep averages. What is the best diagnosis?

A. White-coat hypertension

B. Sustained stage 2 hypertension

C. Masked hypertension

D. Hypertensive emergency

34. A 47-year-old man has office blood pressures around 124/76 mm Hg, but home readings are repeatedly 142-150/84-90 mm Hg and ambulatory monitoring confirms elevated daytime pressure. What is the best diagnosis?

A. White-coat hypertension

B. Normal blood pressure with device artifact

C. Isolated office hypertension

D. Masked hypertension

35. A 72-year-old patient taking three antihypertensive drugs reports dizziness after standing. Supine blood pressure is 146/78 mm Hg; after standing for three minutes it is 118/64 mm Hg with symptoms. Which finding is most important?

A. A symptomatic orthostatic fall that should prompt medication and volume assessment

B. A normal postural response that requires no change in evaluation

C. Proof of pheochromocytoma because pressure changes with posture

D. Evidence of white-coat hypertension because supine pressure is higher

36. A 66-year-old woman has a persistent 28 mm Hg systolic difference between arms on repeated careful measurements. The lower-pressure arm has a diminished radial pulse. What is the most appropriate interpretation?

A. Suspect obstructive disease of the proximal artery supplying the lower-pressure arm

B. Attribute the difference to sequential measurement variability and repeat next year

C. Suspect autonomic neuropathy as the primary cause of the pulse asymmetry

D. Interpret the higher-arm value as a white-coat response confined to that arm

37. A patient is asked to perform home blood-pressure monitoring to clarify control. Which instruction best improves diagnostic reliability?

A. Obtain one seated morning reading each day for a week

B. Use a validated wrist device routinely even when an upper-arm cuff fits

C. Measure immediately after usual exercise to capture peak daily pressure

D. Use a validated upper-arm device and obtain repeated seated readings over multiple days

38. Ambulatory blood-pressure monitoring in a patient with chronic kidney disease shows a normal daytime average but persistently elevated sleep pressure. Which feature is most clinically useful?

A. An isolated morning blood-pressure surge

B. A white-coat hypertension phenotype

C. Masked daytime hypertension with normal sleep pressure

D. Nocturnal hypertension that would be missed by office measurements

39. A patient has hypertension and newly detected albuminuria. Which test best quantifies early hypertensive kidney damage in routine outpatient evaluation?

A. A serum uric acid concentration alone

B. A plasma renin activity measurement alone

C. A spot urine albumin-to-creatinine ratio

D. A random urine sodium concentration alone

40. A 58-year-old patient with long-standing hypertension has voltage criteria for left ventricular hypertrophy on ECG. Which test is more sensitive for defining left ventricular mass and geometry?

A. Transthoracic echocardiography

B. A routine chest radiograph

C. Carotid duplex ultrasonography

D. A standard exercise treadmill test

41. A patient presents with blood pressure of 232/132 mm Hg, confusion, papilledema, and retinal hemorrhages. Which feature makes this presentation a hypertensive emergency rather than severe asymptomatic hypertension?

A. A history of hypertension for more than ten years

B. The presence of chronic left ventricular hypertrophy on an old ECG

C. The absolute systolic pressure being above 180 mm Hg by itself

D. Acute target-organ injury involving the brain and retina

42. A patient with hypertension starts an ACE inhibitor. Serum creatinine rises from 1.0 to 1.2 mg/dL and potassium remains normal. Blood pressure improves and the patient is euvolemic. What is the best interpretation?

A. The ACE inhibitor must be stopped because any creatinine increase indicates nephrotoxicity

B. A modest hemodynamic creatinine rise can be acceptable with continued monitoring

C. The rise proves bilateral renal artery stenosis and requires immediate stenting

D. The finding indicates intrinsic glomerulonephritis caused by ACE inhibition

43. A 69-year-old man has blood pressure of 174/68 mm Hg. Which derived measure is most directly increased and reflects the marked separation between systolic and diastolic pressures?

A. Central venous pressure reflecting right-sided filling pressure

B. Mean arterial pressure as an estimate of average perfusion pressure

C. Pulse pressure calculated as systolic minus diastolic pressure

D. Pulmonary capillary wedge pressure reflecting left-sided filling pressure

44. A patient with frequent premature beats requires repeated office blood-pressure measurements. Which approach is most appropriate?

A. Obtain multiple validated readings because beat-to-beat variability can reduce precision

B. Use the highest of three automated readings as the definitive value

C. Use a single manual auscultatory reading and disregard later variability

D. Replace brachial measurements with a single ankle pressure measurement

45. A 55-year-old woman with apparently resistant hypertension takes seven prescribed medications. Pharmacy refill data are incomplete, and home pressures are variable. Which next step best distinguishes true resistance from pseudoresistance?

A. Verify adherence and obtain reliable out-of-office blood-pressure data

B. Assume adherence because the medication list contains the prescribed drugs

C. Order adrenal venous sampling before any biochemical screening

D. Add a fifth antihypertensive class before reviewing the regimen

46. A patient has an office blood pressure of 136/84 mm Hg but a home average of 129/77 mm Hg on a validated device using correct technique. Which principle is most important when reconciling these measurements?

A. Treat the home average as numerically interchangeable with any office reading

B. Dismiss the home average because it is lower than the clinic measurement

C. Classify the pattern as masked hypertension because the office value is higher

D. Interpret the values in the context of the measurement setting and repeated averages

47. A patient with long-standing hypertension has progressive chronic kidney disease. Which paired measurements are most useful for routine assessment of renal target-organ involvement?

A. Serum creatinine and a screening renal ultrasound

B. Twenty-four-hour urine sodium and serum uric acid

C. Estimated GFR and urine albumin-to-creatinine ratio

D. Plasma renin and aldosterone concentrations

48. A patient asks whether a smartwatch that estimates blood pressure without an inflatable cuff can replace home cuff measurements for titrating medications. What is the best response?

A. Use the smartwatch after calibrating it once against an office cuff

B. Use a validated upper-arm cuff because cuffless smartwatch estimates are not yet reliable enough for titration

C. Use a validated wrist cuff as the preferred replacement for an upper-arm cuff

D. Rely on clinic measurements alone rather than any home monitoring

49. A 29-year-old patient develops abrupt severe hypertension with hypokalemia and no family history of early hypertension. Which diagnostic principle is most appropriate?

A. The young age and biochemical abnormalities justify focused evaluation for secondary hypertension

B. Begin empiric multidrug therapy and defer etiologic evaluation unless control fails

C. Repeat routine office measurements for six months before investigating the hypokalemia

D. Limit the evaluation to structural heart disease because the potassium abnormality is nonspecific

50. A patient has persistent hypertension despite three medications. Direct measurement of prescribed antihypertensive drugs in blood or urine shows several are absent. What is the most accurate classification?

A. Apparent treatment resistance caused by nonadherence

B. True resistant hypertension despite confirmed adherence

C. Refractory hypertension requiring immediate renal denervation

D. White-coat hypertension proven by the drug assay alone

51. A 46-year-old man has newly confirmed hypertension without hypokalemia or resistant disease. According to the 2025 Endocrine Society primary aldosteronism guideline, what screening strategy is suggested?

A. Reserve aldosterone-renin testing for patients with a documented adrenal incidentaloma

B. Begin aldosterone-renin testing after hypokalemia is confirmed on two separate samples

C. Limit aldosterone-renin testing to patients meeting conventional resistant-hypertension criteria

D. Screen for primary aldosteronism with aldosterone and renin measurements

52. A patient with hypertension and spontaneous potassium of 2.9 mmol/L has a nondiagnostic aldosterone-to-renin ratio. Renin is low but aldosterone is lower than expected. What is the best next step before repeating screening?

A. Correct the hypokalemia and repeat aldosterone and renin testing

B. Repeat testing immediately while potassium remains low to preserve the spontaneous phenotype

C. Begin marked sodium restriction for several days before repeating the screening study

D. Proceed directly to adrenal imaging because the low aldosterone excludes a meaningful repeat screen

53. A patient is being screened for primary aldosteronism while taking spironolactone. Why can this medication complicate interpretation of the aldosterone-to-renin ratio?

A. It lowers potassium and thereby consistently increases measured aldosterone concentrations

B. It blocks beta-1 signaling at juxtaglomerular cells and markedly suppresses renin

C. It inhibits cortisol metabolism and creates an apparent mineralocorticoid-excess phenotype

D. It can raise renin and alter aldosterone physiology, potentially obscuring renin suppression

54. A patient with resistant hypertension has potassium 3.2 mmol/L, plasma renin activity 0.2 ng/mL/h, and aldosterone 24 ng/dL. Which pattern best supports primary aldosteronism?

A. High renin with low aldosterone caused by adrenal failure

B. Suppressed renin with aldosterone that is inappropriately high relative to renin

C. High renin and high aldosterone from severe renal sodium loss

D. Parallel suppression of both renin and aldosterone from ENaC activation

55. A patient has resistant hypertension, spontaneous hypokalemia, undetectable renin, and markedly elevated aldosterone. He is willing to receive aldosterone-directed therapy. Which statement about confirmatory suppression testing is most appropriate?

A. It is mandatory before any mineralocorticoid-receptor antagonist can be prescribed

B. It may be unnecessary when overt renin-independent aldosterone production is already demonstrated

C. It should be replaced by renal artery angiography in all such patients

D. It is required even when the patient will not consider adrenal surgery

56. A 52-year-old patient has confirmed primary aldosteronism and wants potentially curative surgery. CT shows a 1.4-cm left adrenal nodule and a normal-appearing right adrenal gland. What is the best next step to determine laterality?

A. Adrenal venous sampling performed by an experienced center

B. Measure renal vein renin levels to localize aldosterone production

C. Proceed directly to left adrenalectomy because CT alone establishes secretion

D. Order a repeat potassium level and treat whichever side appears larger

57. A 29-year-old patient has marked primary aldosteronism, spontaneous hypokalemia, and a solitary 1.8-cm unilateral adrenal adenoma with benign imaging features. Which statement about adrenal venous sampling is most appropriate?

A. It may be omitted whenever CT shows any unilateral adrenal nodule

B. It is unnecessary when hypokalemia corrects with spironolactone before surgery

C. It may be omitted in selected young patients with a highly convincing unilateral phenotype

D. It should generally be omitted in younger patients even when imaging is bilateral or indeterminate

58. A 44-year-old patient has confirmed unilateral aldosterone production by adrenal venous sampling and is a good operative candidate. Which treatment offers the best chance of curing the hormone excess?

A. Laparoscopic removal of the unilateral aldosterone-producing adrenal gland

B. Renal sympathetic denervation as the preferred endocrine treatment

C. Bilateral adrenalectomy followed by chronic glucocorticoid replacement

D. Lifelong hydrochlorothiazide without aldosterone-directed therapy

59. A patient has bilateral primary aldosteronism confirmed during subtype evaluation. Which long-term therapy is most appropriate?

A. Unilateral adrenalectomy directed at the larger adrenal gland on CT

B. A mineralocorticoid-receptor antagonist titrated with potassium and renal monitoring

C. Bilateral adrenalectomy followed by lifelong adrenal-replacement therapy

D. An ACE inhibitor used as the sole therapy directed at aldosterone excess

60. A patient with primary aldosteronism is treated with spironolactone. Blood pressure remains above goal and renin is still suppressed. Which adjustment is most consistent with current guideline logic?

A. Add potassium supplements without reassessing the aldosterone-directed dose

B. Stop mineralocorticoid blockade because renin should remain suppressed during effective therapy

C. Switch to a beta blocker specifically to suppress renin further

D. Increase aldosterone-directed therapy as tolerated while monitoring potassium and kidney function

61. A 34-year-old woman has abrupt-onset hypertension, recurrent headaches, and a right upper abdominal bruit. Kidney function is normal. Which renovascular cause is particularly likely in this demographic?

A. Primary renal amyloidosis

B. Atherosclerotic ostial renal-artery stenosis

C. Bilateral renal cortical necrosis

D. Renal-artery fibromuscular dysplasia

62. A 73-year-old man with diffuse atherosclerosis has resistant hypertension and recurrent episodes of sudden pulmonary edema without major systolic dysfunction. Which secondary cause deserves particular consideration?

A. Hemodynamically significant atherosclerotic renal-artery stenosis

B. Liddle syndrome presenting for the first time in late adulthood

C. Primary hyperparathyroidism without hypercalcemia

D. Isolated mild obstructive sleep apnea as the sole explanation

63. A patient with unsuspected bilateral severe renal-artery stenosis starts lisinopril. Creatinine rises by 55% within a week without another clear cause. What is the most likely mechanism?

A. Loss of angiotensin II-mediated efferent arteriolar constriction reduces glomerular filtration pressure

B. ACE inhibition causes immune-complex glomerulonephritis within days

C. Lisinopril directly obstructs both renal arteries by causing thrombosis

D. ACE inhibition produces afferent arteriolar spasm through excess bradykinin

64. A patient has high clinical suspicion for renal-artery stenosis and preserved kidney function. Which noninvasive study can directly evaluate renal arterial anatomy with high spatial resolution?

A. A routine noncontrast abdominal radiograph

B. CT angiography of the renal arteries

C. A transthoracic echocardiogram

D. A carotid intima-media thickness study

65. A young patient has renovascular hypertension from renal-artery fibromuscular dysplasia with a focal pressure-limiting lesion. Which revascularization strategy is generally preferred when intervention is indicated?

A. Routine drug-eluting stent implantation for every renal lesion

B. Coronary bypass grafting to the affected renal artery

C. Surgical nephrectomy as the first intervention

D. Percutaneous balloon angioplasty without routine stent placement

66. A patient has episodic pounding headaches, diaphoresis, palpitations, and paroxysmal hypertension. Which biochemical test is most appropriate for initial evaluation of pheochromocytoma or paraganglioma?

A. Plasma free metanephrines or appropriately collected urinary fractionated metanephrines

B. A single morning serum cortisol without suppression testing

C. A random serum aldosterone concentration without renin

D. A spot urine vanillylmandelic acid as the preferred modern test

67. A patient with a catecholamine-secreting pheochromocytoma is being prepared for surgery. Which medication sequence is safest?

A. Begin beta blockade first, then add alpha blockade if pressure remains elevated

B. Start alpha and beta blockade simultaneously at full doses on the day before surgery

C. Establish alpha-adrenergic blockade before adding beta blockade if needed

D. Use calcium-channel blockade alone as a universal substitute for preoperative adrenergic preparation

68. A 28-year-old patient with pheochromocytoma has retinal angiomas and a family history of renal cell carcinoma. Which inherited syndrome is most likely?

A. Marfan syndrome

B. Autosomal dominant polycystic kidney disease

C. Familial hypercholesterolemia

D. Von Hippel-Lindau syndrome

69. A patient with hypertension, proximal muscle weakness, easy bruising, and new diabetes is suspected of having endogenous cortisol excess. Which test is an accepted first-line screen for Cushing syndrome?

A. A random morning serum cortisol concentration

B. A 1-mg overnight dexamethasone suppression test

C. A plasma ACTH concentration used as the initial case-detection test

D. A single afternoon serum cortisol concentration after normal activity

70. A patient with hypertension has progressive enlargement of hands and feet, coarse facial features, sleep apnea, and glucose intolerance. Which test is the best initial biochemical screen for acromegaly?

A. Serum insulin-like growth factor 1 concentration

B. Plasma aldosterone-to-renin ratio

C. Urinary fractionated metanephrines

D. Random growth hormone concentration alone

71. A patient has weight loss, tremor, tachycardia, widened pulse pressure, and predominantly systolic hypertension. Which endocrine disorder best fits this pattern?

A. Hyperthyroidism

B. Hypoparathyroidism

C. Primary adrenal insufficiency

D. Primary hypothyroidism

72. A patient has fatigue, cold intolerance, bradycardia, elevated TSH, and new predominantly diastolic hypertension. Which mechanism best explains the pressure pattern?

A. Renal sodium wasting from mineralocorticoid deficiency

B. Marked increase in cardiac output from thyroid-hormone excess

C. Catecholamine surges from an adrenal medullary tumor

D. Increased systemic vascular resistance associated with hypothyroidism

73. A 41-year-old patient has hypertension, recurrent nephrolithiasis, constipation, and serum calcium of 11.6 mg/dL. Which secondary disorder should be evaluated?

A. Central diabetes insipidus

B. Hypopituitarism

C. Primary hyperparathyroidism

D. Primary adrenal insufficiency

74. A 23-year-old patient has hypertension in both arms, delayed femoral pulses, and lower systolic pressure in the legs than the arms. Which diagnosis is most likely?

