PRACTICE EXAM 10: PATHOPHYSIOLOGY (100 QUESTIONS)

This standalone practice exam contains 100 four-option questions covering core pathophysiology across cellular, immune, fluid and electrolyte, hematologic, cardiovascular, respiratory, renal, gastrointestinal, endocrine, and neurologic systems. Select the single best answer for every question. No universal governing body specifies a time limit or passing score for this general course-based study guide.

1. On a medical-surgical unit, during discussion of distributive shock in a 19-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. transudation of fluid into pulmonary interstitium and alveoli
B. peripheral fluid filtration with congestion of abdominal organs
C. reduced systemic vascular resistance despite normal or increased cardiac output
D. compensatory vasoconstriction that raises afterload and worsens congestion

2. On a medical-surgical unit, a 26-year-old patient has heat intolerance, tremor, weight loss, and suppressed pituitary stimulation. Which mechanism best explains this pattern?

A. excess thyroid hormone increasing metabolic and adrenergic activity
B. absolute insulin deficiency from autoimmune beta-cell destruction
C. unrestrained lipolysis generating acidic ketone bodies
D. insufficient thyroid hormone reducing basal metabolic activity

3. On a medical-surgical unit, the care team reviews emphysema in a 33-year-old adult. Which consequence best reflects its established pathophysiology?

A. protein-rich edema with surfactant dysfunction and intrapulmonary shunting
B. premature small-airway collapse with reduced gas-exchange surface area
C. increased alveolar dead space with redistribution of perfusion
D. increased airway resistance that is greatest during expiration

4. On a medical-surgical unit, a 40-year-old patient is being evaluated for multiple sclerosis. Which additional consequence is most consistent with this disorder?

A. falling cerebral perfusion pressure as intracranial pressure approaches arterial pressure
B. declining neuromuscular transmission safety during repetitive stimulation
C. cytotoxic edema with expansion of an irreversibly injured core
D. slowed or blocked saltatory conduction along affected axons

5. On a medical-surgical unit, the pathophysiology of type III hypersensitivity is reviewed in a 47-year-old patient. Which downstream finding should be expected?

A. simultaneous parenchymal destruction and collagen deposition
B. rapid histamine release causing vasodilation and bronchoconstriction
C. neutrophil-rich inflammation damaging vessel walls and adjacent tissue
D. complement activation and antibody-mediated cellular destruction

6. On a medical-surgical unit, a 54-year-old adult has findings compatible with acute respiratory distress syndrome. Which associated change best follows from the underlying mechanism?

A. increased alveolar dead space with redistribution of perfusion
B. protein-rich edema with surfactant dysfunction and intrapulmonary shunting
C. increased airway resistance that is greatest during expiration
D. restrictive mechanics with impaired oxygen transfer across the alveolar membrane

7. On a medical-surgical unit, a 61-year-old adult has diffuse bleeding while laboratory studies show consumption of platelets and clotting factors. Which cellular or physiologic mechanism accounts for these findings?

A. premature rupture of erythrocytes within the circulation
B. insufficient iron limiting hemoglobin synthesis
C. insufficient neutrophil availability for an effective acute response
D. widespread pathologic thrombin generation throughout the microcirculation

8. On a medical-surgical unit, a clinical scenario involving a 68-year-old patient demonstrates gastroesophageal reflux. Which additional finding fits the same causal process?

A. repeated acid exposure causing distal esophageal mucosal injury
B. reflux of water-soluble conjugated bilirubin into blood and urine
C. fat necrosis, edema, and systemic inflammatory mediator release
D. fibrosis and penetrating tracts between bowel and adjacent structures

9. On a medical-surgical unit, during clinical review, a 75-year-old patient has exertional dyspnea, orthopnea, and inspiratory crackles. What is the principal underlying mechanism?

A. elevated left atrial and pulmonary capillary hydrostatic pressure
B. widespread vasodilation and maldistribution of circulating blood flow
C. severe pump failure reducing stroke volume and tissue perfusion
D. intimal inflammation with oxidized lipoprotein uptake by macrophages

10. On a medical-surgical unit, a case involves a 82-year-old adult who has persistent kidney dysfunction after prolonged ischemia with granular casts in urine. Which pathophysiologic process is most directly responsible?

A. inflammatory disruption of glomerular capillary walls
B. increased glomerular permeability to plasma proteins
C. reduced renal perfusion without primary parenchymal damage
D. tubular epithelial damage causing obstruction and filtrate backleak

11. On a medical-surgical unit, the pathophysiology of chronic inflammation is reviewed in a 21-year-old patient. Which downstream finding should be expected?

A. delayed mononuclear inflammation without an antibody requirement
B. simultaneous parenchymal destruction and collagen deposition
C. complement activation and antibody-mediated cellular destruction
D. neutrophil-rich inflammation damaging vessel walls and adjacent tissue

12. On a medical-surgical unit, in a teaching case, a 28-year-old patient develops urticaria and wheezing within minutes of allergen exposure. Which process provides the best explanation?

A. ongoing immune activation occurring alongside tissue repair
B. immune-complex deposition with complement recruitment
C. allergen cross-linking of IgE on sensitized mast cells
D. IgG or IgM binding to cell-surface targets

13. On a medical-surgical unit, during discussion of type II hypersensitivity in a 35-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. delayed mononuclear inflammation without an antibody requirement
B. complement activation and antibody-mediated cellular destruction
C. simultaneous parenchymal destruction and collagen deposition
D. neutrophil-rich inflammation damaging vessel walls and adjacent tissue

14. On a medical-surgical unit, a clinical scenario involving a 42-year-old patient demonstrates portal hypertension in cirrhosis. Which additional finding fits the same causal process?

A. fibrosis and penetrating tracts between bowel and adjacent structures
B. fat necrosis, edema, and systemic inflammatory mediator release
C. portosystemic collateral formation and elevated splanchnic hydrostatic pressure
D. reflux of water-soluble conjugated bilirubin into blood and urine

15. On a medical-surgical unit, the care team reviews acute tubular injury in a 49-year-old adult. Which consequence best reflects its established pathophysiology?

A. persistent parathyroid stimulation with increased bone resorption
B. reduced oncotic pressure with compensatory hepatic lipoprotein synthesis
C. erythrocyte leakage into tubules with sodium and water retention
D. impaired concentrating ability despite reduced glomerular filtration

16. On a medical-surgical unit, a case involves a 56-year-old adult who develops sudden focal neurologic deficits after arterial cerebral blood flow is interrupted. Which pathophysiologic process is most directly responsible?

A. energy failure causing loss of ion gradients and excitotoxic injury
B. immune-mediated destruction of central nervous system myelin
C. antibody-mediated loss or dysfunction of postsynaptic acetylcholine receptors
D. loss of dopaminergic input to basal ganglia motor circuits

17. On a medical-surgical unit, the pathophysiology of primary hyperaldosteronism is reviewed in a 63-year-old patient. Which downstream finding should be expected?

A. water retention that lowers serum sodium without proportional sodium loss
B. movement of fluid from capillaries into the interstitial compartment
C. increased carbonic acid with renal bicarbonate retention if persistent
D. volume expansion with hypokalemia and metabolic alkalosis

18. On a medical-surgical unit, in a teaching case, a 70-year-old patient has hypotension, cool clammy skin, pulmonary congestion, and low cardiac output after myocardial injury. Which process provides the best explanation?

A. severe pump failure reducing stroke volume and tissue perfusion
B. widespread vasodilation and maldistribution of circulating blood flow
C. intimal inflammation with oxidized lipoprotein uptake by macrophages
D. elevated systemic venous pressure from impaired right ventricular output

19. On a medical-surgical unit, during discussion of iron-deficiency anemia in a 77-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. blunted local inflammation despite risk of rapidly progressive infection
B. progressively smaller red cells with reduced oxygen-carrying capacity
C. simultaneous microvascular thrombosis and consumption-related hemorrhage
D. megaloblastic erythropoiesis with neurologic dysfunction

20. On a medical-surgical unit, a 84-year-old patient has hypoventilation with carbon dioxide retention during a severe obstructive exacerbation. Which mechanism best explains this pattern?