A. Obstructive sleep apnea

B. Bilateral renal-artery fibromuscular dysplasia

C. Coarctation of the aorta

D. Primary aldosteronism

75. A patient with obesity, loud snoring, witnessed apneas, and resistant hypertension has a high pretest probability of obstructive sleep apnea. Which test establishes the diagnosis and severity most directly?

A. Overnight pulse oximetry alone without airflow or respiratory-event measurement

B. A daytime aldosterone concentration without evaluation of sleep physiology

C. Renal-artery duplex ultrasonography to look for renovascular hypertension

D. Polysomnography or a validated home sleep apnea test

76. A patient with resistant hypertension and severe obstructive sleep apnea starts effective continuous positive airway pressure therapy. What blood-pressure effect should generally be expected?

A. A typical systolic reduction of 25 to 35 mm Hg in most adherent patients

B. A modest average reduction, with larger benefit in some highly adherent or resistant patients

C. Little average blood-pressure change even when severe apnea is fully treated

D. Near-complete normalization of blood pressure in most patients within the first week

77. A patient with advanced chronic kidney disease develops worsening hypertension and peripheral edema. Which mechanism is most likely to be dominant?

A. Impaired sodium excretion with extracellular-volume expansion

B. A fixed reduction in systemic vascular resistance caused by uremia

C. Obligate sodium wasting from increased nephron mass

D. Chronic suppression of sympathetic and renin-angiotensin activity in all CKD

78. A 36-year-old patient has hypertension, microscopic hematuria, proteinuria, and reduced estimated GFR. Which evaluation is most appropriate before labeling the hypertension primary?

A. Perform a catecholamine-tumor workup before evaluating the abnormal urinalysis

B. Obtain renal-artery imaging first because reduced GFR makes parenchymal disease unlikely

C. Evaluate renal parenchymal disease with urine studies, kidney function, and renal imaging

D. Begin adrenal subtype testing before characterizing the kidney disease

79. A 17-year-old patient with severe early-onset hypertension has hypokalemia, metabolic alkalosis, low renin, and low aldosterone. Genetic testing confirms Liddle syndrome. Which drug directly treats the transport defect?

A. Aliskiren

B. Hydrochlorothiazide

C. Amiloride

D. Spironolactone

80. A patient develops hypertension, hypokalemic alkalosis, low renin, and low aldosterone after consuming large amounts of licorice-containing herbal products. What is the mechanism?

A. Loss of 11-beta-HSD2 activity allows cortisol to stimulate mineralocorticoid receptors

B. Activation of beta-1 receptors causing high-renin secondary aldosteronism

C. Blockade of ENaC causing compensatory sodium retention

D. Direct stimulation of aldosterone synthase by glycyrrhizin

81. A 28-year-old patient has familial hypertension with hyperkalemia and hyperchloremic metabolic acidosis. Which treatment is particularly effective for Gordon syndrome?

A. Fludrocortisone to increase mineralocorticoid activity

B. A low-dose thiazide or thiazide-like diuretic

C. High-dose potassium supplementation

D. Amiloride as obligatory first-line therapy for ENaC gain-of-function

82. A 15-year-old patient has primary aldosteronism and a strong family history of strokes before age 40. Which additional evaluation is most appropriate?

A. Genetic evaluation for a familial form of primary aldosteronism

B. Adrenal venous sampling as the first step before confirming the familial syndrome

C. Renal-artery angiography to look for fibromuscular dysplasia as the inherited cause

D. Chromaffin-tumor genetic testing without first considering familial aldosteronism

83. A 21-year-old patient has severe hypertension, hypokalemia, markedly elevated renin and aldosterone, and a small cortical renal mass. Which rare diagnosis best fits this pattern?

A. Liddle syndrome

B. Apparent mineralocorticoid excess

C. A renin-secreting juxtaglomerular cell tumor

D. Primary aldosteronism from an adrenal adenoma

84. A patient develops abrupt severe hypertension after blunt flank trauma. Imaging shows a large subcapsular renal hematoma compressing the kidney. Which mechanism best explains the hypertension?

A. The hematoma directly secretes aldosterone

B. Renal compression causes systemic vasodilation without hormonal activation

C. Renal compression activates renin release, producing Page kidney physiology

D. Compression suppresses renin and produces isolated cortisol excess

85. A patient with previously controlled hypertension develops higher pressures after beginning daily ibuprofen for arthritis. Which mechanism is most likely?

A. Direct mineralocorticoid-receptor blockade causes compensatory hypertension

B. Reduced renal prostaglandin synthesis promotes sodium retention and blunts antihypertensive effects

C. Irreversible destruction of vascular ACE raises bradykinin indefinitely

D. Inhibition of sympathetic tone triggers reflex renal sodium wasting

86. A 31-year-old woman develops new hypertension several months after starting an estrogen-containing oral contraceptive. Which mechanism can contribute?

A. Estrogen blocks hepatic angiotensinogen synthesis and causes sodium wasting

B. Oral contraceptives directly inhibit ENaC in the collecting duct

C. Estrogen increases hepatic angiotensinogen production and can activate the renin-angiotensin system

D. Estrogen eliminates sympathetic vascular responses

87. A patient with controlled hypertension begins taking an over-the-counter decongestant containing pseudoephedrine and develops palpitations with higher pressures. What is the most likely explanation?

A. Sympathomimetic vasoconstriction and adrenergic stimulation

B. Selective inhibition of the renin-angiotensin system

C. Direct blockade of alpha-1 receptors with reflex hypotension

D. Suppression of norepinephrine release from sympathetic nerve terminals

88. A kidney-transplant recipient develops difficult hypertension after tacrolimus exposure. Which mechanism is a recognized contributor?

A. Persistent renal vasodilation with obligatory salt wasting

B. Direct inhibition of all sympathetic neurotransmission

C. Calcineurin-inhibitor mediated renal vasoconstriction and sodium retention

D. Selective blockade of mineralocorticoid receptors

89. A patient with advanced CKD develops worsening hypertension after the dose of an erythropoiesis-stimulating agent is increased. Which statement is most accurate?

A. The blood-pressure effect occurs primarily through predictable severe sodium wasting

B. Erythropoiesis-stimulating agents can raise blood pressure and worsen preexisting hypertension

C. A pressure rise after dose escalation is more characteristic of new renal-artery stenosis

D. The usual hemodynamic effect is vasodilation with a clinically important pressure reduction

90. A 37-year-old patient presents with severe hypertension, agitation, diaphoresis, mydriasis, and chest pain shortly after recreational drug use. Which exposure is most likely?

A. A monoamine oxidase inhibitor taken with a tyramine-rich meal

B. Abrupt withdrawal of long-term central alpha-2 agonist therapy

C. Cocaine or an amphetamine-type recreational stimulant

D. Heavy use of a sympathomimetic over-the-counter decongestant

91. A patient with stage 1 hypertension asks which dietary change has the strongest evidence as part of a comprehensive lifestyle program. Which recommendation is most appropriate?

A. Adopt a DASH-style eating pattern with reduced sodium intake

B. Adopt a Mediterranean-style pattern while intentionally maintaining a high sodium intake

C. Use a very-low-carbohydrate diet without attention to sodium or food quality

D. Use a high-protein processed-food diet while restricting fruits and vegetables

92. A patient consuming approximately 4,500 mg of sodium daily has hypertension and no contraindication to dietary change. Which counseling point is most appropriate?

A. Keep sodium intake unchanged and focus exclusively on increasing dietary potassium

B. Reducing sodium intake can meaningfully lower blood pressure, especially in salt-sensitive patients

C. Reduce sodium only on days when home systolic pressure exceeds the treatment goal

D. Replace dietary sodium with processed foods labeled low-fat but not lower-sodium

93. A patient with hypertension and normal kidney function asks about increasing dietary potassium. Which advice is most appropriate?

A. Increase potassium supplements routinely even if a mineralocorticoid antagonist is later added

B. Use potassium-chloride salt substitute freely even if kidney function declines

C. Increase potassium-rich foods if safe, but individualize advice when hyperkalemia risk is present

D. Avoid dietary potassium enrichment even when kidney function and serum potassium are normal

94. A patient with obesity and hypertension asks whether weight reduction is likely to affect blood pressure. Which response is most accurate?

A. Weight reduction improves metabolic risk but usually has little effect on blood pressure

B. Blood-pressure improvement generally requires bariatric surgery rather than nonsurgical weight loss

C. Initial weight loss may lower pressure, but sustained loss generally causes pressure rebound above baseline

D. Sustained weight loss generally lowers blood pressure and can reduce medication requirements

95. A sedentary patient with uncomplicated hypertension wants an exercise prescription. Which general strategy is appropriate?

A. Aerobic exercise should be avoided, but moderate resistance exercise can be used alone

B. High-intensity isometric exercise should replace aerobic training in most patients

C. Regular aerobic activity supplemented by resistance exercise as tolerated

D. One prolonged exercise session each week is equivalent to regular activity across the week

96. A patient drinks four to five alcoholic beverages every evening and has poorly controlled hypertension. Which counseling point is most appropriate?

A. Switch from daily alcohol use to the same weekly amount consumed in one or two binges

B. Replace beer with spirits while maintaining the same ethanol intake

C. Reducing or eliminating excess alcohol can improve blood-pressure control

D. Continue the current intake because alcohol-related pressure effects are limited to withdrawal

97. A 59-year-old patient has an average blood pressure of 134/84 mm Hg and a PREVENT 10-year total cardiovascular disease risk of 11%. He has no contraindication to treatment. Which strategy is most consistent with the 2025 ACC/AHA guideline?

A. Begin antihypertensive medication in addition to lifestyle therapy

B. Use lifestyle therapy for 3 to 6 months before considering medication despite the elevated risk

C. Defer medication until average blood pressure reaches at least 140/90 mm Hg

D. Begin three antihypertensive classes because stage 1 hypertension with elevated risk requires triple therapy

98. A 42-year-old patient has repeated average blood pressure of 132/82 mm Hg, no cardiovascular disease, diabetes, or CKD, and PREVENT 10-year CVD risk of 3%. What is the best initial approach?

A. No follow-up because stage 1 hypertension carries no long-term risk

B. Immediate triple-drug therapy despite low predicted risk

C. Lifestyle modification with reassessment after approximately 3 to 6 months

D. Renal denervation before any lifestyle intervention

99. A 54-year-old patient has confirmed average blood pressure of 158/94 mm Hg and no secondary cause. Which initial pharmacologic strategy is preferred by the 2025 ACC/AHA guideline?

A. Begin one first-line agent and defer a second agent until a six-month reassessment

B. Use a short-acting agent twice daily to allow rapid dose-by-dose adjustment

C. Begin a beta blocker plus alpha-1 blocker as the preferred two-drug foundation

D. Two first-line agents of different classes, preferably in a single-pill combination

100. Which group contains only generally accepted first-line drug classes for uncomplicated primary hypertension?

A. Thiazide-type diuretic, ACE inhibitor or ARB, and long-acting calcium-channel blocker

B. Methyldopa, a direct renin inhibitor, and short-acting nifedipine

C. Alpha-1 blocker, long-acting nitrate, and loop diuretic

D. Clonidine, hydralazine, and minoxidil as routine first choices

101. A patient has true resistant hypertension while taking maximally tolerated lisinopril, amlodipine, and hydrochlorothiazide. Volume status and kidney function permit a thiazide-like diuretic. Which change is most appropriate before adding multiple later-line drugs?

A. Stop the diuretic and rely on vasodilators alone

B. Add an alpha blocker while leaving the diuretic regimen unoptimized

C. Replace amlodipine with short-acting nifedipine taken as needed

D. Replace hydrochlorothiazide with a long-acting thiazide-like agent such as chlorthalidone

102. A patient with stage 4 CKD has hypertension, marked edema, and clear extracellular-volume overload despite dietary sodium reduction. Which diuretic strategy is most appropriate for decongestion?

A. Use a thiazide-like diuretic alone regardless of the degree of volume overload

B. Use a mineralocorticoid-receptor antagonist as the primary decongestive drug despite hyperkalemia

C. Use an adequately dosed loop diuretic, with combination nephron blockade if needed

D. Rely on vasodilator therapy first and reserve diuresis until edema becomes refractory

103. A patient develops a persistent dry cough several weeks after starting lisinopril. Kidney function and potassium are normal. Which substitution is most appropriate if renin-angiotensin system blockade remains desired?

A. Switch to a different ACE inhibitor with a shorter half-life

B. Add a low-dose thiazide while continuing lisinopril and observing the cough

C. Switch to an angiotensin receptor blocker, which generally does not reproduce ACE-inhibitor cough

D. Reduce lisinopril to the lowest dose and add a second ACE inhibitor if needed

104. A patient taking an ACE inhibitor develops angioedema with lip and tongue swelling. What is the most appropriate immediate medication decision?

A. Continue the ACE inhibitor at half dose after the swelling improves

B. Add a thiazide and continue the ACE inhibitor because the reaction is dose dependent

C. Switch immediately to another ACE inhibitor in the same class

D. Permanently discontinue the ACE inhibitor and treat the airway risk urgently

105. A woman taking losartan learns that she is eight weeks pregnant. Which action is most appropriate?

A. Continue losartan through the first trimester and switch only after 14 weeks

B. Stop the ARB and transition to a pregnancy-compatible antihypertensive regimen

C. Reduce the losartan dose while adding low-dose aspirin for fetal protection

D. Switch from losartan to an ACE inhibitor because ACE inhibitors have more pregnancy data

106. A patient develops ankle edema after amlodipine is increased. Lungs are clear, jugular venous pressure is normal, and kidney function is unchanged. What is the most likely mechanism?

A. Direct venoconstriction lowers capillary hydrostatic pressure

B. New left ventricular systolic failure caused by dihydropyridine toxicity

C. Preferential arteriolar dilation raises capillary hydrostatic pressure

D. Primary sodium retention from aldosterone excess caused by amlodipine

107. A patient with second-degree atrioventricular block requires additional blood-pressure treatment. Which agent should generally be avoided because it can further slow AV nodal conduction?

A. Losartan

B. Verapamil

C. Amlodipine

D. Chlorthalidone

108. A 76-year-old woman develops confusion and serum sodium of 119 mmol/L two weeks after starting chlorthalidone. She is euvolemic on examination. Which adverse effect is most likely?

A. Calcium-channel blocker induced osmotic diuresis

B. Thiazide-associated hyponatremia

C. Beta-blocker induced diabetes insipidus

D. ACE-inhibitor induced hypernatremia

109. A patient with recurrent gout needs an additional antihypertensive agent. Which drug class can raise serum uric acid and potentially precipitate gout?

A. A thiazide or thiazide-like diuretic used for blood-pressure control

B. Dihydropyridine calcium-channel blockers

C. Angiotensin receptor blockers as a uniform class effect

D. ACE inhibitors through uricosuric action

110. A patient with albuminuric CKD starts an ARB. Creatinine rises from 1.6 to 1.9 mg/dL and then stabilizes; potassium is 4.8 mmol/L. What is the best management?

A. Add an ACE inhibitor to reverse the hemodynamic creatinine change

B. Stop the ARB because any creatinine rise is evidence of kidney injury

C. Start high-dose NSAID therapy to increase renal perfusion

D. Continue the ARB with ongoing monitoring if no other concerning features emerge

111. A patient with diabetic CKD benefits from an ARB but develops potassium of 5.7 mmol/L. Which principle best preserves cardiorenal benefit when feasible?

A. Treat reversible contributors and use potassium-lowering measures to preserve RAAS blockade when feasible

B. Combine the ARB with an ACE inhibitor to lower potassium

C. Add routine potassium supplementation while continuing the same regimen

D. Ignore persistent hyperkalemia because RAAS blockers prevent arrhythmia

112. A patient with hypertension and symptomatic chronic stable angina needs a drug that also reduces myocardial oxygen demand. Which class is particularly useful when no contraindication is present?

A. A beta blocker that lowers heart rate and myocardial oxygen demand

B. A long-acting dihydropyridine calcium-channel blocker that primarily lowers afterload

C. A thiazide-like diuretic that lowers pressure through natriuresis

D. An ACE inhibitor that lowers afterload without directly reducing heart rate

113. A 68-year-old man with hypertension and bothersome benign prostatic hyperplasia asks whether doxazosin can be his only antihypertensive drug. What is the best response?

A. Use doxazosin as sole therapy because its urinary benefit predicts superior cardiovascular outcomes

B. Avoid doxazosin because alpha-1 blockade generally worsens urinary obstruction

C. Use doxazosin as preferred first-line therapy whenever hypertension and BPH coexist

D. An alpha-1 blocker can improve urinary symptoms but is not preferred as sole first-line hypertension therapy

114. A patient taking high-dose clonidine abruptly runs out of medication and presents with severe hypertension, tachycardia, and agitation. What is the likely mechanism?

A. Irreversible blockade of vascular alpha-1 receptors

B. Rebound sympathetic activation after sudden withdrawal of central alpha-2 agonism

C. Acute mineralocorticoid deficiency caused by clonidine cessation

D. A delayed thiazide-like natriuretic effect

115. A patient receiving high-dose hydralazine for difficult hypertension develops arthralgias, fever, and positive antihistone antibodies. Which adverse effect is most likely?

A. Drug-induced lupus syndrome

B. Thiazide-associated hyponatremia

C. Mineralocorticoid-receptor mediated gynecomastia

D. Bradykinin-mediated angioedema

116. A patient with refractory hypertension is started on minoxidil. Which accompanying effects should be anticipated and managed?

A. Severe bradycardia and obligatory potassium wasting

B. Suppression of hair growth and contraction of plasma volume

C. A predictable fall in heart rate with natriuresis

D. Reflex tachycardia and marked sodium-water retention

117. A man taking spironolactone for resistant hypertension develops painful gynecomastia. Which alternative mineralocorticoid-receptor antagonist has fewer antiandrogenic effects?

A. Hydrochlorothiazide

B. Amiloride

C. Eplerenone

D. Clonidine

118. A patient with bilateral primary aldosteronism requires mineralocorticoid-receptor blockade but previously stopped spironolactone because of sexual side effects. Which treatment is most appropriate?

A. Use amiloride as the preferred substitute because it is another mineralocorticoid-receptor antagonist

B. Choose unilateral adrenalectomy based on the larger gland despite bilateral secretion

C. Use dual ACE-inhibitor and ARB therapy as the most specific way to block aldosterone action

D. Eplerenone with careful potassium and kidney-function monitoring

119. A patient with genetically confirmed Liddle syndrome remains hypertensive despite spironolactone. Which change is most mechanistically appropriate?

A. Increase spironolactone because suppression of aldosterone signaling directly closes ENaC

B. Switch to an ACE inhibitor because reducing angiotensin II directly corrects the ENaC mutation

C. Add fludrocortisone because stronger mineralocorticoid signaling suppresses mutant ENaC activity

D. Start an epithelial sodium-channel blocker such as amiloride

120. A patient takes a long-acting calcium-channel blocker, an ACE inhibitor, and an appropriate diuretic at maximally tolerated doses. Adherence is confirmed, technique is correct, and ambulatory blood pressure remains above goal. How is this condition classified?

A. Masked normotension

B. Hypertension cured by medication adherence

C. True resistant hypertension

D. White-coat hypertension

121. A patient is referred for resistant hypertension, but the office uses a cuff that is too small and measures pressure immediately after the patient climbs stairs. What is the most appropriate first step?

A. Proceed directly to renal denervation without confirming pressure

B. Repeat blood pressure using standardized technique before escalating therapy

C. Add spironolactone before repeating any measurements

D. Diagnose refractory hypertension because three drugs are already prescribed

122. A patient prescribed four antihypertensive drugs admits missing several doses each week because the schedule is complicated. Which intervention is most likely to improve control?

A. Split the same total doses into more frequent administrations to smooth blood-pressure control

B. Add a fifth drug before changing the current schedule because dose frequency does not affect adherence

C. Simplify to once-daily and fixed-dose combinations when clinically feasible

D. Convert long-acting drugs to shorter-acting formulations so each missed dose has a smaller duration

123. A treated patient has office pressure of 152/88 mm Hg but repeated ambulatory averages are at goal. Adherence is confirmed. What is the best interpretation?

A. Masked uncontrolled hypertension

B. White-coat effect causing apparent treatment resistance

C. Definite secondary hypertension requiring adrenal surgery

D. Hypertensive emergency

124. A patient with resistant hypertension has mild edema, high dietary sodium intake, and a regimen containing hydrochlorothiazide 12.5 mg daily. Which intervention most directly addresses a common mechanism of resistance?

A. Increase amlodipine while leaving sodium intake and diuretic intensity unchanged

B. Intensify sodium restriction and optimize diuretic therapy

C. Add an alpha-1 blocker before addressing the clinical evidence of volume excess

D. Add a central alpha-2 agonist while leaving hydrochlorothiazide at the current dose

125. A patient has true resistant hypertension on an ACE inhibitor, amlodipine, and chlorthalidone. Potassium is 4.1 mmol/L and estimated GFR is 65 mL/min/1.73 m2. Which drug is the preferred usual fourth-line addition?

A. Spironolactone added as mineralocorticoid-receptor blockade

B. Doxazosin added as an alpha-1 adrenergic blocker

C. Hydralazine added as a direct arteriolar vasodilator

D. Short-acting nifedipine used intermittently as needed

126. A patient has confirmed resistant hypertension, obesity, loud snoring, daytime sleepiness, and a nondipping ambulatory pattern. Which coexisting disorder should be actively evaluated because treatment may improve overall risk and modestly lower pressure?

A. Primary aldosteronism with autonomous mineralocorticoid activity

B. Obstructive sleep apnea with recurrent nocturnal upper-airway collapse

C. Renovascular hypertension from hemodynamically important renal-artery disease

D. Chronic kidney disease with impaired sodium excretion and volume expansion

127. A 61-year-old patient has sustained uncontrolled hypertension despite intensive lifestyle treatment and a well-tolerated multidrug regimen. Secondary causes and white-coat effect have been addressed, and the patient strongly prefers a device-based adjunct. Which option can now be considered through shared decision-making at an experienced center?

A. Baroreflex-activation therapy with an implanted carotid-sinus device as routine first choice

B. Creation of a central arteriovenous anastomosis as standard adjunctive therapy

C. Endovascular carotid stenting to reduce sympathetic outflow in the absence of carotid stenosis

D. Catheter-based renal sympathetic denervation used as an adjunct to ongoing lifestyle and medication therapy

128. A health system wants to improve hypertension control across a large population. Which strategy is most consistent with modern guideline-based care?

A. Increase visit frequency but keep medication changes restricted to physician-only office encounters

B. Use home monitoring but leave treatment changes to unstructured individual clinician preference

C. Create a standardized titration protocol without home measurements or multidisciplinary follow-up

D. Combine home blood-pressure monitoring with standardized protocols and team-based follow-up

129. A patient with stage 2 hypertension is prescribed an ARB and long-acting calcium-channel blocker. Which formulation can improve adherence when doses are appropriate?

A. Two separate once-daily pills dispensed in different monthly refill cycles

B. Two extended-release agents taken at different times to preserve regimen flexibility

C. A single-pill fixed-dose combination containing both prescribed long-acting antihypertensive agents

D. Alternating one drug on even days and the other on odd days

130. A patient asks whether every antihypertensive medication should be moved to bedtime to prevent cardiovascular events. Which response is most appropriate?

A. Move all agents to bedtime whenever ambulatory monitoring shows any nondipping pattern

B. Move all agents to bedtime because nighttime dosing has proven universal event reduction

C. Keep all agents in the morning because nighttime dosing increases cardiovascular events

D. Dose timing should be individualized; routine universal bedtime dosing is not required

131. An 82-year-old patient has repeated blood pressures of 164/64 mm Hg with preserved functional status. Which treatment principle is most appropriate?

A. Lower systolic pressure below 110 mm Hg rapidly because isolated systolic hypertension is an emergency

B. Prefer short-acting agents so the low diastolic pressure can recover between doses

C. Treat systolic hypertension while monitoring orthostasis and excessive diastolic lowering

D. Withhold antihypertensive therapy because a diastolic value below 70 mm Hg eliminates systolic risk

132. A 76-year-old man with known coronary artery disease has blood pressure of 142/54 mm Hg while taking several vasodilators and reports exertional chest discomfort. Which concern is most relevant when intensifying therapy?

A. The low diastolic pressure is reassuring because coronary perfusion is determined primarily by systolic pressure

B. Further diastolic lowering should improve angina by reducing left-ventricular filling pressure

C. The systolic pressure should be left untreated whenever diastolic pressure is below 60 mm Hg

D. Further reduction in diastolic pressure may compromise coronary perfusion in a susceptible patient

133. A patient with hypertension has no compelling comorbidity and asks whether treatment should be selected solely from self-identified race. Which principle best reflects contemporary care?

A. Select the same first-line class for everyone and adjust only after treatment failure

B. Use self-identified race as the principal determinant whenever there is no compelling comorbidity

C. Choose effective first-line therapy using comorbidities, tolerability, response, and patient factors rather than race alone

D. Use genetic-ancestry testing to predict the single best antihypertensive class before treatment

134. A patient with diabetes, hypertension, eGFR 58 mL/min/1.73 m2, and urine albumin-to-creatinine ratio of 480 mg/g needs antihypertensive therapy. Which class is particularly indicated for kidney protection?

A. A long-acting dihydropyridine calcium-channel blocker as the sole kidney-protective agent

B. A thiazide-like diuretic because natriuresis specifically reduces albuminuria-related kidney risk

C. A beta blocker because heart-rate reduction is the principal therapy for albuminuric diabetic CKD

D. An ACE inhibitor or angiotensin receptor blocker titrated with appropriate kidney-function and potassium monitoring

135. A patient with diabetes and hypertension has normal eGFR and no albuminuria or coronary disease. Which statement about initial antihypertensive drug choice is most accurate?

A. An ACE inhibitor is preferred over every other class even without albuminuria or coronary disease

B. Several standard first-line classes are reasonable, selected according to the overall clinical profile

C. A beta blocker should be the initial drug because diabetes itself is a compelling beta-blocker indication

D. A thiazide-type diuretic should be avoided because any glucose effect outweighs cardiovascular benefit

136. A patient with nondiabetic CKD has hypertension and heavy albuminuria. Which therapy is most appropriate as part of the regimen if tolerated?

A. Use an ACE inhibitor or ARB and titrate with potassium and creatinine monitoring

B. Use a dihydropyridine calcium-channel blocker first because albuminuria removes the role of RAAS blockade

C. Use an alpha-1 blocker because lowering systemic pressure is sufficient to address heavy albuminuria

D. Combine an ACE inhibitor and ARB to achieve more complete renin-angiotensin blockade

137. A patient with proteinuric CKD is taking lisinopril. Another clinician proposes adding losartan solely to lower albuminuria further. What is the best response?

A. Avoid routine dual ACE-inhibitor and ARB therapy because harm outweighs added benefit

B. Stop all blood-pressure treatment because albuminuria should not influence therapy

C. Add losartan because dual blockade reduces acute kidney injury and hyperkalemia

D. Add a direct renin inhibitor to create triple renin-angiotensin blockade

138. A patient with hypertension and recurrent gout needs an ARB. Which agent may be advantageous because it has a modest uricosuric effect?

A. Olmesartan

B. Candesartan

C. Valsartan

D. Losartan

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

A. A direct renin inhibitor such as aliskiren

B. A loop diuretic such as furosemide

C. A thiazide or thiazide-like diuretic

D. A central alpha-2 agonist such as clonidine

140. A patient has excellent pressure reduction with amlodipine but bothersome dependent edema. Which addition can sometimes reduce the edema while providing complementary blood-pressure control?

A. A large sodium supplement

B. A short-acting alpha agonist used intermittently

C. An ACE inhibitor or angiotensin receptor blocker

D. A second dihydropyridine calcium-channel blocker

141. A patient with hypertension also has frequent migraine and no asthma, bradycardia, or conduction disease. Which antihypertensive class can provide migraine prophylaxis in selected patients?

A. A beta blocker such as propranolol

B. An alpha-1 blocker as the preferred migraine prophylaxis

C. A loop diuretic used for routine migraine prevention

D. Hydralazine as a standard migraine preventive agent

142. A patient has hypertension and disabling essential tremor. Which medication can treat both conditions when no contraindication is present?

A. Propranolol

B. Hydralazine

C. Nifedipine immediate-release

D. Spironolactone

143. An older man has hypertension and symptomatic benign prostatic hyperplasia. Which drug can improve urinary flow while also lowering blood pressure, although it is not preferred as sole foundational antihypertensive therapy?

A. A short-acting nitrate used chronically as monotherapy

B. An alpha-1 blocker such as doxazosin

C. A mineralocorticoid receptor agonist

D. A direct renin activator

144. A pregnant patient with chronic hypertension requires oral pharmacologic treatment. Which medication is commonly used because of an established pregnancy safety profile?

A. Losartan, an angiotensin-receptor blocker

B. Lisinopril, an angiotensin-converting-enzyme inhibitor

C. Aliskiren, a direct renin inhibitor

D. Extended-release nifedipine for chronic blood-pressure therapy

145. A woman of childbearing potential with hypertension is taking an ACE inhibitor and is planning pregnancy. Which counseling is most appropriate?

A. Stop all blood-pressure monitoring until the second trimester

B. Add an ARB before conception to reduce fetal angiotensin II exposure

C. Transition to a pregnancy-compatible regimen before conception when feasible

D. Continue the ACE inhibitor through pregnancy because first-trimester exposure is protective

146. A patient has uncontrolled hypertension because three prescribed medications are taken at four different times each day and doses are frequently missed. Which redesign is most likely to improve adherence?

A. Use separate short-acting agents timed before meals to make each dose easier to remember

B. Use long-acting once-daily agents and fixed-dose combinations where possible

C. Keep the same drugs but divide each dose further to reduce peak adverse effects

D. Add one additional tablet while leaving the four-times-daily schedule unchanged

147. A patient taking an ACE inhibitor and thiazide develops higher blood pressure and a creatinine rise after starting naproxen. Which interaction is most likely?

A. Naproxen converts the thiazide into a loop diuretic

B. NSAID-related renal prostaglandin inhibition blunts pressure control and can reduce GFR

C. NSAIDs directly block aldosterone receptors and lower potassium

D. Naproxen potentiates ACE inhibition and causes excessive natriuresis

148. A patient takes an ACE inhibitor and diuretic and becomes volume depleted from gastroenteritis while continuing high-dose ibuprofen. Which complication is especially concerning?

A. Direct immune-mediated nephritis caused specifically by the three-drug combination

B. Permanent hypertension cure from transient sodium loss

C. Severe hyperfiltration caused by simultaneous afferent and efferent dilation

D. Acute kidney injury from combined efferent dilation, reduced volume, and afferent vasoconstriction

149. A patient with resistant hypertension is using a potassium-chloride salt substitute while taking lisinopril and spironolactone. Which laboratory finding would most strongly prompt stopping the salt substitute and reassessing the regimen?

A. A serum potassium concentration of 6.1 mmol/L

B. A serum sodium concentration of 133 mmol/L

C. A serum magnesium concentration of 1.6 mg/dL

D. A serum uric acid concentration of 8.2 mg/dL

150. A patient with refractory hypertension is being considered for hydralazine after optimization of diuretics and other agents. Which co-therapy is often needed because of hydralazine physiology?

A. An NSAID to preserve renal prostaglandin inhibition

B. Potassium supplementation and a stimulant to prevent bradycardia

C. A second direct arteriolar vasodilator without any diuretic

D. A diuretic and a rate-controlling agent to limit fluid retention and reflex tachycardia

151. A patient with bilateral severe renal-artery stenosis has pressure controlled on other agents but develops recurrent large creatinine rises whenever an ACE inhibitor is introduced. Which principle is most appropriate?

A. Avoid further routine RAAS blockade if the hemodynamic kidney response is repeatedly unacceptable

B. Continue the ACE inhibitor at a lower dose despite recurrent large creatinine rises because the response is expected

C. Switch from an ACE inhibitor to dual ACE-inhibitor and ARB therapy to stabilize glomerular pressure

D. Use a direct renin inhibitor together with the ACE inhibitor to preserve efferent tone

152. A patient starts chlorthalidone and spironolactone for resistant hypertension. Which laboratory surveillance is most important after initiation and dose changes?

A. Serum sodium and creatinine, with potassium checked only if symptoms develop

B. Serum potassium and uric acid, with kidney function checked only after several months

C. Serum sodium and potassium without reassessing kidney function after dose changes

D. Serum sodium, serum potassium, and kidney function checked after initiation and clinically important dose changes

153. A medication lowers office blood pressure substantially but has little effect on a patient's elevated 24-hour average because its action wears off overnight. Which pharmacologic property would be most useful in a replacement drug?

A. A rapid peak concentration that maximizes the office pre-visit effect

B. A short elimination half-life that permits symptom-triggered dosing

C. A long duration of action that provides sustained 24-hour control

D. A formulation with high peak-to-trough variability to avoid nocturnal exposure

154. A patient with true resistant hypertension consumes a high-sodium diet despite four medications. Which lifestyle intervention is likely to have particularly meaningful benefit?