A. reduced plasma colloid oncotic pressure favoring filtration
B. excess mineralocorticoid-driven sodium reabsorption in the distal nephron
C. alveolar ventilation inadequate for carbon dioxide production
D. gastrointestinal loss of bicarbonate-rich fluid

21. On a medical-surgical unit, the care team reviews myasthenia gravis in a 23-year-old adult. Which consequence best reflects its established pathophysiology?

A. cytotoxic edema with expansion of an irreversibly injured core
B. declining neuromuscular transmission safety during repetitive stimulation
C. reduced facilitation of intended movement with excessive inhibitory output
D. slowed or blocked saltatory conduction along affected axons

22. On a medical-surgical unit, a 30-year-old patient is being evaluated for pulmonary embolic VQ mismatch. Which additional consequence is most consistent with this disorder?

A. protein-rich edema with surfactant dysfunction and intrapulmonary shunting
B. premature small-airway collapse with reduced gas-exchange surface area
C. restrictive mechanics with impaired oxygen transfer across the alveolar membrane
D. increased alveolar dead space with redistribution of perfusion

23. On a medical-surgical unit, the pathophysiology of pulmonary fibrosis is reviewed in a 37-year-old patient. Which downstream finding should be expected?

A. restrictive mechanics with impaired oxygen transfer across the alveolar membrane
B. premature small-airway collapse with reduced gas-exchange surface area
C. protein-rich edema with surfactant dysfunction and intrapulmonary shunting
D. increased alveolar dead space with redistribution of perfusion

24. On a medical-surgical unit, in a teaching case, a 44-year-old patient develops bilateral pulmonary opacities and severe hypoxemia after a major inflammatory insult. Which process provides the best explanation?

A. protease-mediated loss of alveolar septa and supporting elastic tissue
B. abrupt loss of regional pulmonary blood flow
C. diffuse alveolar-capillary barrier injury increasing permeability
D. bronchial smooth-muscle constriction plus mucosal inflammation

25. On a medical-surgical unit, a 51-year-old adult has macrocytic anemia accompanied by paresthesias and impaired vibration sensation. Which cellular or physiologic mechanism accounts for these findings?

A. impaired DNA synthesis plus defective myelin maintenance
B. insufficient neutrophil availability for an effective acute response
C. insufficient iron limiting hemoglobin synthesis
D. widespread pathologic thrombin generation throughout the microcirculation

26. On a medical-surgical unit, a clinical scenario involving a 58-year-old patient demonstrates primary hyperthyroidism. Which additional finding fits the same causal process?

A. reduced cellular glucose uptake with increased lipolysis and ketogenesis
B. reduced TSH through negative feedback despite increased tissue stimulation
C. high-anion-gap metabolic acidosis with respiratory compensation
D. muscle wasting with impaired connective tissue maintenance

27. On a medical-surgical unit, during clinical review, a 65-year-old patient has heavy protein loss in urine, hypoalbuminemia, edema, and hyperlipidemia. What is the principal underlying mechanism?

A. tubular epithelial damage causing obstruction and filtrate backleak
B. inflammatory disruption of glomerular capillary walls
C. phosphate retention and reduced renal activation of vitamin D
D. increased glomerular permeability to plasma proteins

28. On a medical-surgical unit, a case involves a 72-year-old adult who has severe epigastric pain after premature activation of pancreatic digestive enzymes. Which pathophysiologic process is most directly responsible?

A. transient or chronically reduced lower esophageal sphincter competence
B. increased resistance to portal flow through a scarred nodular liver
C. intrapancreatic enzyme activation causing autodigestion
D. discontinuous immune-mediated inflammation extending through the bowel wall

29. On a medical-surgical unit, the pathophysiology of epithelial metaplasia is reviewed in a 79-year-old patient. Which downstream finding should be expected?

A. intracellular sodium and water accumulation with cell swelling
B. loss of nuclei while the basic tissue architecture remains visible
C. lipid peroxidation with increased membrane permeability
D. greater injury resistance accompanied by reduced mucociliary clearance

30. On a medical-surgical unit, in a teaching case, a 18-year-old patient has ascites, splenomegaly, and esophageal varices with advanced hepatic fibrosis. Which process provides the best explanation?

A. increased resistance to portal flow through a scarred nodular liver
B. failure of conjugated bilirubin and bile salts to reach the intestine
C. transient or chronically reduced lower esophageal sphincter competence
D. intrapancreatic enzyme activation causing autodigestion

31. On a medical-surgical unit, during discussion of left-sided heart failure in a 25-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. reduced systemic vascular resistance despite normal or increased cardiac output
B. transudation of fluid into pulmonary interstitium and alveoli
C. peripheral fluid filtration with congestion of abdominal organs
D. compensatory vasoconstriction that raises afterload and worsens congestion

32. On a medical-surgical unit, a clinical scenario involving a 32-year-old patient demonstrates ischemic stroke. Which additional finding fits the same causal process?

A. reduced facilitation of intended movement with excessive inhibitory output
B. slowed or blocked saltatory conduction along affected axons
C. cytotoxic edema with expansion of an irreversibly injured core
D. declining neuromuscular transmission safety during repetitive stimulation

33. On a medical-surgical unit, during clinical review, a 39-year-old patient has persistent tissue injury with macrophages, lymphocytes, fibrosis, and attempted repair. What is the principal underlying mechanism?

A. immune-complex deposition with complement recruitment
B. sensitized T lymphocytes releasing cytokines and activating macrophages
C. IgG or IgM binding to cell-surface targets
D. ongoing immune activation occurring alongside tissue repair

34. On a medical-surgical unit, a 46-year-old patient is being evaluated for nephritic syndrome. Which additional consequence is most consistent with this disorder?

A. erythrocyte leakage into tubules with sodium and water retention
B. persistent parathyroid stimulation with increased bone resorption
C. impaired concentrating ability despite reduced glomerular filtration
D. avid renal sodium and water conservation with concentrated urine

35. On a medical-surgical unit, the pathophysiology of oxidative cellular injury is reviewed in a 53-year-old patient. Which downstream finding should be expected?

A. greater injury resistance accompanied by reduced mucociliary clearance
B. formation of membrane-bound fragments cleared by phagocytes
C. lipid peroxidation with increased membrane permeability
D. loss of nuclei while the basic tissue architecture remains visible

36. On a medical-surgical unit, in a teaching case, a 60-year-old patient has hyperglycemia after immune-mediated loss of pancreatic beta cells. Which process provides the best explanation?

A. glucocorticoid excess promoting protein catabolism and gluconeogenesis
B. absolute insulin deficiency from autoimmune beta-cell destruction
C. unrestrained lipolysis generating acidic ketone bodies
D. excess thyroid hormone increasing metabolic and adrenergic activity

37. On a medical-surgical unit, during discussion of Parkinson disease in a 67-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. reduced facilitation of intended movement with excessive inhibitory output
B. falling cerebral perfusion pressure as intracranial pressure approaches arterial pressure
C. slowed or blocked saltatory conduction along affected axons
D. cytotoxic edema with expansion of an irreversibly injured core

38. On a medical-surgical unit, a clinical scenario involving a 74-year-old patient demonstrates type 1 diabetes mellitus. Which additional finding fits the same causal process?

A. reduced TSH through negative feedback despite increased tissue stimulation
B. muscle wasting with impaired connective tissue maintenance
C. reduced cellular glucose uptake with increased lipolysis and ketogenesis
D. increased TSH when the pituitary-thyroid feedback loop is intact

39. On a medical-surgical unit, the care team reviews bicarbonate-loss metabolic acidosis in a 81-year-old adult. Which consequence best reflects its established pathophysiology?

A. volume expansion with hypokalemia and metabolic alkalosis
B. compensatory hyperventilation that lowers arterial carbon dioxide
C. movement of fluid from capillaries into the interstitial compartment
D. increased carbonic acid with renal bicarbonate retention if persistent

40. On a medical-surgical unit, a case involves a 20-year-old adult who has chronic kidney dysfunction with disturbed calcium and phosphate balance. Which pathophysiologic process is most directly responsible?