A. Increasing processed-food intake to suppress aldosterone

B. Eliminating dietary potassium despite normal kidney function

C. Avoiding all physical activity to prevent transient pressure rises

D. Substantial dietary sodium reduction to address salt-sensitive volume expansion

155. A patient with eGFR 22 mL/min/1.73 m2 and potassium 5.5 mmol/L asks whether a potassium-rich salt substitute is advisable for hypertension. What is the best recommendation?

A. Use a potassium-chloride salt substitute but reduce the amount if weakness develops

B. Increase potassium intake through supplements rather than foods to allow exact dosing

C. Avoid unsupervised potassium loading because advanced CKD and hyperkalemia make it unsafe

D. Replace sodium with potassium freely after holding the RAAS blocker on high-potassium days

156. A patient with type 2 diabetes, albuminuric CKD, and hypertension is already taking an ARB. An SGLT2 inhibitor is added for kidney and cardiovascular protection. What blood-pressure effect is typically expected?

A. A consistent large increase in blood pressure from sodium retention

B. No hemodynamic effect of any kind in patients with kidney disease

C. A modest additional reduction rather than replacement of dedicated antihypertensive therapy

D. A guaranteed 40-mm Hg systolic fall that permits stopping all other drugs

157. A patient with obesity and hypertension loses substantial weight during evidence-based obesity treatment. Blood pressure falls to 108/64 mm Hg with lightheadedness while the same four antihypertensive drugs are continued. What is the best next step?

A. Stop measuring blood pressure during weight loss

B. Reassess and de-intensify antihypertensive therapy according to current pressure and symptoms

C. Continue all doses unchanged because prior hypertension prevents overtreatment

D. Add another medication to prevent rebound hypertension

158. A patient with diabetic kidney disease is taking finerenone for cardiorenal risk reduction. Which statement best characterizes its role in hypertension treatment?

A. It is a rapid arterial vasodilator used when hypertensive emergency accompanies diabetic kidney disease

B. It lowers blood pressure without materially affecting potassium because it lacks mineralocorticoid-receptor activity

C. It may modestly lower pressure but is not a substitute for standard antihypertensive therapy

D. It should replace conventional first-line antihypertensive therapy whenever diabetic kidney disease is present

159. A patient with diabetes is taking lisinopril. A clinician proposes adding aliskiren to intensify renin-angiotensin blockade. What is the best response?

A. Avoid the combination because dual RAAS blockade increases renal and potassium-related harm

B. Replace blood-pressure monitoring with renin levels after combination therapy

C. Add aliskiren because triple RAAS blockade is preferred in diabetes

D. Add aliskiren because the combination prevents hyperkalemia

160. A patient with severe refractory hypertension requires minoxidil after multiple other therapies have been optimized. Which combination best anticipates the drug's predictable compensatory effects?

A. Minoxidil plus withdrawal of all diuretics and beta blockade

B. Minoxidil plus a potent diuretic and an agent that controls reflex tachycardia

C. Minoxidil plus a high-sodium diet and stimulant medication

D. Minoxidil alone without monitoring for fluid retention

161. A 64-year-old patient has repeated blood pressure of 204/116 mm Hg but no chest pain, neurologic deficit, papilledema, acute kidney injury, heart failure, or other acute target-organ injury. What is the most appropriate general approach?

A. Admit for intravenous therapy because any pressure above 200 mm Hg constitutes hypertensive emergency

B. Lower mean arterial pressure by about 25 percent in the first hour despite the absence of acute organ injury

C. Treat as severe hypertension with timely oral medication adjustment and close outpatient follow-up

D. Observe without medication change unless acute target-organ injury appears later

162. A patient has hypertensive encephalopathy with blood pressure of 238/142 mm Hg. There is no aortic dissection or other condition requiring a different target. What is the usual initial reduction strategy?

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

B. Reduce systolic pressure below 90 mm Hg as quickly as possible

C. Reduce mean arterial pressure by roughly 20 to 25 percent during the first hour

D. Avoid any pressure reduction for the first 24 hours

163. A patient presents with acute type B aortic dissection, severe tearing pain, heart rate 112/min, and blood pressure 210/118 mm Hg. Which treatment principle is most important initially?

A. Use oral thiazide monotherapy because intravenous therapy is unnecessary

B. Rapid anti-impulse therapy with intravenous beta blockade followed by vasodilator therapy if needed

C. Begin a pure arteriolar vasodilator before controlling heart rate

D. Allow severe hypertension for 24 hours to preserve renal perfusion

164. A patient with acute ischemic stroke is not a candidate for thrombolysis or thrombectomy and has blood pressure of 198/112 mm Hg without another hypertensive emergency. Which approach is generally appropriate in the first day?

A. Lower systolic pressure to 140 mm Hg immediately to match the usual chronic treatment goal

B. Avoid routine aggressive lowering because permissive hypertension may support cerebral perfusion

C. Reduce systolic pressure by about 25 percent in the first hour as for hypertensive encephalopathy

D. Use anti-impulse therapy to reach systolic pressure near 120 mm Hg even without aortic disease

165. A patient with acute ischemic stroke is otherwise eligible for intravenous thrombolysis, but blood pressure is 198/116 mm Hg. What must occur before thrombolytic therapy?

A. Lower blood pressure below the accepted pretreatment threshold, approximately 185/110 mm Hg

B. Delay all pressure treatment until 24 hours after thrombolysis

C. Increase systolic pressure above 220 mm Hg to improve penumbral perfusion

D. Normalize blood pressure below 100/60 mm Hg before treatment

166. After successful intravenous thrombolysis for acute ischemic stroke, which systolic blood-pressure strategy is most consistent with the 2026 AHA/ASA guideline during the first 24 hours?

A. Allow systolic pressure to rise above 220 mm Hg to preserve reperfusion

B. Maintain systolic pressure in a range of approximately 140 to 180 mm Hg with frequent monitoring

C. Lower systolic pressure below 120 mm Hg as rapidly as possible

D. Stop blood-pressure monitoring once the thrombolytic infusion is complete

167. A patient with spontaneous intracerebral hemorrhage presents with systolic blood pressure of 188 mm Hg. Which general pressure-lowering approach is preferred when there is no contraindication?

A. Smooth intravenous lowering toward systolic 140 mm Hg while avoiding excessive reduction

B. Lower systolic pressure to 160–180 mm Hg and avoid further treatment during the first day

C. Reduce systolic pressure below 110 mm Hg to minimize hematoma expansion

D. Maintain systolic pressure near the presenting value unless neurologic status worsens

168. A patient with hypertensive emergency has acute pulmonary edema, severe dyspnea, diffuse crackles, and blood pressure of 224/128 mm Hg. Which intravenous strategy is particularly useful?

A. Immediate large-volume saline infusion to increase preload

B. Long-acting oral therapy alone despite respiratory distress

C. Titrated intravenous nitroglycerin, with loop diuresis if volume overloaded

D. Pure beta blockade without addressing severe afterload or congestion

169. A patient with acute coronary syndrome has severe hypertension and ongoing ischemic chest pain without acute heart failure. Which treatment combination can reduce pressure and myocardial oxygen demand when no contraindication is present?

A. Intravenous nitroglycerin with appropriate beta blockade and standard ACS therapy

B. High-dose NSAID therapy to reduce vascular inflammation

C. Immediate mineralocorticoid infusion to increase coronary perfusion

D. Hydralazine alone despite reflex tachycardia

170. A patient with known pheochromocytoma develops a hypertensive crisis with severe headache and a pressure of 240/130 mm Hg. Which intravenous agent most directly reverses catecholamine-mediated vasoconstriction through alpha-adrenergic blockade?

A. Intravenous nicardipine, a titratable dihydropyridine vasodilator

B. Intravenous clevidipine, an ultrashort-acting arterial vasodilator

C. Intravenous esmolol, an ultrashort-acting beta-1 adrenergic blocker

D. Intravenous phentolamine, a rapidly acting alpha-adrenergic antagonist

171. A pregnant patient at 34 weeks has persistent blood pressure of 172/114 mm Hg with headache. Which treatment principle is most appropriate?

A. Treat acute severe hypertension promptly with a pregnancy-appropriate rapid-acting agent

B. Begin oral maintenance therapy and reassess after several hours before treating the severe range

C. Use an ACE inhibitor because renin-angiotensin blockade is effective for rapid pressure control

D. Prioritize magnesium sulfate alone and defer antihypertensive treatment unless a seizure occurs

172. A woman at 18 weeks gestation has chronic hypertension with repeated blood pressures of 146/94 mm Hg and no acute symptoms. Based on contemporary post-CHAP guidance, which strategy is appropriate?

A. Withhold all treatment until severe hypertension develops

B. Initiate or titrate pregnancy-compatible therapy at a threshold around 140/90 mm Hg

C. Target immediate systolic pressure below 90 mm Hg

D. Use losartan because it is preferred in the second trimester

173. A patient with preeclampsia develops a generalized tonic-clonic seizure. Which medication is the treatment of choice for seizure prevention and recurrence while hypertension is managed separately?

A. Intravenous lorazepam as the routine first-line prophylactic anticonvulsant

B. Intravenous magnesium sulfate for treatment and prevention of recurrent eclamptic seizures

C. Intravenous levetiracetam as preferred prophylaxis for all patients with preeclampsia

D. Intravenous phenytoin as the standard first-line therapy for eclampsia

174. A breastfeeding patient has persistent postpartum hypertension and a compelling indication for renin-angiotensin system blockade. Which agent has the strongest established lactation experience among the following choices?

A. Aliskiren, an oral direct renin inhibitor

B. Losartan, an angiotensin II receptor blocker

C. Enalapril, an angiotensin-converting enzyme inhibitor

D. Sacubitril/valsartan, an angiotensin receptor–neprilysin inhibitor

175. A patient with non-dialysis CKD asks about the KDIGO blood-pressure target. Which statement accurately reflects the 2021 KDIGO recommendation when standardized office measurement is used and treatment is tolerated?

A. Target systolic blood pressure below 120 mm Hg in many adults, with individualization

B. Use a target below 130/80 mm Hg because KDIGO does not depend on standardized measurement technique

C. Target systolic pressure below 140 mm Hg because lower targets are discouraged in CKD

D. Apply the below-120-mm Hg target equally to casual nonstandardized readings and standardized readings

176. A hemodialysis patient has persistent hypertension, large interdialytic weight gains, and edema despite multiple antihypertensive drugs. Which intervention addresses the most likely dominant mechanism?

A. Stop ultrafiltration because volume does not influence dialysis hypertension

B. Reassess dry weight and reduce sodium and volume excess through the dialysis prescription and diet

C. Add medications indefinitely without reassessing extracellular volume

D. Increase dietary sodium to reduce intradialytic symptoms

177. A kidney-transplant recipient taking tacrolimus has hypertension with stable graft function and no edema. Which antihypertensive class is often useful because it counteracts calcineurin-inhibitor-related vasoconstriction?

A. A direct mineralocorticoid agonist

B. A short-acting nitrate as sole chronic therapy

C. A high-dose sympathomimetic

D. A dihydropyridine calcium-channel blocker

178. A patient with hypertension and heart failure with reduced ejection fraction needs treatment. Which principle best guides the antihypertensive regimen?

A. Choose antihypertensives primarily by pressure effect even if they lack heart-failure outcome benefit

B. Prioritize guideline-directed heart-failure therapies that also lower blood pressure and improve outcomes

C. Avoid renin-angiotensin and beta-blocking therapy because lower pressure can reduce cardiac output

D. Use a dihydropyridine calcium-channel blocker as the foundational mortality-reducing heart-failure therapy

179. A patient has heart failure with preserved ejection fraction and persistent blood pressure of 152/86 mm Hg. Which treatment goal is most appropriate?

A. Treat congestion with diuretics but leave systolic hypertension untreated to preserve ventricular filling

B. Control hypertension aggressively while treating congestion and associated comorbidities

C. Prioritize heart-rate acceleration to maintain cardiac output while accepting systolic pressure above 160 mm Hg

D. Use intermittent short-acting vasodilators during dyspnea rather than sustained blood-pressure control

180. A patient with chronic coronary disease and hypertension has frequent exertional angina and a resting heart rate of 88/min. Which antihypertensive most directly reduces heart rate and myocardial oxygen demand while also lowering blood pressure, when no contraindication is present?

A. A beta blocker with proven antianginal heart-rate-lowering effects

B. A thiazide-like diuretic used primarily for natriuresis and pressure reduction

C. An ACE inhibitor used primarily for renin-angiotensin system blockade

D. A dihydropyridine calcium-channel blocker that primarily lowers vascular resistance

181. A patient with recent myocardial infarction has hypertension and preserved hemodynamics. Which class is particularly useful early when there is no contraindication?

A. A mineralocorticoid agonist to retain sodium

B. A high-dose alpha agonist to raise afterload

C. A beta blocker as part of appropriate post-infarction therapy

D. A pure direct arteriolar vasodilator that provokes tachycardia

182. A patient has hypertension, persistent atrial fibrillation with rapid ventricular response, and preserved ejection fraction. Which drug can treat both the ventricular rate and blood pressure?

A. Diltiazem

B. Hydrochlorothiazide

C. Spironolactone

D. Hydralazine

183. A patient with thoracic aortic aneurysm has hypertension and a resting heart rate of 92/min. Which therapeutic principle is most relevant to reducing aortic wall stress?

A. Encourage marked uncontrolled hypertension to maintain aortic perfusion

B. Use a stimulant to increase heart rate and contractility

C. Control blood pressure and reduce impulse with beta blockade when appropriate

D. Avoid all antihypertensive therapy until dissection occurs

184. A patient with hypertension and symptomatic peripheral artery disease has an indication for beta blockade after myocardial infarction. Which statement is most accurate?

A. Avoid beta blockade and substitute an alpha-1 blocker because peripheral vasodilation is required in PAD

B. Use beta blockade only after limb revascularization because it otherwise predictably worsens claudication

C. Peripheral artery disease alone is not a reason to withhold an otherwise indicated beta blocker

D. Replace the post-infarction beta blocker with a direct vasodilator solely to preserve limb perfusion

185. A patient with long-standing diabetes has supine blood pressure of 158/82 mm Hg but falls to 116/64 mm Hg on standing with dizziness and minimal heart-rate increase. Which mechanism is most likely?

A. Renal artery stenosis causing obligatory tachycardia on standing

B. Diabetic autonomic neuropathy with impaired reflex sympathetic compensation

C. Normal baroreflex function with exaggerated sympathetic activation

D. Primary aldosteronism causing isolated postural hypotension

186. An 89-year-old nursing-home resident has advanced dementia, recurrent falls, orthostatic symptoms, and seated blood pressure of 132/70 mm Hg on four medications. Which management principle is most appropriate?

A. Maintain the current four-drug regimen because seated systolic pressure above 130 mm Hg warrants treatment regardless of frailty

B. Intensify therapy because recurrent falls are more likely from uncontrolled pressure than medication effects

C. Add a fifth drug while using standing pressure only to monitor safety after the target is reached

D. Individualize the target and consider de-intensification to reduce treatment-related harm

187. A patient treated for hypertension reports recurrent presyncope on standing. Which medication review strategy is most appropriate?

A. Stop measuring standing blood pressure because it confuses management

B. Add an alpha-1 blocker at bedtime without reviewing the current regimen

C. Increase all vasodilator doses because orthostatic symptoms indicate undertreatment

D. Identify drugs that worsen orthostasis and reduce or retime them while preserving necessary pressure control

188. A patient with severe autonomic failure has disabling daytime orthostatic hypotension and marked supine hypertension at night. Which nonpharmacologic measure can reduce nocturnal supine pressure without worsening daytime standing pressure?

A. Increase evening sodium and fluid intake substantially

B. Lie completely flat for the entire night

C. Sleep with the head of the bed elevated

D. Take a long-acting daytime vasodilator immediately before standing

189. A patient has recurrent paroxysmal hypertension, flushing, palpitations, and fear. Repeated high-quality plasma and urinary metanephrine testing is normal, and imaging is unrevealing. Episodes follow emotional stress. Which next concept is most appropriate?

A. Treat empirically for pheochromocytoma indefinitely despite repeated normal high-quality biochemical testing

B. Consider panic-related or pseudopheochromocytoma syndromes after true catecholamine tumor has been excluded

C. Proceed to adrenal venous sampling to localize occult catecholamine secretion

D. Diagnose primary aldosteronism as the explanation for the episodic adrenergic symptoms

190. A 10-year-old child has repeated auscultatory blood pressures above the 95th percentile for age, sex, and height. Which principle is correct for classifying pediatric hypertension at this age?

A. Use the adult 130/80-mm Hg threshold because percentile-based classification stops after age 6

B. Use the adult 140/90-mm Hg threshold because any lower value is normal before adolescence

C. Classify from a single oscillometric value if it exceeds the 95th percentile by at least 10 mm Hg

D. Use age-, sex-, and height-based percentile thresholds rather than adult cut points alone

191. A 12-year-old has persistently elevated office blood pressures on three visits but no symptoms. Which test is particularly useful to confirm sustained hypertension and exclude a white-coat pattern?

A. Coronary angiography

B. A single home smartwatch reading

C. Ambulatory blood-pressure monitoring

D. Adrenal venous sampling

192. A 7-year-old child has confirmed stage 2 hypertension, poor growth, and abnormal urinalysis. Which approach is most appropriate?

A. Begin lifestyle therapy first and defer renal evaluation unless hypertension persists for six months

B. Evaluate only for endocrine causes because abnormal urinalysis is common in primary pediatric hypertension

C. Perform a thorough evaluation for secondary causes, especially renal disease

D. Treat pharmacologically first and postpone etiologic evaluation until blood pressure is controlled

193. A 15-year-old with obesity, family history of hypertension, normal urinalysis, and normal kidney function has repeated blood pressures of 136/84 mm Hg. Which cause is most likely after appropriate confirmation?

A. Primary hypertension associated with obesity and cardiometabolic risk

B. Pheochromocytoma as the most likely cause in every obese adolescent

C. Liddle syndrome despite normal potassium and no early family pattern

D. Coarctation despite normal femoral pulses and no pressure gradient

194. A 13-year-old has hypertension in both arms, weak femoral pulses, and a systolic murmur over the back. Which next test is most appropriate?

A. Renal ultrasonography to evaluate renal parenchymal causes before addressing the pulse discrepancy

B. Adrenal venous sampling to exclude unilateral primary aldosteronism

C. CT angiography of the renal arteries as the first test for the upper-to-lower-extremity gradient

D. Echocardiography to assess the aortic arch and associated cardiac anatomy

195. A pregnant patient with hypertension is mistakenly continued on an ACE inhibitor into the second trimester. Which fetal complication is a major concern?

A. Congenital hyperthyroidism caused by ACE inhibition

B. Neonatal polycythemia from excessive erythropoietin stimulation

C. Fetal renal injury with oligohydramnios and related developmental complications

D. Fetal hyperinsulinemia caused by increased angiotensin II

196. A patient with stage 4 CKD has resistant hypertension on an ARB, amlodipine, and loop diuretic. Potassium is 5.4 mmol/L, making spironolactone difficult to use. Which additional option has evidence for lowering blood pressure even in advanced CKD?

A. Immediate bilateral nephrectomy for pharmacologic resistance

B. A carefully monitored thiazide-like diuretic such as chlorthalidone

C. Routine dual ACE-inhibitor and ARB therapy

D. High-dose potassium supplementation before adding a diuretic

197. A patient asks whether renal denervation should replace all antihypertensive medication. Which statement best reflects current evidence?

A. Use renal denervation before confirming sustained out-of-office hypertension because office pressure determines candidacy

B. Adjunctive therapy for selected uncontrolled patients; medications often remain necessary

C. Reserve it for patients who have never tolerated any antihypertensive medication, regardless of pressure burden

D. Expect most treated patients to discontinue all antihypertensive medications if the procedure is technically successful

198. A patient has extreme office blood-pressure variability linked to severe anxiety, but repeated ambulatory averages are controlled and there is no acute organ injury. Which management approach is most appropriate?

A. Treat the office values as the primary therapeutic target because anxiety-related sympathetic activation carries the same exposure as sustained hypertension

B. Add a short-acting rescue antihypertensive for each anxiety-related office spike

C. Escalate the chronic regimen until the highest office readings reach goal despite controlled ambulatory averages

D. Use out-of-office data to avoid overtreatment while addressing anxiety and measurement conditions

199. A patient has episodic palpitations and hypertension. Which finding most strongly favors panic attacks rather than pheochromocytoma after appropriate evaluation?

A. Repeated normal metanephrine testing during a well-conducted endocrine evaluation

B. A pathogenic catecholamine-tumor syndrome with compatible biochemical findings

C. Markedly elevated plasma free metanephrines on repeated testing

D. A biochemically active adrenal chromaffin tumor on imaging and laboratory testing

200. A patient with uncontrolled hypertension cannot afford several prescriptions, has difficulty arranging transportation, and misses follow-up visits. Which intervention is most likely to improve long-term control?

A. Refer directly for renal denervation before addressing medication access and missed doses

B. Use team-based care to simplify therapy and address medication access, monitoring, and follow-up barriers

C. Add another long-acting antihypertensive while leaving affordability and transportation barriers unchanged

D. Begin an extensive secondary-hypertension workup before first clarifying adherence and access problems

Answers

1. A. Chronic hypertension shifts both arterial baroreflex operating points and cerebral autoregulation toward higher pressures. A rapid reduction to a pressure that is numerically normal can therefore transiently fall below the adapted autoregulatory range and reduce cerebral perfusion. This is one reason hypertensive emergencies are generally treated with controlled rather than abrupt pressure reduction, except in selected conditions with different targets.

2. D. Long-term arterial pressure is tightly linked to renal sodium handling. In salt-sensitive hypertension, the kidney requires a higher perfusion pressure to achieve sodium balance, producing a rightward shift of the pressure-natriuresis relationship. Persistent sodium retention expands extracellular volume until arterial pressure rises enough to restore natriuresis and equilibrium.

3. D. Dietary sodium restriction reduces effective circulating volume and renal chloride delivery to the macula densa, increasing renin release. Renin generates angiotensin II and stimulates aldosterone, which together promote vasoconstriction and renal sodium retention. The coordinated renin-angiotensin-aldosterone system response helps maintain perfusion and extracellular volume.

4. B. Angiotensin II preferentially constricts the efferent arteriole at physiologic concentrations. This raises intraglomerular hydraulic pressure and helps preserve filtration when renal perfusion falls. Marked blockade of this response with an ACE inhibitor or ARB can therefore cause a substantial

creatinine rise when glomerular filtration depends heavily on efferent constriction, such as bilateral severe renal artery stenosis.

5. D. Aldosterone acts through the mineralocorticoid receptor in collecting-duct principal cells to increase ENaC activity and basolateral sodium-potassium ATPase function. Sodium reabsorption creates a lumen-negative potential that favors potassium and hydrogen secretion. This mechanism explains the hypertension and hypokalemic alkalosis that can accompany mineralocorticoid excess.

6. B. Renal sympathetic nerves stimulate beta-1 adrenergic receptors on juxtaglomerular cells, increasing renin secretion. Sympathetic activation also raises vascular resistance and enhances tubular sodium reabsorption. Beta blockade can therefore lower pressure partly by reducing cardiac output and suppressing renin release.

7. A. Nitric oxide activates soluble guanylate cyclase in vascular smooth muscle, increasing cyclic GMP and protein kinase G signaling. The resulting fall in intracellular calcium and reduced contractile signaling produces vasodilation. Endothelial dysfunction in hypertension can impair this nitric-oxide pathway and favor increased vascular resistance.

8. A. Endothelin-1 is among the most potent endogenous vasoconstrictors and also has trophic effects on vascular smooth muscle. By contrast, atrial natriuretic peptide, bradykinin, and prostacyclin generally favor vasodilation or natriuresis. Endothelin signaling contributes to several forms of vascular disease, although endothelin antagonists are not routine therapy for primary systemic hypertension.

9. B. With aging, fragmentation of elastin, collagen deposition, calcification, and other structural changes stiffen the aorta and large arteries. Reduced arterial compliance raises systolic pressure and pulse pressure, while reflected pressure waves return earlier in systole. This physiology is central to isolated systolic hypertension in older adults.

10. B. Stiffer arteries conduct pulse waves more rapidly, causing reflected waves to return during late systole rather than diastole. This augments central systolic pressure and left ventricular afterload and can reduce the diastolic pressure support that otherwise favors coronary perfusion. The effect contributes to systolic hypertension and ventricular hypertrophy.

11. C. For a pressurized chamber, wall stress rises with pressure and radius and falls as wall thickness increases. Chronic systemic pressure overload therefore promotes parallel addition of sarcomeres and concentric wall thickening, which initially reduces wall stress. Over time, the same remodeling can impair relaxation, increase myocardial oxygen demand, and contribute to heart failure.

12. A. Chronic hypertension causes structural and functional adaptation of cerebral resistance vessels, shifting the autoregulatory curve to the right. Cerebral perfusion can remain stable at higher pressures, but overly rapid normalization may cross the shifted lower limit and cause ischemia. The curve is not abolished; rather, its operating range is altered.

13. D. Salt sensitivity refers to a substantial blood-pressure response to changes in sodium intake. It reflects interactions among renal sodium handling, the renin-angiotensin-aldosterone system, sympathetic activity, vascular function, age, kidney disease, and genetic factors. It is not synonymous with any particular office or ambulatory blood-pressure pattern.

14. D. Obesity can increase sympathetic nervous-system activity, activate the renin-angiotensin-aldosterone system, alter renal pressure-natriuresis, and promote sodium retention. Adipose-derived signals, including leptin-related pathways, contribute to sympathetic activation. Obstructive sleep apnea and metabolic kidney effects can amplify these mechanisms.

15. B. Chronic hypertension is associated with eutrophic or hypertrophic remodeling of small arteries, producing a higher media-to-lumen ratio. Even modest narrowing of the vessel lumen markedly increases resistance because resistance varies approximately with the inverse fourth power of radius. Remodeling also helps sustain hypertension after the initiating stimulus has changed.

16. C. A lower nephron number can increase sodium-retaining pressure requirements and place greater filtration and hemodynamic demand on each nephron. This concept is one proposed link between adverse fetal growth and adult hypertension. It illustrates how renal developmental reserve can influence the pressure-natriuresis relationship later in life.

17. C. ACE degrades bradykinin as well as converting angiotensin I to angiotensin II. ACE inhibition therefore increases bradykinin, contributing to vasodilation but also to cough and, rarely, angioedema. Angiotensin receptor blockers do not inhibit bradykinin breakdown, which explains their much lower incidence of cough.

18. B. ACE2 metabolizes angiotensin peptides to generate angiotensin-(1-7), which signals through the Mas receptor and generally opposes vasoconstrictor, proliferative, and sodium-retaining actions of the classic ACE-angiotensin II-AT1 pathway. The balance among these pathways influences vascular and renal homeostasis.

19. B. Atrial natriuretic peptide is released in response to myocardial stretch and promotes natriuresis, vasodilation, and suppression of renin and aldosterone. These actions oppose volume expansion. Natriuretic peptide systems are therefore physiologic counter-regulators of sodium-retaining and vasoconstrictor pathways.

20. D. Renal dopamine is produced locally from filtered or circulating precursors and inhibits sodium transport in several nephron segments, helping excrete a sodium load. Impaired renal dopaminergic signaling has been studied as one contributor to salt-sensitive hypertension. Its renal role differs from the systemic catecholamine effects of dopamine at pharmacologic doses.

21. C. Liddle syndrome is caused by activating abnormalities of ENaC, leading to excessive distal sodium reabsorption, volume expansion, hypertension, potassium loss, and metabolic alkalosis. Because

the volume expansion suppresses the renin-angiotensin-aldosterone system, both renin and aldosterone are low. Amiloride or triamterene directly blocks ENaC and is mechanistically appropriate treatment.

22. D. Gordon syndrome, or pseudohypoaldosteronism type II, results from abnormalities in the WNK-SPAK/OSR1 pathway that increase thiazide-sensitive NCC activity. The characteristic combination is hypertension with hyperkalemia and hyperchloremic metabolic acidosis. Low-dose thiazide therapy is particularly effective because it targets the overactive transporter.

23. B. 11-beta-hydroxysteroid dehydrogenase type 2 converts cortisol to cortisone in mineralocorticoid target tissues. Deficiency allows abundant cortisol to activate mineralocorticoid receptors, producing apparent mineralocorticoid excess with sodium retention, hypokalemia, metabolic alkalosis, suppressed renin, and suppressed aldosterone. Licorice can produce a similar acquired phenotype by inhibiting this enzyme.

24. A. Familial hyperaldosteronism type I, also called glucocorticoid-remediable aldosteronism, results from a chimeric gene that places aldosterone synthase activity under ACTH control. Suppressing ACTH with low-dose glucocorticoid can reduce aldosterone production. The biochemical pattern remains one of renin suppression with inappropriate aldosterone production.

25. B. Norepinephrine and epinephrine can produce intense vasoconstriction through vascular alpha-1 receptors, while beta-1 stimulation increases heart rate and contractility. This receptor physiology explains the treatment sequence for catecholamine-producing tumors: establish alpha blockade before introducing beta blockade, because unopposed alpha vasoconstriction can otherwise worsen hypertension.

26. D. Glucocorticoid excess can raise blood pressure through several mechanisms, including increased vascular reactivity, effects on renal sodium handling, and at sufficiently high concentrations mineralocorticoid-receptor activation when local cortisol inactivation is overwhelmed. The result is often volume expansion and increased systemic vascular resistance rather than sodium wasting.

27. B. Repetitive upper-airway obstruction produces intermittent hypoxemia, arousals, large intrathoracic pressure swings, oxidative stress, and recurrent sympathetic surges. These effects can persist into daytime and contribute to resistant and nocturnal hypertension. Obesity and aldosterone-related fluid shifts may further reinforce the association.

28. D. The compliant aorta normally buffers pulsatile ejection by expanding during systole and recoiling during diastole. Age-related stiffening reduces this Windkessel function, raising systolic and pulse pressures and accelerating pulse-wave velocity. Cardiac output does not need to increase for systolic pressure to rise.

29. A. Blood pressure normally falls during sleep. A decline of less than about 10% from daytime to nighttime average is commonly termed a nondipping or blunted-dipping pattern. Nondipping is

associated with conditions such as sleep apnea, chronic kidney disease, autonomic dysfunction, and higher cardiovascular risk, although the pattern alone does not identify a specific cause.

30. D. Masked hypertension exposes the vasculature, heart, brain, and kidneys to elevated pressure outside the clinic even though office readings appear normal. Because the true pressure burden is missed by clinic measurement, untreated risk can approach that of sustained hypertension. Home or ambulatory monitoring is therefore important when the phenotype is suspected.

31. C. Using a cuff that is too small can overestimate blood pressure. Proper technique requires a validated device and a cuff with bladder dimensions appropriate for the measured arm circumference. A fixed arithmetic correction is not reliable, and wrist measurement is not the preferred substitute when an appropriately sized upper-arm cuff can be used.

32. B. Standardized office measurement includes a period of seated rest, back and feet support, an arm supported at heart level, an appropriately sized cuff on bare skin, and repeated measurements that are averaged. Technique errors can produce clinically important bias and can create apparent treatment resistance or obscure true hypertension.

33. A. White-coat hypertension is characterized by elevated office pressure with normal out-of-office measurements in an untreated patient. Ambulatory or validated home monitoring is used to confirm the phenotype. It is distinct from white-coat effect in a treated patient and from masked hypertension, in which office pressure is normal but out-of-office pressure is elevated.

34. D. Masked hypertension is normal blood pressure in the clinic with elevated pressure outside the clinic. It is clinically important because the patient experiences a sustained pressure burden that routine office measurement misses. Ambulatory or validated home monitoring confirms the diagnosis and helps avoid false reassurance from normal clinic readings.

35. A. A sustained fall in systolic pressure of at least 20 mm Hg or diastolic pressure of at least 10 mm Hg within three minutes of standing is consistent with orthostatic hypotension, particularly when symptoms occur. Evaluation should consider volume depletion, autonomic dysfunction, and medications while balancing the need for control of seated or supine hypertension.

36. A. A reproducible, substantial inter-arm pressure difference with pulse asymmetry suggests subclavian or other proximal arterial obstruction. Initial assessment should measure both arms; subsequent management generally uses the arm with the higher pressure unless a specific vascular issue dictates otherwise. A persistent difference can also be a marker of vascular disease.

37. D. Reliable home monitoring uses a validated upper-arm cuff, standardized seated technique, and repeated measurements over several days rather than symptom-triggered spot checks. Averaging multiple readings reduces random variation. Current hypertension guidance advises against relying on cuffless devices for clinical decisions until their precision and reliability are adequately established.

38. D. Ambulatory monitoring uniquely characterizes sleep blood pressure and circadian patterns. Nocturnal hypertension and nondipping are common in chronic kidney disease and sleep apnea and can carry prognostic information independent of office readings. The finding does not by itself identify a specific secondary cause.

39. C. A urine albumin-to-creatinine ratio provides a practical quantitative assessment of albuminuria and is central to chronic kidney disease staging and cardiovascular risk assessment. Serum creatinine and estimated GFR complement it, because kidney damage may be present even when filtration remains relatively preserved.