A. inflammatory disruption of glomerular capillary walls
B. increased glomerular permeability to plasma proteins
C. reduced renal perfusion without primary parenchymal damage
D. phosphate retention and reduced renal activation of vitamin D

41. On a medical-surgical unit, the clinician evaluates a 27-year-old patient who develops anemia, elevated indirect bilirubin, reduced haptoglobin, and hemoglobinuria. Which mechanism should be identified as primary?

A. premature rupture of erythrocytes within the circulation
B. widespread pathologic thrombin generation throughout the microcirculation
C. insufficient iron limiting hemoglobin synthesis
D. impaired DNA synthesis plus defective myelin maintenance

42. On a medical-surgical unit, in a teaching case, a 34-year-old patient has hyperglycemia, dehydration, ketones, and deep rapid respirations. Which process provides the best explanation?

A. insufficient thyroid hormone reducing basal metabolic activity
B. glucocorticoid excess promoting protein catabolism and gluconeogenesis
C. unrestrained lipolysis generating acidic ketone bodies
D. excess thyroid hormone increasing metabolic and adrenergic activity

43. On a medical-surgical unit, a 41-year-old adult has fatigue with microcytic, hypochromic erythrocytes after chronic blood loss. Which cellular or physiologic mechanism accounts for these findings?

A. impaired DNA synthesis plus defective myelin maintenance
B. insufficient neutrophil availability for an effective acute response
C. premature rupture of erythrocytes within the circulation
D. insufficient iron limiting hemoglobin synthesis

44. On a medical-surgical unit, a 48-year-old patient develops oliguria after substantial volume depletion while tubular function remains initially intact. Which mechanism best explains this pattern?

A. increased glomerular permeability to plasma proteins
B. reduced renal perfusion without primary parenchymal damage
C. phosphate retention and reduced renal activation of vitamin D
D. inflammatory disruption of glomerular capillary walls

45. On a medical-surgical unit, the care team reviews cortisol excess in a 55-year-old adult. Which consequence best reflects its established pathophysiology?

A. high-anion-gap metabolic acidosis with respiratory compensation
B. muscle wasting with impaired connective tissue maintenance
C. reduced TSH through negative feedback despite increased tissue stimulation
D. increased TSH when the pituitary-thyroid feedback loop is intact

46. On a medical-surgical unit, a case involves a 62-year-old adult who has jugular venous distention, dependent edema, and hepatic congestion. Which pathophysiologic process is most directly responsible?

A. elevated systemic venous pressure from impaired right ventricular output
B. severe pump failure reducing stroke volume and tissue perfusion
C. widespread vasodilation and maldistribution of circulating blood flow
D. intimal inflammation with oxidized lipoprotein uptake by macrophages

47. On a medical-surgical unit, the pathophysiology of acute pancreatitis is reviewed in a 69-year-old patient. Which downstream finding should be expected?

A. fibrosis and penetrating tracts between bowel and adjacent structures
B. reflux of water-soluble conjugated bilirubin into blood and urine
C. fat necrosis, edema, and systemic inflammatory mediator release
D. portosystemic collateral formation and elevated splanchnic hydrostatic pressure

48. On a medical-surgical unit, in a teaching case, a 76-year-old patient has concentrated urine despite low plasma osmolality and dilutional hyponatremia. Which process provides the best explanation?

A. alveolar ventilation inadequate for carbon dioxide production
B. reduced plasma colloid oncotic pressure favoring filtration
C. excess mineralocorticoid-driven sodium reabsorption in the distal nephron
D. continued antidiuretic hormone effect despite hypo-osmolality

49. On a medical-surgical unit, a 83-year-old adult develops vasculitic injury where circulating antigen-antibody complexes lodge in small vessels. Which cellular or physiologic mechanism accounts for these findings?

A. immune-complex deposition with complement recruitment
B. sensitized T lymphocytes releasing cytokines and activating macrophages
C. ongoing immune activation occurring alongside tissue repair
D. allergen cross-linking of IgE on sensitized mast cells

50. On a medical-surgical unit, a clinical scenario involving a 22-year-old patient demonstrates atherosclerotic plaque formation. Which additional finding fits the same causal process?

A. transudation of fluid into pulmonary interstitium and alveoli
B. compensatory vasoconstriction that raises afterload and worsens congestion
C. reduced systemic vascular resistance despite normal or increased cardiac output
D. foam-cell formation followed by a fibrous cap over a lipid core

51. On a medical-surgical unit, during clinical review, a 29-year-old patient has isolated damaged cells that shrink without provoking surrounding inflammation. What is the principal underlying mechanism?

A. ATP depletion causing failure of membrane ion pumps
B. reprogramming of local stem cells toward a stress-resistant phenotype
C. caspase-mediated programmed cellular self-destruction
D. ischemic protein denaturation preserving tissue outlines temporarily

52. On a medical-surgical unit, a case involves a 36-year-old adult who has hypotension with warm extremities early in a severe systemic inflammatory state. Which pathophysiologic process is most directly responsible?

A. elevated left atrial and pulmonary capillary hydrostatic pressure
B. widespread vasodilation and maldistribution of circulating blood flow
C. elevated systemic venous pressure from impaired right ventricular output
D. intimal inflammation with oxidized lipoprotein uptake by macrophages

53. On a medical-surgical unit, the pathophysiology of hypoalbuminemic edema is reviewed in a 43-year-old patient. Which downstream finding should be expected?

A. increased carbonic acid with renal bicarbonate retention if persistent
B. compensatory hyperventilation that lowers arterial carbon dioxide
C. movement of fluid from capillaries into the interstitial compartment
D. water retention that lowers serum sodium without proportional sodium loss

54. On a medical-surgical unit, a 50-year-old adult has findings compatible with severe neutropenia. Which associated change best follows from the underlying mechanism?

A. simultaneous microvascular thrombosis and consumption-related hemorrhage
B. progressively smaller red cells with reduced oxygen-carrying capacity
C. megaloblastic erythropoiesis with neurologic dysfunction
D. blunted local inflammation despite risk of rapidly progressive infection

55. On a medical-surgical unit, a 57-year-old adult has a pale wedge-shaped renal infarct after arterial occlusion. Which cellular or physiologic mechanism accounts for these findings?

A. ischemic protein denaturation preserving tissue outlines temporarily
B. reactive oxygen species attacking lipids, proteins, and DNA
C. ATP depletion causing failure of membrane ion pumps
D. reprogramming of local stem cells toward a stress-resistant phenotype

56. On a medical-surgical unit, a 64-year-old patient has hypertension, potassium depletion, and increased renal hydrogen ion secretion. Which mechanism best explains this pattern?

A. alveolar ventilation inadequate for carbon dioxide production
B. excess mineralocorticoid-driven sodium reabsorption in the distal nephron
C. gastrointestinal loss of bicarbonate-rich fluid
D. continued antidiuretic hormone effect despite hypo-osmolality

57. On a medical-surgical unit, during clinical review, a 71-year-old patient has proximal muscle weakness, hyperglycemia, thin skin, and central fat accumulation. What is the principal underlying mechanism?

A. unrestrained lipolysis generating acidic ketone bodies
B. absolute insulin deficiency from autoimmune beta-cell destruction
C. excess thyroid hormone increasing metabolic and adrenergic activity
D. glucocorticoid excess promoting protein catabolism and gluconeogenesis

58. On a medical-surgical unit, a 78-year-old patient is being evaluated for cardiogenic shock. Which additional consequence is most consistent with this disorder?

A. compensatory vasoconstriction that raises afterload and worsens congestion
B. reduced systemic vascular resistance despite normal or increased cardiac output
C. peripheral fluid filtration with congestion of abdominal organs
D. transudation of fluid into pulmonary interstitium and alveoli

59. On a medical-surgical unit, the clinician evaluates a 85-year-old patient who develops sudden hypoxemia when a clot blocks perfusion to ventilated alveoli. Which mechanism should be identified as primary?

A. bronchial smooth-muscle constriction plus mucosal inflammation
B. abrupt loss of regional pulmonary blood flow
C. diffuse alveolar-capillary barrier injury increasing permeability
D. protease-mediated loss of alveolar septa and supporting elastic tissue

60. On a medical-surgical unit, in a teaching case, a 24-year-old patient has antibodies directed against antigens fixed on red blood cell membranes. Which process provides the best explanation?

A. sensitized T lymphocytes releasing cytokines and activating macrophages
B. immune-complex deposition with complement recruitment
C. IgG or IgM binding to cell-surface targets
D. allergen cross-linking of IgE on sensitized mast cells

61. On a medical-surgical unit, a 31-year-old adult has chronic airway irritation and replacement of ciliated columnar cells by squamous cells. Which cellular or physiologic mechanism accounts for these findings?

A. reactive oxygen species attacking lipids, proteins, and DNA
B. reprogramming of local stem cells toward a stress-resistant phenotype
C. ischemic protein denaturation preserving tissue outlines temporarily
D. ATP depletion causing failure of membrane ion pumps

62. On a medical-surgical unit, a clinical scenario involving a 38-year-old patient demonstrates type I hypersensitivity. Which additional finding fits the same causal process?

A. neutrophil-rich inflammation damaging vessel walls and adjacent tissue
B. simultaneous parenchymal destruction and collagen deposition
C. rapid histamine release causing vasodilation and bronchoconstriction
D. delayed mononuclear inflammation without an antibody requirement

63. On a medical-surgical unit, the care team reviews type IV hypersensitivity in a 45-year-old adult. Which consequence best reflects its established pathophysiology?

A. delayed mononuclear inflammation without an antibody requirement
B. neutrophil-rich inflammation damaging vessel walls and adjacent tissue
C. complement activation and antibody-mediated cellular destruction
D. rapid histamine release causing vasodilation and bronchoconstriction

64. On a medical-surgical unit, a case involves a 52-year-old adult who has hyperinflation and reduced elastic recoil after destruction of alveolar walls. Which pathophysiologic process is most directly responsible?

A. diffuse alveolar-capillary barrier injury increasing permeability
B. abrupt loss of regional pulmonary blood flow
C. excess collagen deposition thickening the pulmonary interstitium
D. protease-mediated loss of alveolar septa and supporting elastic tissue

65. On a medical-surgical unit, the clinician evaluates a 59-year-old patient who develops acidemia after prolonged high-volume diarrhea. Which mechanism should be identified as primary?

A. gastrointestinal loss of bicarbonate-rich fluid
B. excess mineralocorticoid-driven sodium reabsorption in the distal nephron
C. reduced plasma colloid oncotic pressure favoring filtration
D. alveolar ventilation inadequate for carbon dioxide production

66. On a medical-surgical unit, a 66-year-old adult has findings compatible with coagulative necrosis. Which associated change best follows from the underlying mechanism?

A. greater injury resistance accompanied by reduced mucociliary clearance
B. lipid peroxidation with increased membrane permeability
C. loss of nuclei while the basic tissue architecture remains visible
D. formation of membrane-bound fragments cleared by phagocytes

67. On a medical-surgical unit, a 73-year-old adult has jaundice, dark urine, pale stools, and pruritus from blocked bile flow. Which cellular or physiologic mechanism accounts for these findings?

A. transient or chronically reduced lower esophageal sphincter competence
B. intrapancreatic enzyme activation causing autodigestion
C. increased resistance to portal flow through a scarred nodular liver
D. failure of conjugated bilirubin and bile salts to reach the intestine

68. On a medical-surgical unit, a 80-year-old patient has progressive arterial narrowing after endothelial injury and lipid accumulation. Which mechanism best explains this pattern?

A. severe pump failure reducing stroke volume and tissue perfusion
B. intimal inflammation with oxidized lipoprotein uptake by macrophages
C. widespread vasodilation and maldistribution of circulating blood flow
D. elevated left atrial and pulmonary capillary hydrostatic pressure

69. On a medical-surgical unit, during clinical review, a 19-year-old patient has neurologic deficits separated in time and location within the central nervous system. What is the principal underlying mechanism?

A. antibody-mediated loss or dysfunction of postsynaptic acetylcholine receptors
B. fixed cranial volume forcing compensatory displacement before pressure rises steeply
C. loss of dopaminergic input to basal ganglia motor circuits
D. immune-mediated destruction of central nervous system myelin

70. On a medical-surgical unit, a case involves a 26-year-old adult who has progressive exertional dyspnea with stiff lungs and reduced diffusion capacity. Which pathophysiologic process is most directly responsible?

A. excess collagen deposition thickening the pulmonary interstitium
B. protease-mediated loss of alveolar septa and supporting elastic tissue
C. abrupt loss of regional pulmonary blood flow
D. bronchial smooth-muscle constriction plus mucosal inflammation

71. On a medical-surgical unit, the pathophysiology of disseminated intravascular coagulation is reviewed in a 33-year-old patient. Which downstream finding should be expected?

A. blunted local inflammation despite risk of rapidly progressive infection
B. simultaneous microvascular thrombosis and consumption-related hemorrhage
C. progressively smaller red cells with reduced oxygen-carrying capacity
D. free hemoglobin binding haptoglobin until circulating stores are depleted

72. On a medical-surgical unit, a 40-year-old adult has findings compatible with vitamin B12 deficiency. Which associated change best follows from the underlying mechanism?

A. simultaneous microvascular thrombosis and consumption-related hemorrhage
B. progressively smaller red cells with reduced oxygen-carrying capacity
C. megaloblastic erythropoiesis with neurologic dysfunction
D. blunted local inflammation despite risk of rapidly progressive infection

73. On a medical-surgical unit, during discussion of diabetic ketoacidosis in a 47-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. reduced cellular glucose uptake with increased lipolysis and ketogenesis
B. high-anion-gap metabolic acidosis with respiratory compensation
C. muscle wasting with impaired connective tissue maintenance
D. increased TSH when the pituitary-thyroid feedback loop is intact

74. On a medical-surgical unit, a 54-year-old patient has patchy transmural intestinal inflammation with strictures and fistulas. Which mechanism best explains this pattern?

A. increased resistance to portal flow through a scarred nodular liver
B. failure of conjugated bilirubin and bile salts to reach the intestine
C. discontinuous immune-mediated inflammation extending through the bowel wall
D. transient or chronically reduced lower esophageal sphincter competence

75. On a medical-surgical unit, during clinical review, a 61-year-old patient has burning retrosternal discomfort after meals and when lying flat. What is the principal underlying mechanism?

A. discontinuous immune-mediated inflammation extending through the bowel wall
B. increased resistance to portal flow through a scarred nodular liver
C. failure of conjugated bilirubin and bile salts to reach the intestine
D. transient or chronically reduced lower esophageal sphincter competence

76. On a medical-surgical unit, a 68-year-old patient is being evaluated for intravascular hemolysis. Which additional consequence is most consistent with this disorder?

A. free hemoglobin binding haptoglobin until circulating stores are depleted
B. megaloblastic erythropoiesis with neurologic dysfunction
C. blunted local inflammation despite risk of rapidly progressive infection
D. progressively smaller red cells with reduced oxygen-carrying capacity

77. On a medical-surgical unit, the clinician evaluates a 75-year-old patient who has episodic wheezing with reversible expiratory airflow limitation. Which mechanism should be identified as primary?

A. bronchial smooth-muscle constriction plus mucosal inflammation
B. protease-mediated loss of alveolar septa and supporting elastic tissue
C. abrupt loss of regional pulmonary blood flow
D. diffuse alveolar-capillary barrier injury increasing permeability

78. On a medical-surgical unit, a 82-year-old adult has findings compatible with SIADH. Which associated change best follows from the underlying mechanism?

A. volume expansion with hypokalemia and metabolic alkalosis
B. water retention that lowers serum sodium without proportional sodium loss
C. increased carbonic acid with renal bicarbonate retention if persistent
D. movement of fluid from capillaries into the interstitial compartment

79. On a medical-surgical unit, during discussion of apoptosis in a 21-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. greater injury resistance accompanied by reduced mucociliary clearance
B. intracellular sodium and water accumulation with cell swelling
C. formation of membrane-bound fragments cleared by phagocytes
D. loss of nuclei while the basic tissue architecture remains visible

80. On a medical-surgical unit, a 28-year-old patient develops an indurated skin reaction two days after antigen exposure. Which mechanism best explains this pattern?