40. A. ECG evidence of left ventricular hypertrophy is specific in some settings but relatively insensitive. Echocardiography can quantify left ventricular mass, wall thickness, chamber geometry, systolic function, and diastolic abnormalities. It is not required for every patient with hypertension, but it provides more direct characterization when target-organ assessment will affect management.

41. D. Hypertensive emergency is defined by severe blood-pressure elevation accompanied by acute hypertension-mediated target-organ injury. Neurologic dysfunction and advanced acute retinopathy support that diagnosis. The pressure number alone does not distinguish emergency from severe hypertension without acute organ injury, which is generally managed with oral therapy rather than rapid intravenous lowering.

42. B. ACE inhibitors reduce efferent arteriolar tone and can produce a modest early fall in intraglomerular pressure, causing a small creatinine increase. A rise of this magnitude can be acceptable if it stabilizes and potassium and volume status are monitored. A larger progressive increase should prompt evaluation for volume depletion, NSAID use, renal artery disease, or other contributors.

43. C. Pulse pressure is the difference between systolic and diastolic blood pressure. It commonly widens with large-artery stiffness and isolated systolic hypertension in older adults. Although mean arterial pressure is also relevant to perfusion, it does not specifically capture the systolic-diastolic separation highlighted in this case.

44. A. Irregular rhythms can increase beat-to-beat variability and reduce the precision of a single oscillometric reading. Repeated measurements with a validated device, careful technique, and confirmation by auscultation when needed are appropriate. Treatment decisions should not be based on a single extreme value when measurement reliability is uncertain.

45. A. True resistant hypertension requires confirmation that prescribed therapy is actually being taken at appropriate doses and that elevated pressure persists outside the clinic. Medication reconciliation, refill history, direct discussion, and in selected cases biochemical drug testing can help assess adherence, while home or ambulatory monitoring excludes a white-coat effect.

46. D. Blood-pressure values depend on the measurement setting and technique. Diagnosis and management should use repeated standardized measurements and, when appropriate, out-of-office data

to identify white-coat or masked patterns. A single value is less informative than a reliable average obtained with a validated method.

47. C. Kidney involvement is characterized by both filtration and structural or glomerular injury. Estimated GFR assesses filtration, while albuminuria quantified by urine albumin-to-creatinine ratio is a key marker of kidney damage and cardiovascular risk. Secondary-cause testing is guided by clinical suspicion rather than performed universally at this level of intensity.

48. B. Current hypertension guidance supports validated cuff-based home monitoring and cautions against relying on cuffless devices, including smartwatch estimates, for diagnosis or medication titration until accuracy and reliability are better established. Convenience does not substitute for validation against accepted measurement standards.

49. A. Early onset, abrupt worsening, resistant hypertension, hypokalemia, unusual severity, and suggestive symptoms or examination findings increase the likelihood of a secondary cause. Evaluation should be targeted to the clinical pattern rather than indiscriminate, but this presentation warrants specific assessment for causes such as primary aldosteronism and renovascular or renal disease.

50. A. Resistant hypertension cannot be diagnosed until adherence and out-of-office pressure are established. Objective drug testing can reveal partial or complete nonadherence when the clinical picture is uncertain. If prescribed medications are absent, the case is pseudoresistance or apparent treatment-resistant hypertension rather than proven true resistance.

51. D. The 2025 Endocrine Society guideline broadened case detection by suggesting primary aldosteronism screening in all individuals with hypertension, while recognizing that implementation depends on resources and local practice. Screening uses plasma or serum aldosterone with renin and calculation of an aldosterone-to-renin ratio. Potassium is measured alongside these tests because hypokalemia can suppress aldosterone and obscure the result.

52. A. Hypokalemia can suppress aldosterone secretion and produce a falsely low aldosterone concentration. When suspicion remains and potassium is low, potassium should be corrected into the reference range before repeating screening. Sodium restriction immediately before testing can also raise renin and reduce sensitivity, so patients are generally advised not to restrict sodium for the screening measurement.

53. D. Mineralocorticoid-receptor antagonists interfere substantially with renin and aldosterone physiology, often increasing renin and changing the screening ratio. The 2025 guideline allows medication-management strategies tailored to safety and feasibility rather than a single rigid washout for every patient. Results must be interpreted in the context of interfering drugs and pretest probability.

54. B. The hallmark of primary aldosteronism is renin suppression with aldosterone production that is inappropriate for the sodium and volume state. Hypokalemia strengthens the clinical suspicion but is not

required. The aldosterone-to-renin ratio is useful because it integrates the inappropriate aldosterone concentration with the suppressed renin signal.

55. B. In a patient with a high pretest probability and overt biochemical evidence of renin-independent aldosterone production, the 2025 Endocrine Society guideline notes that aldosterone-suppression testing may be unnecessary. Empiric mineralocorticoid-receptor antagonist therapy can also be reasonable when a patient will not pursue adrenal venous sampling or surgery. Confirmation is most useful when it will change a decision about surgical eligibility.

56. A. Adrenal nodules are common and CT anatomy does not reliably identify which adrenal gland is producing excess aldosterone. In surgical candidates, adrenal venous sampling is generally recommended to distinguish unilateral from bilateral secretion. CT remains important for anatomy and excluding concerning masses, but it is not a functional lateralization test.

57. C. Guidelines allow a potential exception to adrenal venous sampling in selected patients younger than about 35 years who have marked biochemical primary aldosteronism, spontaneous hypokalemia, and a clear unilateral adrenal adenoma. The exception is narrow; most surgical candidates still benefit from functional lateralization because incidental nonfunctioning nodules are common.

58. A. For lateralizing primary aldosteronism in a patient who desires and can undergo surgery, unilateral adrenalectomy is the preferred targeted therapy. It can normalize hypokalemia and substantially improve or cure hypertension. Bilateral adrenalectomy is not appropriate for routine unilateral disease, and nonspecific antihypertensive therapy does not address the excess aldosterone exposure as directly.

59. B. Bilateral primary aldosteronism is usually treated medically with a mineralocorticoid-receptor antagonist such as spironolactone or eplerenone. Therapy targets blood pressure, potassium, and the adverse effects of aldosterone excess. Unilateral adrenalectomy is appropriate only when aldosterone secretion clearly lateralizes and surgery is desired.

60. D. In medically treated primary aldosteronism, persistent renin suppression with uncontrolled blood pressure can indicate insufficient blockade of aldosterone-mediated sodium retention. The 2025 Endocrine Society guideline suggests increasing PA-specific medical therapy to produce a rise in renin when hypertension remains uncontrolled, with close monitoring of potassium, renal function, and blood pressure.

61. D. Fibromuscular dysplasia is a nonatherosclerotic, noninflammatory arterial disorder that commonly affects renal arteries and can cause renovascular hypertension, often in younger or middle-aged women. Atherosclerotic renal-artery disease is more typical in older patients with diffuse atherosclerotic risk and ostial lesions.

62. A. Recurrent flash pulmonary edema, rapidly worsening kidney function, and difficult hypertension in a patient with extensive atherosclerosis are classic clues to significant renovascular disease. Most

atherosclerotic renal-artery stenoses are managed medically, but selected high-risk syndromes such as recurrent pulmonary edema can prompt consideration of revascularization after careful anatomic and clinical assessment.

63. A. When renal perfusion is severely reduced, angiotensin II-mediated efferent constriction helps maintain intraglomerular pressure. ACE inhibition removes that support, so GFR can fall sharply in bilateral significant stenosis or stenosis to a solitary functioning kidney. A large creatinine rise should prompt assessment for renovascular disease, volume depletion, NSAID exposure, and other contributors.

64. B. CT angiography can define renal-artery anatomy and is useful when kidney function and contrast risk are acceptable. Duplex ultrasonography and MR angiography are alternatives depending on expertise, body habitus, kidney function, and local resources. The best test is individualized rather than universal.

65. D. For renal fibromuscular dysplasia, percutaneous transluminal angioplasty is the usual revascularization approach when intervention is appropriate. Stents are generally reserved for complications or unsuccessful angioplasty rather than used routinely. This differs from management of atherosclerotic disease, where intervention is selective and medical therapy remains central.

66. A. Catecholamine-producing tumors continuously metabolize catecholamines to metanephrines, making plasma free or urinary fractionated metanephrines sensitive screening tests. Sampling conditions and medications can influence false-positive results. Imaging is generally pursued after biochemical evidence supports the diagnosis rather than used as the initial screening step.

67. C. Preoperative alpha blockade reduces catecholamine-mediated vasoconstriction and helps prevent severe perioperative hypertension. Beta blockade can be added after adequate alpha blockade for persistent tachycardia. Starting beta blockade first risks unopposed alpha-adrenergic vasoconstriction and potentially dangerous hypertension.

68. D. Von Hippel-Lindau syndrome can include pheochromocytoma or paraganglioma along with retinal and central nervous system hemangioblastomas, renal cell carcinoma, and other tumors. Pheochromocytoma also occurs with MEN2, neurofibromatosis type 1, and pathogenic variants affecting succinate dehydrogenase pathways, so genetic evaluation is important in appropriate patients.

69. B. Accepted screening approaches for Cushing syndrome include the 1-mg overnight dexamethasone suppression test, late-night salivary cortisol, or 24-hour urinary free cortisol in appropriate settings. The phenotype in this case raises suspicion for glucocorticoid excess, which can cause hypertension through vascular and renal mechanisms.

70. A. Insulin-like growth factor 1 is the preferred initial biochemical screen for acromegaly because it reflects integrated growth-hormone activity more reliably than a random growth-hormone level. Acromegaly can contribute to hypertension through sodium retention, vascular changes, insulin resistance, and obstructive sleep apnea.

71. A. Thyroid hormone excess increases heart rate, contractility, and cardiac output and can lower systemic vascular resistance, producing a widened pulse pressure and systolic hypertension. Hypothyroidism more often increases systemic vascular resistance and may produce predominantly diastolic hypertension.

72. D. Hypothyroidism can increase systemic vascular resistance and is associated with diastolic hypertension in some patients. Treating the thyroid disorder may improve pressure. The pattern contrasts with hyperthyroidism, which more commonly causes tachycardia, increased cardiac output, and systolic hypertension with widened pulse pressure.

73. C. Primary hyperparathyroidism can be associated with hypertension, although the mechanism is multifactorial and blood pressure may not normalize after parathyroid treatment. Hypercalcemia with kidney stones strongly supports evaluation with parathyroid hormone measurement. The accompanying biochemical clues are essential because hypertension alone is nonspecific.

74. C. Coarctation causes upper-body hypertension with reduced or delayed lower-extremity pulses and a pressure gradient between arms and legs. A murmur over the back or collateral vessels may be present. Imaging with echocardiography, CT angiography, or MR angiography defines the anatomy and associated cardiac abnormalities.

75. D. Obstructive sleep apnea is diagnosed with objective sleep testing in the appropriate clinical setting. Polysomnography is the reference laboratory study, while validated home sleep apnea testing is appropriate for selected uncomplicated patients. Identifying apnea is important in resistant hypertension, but symptoms and questionnaires alone do not establish severity.

76. B. Treating obstructive sleep apnea improves airway obstruction and has cardiovascular and symptomatic benefits, but the average blood-pressure reduction from CPAP is usually modest. The effect can be greater with good nightly adherence, high baseline pressure, severe apnea, or resistant hypertension. Antihypertensive therapy and lifestyle management therefore usually remain necessary.

77. A. As nephron function declines, the kidney may require higher pressures and greater fractional sodium excretion to maintain sodium balance. Sodium and volume retention are common major contributors to hypertension in advanced CKD, although sympathetic and renin-angiotensin activation can also contribute. Assessing volume status is therefore central to treatment.

78. C. Abnormal urinalysis, proteinuria, and impaired filtration point toward renal parenchymal disease as a possible cause or consequence of hypertension. Evaluation includes kidney function, quantified albuminuria or proteinuria, urine sediment, and renal imaging or disease-specific studies when indicated. The findings should not be attributed automatically to primary hypertension.

79. C. Liddle syndrome is caused by constitutive ENaC activation. Amiloride or triamterene directly blocks ENaC and reverses sodium retention and potassium wasting. Spironolactone is often ineffective

because aldosterone is already suppressed and the abnormal channel activity is independent of mineralocorticoid-receptor stimulation.

80. A. Glycyrrhetinic acid from licorice inhibits 11-beta-HSD2, reducing conversion of cortisol to cortisone in mineralocorticoid-sensitive tissues. Cortisol then activates mineralocorticoid receptors, causing sodium retention, hypertension, hypokalemia, and metabolic alkalosis with suppression of both renin and aldosterone.

81. B. Gordon syndrome results from increased activity of the thiazide-sensitive sodium-chloride cotransporter through WNK-pathway abnormalities. Thiazide therapy directly blocks NCC and can correct both hypertension and hyperkalemia. The phenotype is therefore almost a mirror image of Gitelman syndrome, in which NCC function is reduced.

82. A. Very young onset of primary aldosteronism or a family history of early aldosteronism or premature stroke raises concern for familial hyperaldosteronism. Current guidance supports genetic evaluation in these settings. Identifying a heritable form can alter treatment and enables appropriate testing of first-degree relatives.

83. C. A juxtaglomerular cell tumor, or reninoma, can cause severe high-renin hypertension with secondary hyperaldosteronism, hypokalemia, and metabolic alkalosis in a young patient. The renal mass and high renin distinguish it from primary aldosteronism and monogenic low-renin syndromes.

84. C. External compression of renal parenchyma can reduce local perfusion, activating the renin-angiotensin-aldosterone system and causing secondary hypertension. This Page kidney phenomenon can follow trauma, biopsy, transplantation, or spontaneous subcapsular bleeding. Management depends on severity, kidney function, and whether the compressive lesion resolves.

85. B. NSAIDs inhibit cyclooxygenase-dependent renal prostaglandin synthesis, which can reduce renal blood flow in susceptible patients, promote sodium retention, and attenuate the effects of several antihypertensive agents. They are common contributors to difficult blood-pressure control and can also increase acute kidney injury risk in certain drug combinations.

86. C. Estrogen-containing contraceptives can raise blood pressure in susceptible patients, partly through increased hepatic angiotensinogen production and activation of renin-angiotensin signaling. Medication review is therefore an important part of evaluating new or worsening hypertension, particularly when the timing of onset follows a new agent.

87. A. Pseudoephedrine and related sympathomimetics can increase blood pressure and heart rate through adrenergic stimulation and vasoconstriction. Prescription stimulants and certain weight-loss agents can have similar effects. A complete medication and supplement history is essential in apparent treatment-resistant hypertension.

88. C. Calcineurin inhibitors such as tacrolimus and cyclosporine can cause or worsen hypertension through renal vasoconstriction, sodium retention, sympathetic effects, and chronic nephrotoxicity.

Management includes attention to drug exposure, kidney function, sodium balance, and appropriate antihypertensive therapy while preserving transplant immunosuppression.

89. B. Erythropoiesis-stimulating agents can increase blood pressure through mechanisms that may include increased vascular resistance and altered endothelial function. Hypertension should be controlled before and during therapy, and a temporal pressure rise should prompt reassessment of dose, volume status, and the overall antihypertensive regimen.

90. C. Cocaine and amphetamine-type stimulants increase catecholaminergic activity and can produce abrupt severe hypertension, agitation, mydriasis, diaphoresis, chest pain, coronary vasospasm, hyperthermia, and neurologic complications. MAOI–tyramine interactions, clonidine withdrawal, and sympathomimetic decongestants can also cause marked hypertension, but the recreational-drug context with this sympathomimetic toxidrome most strongly supports cocaine or an amphetamine-type stimulant.

91. A. A DASH-style pattern emphasizes fruits, vegetables, whole grains, legumes, low-fat dairy, and reduced saturated fat, and blood-pressure benefit is enhanced by sodium reduction. Lifestyle treatment also includes healthy weight, physical activity, moderation or elimination of alcohol, adequate dietary potassium when safe, and attention to sleep and stress.

92. B. Reducing dietary sodium is a core nonpharmacologic strategy for prevention and treatment of hypertension. The magnitude of response varies, with larger effects often seen in older adults, chronic kidney disease, resistant hypertension, and other salt-sensitive states. Counseling should focus on processed foods and realistic sustained reductions rather than only table salt.

93. C. Higher dietary potassium intake can lower blood pressure in many patients and is part of guideline-supported lifestyle management. However, potassium supplementation or high-potassium diets can be hazardous in advanced kidney disease, hypoaldosteronism, or during medications that predispose to hyperkalemia. Safety therefore depends on kidney function, baseline potassium, and the medication regimen.