A. ongoing immune activation occurring alongside tissue repair
B. allergen cross-linking of IgE on sensitized mast cells
C. immune-complex deposition with complement recruitment
D. sensitized T lymphocytes releasing cytokines and activating macrophages

81. On a medical-surgical unit, the care team reviews primary hypothyroidism in a 35-year-old adult. Which consequence best reflects its established pathophysiology?

A. muscle wasting with impaired connective tissue maintenance
B. high-anion-gap metabolic acidosis with respiratory compensation
C. increased TSH when the pituitary-thyroid feedback loop is intact
D. reduced cellular glucose uptake with increased lipolysis and ketogenesis

82. On a medical-surgical unit, a case involves a 42-year-old adult who has fatigue, cold intolerance, slowed reflexes, and elevated pituitary stimulation. Which pathophysiologic process is most directly responsible?

A. insufficient thyroid hormone reducing basal metabolic activity
B. glucocorticoid excess promoting protein catabolism and gluconeogenesis
C. unrestrained lipolysis generating acidic ketone bodies
D. absolute insulin deficiency from autoimmune beta-cell destruction

83. On a medical-surgical unit, the clinician evaluates a 49-year-old patient who has bradykinesia, rigidity, and resting tremor from degeneration in the substantia nigra. Which mechanism should be identified as primary?

A. immune-mediated destruction of central nervous system myelin
B. antibody-mediated loss or dysfunction of postsynaptic acetylcholine receptors
C. fixed cranial volume forcing compensatory displacement before pressure rises steeply
D. loss of dopaminergic input to basal ganglia motor circuits

84. On a medical-surgical unit, a 56-year-old adult has findings compatible with extrahepatic biliary obstruction. Which associated change best follows from the underlying mechanism?

A. repeated acid exposure causing distal esophageal mucosal injury
B. reflux of water-soluble conjugated bilirubin into blood and urine
C. portosystemic collateral formation and elevated splanchnic hydrostatic pressure
D. fibrosis and penetrating tracts between bowel and adjacent structures

85. On a medical-surgical unit, during discussion of acute asthma in a 63-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. increased alveolar dead space with redistribution of perfusion
B. increased airway resistance that is greatest during expiration
C. protein-rich edema with surfactant dysfunction and intrapulmonary shunting
D. restrictive mechanics with impaired oxygen transfer across the alveolar membrane

86. On a medical-surgical unit, a 70-year-old patient has hematuria, red-cell casts, hypertension, and reduced filtration. Which mechanism best explains this pattern?

A. phosphate retention and reduced renal activation of vitamin D
B. tubular epithelial damage causing obstruction and filtrate backleak
C. reduced renal perfusion without primary parenchymal damage
D. inflammatory disruption of glomerular capillary walls

87. On a medical-surgical unit, the care team reviews nephrotic syndrome in a 77-year-old adult. Which consequence best reflects its established pathophysiology?

A. erythrocyte leakage into tubules with sodium and water retention
B. avid renal sodium and water conservation with concentrated urine
C. reduced oncotic pressure with compensatory hepatic lipoprotein synthesis
D. persistent parathyroid stimulation with increased bone resorption

88. On a medical-surgical unit, a case involves a 84-year-old adult who has fluctuating skeletal muscle weakness that worsens with repeated activity. Which pathophysiologic process is most directly responsible?

A. antibody-mediated loss or dysfunction of postsynaptic acetylcholine receptors
B. fixed cranial volume forcing compensatory displacement before pressure rises steeply
C. immune-mediated destruction of central nervous system myelin
D. loss of dopaminergic input to basal ganglia motor circuits

89. On a medical-surgical unit, the pathophysiology of right-sided heart failure is reviewed in a 23-year-old patient. Which downstream finding should be expected?

A. peripheral fluid filtration with congestion of abdominal organs
B. compensatory vasoconstriction that raises afterload and worsens congestion
C. reduced systemic vascular resistance despite normal or increased cardiac output
D. transudation of fluid into pulmonary interstitium and alveoli

90. On a medical-surgical unit, a 30-year-old adult has findings compatible with prerenal acute kidney injury. Which associated change best follows from the underlying mechanism?

A. erythrocyte leakage into tubules with sodium and water retention
B. reduced oncotic pressure with compensatory hepatic lipoprotein synthesis
C. persistent parathyroid stimulation with increased bone resorption
D. avid renal sodium and water conservation with concentrated urine

91. On a medical-surgical unit, during discussion of Crohn disease in a 37-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. repeated acid exposure causing distal esophageal mucosal injury
B. portosystemic collateral formation and elevated splanchnic hydrostatic pressure
C. fibrosis and penetrating tracts between bowel and adjacent structures
D. reflux of water-soluble conjugated bilirubin into blood and urine

92. On a medical-surgical unit, a 44-year-old patient develops generalized edema after a marked fall in plasma albumin. Which mechanism best explains this pattern?

A. continued antidiuretic hormone effect despite hypo-osmolality
B. reduced plasma colloid oncotic pressure favoring filtration
C. gastrointestinal loss of bicarbonate-rich fluid
D. excess mineralocorticoid-driven sodium reabsorption in the distal nephron

93. On a medical-surgical unit, during clinical review, a 51-year-old patient experiences reperfusion of previously ischemic tissue followed by additional membrane damage. What is the principal underlying mechanism?

A. reprogramming of local stem cells toward a stress-resistant phenotype
B. reactive oxygen species attacking lipids, proteins, and DNA
C. caspase-mediated programmed cellular self-destruction
D. ischemic protein denaturation preserving tissue outlines temporarily

94. On a medical-surgical unit, a case involves a 58-year-old adult who develops fever with minimal pus formation during a bacterial infection. Which pathophysiologic process is most directly responsible?

A. impaired DNA synthesis plus defective myelin maintenance
B. widespread pathologic thrombin generation throughout the microcirculation
C. insufficient neutrophil availability for an effective acute response
D. insufficient iron limiting hemoglobin synthesis

95. On a medical-surgical unit, the pathophysiology of reversible hypoxic cell injury is reviewed in a 65-year-old patient. Which downstream finding should be expected?

A. formation of membrane-bound fragments cleared by phagocytes
B. lipid peroxidation with increased membrane permeability
C. loss of nuclei while the basic tissue architecture remains visible
D. intracellular sodium and water accumulation with cell swelling

96. On a medical-surgical unit, in a teaching case, a 72-year-old patient develops cellular swelling shortly after a brief episode of tissue hypoxia. Which process provides the best explanation?

A. ATP depletion causing failure of membrane ion pumps
B. reprogramming of local stem cells toward a stress-resistant phenotype
C. ischemic protein denaturation preserving tissue outlines temporarily
D. caspase-mediated programmed cellular self-destruction

97. On a medical-surgical unit, during discussion of secondary hyperparathyroidism in chronic kidney disease in a 79-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. persistent parathyroid stimulation with increased bone resorption
B. reduced oncotic pressure with compensatory hepatic lipoprotein synthesis
C. avid renal sodium and water conservation with concentrated urine
D. erythrocyte leakage into tubules with sodium and water retention

98. On a medical-surgical unit, a clinical scenario involving a 18-year-old patient demonstrates respiratory acidosis. Which additional finding fits the same causal process?

A. movement of fluid from capillaries into the interstitial compartment
B. volume expansion with hypokalemia and metabolic alkalosis
C. water retention that lowers serum sodium without proportional sodium loss
D. increased carbonic acid with renal bicarbonate retention if persistent

99. On a medical-surgical unit, during clinical review, a 25-year-old patient develops declining consciousness as an expanding intracranial lesion reduces cerebral perfusion. What is the principal underlying mechanism?

A. antibody-mediated loss or dysfunction of postsynaptic acetylcholine receptors
B. fixed cranial volume forcing compensatory displacement before pressure rises steeply
C. energy failure causing loss of ion gradients and excitotoxic injury
D. loss of dopaminergic input to basal ganglia motor circuits

100. On a medical-surgical unit, a 32-year-old patient is being evaluated for increased intracranial pressure. Which additional consequence is most consistent with this disorder?

A. declining neuromuscular transmission safety during repetitive stimulation
B. reduced facilitation of intended movement with excessive inhibitory output
C. falling cerebral perfusion pressure as intracranial pressure approaches arterial pressure
D. cytotoxic edema with expansion of an irreversibly injured core

Practice Exam 10: Answer Key And Explanations

1. C — Inflammatory mediators can cause profound arteriolar dilation and abnormal vascular permeability. Effective perfusion falls because vascular capacity expands and flow is maldistributed, even when the heart initially maintains or increases output. Therefore, reduced systemic vascular resistance despite normal or increased cardiac output is the expected consequence. The other choices follow different disease processes and do not arise from.

2. A — Excess thyroid hormone raises metabolic turnover and increases responsiveness to catecholamines, producing heat intolerance, tremor, and weight loss. Negative feedback suppresses pituitary thyroid-stimulating hormone when the excess originates in the thyroid. Therefore, excess thyroid hormone increasing metabolic and adrenergic activity is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the.

3. B — Destruction of alveolar septa removes both capillary surface and elastic support for small airways. Reduced recoil favors expiratory collapse and air trapping, while the lost interface decreases diffusing capacity. Therefore, premature small-airway collapse with reduced gas-exchange surface area is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting.

4. D — Inflammatory demyelination disrupts oligodendrocyte myelin in scattered central nervous system sites. Loss of insulation slows or blocks impulse conduction, producing episodic deficits whose location depends on the plaques involved. Therefore, slowed or blocked saltatory conduction along affected axons is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting.

5. C — In type III hypersensitivity, soluble immune complexes deposit in tissues and activate complement. Recruited neutrophils release enzymes and oxidants, producing inflammation at sites such as vessels, glomeruli, or joints. Therefore, neutrophil-rich inflammation damaging vessel walls and adjacent tissue is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting.

6. B — Inflammatory injury disrupts pulmonary endothelial and epithelial barriers, allowing protein-rich fluid into alveoli. Surfactant impairment promotes collapse, and perfusion of poorly ventilated units creates shunt physiology that can resist supplemental oxygen. Therefore, protein-rich edema with surfactant dysfunction and intrapulmonary shunting is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism.

7. D — Systemic coagulation activation produces fibrin-rich microthrombi and consumes platelets and clotting proteins. Secondary fibrinolysis adds further bleeding risk, so thrombosis, organ ischemia, and hemorrhage can coexist. Therefore, widespread pathologic thrombin generation throughout the microcirculation is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in the stem. Tracing.

8. A — An ineffective antireflux barrier allows acidic gastric contents to enter the esophagus, especially when pressure relationships favor reflux. Repeated exposure injures squamous mucosa and produces burning discomfort and inflammation. Therefore, repeated acid exposure causing distal esophageal mucosal injury is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting.

9. A — Failure of left ventricular forward pumping raises upstream left atrial and pulmonary venous pressures. Increased pulmonary capillary hydrostatic pressure drives fluid into lung tissue, reducing compliance and impairing gas exchange. Therefore, elevated left atrial and pulmonary capillary hydrostatic pressure is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire.

10. D — Prolonged ischemia injures tubular epithelial cells. Sloughed cells obstruct lumens, damaged epithelium permits filtrate backleak, and abnormal tubuloglomerular feedback reduces filtration; injured tubules also lose efficient reabsorptive and concentrating function. Therefore, tubular epithelial damage causing obstruction and filtrate backleak is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire.

11. B — Chronic inflammation combines prolonged injurious signaling with repair. Macrophages and lymphocytes sustain cytokine activity while growth factors stimulate angiogenesis and fibrosis, producing tissue destruction and scarring at the same time. Therefore, simultaneous parenchymal destruction and collagen deposition is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting the.

12. C — Immediate hypersensitivity follows IgE-mediated mast-cell activation. Granule mediators such as histamine rapidly increase vascular permeability and contract bronchial smooth muscle, explaining the abrupt skin and airway manifestations. Therefore, allergen cross-linking of ige on sensitized mast cells is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in the.

13. B — Type II reactions involve antibodies against antigens on cells or extracellular structures. Complement, opsonization, or receptor alteration then causes tissue-specific injury rather than deposition of circulating antigen-antibody complexes. Therefore, complement activation and antibody-mediated cellular destruction is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting the initiating abnormality.

14. C — Fibrosis and regenerative nodules distort hepatic vascular channels and raise resistance to portal inflow. Pressure is transmitted backward, promoting collateral vessels, splenic congestion, and filtration of fluid into the peritoneal cavity. Therefore, portosystemic collateral formation and elevated splanchnic hydrostatic pressure is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism.

15. D — Prolonged ischemia injures tubular epithelial cells. Sloughed cells obstruct lumens, damaged epithelium permits filtrate backleak, and abnormal tubuloglomerular feedback reduces filtration; injured tubules also lose efficient reabsorptive and concentrating function. Therefore, impaired concentrating ability despite reduced glomerular filtration is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting.

16. A — Interrupted oxygen and glucose delivery rapidly depletes neuronal ATP. Membrane depolarization, calcium influx, and excitatory neurotransmitter release amplify injury, producing cytotoxic edema and cell death in the most severely underperfused tissue. Therefore, energy failure causing loss of ion gradients and excitotoxic injury is the best answer. The competing choices represent mechanisms from related disorders, but they do not.

17. D — Aldosterone increases distal sodium reabsorption while promoting potassium and hydrogen secretion. Excess activity expands extracellular volume and raises blood pressure while creating potassium depletion and an alkalotic tendency. Therefore, volume expansion with hypokalemia and metabolic alkalosis is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting the initiating.

18. A — Major myocardial dysfunction lowers stroke volume and cardiac output. Sympathetic vasoconstriction attempts to preserve arterial pressure but increases afterload, while blood backs up into the pulmonary circulation and tissue perfusion remains inadequate. Therefore, severe pump failure reducing stroke volume and tissue perfusion is the best answer. The competing choices represent mechanisms from related disorders, but they do not.

19. B — Iron is required for heme production. Depleted stores restrict hemoglobin synthesis, causing developing erythrocytes to undergo additional divisions and emerge smaller and paler, with diminished capacity to deliver oxygen. Therefore, progressively smaller red cells with reduced oxygen-carrying capacity is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting.

20. C — When effective alveolar ventilation falls, carbon dioxide accumulates and shifts the carbonic acid equilibrium toward hydrogen ions. With persistence, the kidneys compensate by conserving bicarbonate and increasing acid excretion. Therefore, alveolar ventilation inadequate for carbon dioxide production is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in.

21. B — Autoantibodies impair the postsynaptic neuromuscular membrane and reduce functional acetylcholine receptors. Repeated stimulation exhausts the available transmission margin, so weakness becomes more apparent with activity and improves with rest. Therefore, declining neuromuscular transmission safety during repetitive stimulation is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting the.

22. D — An embolus creates regions that continue receiving ventilation but little or no perfusion. This increases dead-space ventilation and redirects blood to other lung units, where mismatch can contribute to arterial hypoxemia. Therefore, increased alveolar dead space with redistribution of perfusion is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism.

23. A — Interstitial collagen makes the lung less compliant and increases the diffusion distance between alveolar gas and capillary blood. Patients therefore require greater effort to expand the lungs and develop impaired oxygen transfer. Therefore, restrictive mechanics with impaired oxygen transfer across the alveolar membrane is the expected consequence. The other choices follow different disease processes and do not arise.

24. C — Inflammatory injury disrupts pulmonary endothelial and epithelial barriers, allowing protein-rich fluid into alveoli. Surfactant impairment promotes collapse, and perfusion of poorly ventilated units creates shunt physiology that can resist supplemental oxygen. Therefore, diffuse alveolar-capillary barrier injury increasing permeability is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern.

25. A — Vitamin B12 deficiency disrupts nucleotide synthesis, producing ineffective megaloblastic hematopoiesis. It also permits metabolites associated with myelin injury to accumulate, which distinguishes it from folate deficiency when neurologic findings are present. Therefore, impaired dna synthesis plus defective myelin maintenance is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire.