94. D. Excess adiposity contributes to hypertension through sympathetic activation, renal sodium retention, sleep apnea, insulin resistance, and renin-angiotensin-aldosterone effects. Sustained weight reduction can lower pressure and improve multiple cardiovascular risk factors. The benefit occurs across lifestyle, pharmacologic weight-management, and surgical approaches when clinically appropriate.

95. C. Regular moderate aerobic activity lowers resting blood pressure and improves cardiovascular risk, while resistance exercise can provide additional benefit when appropriately prescribed. Exercise recommendations should consider symptoms, comorbid cardiovascular disease, functional capacity, and baseline pressure, but uncomplicated hypertension is not a reason to avoid physical activity.

96. C. Excess alcohol intake can increase blood pressure and interfere with treatment. Reducing or eliminating alcohol is part of current lifestyle recommendations for hypertension. The relevant exposure

is ethanol amount rather than beverage type, and counseling should also consider dependence risk and safe approaches to reduction.

97. A. The 2025 ACC/AHA guideline recommends pharmacologic treatment for selected adults with average blood pressure at least 130/80 mm Hg who have increased cardiovascular risk, including a PREVENT 10-year total CVD risk of at least 7.5%. Lifestyle therapy remains essential, but risk is used to determine when medication should be added in stage 1 hypertension.

98. C. For lower-risk adults with stage 1 hypertension, the 2025 ACC/AHA guideline emphasizes an initial period of lifestyle modification. If average blood pressure remains at least 130/80 mm Hg after roughly 3 to 6 months, medication therapy is recommended. This approach differs from patients with established CVD, diabetes, CKD, prior stroke, or higher PREVENT risk.

99. D. Stage 2 hypertension is generally treated promptly with pharmacologic therapy in addition to lifestyle measures. The 2025 guideline prefers two first-line agents of different classes in a single-pill fixed-dose combination when possible, which improves adherence and speeds control. Choice among first-line classes is individualized to comorbidity and tolerance.

100. A. Core first-line classes for primary hypertension include thiazide or thiazide-like diuretics, renin-angiotensin system blockers with either an ACE inhibitor or ARB, and long-acting calcium-channel blockers. Other agents have important roles in compelling indications or resistant disease but are not routine first-line choices for uncomplicated hypertension.

101. D. In resistant hypertension, the foundational three-drug regimen should be optimized before layering on additional agents. A long-acting thiazide-like diuretic such as chlorthalidone or indapamide often provides stronger and more sustained natriuresis than hydrochlorothiazide. Volume status, kidney function, electrolytes, and tolerance guide the choice and dose.

102. C. Loop diuretics remain central for clinically significant volume overload in advanced CKD because higher doses can deliver drug to the tubular lumen despite reduced GFR. Thiazide or thiazide-like agents can still lower blood pressure at low GFR and may be combined for sequential nephron blockade, but electrolyte and kidney-function monitoring becomes especially important.

103. C. ACE-inhibitor cough is related in part to accumulation of bradykinin and other peptides. ARBs provide renin-angiotensin system blockade without inhibiting bradykinin degradation and therefore have a much lower incidence of cough. Combining two renin-angiotensin blockers is not recommended for routine hypertension because adverse renal and potassium effects increase without added outcome benefit.

104. D. ACE-inhibitor angioedema can threaten the airway and requires prompt clinical assessment and permanent discontinuation of the ACE inhibitor. Rechallenge with another ACE inhibitor is inappropriate because the reaction is a class effect rather than a predictable dose-related adverse effect. Future antihypertensive selection should account for the prior event.

105. B. ACE inhibitors, ARBs, and direct renin inhibitors are avoided in pregnancy because fetal renin-angiotensin system blockade can cause serious renal and developmental toxicity, particularly with later gestational exposure. A pregnancy-compatible agent such as labetalol or extended-release nifedipine is commonly used, with treatment individualized to maternal and fetal circumstances.

106. C. Dihydropyridine calcium-channel blockers dilate arterioles more than venules, increasing intracapillary hydrostatic pressure and promoting dependent edema. The edema is not necessarily a sign of total-body volume overload and often responds poorly to simply adding a diuretic. Dose reduction or combination with a venodilating renin-angiotensin blocker can reduce the effect in appropriate patients.

107. B. Non-dihydropyridine calcium-channel blockers such as verapamil and diltiazem slow AV nodal conduction and can worsen clinically significant bradycardia or heart block. Dihydropyridine agents such as amlodipine have much less effect on AV nodal conduction and are used primarily for vascular smooth-muscle relaxation.

108. B. Thiazide and thiazide-like diuretics can cause clinically important hyponatremia, particularly in older adults and those with low solute intake or high free-water intake. The drug should be stopped in significant symptomatic cases while sodium is corrected carefully. Electrolyte monitoring after initiation or dose escalation helps identify this complication early.

109. A. Thiazide-type diuretics reduce renal urate excretion and can increase serum uric acid, which may precipitate gout in susceptible patients. This is a relative consideration rather than an absolute prohibition; cardiovascular benefit, gout severity, dose, and alternative agents should all be weighed. Losartan is unusual among ARBs because it has a modest uricosuric effect.

110. D. A modest creatinine increase can occur after starting ACE-inhibitor or ARB therapy because efferent arteriolar dilation lowers intraglomerular pressure. If the increase is limited and stabilizes, and potassium and volume status are acceptable, therapy can often continue. A larger or progressive rise should prompt evaluation for volume depletion, NSAIDs, renovascular disease, or other causes.

111. A. When renin-angiotensin system blockade has a strong indication, mild to moderate hyperkalemia should prompt review of diet, supplements, NSAIDs, kidney function, volume status, and other potassium-raising drugs. Diuretic adjustment or potassium binders may permit continued therapy in selected patients. Severe or refractory hyperkalemia still requires dose reduction or discontinuation for safety.

112. A. Beta blockers reduce heart rate, contractility, and myocardial oxygen demand and are useful when hypertension coexists with angina or certain other cardiac indications. They are not generally the preferred first-line drug for uncomplicated hypertension without a compelling indication, but comorbidity can make them an appropriate choice.

113. D. Alpha-1 blockers can lower blood pressure and improve lower urinary tract symptoms from benign prostatic hyperplasia, but they are not preferred as sole first-line therapy for hypertension

because outcome data favor other foundational classes. Orthostatic hypotension is also a concern, especially in older adults.

114. B. Clonidine suppresses central sympathetic outflow through alpha-2 agonism. Abrupt discontinuation after chronic therapy can produce rebound sympathetic activation with marked hypertension, tachycardia, and other adrenergic symptoms. When possible, clonidine should be tapered rather than stopped suddenly.

115. A. Hydralazine can cause a dose- and exposure-related drug-induced lupus syndrome, with risk influenced by acetylator status and cumulative exposure. Hydralazine also can cause reflex tachycardia and sodium retention, so it is usually combined with appropriate background therapy when used for refractory hypertension.

116. D. Minoxidil is a potent direct arteriolar vasodilator that can produce reflex sympathetic activation and substantial sodium and water retention. It is generally given with a beta blocker or other rate-controlling agent and a potent diuretic. Hypertrichosis and, rarely, pericardial effusion are additional adverse effects.

117. C. Eplerenone is more selective for the mineralocorticoid receptor and has fewer androgen- and progesterone-receptor effects than spironolactone. It can therefore be useful when spironolactone causes gynecomastia or sexual adverse effects, although dosing frequency, cost, kidney function, and hyperkalemia risk must still be considered.

118. D. Eplerenone is an appropriate alternative mineralocorticoid-receptor antagonist when spironolactone is poorly tolerated because of antiandrogenic effects. It remains aldosterone-directed treatment and requires monitoring for hyperkalemia and changes in kidney function. Bilateral disease is generally managed medically rather than with adrenalectomy.

119. D. The sodium retention in Liddle syndrome is caused by constitutively active ENaC and occurs independently of aldosterone. Therefore, mineralocorticoid-receptor antagonists do not directly correct the abnormal channel activity. Amiloride or triamterene blocks ENaC and is the targeted therapy.

120. C. True resistant hypertension is blood pressure above goal despite concurrent use of three appropriately chosen antihypertensive agents at maximally tolerated doses, typically including a long-acting calcium-channel blocker, an ACE inhibitor or ARB, and a diuretic, after adherence and out-of-office elevation are confirmed. Controlled pressure requiring four or more drugs also falls within resistant-hypertension definitions.

121. B. Measurement error is a common cause of pseudoresistance. Proper cuff size, seated rest, supported positioning, repeated readings, and validated equipment are prerequisites before concluding that medication therapy has failed. Escalation based on systematically inflated measurements can cause overtreatment and orthostatic symptoms.

122. C. Medication nonadherence is a major contributor to uncontrolled and apparent resistant hypertension. Simplifying regimens, using once-daily dosing and single-pill combinations, reducing cost barriers, and engaging in nonjudgmental shared problem solving can improve persistence and control. Adding more complexity without addressing adherence often worsens the problem.

123. B. In a treated patient, elevated office pressure with controlled out-of-office blood pressure is termed a white-coat effect. It can create apparent treatment resistance if ambulatory or home measurements are not obtained. Treatment decisions should use reliable out-of-office data to avoid excessive medication and orthostatic adverse effects.

124. B. Excess dietary sodium and inadequate diuresis are frequent contributors to resistant hypertension. Management includes meaningful sodium restriction and an effective long-acting diuretic strategy, adjusted for kidney function and volume status. Correcting volume excess can substantially improve the response to the rest of the regimen.

125. A. Mineralocorticoid-receptor antagonism with spironolactone is highly effective as fourth-line therapy for many patients with resistant hypertension, reflecting the important role of sodium retention and aldosterone physiology. Potassium and kidney function must be monitored, and eplerenone or other strategies may be needed when spironolactone is not tolerated or hyperkalemia risk is prohibitive.

126. B. Obstructive sleep apnea is highly prevalent in resistant hypertension and is particularly suggested by obesity, loud snoring, daytime sleepiness, and a nondipping ambulatory pattern. Primary aldosteronism, renovascular disease, and chronic kidney disease are also important causes or contributors to resistant hypertension, but they do not specifically explain this sleep-related symptom cluster. Treating sleep apnea can improve symptoms and modestly lower blood pressure, especially with good adherence.

127. D. Contemporary radiofrequency and ultrasound renal denervation systems are FDA-approved as adjunctive therapies for selected patients whose hypertension remains inadequately controlled despite lifestyle measures and medications. The average pressure reduction is modest and individual response varies. Appropriate selection requires confirmation of sustained hypertension, evaluation for secondary causes, and multidisciplinary shared decision-making.

128. D. Home monitoring is most effective when embedded in a structured system that includes validated devices, standardized measurement, timely feedback, and protocol-driven treatment adjustments. Multidisciplinary care involving physicians, pharmacists, nurses, and other professionals can reduce therapeutic inertia and improve access, adherence, and rates of blood-pressure control.

129. C. Single-pill combinations reduce pill burden and can improve adherence and speed blood-pressure control. The 2025 ACC/AHA guideline prefers a single-pill fixed-dose combination of two first-line classes for many patients with stage 2 hypertension. Component doses and contraindications still require individualization.

130. D. Large contemporary trials have not established a universal cardiovascular advantage to moving all antihypertensive medications to bedtime. Timing should be individualized to pharmacokinetics, adherence, side effects, orthostatic symptoms, nocturnal hypertension, and the clinical regimen. Consistent adherence is generally more important than a rigid clock-time rule.

131. C. Isolated systolic hypertension is common in older adults because of arterial stiffness and remains associated with cardiovascular risk. Treatment can reduce events, but therapy should be titrated carefully with attention to orthostatic symptoms, falls, kidney function, and very low diastolic pressure, especially in patients with coronary disease.

132. D. Coronary perfusion occurs predominantly during diastole, and very low diastolic pressure can be concerning in patients with obstructive coronary disease, particularly when pulse pressure is wide. Blood-pressure goals still matter, but symptoms and the overall hemodynamic context should guide individualized titration rather than pursuing a systolic number without regard to diastolic pressure.

133. C. Population-level differences in average drug response do not reliably determine the best treatment for an individual. Contemporary care emphasizes proven first-line agents, comorbid conditions, kidney function, adverse-effect profiles, access, adherence, and observed response. Race is a social construct and should not substitute for individualized clinical assessment.

134. D. In diabetes with significant albuminuria, ACE inhibitors or ARBs reduce intraglomerular pressure, albuminuria, and progression of kidney disease and are foundational antihypertensive therapy when tolerated. Potassium and creatinine should be monitored after initiation and dose changes. Additional agents are commonly needed to reach blood-pressure goals.

135. B. When diabetes is present without albuminuric kidney disease or another specific indication, multiple first-line antihypertensive classes can be effective. The regimen should be individualized to blood pressure, comorbidities, adverse effects, kidney function, and metabolic considerations. Renin-angiotensin blockade becomes particularly important when albuminuria is present.

136. A. Renin-angiotensin system blockade is recommended in albuminuric CKD because it lowers blood pressure and reduces intraglomerular pressure and albuminuria. The drug should be titrated thoughtfully with monitoring of creatinine and potassium. Combining an ACE inhibitor with an ARB is not recommended because adverse renal and hyperkalemic effects outweigh incremental surrogate benefits.

137. A. Dual blockade of the renin-angiotensin system can reduce albuminuria but increases acute kidney injury, hypotension, and hyperkalemia without sufficient outcome benefit to justify routine use. Choose either an ACE inhibitor or an ARB at an appropriate tolerated dose and add other complementary classes for blood-pressure control.

138. D. Losartan is unusual among ARBs because it has a modest uricosuric effect and can lower serum uric acid somewhat. This property may be useful in a patient with gout, although it does not replace

appropriate gout management and should not override more important considerations such as kidney function, blood-pressure response, and tolerability.

139. C. Thiazide-type diuretics increase distal calcium reabsorption and reduce urinary calcium excretion, which can be useful in selected patients with recurrent calcium stones and hypercalciuria. Loop diuretics have the opposite effect on urinary calcium. Electrolytes, uric acid, glucose, and kidney function should still be monitored during thiazide therapy.

140. C. Dihydropyridine edema reflects preferential arteriolar dilation and elevated capillary hydrostatic pressure. Adding an ACE inhibitor or ARB can provide some postcapillary or venous hemodynamic counterbalance and often reduces edema, while also improving blood-pressure control. Dose reduction or switching agents may also be appropriate.

141. A. Certain beta blockers, including propranolol and metoprolol, have evidence for migraine prevention and can be useful when hypertension coexists. The choice should account for heart rate, asthma, conduction disease, exercise tolerance, and other indications. A dual-purpose drug can simplify therapy when it fits the clinical profile.

142. A. Propranolol is effective for essential tremor and also lowers blood pressure through beta blockade. It may therefore be a useful dual-purpose agent in an appropriate patient. Bradycardia, asthma, conduction disease, and other contraindications must be considered before selecting a nonselective beta blocker.

143. B. Alpha-1 blockers relax smooth muscle in the prostate and bladder neck and lower peripheral vascular resistance. They can be useful as add-on therapy when benign prostatic hyperplasia coexists, but they are not preferred as sole first-line hypertension treatment and can cause orthostatic hypotension, especially after the first dose or in older patients.

144. D. Extended-release nifedipine and labetalol are common first-line oral agents for chronic hypertension in pregnancy, with methyldopa used in selected situations. ACE inhibitors, ARBs, and direct renin inhibitors are avoided because fetal renin-angiotensin system blockade can cause serious toxicity.

145. C. For a patient planning pregnancy, medications with known fetal toxicity should be reviewed before conception. ACE inhibitors and ARBs should generally be replaced with pregnancy-compatible agents when feasible, while maintaining adequate maternal blood-pressure control. Preconception counseling also allows review of comorbidities and baseline kidney function.

146. B. Regimen complexity predicts lower adherence. Long-acting once-daily drugs, single-pill combinations, synchronized refills, affordability interventions, and clear written plans can improve persistence. Simplifying therapy is particularly important before labeling a patient as having pharmacologically resistant hypertension.

147. B. NSAIDs can promote sodium retention, increase blood pressure, and reduce the efficacy of several antihypertensive agents. In a patient taking a renin-angiotensin blocker and diuretic, prostaglandin inhibition can also contribute to a marked fall in GFR, particularly during volume depletion. Medication review is essential when control unexpectedly worsens.

148. D. The combination of a renin-angiotensin blocker, diuretic-associated or illness-related volume depletion, and an NSAID can sharply reduce glomerular perfusion. ACE inhibition or ARB therapy dilates the efferent arteriole, while NSAIDs reduce prostaglandin-mediated afferent dilation. The combined hemodynamic effect can precipitate acute kidney injury.