26. B — Excess thyroid hormone raises metabolic turnover and increases responsiveness to catecholamines, producing heat intolerance, tremor, and weight loss. Negative feedback suppresses pituitary thyroid-stimulating hormone when the excess originates in the thyroid. Therefore, reduced tsh through negative feedback despite increased tissue stimulation is the expected consequence. The other choices follow different disease processes and do not arise from the.

27. D — Glomerular barrier injury permits substantial albumin loss. Falling plasma oncotic pressure promotes edema, while the liver increases protein and lipoprotein synthesis; urinary loss of anticoagulant proteins can also favor thrombosis. Therefore, increased glomerular permeability to plasma proteins is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in.

28. C — Digestive enzymes activated within the pancreas injure acinar tissue and vessels. Local autodigestion produces edema and fat necrosis, while released inflammatory mediators can cause capillary leak and distant organ dysfunction. Therefore, intrapancreatic enzyme activation causing autodigestion is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in the.

29. D — Metaplasia is an adaptive, potentially reversible change in cell differentiation. Squamous epithelium tolerates irritation better than ciliated epithelium, but the tradeoff is loss of specialized mucus-clearing function and increased vulnerability to further change. Therefore, greater injury resistance accompanied by reduced mucociliary clearance is the expected consequence. The other choices follow different disease processes and do not arise from.

30. A — Fibrosis and regenerative nodules distort hepatic vascular channels and raise resistance to portal inflow. Pressure is transmitted backward, promoting collateral vessels, splenic congestion, and filtration of fluid into the peritoneal cavity. Therefore, increased resistance to portal flow through a scarred nodular liver is the best answer. The competing choices represent mechanisms from related disorders, but they do not.

31. B — Failure of left ventricular forward pumping raises upstream left atrial and pulmonary venous pressures. Increased pulmonary capillary hydrostatic pressure drives fluid into lung tissue, reducing compliance and impairing gas exchange. Therefore, transudation of fluid into pulmonary interstitium and alveoli is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described..

32. C — Interrupted oxygen and glucose delivery rapidly depletes neuronal ATP. Membrane depolarization, calcium influx, and excitatory neurotransmitter release amplify injury, producing cytotoxic edema and cell death in the most severely underperfused tissue. Therefore, cytotoxic edema with expansion of an irreversibly injured core is the expected consequence. The other choices follow different disease processes and do not arise from the.

33. D — Chronic inflammation combines prolonged injurious signaling with repair. Macrophages and lymphocytes sustain cytokine activity while growth factors stimulate angiogenesis and fibrosis, producing tissue destruction and scarring at the same time. Therefore, ongoing immune activation occurring alongside tissue repair is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern.

34. A — Glomerular inflammation damages capillary walls, allowing red cells to enter filtrate and form casts. Reduced filtration and activation of sodium-retaining pathways expand volume, contributing to edema and hypertension. Therefore, erythrocyte leakage into tubules with sodium and water retention is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting.

35. C — Reintroduced oxygen can generate reactive oxygen species faster than antioxidant systems neutralize them. Free radicals initiate lipid peroxidation, alter proteins, and damage DNA, thereby worsening membrane and organelle injury after perfusion returns. Therefore, lipid peroxidation with increased membrane permeability is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described..

36. B — Loss of beta cells removes insulin's anabolic signals. Glucose uptake falls in insulin-dependent tissues while hepatic glucose output, fat breakdown, and ketone production rise, creating hyperglycemia and a risk of ketoacidosis. Therefore, absolute insulin deficiency from autoimmune beta-cell destruction is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire.

37. A — Degeneration of nigrostriatal dopamine neurons disrupts the balance of basal ganglia pathways. Reduced activation of movement-facilitating circuits and relative dominance of inhibitory signaling make movement initiation slow and produce characteristic rigidity and tremor. Therefore, reduced facilitation of intended movement with excessive inhibitory output is the expected consequence. The other choices follow different disease processes and do not arise.

38. C — Loss of beta cells removes insulin's anabolic signals. Glucose uptake falls in insulin-dependent tissues while hepatic glucose output, fat breakdown, and ketone production rise, creating hyperglycemia and a risk of ketoacidosis. Therefore, reduced cellular glucose uptake with increased lipolysis and ketogenesis is the expected consequence. The other choices follow different disease processes and do not arise from the.

39. B — Lower intestinal fluid contains bicarbonate, so sustained diarrhea reduces the body's base reserve and produces metabolic acidosis. Chemoreceptor stimulation increases ventilation, lowering carbon dioxide as respiratory compensation rather than correcting the original loss. Therefore, compensatory hyperventilation that lowers arterial carbon dioxide is the expected consequence. The other choices follow different disease processes and do not arise from the.

40. D — Failing kidneys retain phosphate and produce less active vitamin D, decreasing intestinal calcium absorption. The resulting signals stimulate parathyroid hormone release, which promotes skeletal mineral mobilization and contributes to renal bone disease. Therefore, phosphate retention and reduced renal activation of vitamin d is the best answer. The competing choices represent mechanisms from related disorders, but they do not.

41. A — Intravascular red-cell destruction releases hemoglobin directly into plasma. Haptoglobin binds the free protein and becomes depleted; excess filtered hemoglobin may enter urine while heme breakdown increases unconjugated bilirubin. Therefore, premature rupture of erythrocytes within the circulation is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in the.

42. C — Severe insulin deficiency and excess counterregulatory hormones drive fatty-acid release and hepatic ketogenesis. Accumulated ketoacids consume bicarbonate; osmotic diuresis causes dehydration, and deep ventilation lowers carbon dioxide as compensation. Therefore, unrestrained lipolysis generating acidic ketone bodies is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in the.

43. D — Iron is required for heme production. Depleted stores restrict hemoglobin synthesis, causing developing erythrocytes to undergo additional divisions and emerge smaller and paler, with diminished capacity to deliver oxygen. Therefore, insufficient iron limiting hemoglobin synthesis is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in the stem..

44. B — Reduced effective circulating volume lowers glomerular perfusion. Intact tubules respond by reabsorbing sodium and water under sympathetic and hormonal influence, producing concentrated urine while attempting to restore intravascular volume. Therefore, reduced renal perfusion without primary parenchymal damage is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in.

45. B — Sustained cortisol excess mobilizes amino acids through protein breakdown and increases hepatic glucose production. Catabolism weakens proximal muscles and thins skin, while insulin resistance and characteristic fat redistribution alter body composition. Therefore, muscle wasting with impaired connective tissue maintenance is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described..

46. A — Right ventricular pump failure raises pressure in the systemic venous circulation. The resulting hydrostatic force promotes dependent edema and distends neck veins while venous backup congests the liver and other organs. Therefore, elevated systemic venous pressure from impaired right ventricular output is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain.

47. C — Digestive enzymes activated within the pancreas injure acinar tissue and vessels. Local autodigestion produces edema and fat necrosis, while released inflammatory mediators can cause capillary leak and distant organ dysfunction. Therefore, fat necrosis, edema, and systemic inflammatory mediator release is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described..

48. D — Inappropriate antidiuretic hormone activity increases collecting-duct water reabsorption. Retained free water dilutes plasma sodium, suppresses plasma osmolality, and leaves urine inappropriately concentrated for the patient's volume state. Therefore, continued antidiuretic hormone effect despite hypo-osmolality is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in the stem. Tracing.

49. A — In type III hypersensitivity, soluble immune complexes deposit in tissues and activate complement. Recruited neutrophils release enzymes and oxidants, producing inflammation at sites such as vessels, glomeruli, or joints. Therefore, immune-complex deposition with complement recruitment is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in the stem..

50. D — Endothelial dysfunction permits lipoprotein entry and inflammatory-cell recruitment. Macrophages ingest modified lipids to become foam cells, while smooth-muscle migration and matrix deposition create a fibrous plaque that can narrow or rupture. Therefore, foam-cell formation followed by a fibrous cap over a lipid core is the expected consequence. The other choices follow different disease processes and do not arise.

51. C — Apoptosis activates regulated caspase pathways. The cell condenses and fragments into membrane-bound apoptotic bodies, allowing phagocytosis without leakage of intracellular contents or a prominent inflammatory response. Therefore, caspase-mediated programmed cellular self-destruction is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in the stem. Tracing the sequence from.