149. A. A potassium-containing salt substitute can deliver substantial potassium. Combined with an ACE inhibitor and a mineralocorticoid-receptor antagonist—especially in chronic kidney disease—this can produce clinically important hyperkalemia. A potassium of 6.1 mmol/L warrants prompt reassessment of potassium intake and medications, along with evaluation of renal function and electrocardiographic risk as clinically indicated.

150. D. Hydralazine directly dilates arterioles, provoking baroreflex sympathetic activation and sodium-water retention. When used for difficult hypertension, it is typically layered onto an effective diuretic regimen and often a beta blocker or other appropriate rate-control therapy. This minimizes reflex tachycardia and fluid accumulation.

151. A. Renin-angiotensin blockade can be beneficial in many patients with vascular disease, but severe bilateral renal-artery stenosis can make filtration highly dependent on angiotensin II-mediated efferent constriction. A recurrent large creatinine rise despite correction of volume and other factors indicates poor tolerance. Alternative antihypertensive therapy and evaluation of the renovascular syndrome are appropriate.

152. D. Diuretics and mineralocorticoid-receptor antagonists can alter sodium, potassium, and kidney function. Chlorthalidone can cause hyponatremia and hypokalemia, while spironolactone can cause hyperkalemia, particularly in CKD or with renin-angiotensin blockers. Early and repeated laboratory monitoring supports safe titration.

153. C. Sustained blood-pressure control throughout the dosing interval is important because risk reflects cumulative pressure exposure rather than a single clinic value. Long-acting agents improve trough control and often simplify adherence. Ambulatory or home measurements can reveal loss of effect that is not apparent from office readings alone.

154. D. Resistant hypertension is often highly salt sensitive, and excess sodium can blunt the effect of multiple antihypertensive drugs. Meaningful sodium reduction can produce a clinically important pressure decrease, especially when combined with optimized diuretic therapy. Counseling should identify hidden sodium sources in processed and restaurant foods.

155. C. Although dietary potassium can lower blood pressure in many people, advanced CKD and existing hyperkalemia substantially increase the risk from potassium-rich salt substitutes or supplements. Sodium reduction can still be pursued through other strategies. Dietary advice must be individualized to kidney function, serum potassium, and potassium-raising medications.

156. C. SGLT2 inhibitors provide important cardiorenal benefits in appropriate patients and usually produce a modest reduction in blood pressure through natriuretic, osmotic, vascular, and weight-related effects. They are not primarily prescribed as antihypertensive drugs and usually do not replace standard blood-pressure therapy when hypertension remains above goal.

157. B. Meaningful weight loss can reduce blood pressure and medication requirements. When symptoms and low readings develop, the regimen should be reassessed to prevent falls, kidney hypoperfusion, and other adverse effects. Hypertension treatment is dynamic and should respond to changes in weight, diet, kidney function, activity, and other determinants of pressure.

158. C. Finerenone is a nonsteroidal mineralocorticoid-receptor antagonist used in selected patients with diabetic CKD to reduce cardiorenal events. Its blood-pressure effect is generally modest, and hyperkalemia remains an important safety consideration. Dedicated antihypertensive agents are still required when pressure is above goal.

159. A. Combining a direct renin inhibitor with an ACE inhibitor or ARB is not recommended in patients with diabetes and is generally avoided because dual renin-angiotensin blockade increases adverse renal outcomes, hypotension, and hyperkalemia without adequate outcome benefit. Complementary antihypertensive classes should be used instead.

160. B. Minoxidil opens potassium channels in arteriolar smooth muscle and is a potent direct vasodilator. Reflex sympathetic activation and sodium-water retention can be substantial, so it is generally paired with an effective diuretic and a beta blocker or another appropriate rate-controlling strategy. It is reserved for highly selected refractory cases.

161. C. Severe blood pressure elevation without acute target-organ injury is not a hypertensive emergency. The 2025 ACC/AHA guideline supports outpatient evaluation with initiation, reinstitution, or intensification of oral therapy in a timely fashion, while addressing adherence and precipitating factors. Rapid intravenous lowering can cause ischemia when no emergency indication exists.

162. C. In most hypertensive emergencies, the goal is controlled reduction rather than immediate normalization because chronic hypertension shifts autoregulation. A common strategy is to reduce mean arterial pressure by about 20% to 25% in the first hour, then toward approximately 160/100-110 mm Hg over the next several hours if the patient remains stable. Aortic dissection and selected other syndromes use different targets.

163. B. Aortic dissection requires rapid reduction of aortic wall shear stress. Intravenous beta blockade, such as esmolol or labetalol, is used first to reduce heart rate and contractility; an arterial vasodilator can

be added if blood pressure remains high. Starting a vasodilator alone can trigger reflex tachycardia and increase shear stress.

164. B. In acute ischemic stroke without reperfusion therapy, blood pressure is often left untreated unless it is extremely high or another acute indication exists, because cerebral autoregulation is impaired and sudden reduction can worsen ischemia. Common stroke guidance uses a threshold around 220/120 mm Hg for considering modest reduction in otherwise uncomplicated cases.

165. A. Intravenous thrombolysis for acute ischemic stroke requires blood pressure below the established pretreatment safety threshold, commonly less than 185/110 mm Hg, to reduce hemorrhagic risk. After thrombolysis, pressure is maintained below a somewhat lower post-treatment ceiling. The specific acute stroke protocol should be followed closely.

166. B. After intravenous thrombolysis, blood pressure should remain controlled with frequent neurologic and blood-pressure monitoring. The 2026 AHA/ASA acute ischemic stroke guideline supports maintaining systolic blood pressure in the approximate 140–180 mm Hg range after thrombolysis and does not support routine intensive lowering below 140 mm Hg to improve functional outcome. Excessive hypertension increases hemorrhagic risk, whereas overly aggressive reduction can threaten cerebral perfusion.

167. A. Acute intracerebral hemorrhage management favors early, smooth, sustained blood-pressure control because marked hypertension can promote hematoma expansion, while excessive reduction can impair perfusion. Contemporary stroke guidance generally supports targeting near 140 mm Hg in many patients presenting between about 150 and 220 mm Hg, while avoiding overshoot below roughly 130 mm Hg.

168. C. Hypertensive acute pulmonary edema is driven by severe afterload and redistribution or accumulation of pulmonary fluid. Rapidly titratable intravenous vasodilators such as nitroglycerin can reduce preload and afterload quickly, while loop diuretics are used when volume overload is present. Isolated beta blockade can worsen decompensated pulmonary edema.

169. A. In acute coronary ischemia with severe hypertension, nitrates reduce preload and myocardial oxygen demand and can help control pressure, while beta blockade can reduce heart rate and contractility when appropriate. Treatment must be integrated with the full acute coronary syndrome protocol and individualized to heart failure, conduction status, and hemodynamics.

170. D. Phentolamine directly antagonizes alpha-adrenergic receptors and is therefore particularly suited to catecholamine-mediated vasoconstriction. Nicardipine and clevidipine can also lower blood pressure in hypertensive emergencies but do not directly block alpha receptors. A beta blocker should not be used as the initial sole adrenergic blocker in pheochromocytoma because unopposed alpha-mediated vasoconstriction can worsen the crisis.

171. A. Persistent severe hypertension in pregnancy, typically at least 160 systolic or 110 diastolic, requires urgent treatment to reduce maternal risk of stroke and other complications. Intravenous labetalol, intravenous hydralazine, or immediate-release oral nifedipine are commonly used protocols. Evaluation for preeclampsia and fetal status proceeds simultaneously.

172. B. The CHAP trial changed management of mild chronic hypertension in pregnancy by supporting active treatment beginning around 140/90 mm Hg rather than waiting for severe hypertension. ACOG guidance was reaffirmed in 2025. Labetalol and extended-release nifedipine are common choices, with therapy individualized to contraindications and maternal-fetal status.

173. B. A generalized seizure in a patient with preeclampsia is eclampsia. Magnesium sulfate is the standard first-line medication for treatment and prevention of recurrent eclamptic seizures. Benzodiazepines or other antiseizure drugs may be required for refractory or alternative-cause seizures, but they do not replace magnesium as routine first-line eclampsia therapy. Severe hypertension requires separate prompt treatment with pregnancy-appropriate antihypertensive medication.

174. C. Enalapril has extensive postpartum experience, and LactMed reports very low concentrations in breast milk with small infant exposure and no reported adverse effects in the limited available cases. In contrast, lactation data for many other renin-angiotensin system agents are more limited. Postpartum drug choice should also account for renal function, infant factors, future pregnancy plans, and the patient's other cardiovascular indications.

175. A. KDIGO 2021 suggests a standardized office systolic target below 120 mm Hg for many adults with non-dialysis CKD when tolerated. The recommendation depends on standardized measurement similar to that used in SPRINT and should not be mechanically applied to casual clinic readings. Frailty, symptoms, transplant status, dialysis, and other circumstances require individualization.

176. B. Volume excess is a major driver of hypertension in many dialysis patients. Achieving an appropriate dry weight, limiting sodium intake, and optimizing ultrafiltration and dialysis delivery can lower pressure and reduce medication burden. Antihypertensive drugs remain important, but failure to address chronic sodium and volume excess can make pharmacologic treatment appear resistant.

177. D. Dihydropyridine calcium-channel blockers are commonly effective after kidney transplantation and can counteract calcineurin-inhibitor-associated vasoconstriction. Drug interactions, edema, graft function, proteinuria, potassium, and immunosuppressant levels all influence regimen selection. Hypertension in a transplant recipient also warrants attention to transplant renal-artery stenosis and native kidney disease when clinically suggested.

178. B. In HFrEF, the regimen should be built around therapies that improve heart-failure outcomes, such as evidence-based beta blockers, renin-angiotensin system or ARNI therapy, mineralocorticoid-receptor antagonists, and SGLT2 inhibitors when appropriate. Additional agents are added for residual hypertension after the core disease-modifying regimen is optimized.

179. B. Hypertension is a major contributor to HFpEF development and decompensation. Blood-pressure control is therefore central, along with management of volume status, atrial fibrillation, ischemia, obesity, sleep apnea, diabetes, and CKD. Therapy should avoid excessive hypotension while reducing chronic pressure and filling-pressure burden.

180. A. A beta blocker can lower blood pressure while reducing heart rate, contractility, and myocardial oxygen demand, which directly addresses exertional angina in an appropriate patient. Thiazide-like diuretics and ACE inhibitors can be valuable for blood-pressure and cardiovascular-risk management but do not provide the same heart-rate-lowering antianginal mechanism. Dihydropyridine calcium-channel blockers can treat angina and hypertension but generally do not lower heart rate.

181. C. Beta blockers reduce heart rate, blood pressure, and myocardial oxygen demand and are commonly used after myocardial infarction when clinically appropriate. The full post-MI regimen depends on ventricular function, ongoing ischemia, arrhythmia, kidney function, and contemporary coronary guidelines, but hypertension treatment should support rather than conflict with those outcome goals.

182. A. A non-dihydropyridine calcium-channel blocker such as diltiazem can slow AV nodal conduction and lower blood pressure, making it useful for rate control in atrial fibrillation when ventricular systolic function is preserved and no conduction contraindication exists. These agents are generally avoided in significant HFrEF because of negative inotropic effects.

183. C. Chronic aortic disease management emphasizes blood-pressure control and reduction of hemodynamic stress on the aortic wall. Beta blockers are often useful because they reduce heart rate and contractility as well as pressure, while other agents are added to achieve control. Acute dissection requires much more rapid intravenous anti-impulse therapy.

184. C. Modern evidence does not support withholding cardioselective beta blockers solely because a patient has peripheral artery disease when there is a strong cardiac indication. Blood-pressure reduction lowers overall cardiovascular risk. Limb symptoms, perfusion, heart rate, and other comorbidities should still be monitored as therapy is individualized.

185. B. Autonomic neuropathy can impair the sympathetic vasoconstrictor and heart-rate responses required to maintain pressure on standing. The result may be coexisting supine hypertension and orthostatic hypotension, which makes treatment challenging. Management focuses on symptoms, falls, volume status, medication contributors, and selective treatment of severe supine hypertension.

186. D. Although the general adult blood-pressure goal is below 130/80 mm Hg, contemporary guidance recognizes the need for individualization in people with limited life expectancy, institutional care, frailty, or substantial treatment-related symptoms. Preventing falls, syncope, kidney injury, and loss of function may justify de-intensifying therapy when the risk-benefit balance changes.

187. D. Orthostatic symptoms should trigger measurement of supine or seated and standing pressures, assessment of volume status and autonomic disease, and review of medications. Alpha blockers, central sympatholytics, nitrates, diuretics, and other agents can contribute. The goal is to reduce symptoms and falls while still treating clinically important hypertension.

188. C. Head-up sleeping can reduce nocturnal supine hypertension and may lessen pressure natriuresis that worsens morning orthostasis. Management of autonomic failure often requires balancing two opposing problems: raising standing pressure enough to prevent symptoms while limiting severe supine hypertension. Short-acting nighttime therapy may be considered in selected cases.

189. B. Paroxysmal hypertension has a broad differential. Once appropriate biochemical testing has convincingly excluded pheochromocytoma or paraganglioma, clinicians should consider medication effects, panic disorder, trauma-related syndromes, baroreflex failure, sleep disorders, and pseudopheochromocytoma. Careful recognition can prevent repeated invasive testing and support targeted behavioral and pharmacologic treatment.

190. D. For children younger than 13 years, blood-pressure classification uses normative percentiles based on age, sex, and height, with confirmation by auscultation and repeated visits as appropriate. Adolescents 13 years and older use fixed thresholds aligned more closely with adult categories. Pediatric technique and cuff sizing are especially important.

191. C. Ambulatory blood-pressure monitoring is important in pediatric hypertension for confirming persistent hypertension, identifying white-coat and masked patterns, and characterizing nocturnal pressure. Interpretation uses pediatric-specific standards and should be performed with an appropriate validated monitor and cuff.

192. C. Secondary hypertension is more common in younger children than in adolescents, and severe hypertension, poor growth, abnormal examination findings, or abnormal urinalysis further increase suspicion. Renal parenchymal and renovascular disorders are important causes. Evaluation and treatment should involve clinicians experienced in pediatric hypertension.

193. A. Primary hypertension has become increasingly common in adolescents, particularly with obesity, family history, sleep-disordered breathing, and other cardiometabolic risk factors. Secondary causes still require consideration based on age, severity, examination, and laboratory clues, but a typical adolescent phenotype can be primary after appropriate confirmation and basic evaluation.

194. D. Arm-leg pressure discrepancy, weak or delayed femoral pulses, and a posterior systolic murmur strongly suggest aortic coarctation. Echocardiography is an appropriate initial imaging test in children and can assess the arch and associated cardiac lesions; CT or MR angiography may further define anatomy when needed.

195. C. Fetal renin-angiotensin system activity is critical for kidney development and perfusion. ACE inhibitors and ARBs can cause fetal renal failure, oligohydramnios, pulmonary hypoplasia, skull

abnormalities, and neonatal complications, particularly with exposure later in pregnancy. They should be stopped and replaced with pregnancy-compatible therapy.

196. B. Thiazide-like diuretics can retain antihypertensive efficacy even in advanced CKD, as demonstrated with chlorthalidone, and can be useful when mineralocorticoid blockade is limited by hyperkalemia. Careful monitoring is essential because volume depletion, electrolyte abnormalities, creatinine changes, and gout can occur, particularly when combined with loop diuretics.

197. B. Modern renal denervation can lower blood pressure across several hypertension phenotypes, but the average effect is comparable to adding an effective medication rather than a guaranteed cure. Patient response varies. Selection should confirm sustained uncontrolled hypertension, address secondary causes and adherence, and use shared decision-making at experienced centers.

198. D. Emotional distress and the measurement environment can produce large transient sympathetic pressure elevations. When high-quality ambulatory data show adequate average control, repeatedly escalating chronic therapy in response to office spikes can cause hypotension outside the clinic. Anxiety, panic, pain, sleep, and medication effects should be addressed as part of the overall plan.

199. A. Symptoms overlap substantially between panic attacks and catecholamine-producing tumors, so symptom quality alone is insufficient. Repeated normal high-quality metanephrine testing in an appropriate clinical context makes pheochromocytoma much less likely and should redirect attention toward panic, pseudopheochromocytoma, medication effects, or other causes rather than continued indiscriminate tumor imaging.

200. B. Hypertension control depends on both effective pharmacotherapy and the ability to obtain, take, monitor, and follow up on that therapy. Cost and transportation barriers make a team-based strategy—simplifying the regimen, assisting with medication access, supporting home monitoring, and coordinating follow-up—the most direct intervention. Device therapy, additional drugs, or an extensive secondary-cause evaluation may be appropriate in selected patients, but they should not substitute for correcting clear adherence and access barriers.