52. B — Inflammatory mediators can cause profound arteriolar dilation and abnormal vascular permeability. Effective perfusion falls because vascular capacity expands and flow is maldistributed, even when the heart initially maintains or increases output. Therefore, widespread vasodilation and maldistribution of circulating blood flow is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the.

53. C — Albumin supplies much of plasma oncotic pressure. When albumin falls substantially, capillary reabsorption is reduced and net filtration into tissues increases, producing interstitial edema even when total body water is not initially excessive. Therefore, movement of fluid from capillaries into the interstitial compartment is the expected consequence. The other choices follow different disease processes and do not arise.

54. D — Neutrophils are essential early phagocytes in bacterial defense and major contributors to pus. Profound deficiency can mute redness or purulence even while organisms spread, making systemic signs such as fever especially important. Therefore, blunted local inflammation despite risk of rapidly progressive infection is the expected consequence. The other choices follow different disease processes and do not arise from.

55. A — Ischemia in most solid organs denatures structural proteins and enzymes. Proteolysis is delayed, so ghostlike cell outlines persist even though nuclei disappear and the affected cells are irreversibly injured. Therefore, ischemic protein denaturation preserving tissue outlines temporarily is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in.

56. B — Aldosterone increases distal sodium reabsorption while promoting potassium and hydrogen secretion. Excess activity expands extracellular volume and raises blood pressure while creating potassium depletion and an alkalotic tendency. Therefore, excess mineralocorticoid-driven sodium reabsorption in the distal nephron is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in.

57. D — Sustained cortisol excess mobilizes amino acids through protein breakdown and increases hepatic glucose production. Catabolism weakens proximal muscles and thins skin, while insulin resistance and characteristic fat redistribution alter body composition. Therefore, glucocorticoid excess promoting protein catabolism and gluconeogenesis is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire.

58. A — Major myocardial dysfunction lowers stroke volume and cardiac output. Sympathetic vasoconstriction attempts to preserve arterial pressure but increases afterload, while blood backs up into the pulmonary circulation and tissue perfusion remains inadequate. Therefore, compensatory vasoconstriction that raises afterload and worsens congestion is the expected consequence. The other choices follow different disease processes and do not arise from the.

59. B — An embolus creates regions that continue receiving ventilation but little or no perfusion. This increases dead-space ventilation and redirects blood to other lung units, where mismatch can contribute to arterial hypoxemia. Therefore, abrupt loss of regional pulmonary blood flow is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire.

60. C — Type II reactions involve antibodies against antigens on cells or extracellular structures. Complement, opsonization, or receptor alteration then causes tissue-specific injury rather than deposition of circulating antigen-antibody complexes. Therefore, igg or igm binding to cell-surface targets is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in the.

61. B — Metaplasia is an adaptive, potentially reversible change in cell differentiation. Squamous epithelium tolerates irritation better than ciliated epithelium, but the tradeoff is loss of specialized mucus-clearing function and increased vulnerability to further change. Therefore, reprogramming of local stem cells toward a stress-resistant phenotype is the best answer. The competing choices represent mechanisms from related disorders, but they do.

62. C — Immediate hypersensitivity follows IgE-mediated mast-cell activation. Granule mediators such as histamine rapidly increase vascular permeability and contract bronchial smooth muscle, explaining the abrupt skin and airway manifestations. Therefore, rapid histamine release causing vasodilation and bronchoconstriction is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting the initiating abnormality.

63. A — Delayed hypersensitivity is mediated by antigen-specific T cells rather than antibodies. Cytokine release recruits and activates macrophages, so induration and tissue injury evolve over many hours instead of appearing immediately. Therefore, delayed mononuclear inflammation without an antibody requirement is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting.

64. D — Destruction of alveolar septa removes both capillary surface and elastic support for small airways. Reduced recoil favors expiratory collapse and air trapping, while the lost interface decreases diffusing capacity. Therefore, protease-mediated loss of alveolar septa and supporting elastic tissue is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire.

65. A — Lower intestinal fluid contains bicarbonate, so sustained diarrhea reduces the body's base reserve and produces metabolic acidosis. Chemoreceptor stimulation increases ventilation, lowering carbon dioxide as respiratory compensation rather than correcting the original loss. Therefore, gastrointestinal loss of bicarbonate-rich fluid is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire.

66. C — Ischemia in most solid organs denatures structural proteins and enzymes. Proteolysis is delayed, so ghostlike cell outlines persist even though nuclei disappear and the affected cells are irreversibly injured. Therefore, loss of nuclei while the basic tissue architecture remains visible is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism.

67. D — Obstruction prevents bile from entering the intestine. Conjugated bilirubin returns to the circulation and is filtered into urine, while less pigment reaches stool; retained bile salts contribute to itching. Therefore, failure of conjugated bilirubin and bile salts to reach the intestine is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain.

68. B — Endothelial dysfunction permits lipoprotein entry and inflammatory-cell recruitment. Macrophages ingest modified lipids to become foam cells, while smooth-muscle migration and matrix deposition create a fibrous plaque that can narrow or rupture. Therefore, intimal inflammation with oxidized lipoprotein uptake by macrophages is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the.

69. D — Inflammatory demyelination disrupts oligodendrocyte myelin in scattered central nervous system sites. Loss of insulation slows or blocks impulse conduction, producing episodic deficits whose location depends on the plaques involved. Therefore, immune-mediated destruction of central nervous system myelin is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in.

70. A — Interstitial collagen makes the lung less compliant and increases the diffusion distance between alveolar gas and capillary blood. Patients therefore require greater effort to expand the lungs and develop impaired oxygen transfer. Therefore, excess collagen deposition thickening the pulmonary interstitium is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the.

71. B — Systemic coagulation activation produces fibrin-rich microthrombi and consumes platelets and clotting proteins. Secondary fibrinolysis adds further bleeding risk, so thrombosis, organ ischemia, and hemorrhage can coexist. Therefore, simultaneous microvascular thrombosis and consumption-related hemorrhage is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting the initiating abnormality to its.

72. C — Vitamin B12 deficiency disrupts nucleotide synthesis, producing ineffective megaloblastic hematopoiesis. It also permits metabolites associated with myelin injury to accumulate, which distinguishes it from folate deficiency when neurologic findings are present. Therefore, megaloblastic erythropoiesis with neurologic dysfunction is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting the.

73. B — Severe insulin deficiency and excess counterregulatory hormones drive fatty-acid release and hepatic ketogenesis. Accumulated ketoacids consume bicarbonate; osmotic diuresis causes dehydration, and deep ventilation lowers carbon dioxide as compensation. Therefore, high-anion-gap metabolic acidosis with respiratory compensation is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting the initiating.

74. C — Crohn disease produces skip lesions and transmural inflammation rather than continuous mucosal disease. Deep injury can penetrate to create fistulas, while repeated inflammation and repair narrow the lumen through fibrosis. Therefore, discontinuous immune-mediated inflammation extending through the bowel wall is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire.

75. D — An ineffective antireflux barrier allows acidic gastric contents to enter the esophagus, especially when pressure relationships favor reflux. Repeated exposure injures squamous mucosa and produces burning discomfort and inflammation. Therefore, transient or chronically reduced lower esophageal sphincter competence is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern.

76. A — Intravascular red-cell destruction releases hemoglobin directly into plasma. Haptoglobin binds the free protein and becomes depleted; excess filtered hemoglobin may enter urine while heme breakdown increases unconjugated bilirubin. Therefore, free hemoglobin binding haptoglobin until circulating stores are depleted is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting.

77. A — Asthma combines airway inflammation, hyperresponsiveness, bronchoconstriction, and mucus production. Narrowed intrathoracic airways impede expiratory flow particularly strongly, producing wheezing and air trapping that can improve when the airway obstruction reverses. Therefore, bronchial smooth-muscle constriction plus mucosal inflammation is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in.

78. B — Inappropriate antidiuretic hormone activity increases collecting-duct water reabsorption. Retained free water dilutes plasma sodium, suppresses plasma osmolality, and leaves urine inappropriately concentrated for the patient's volume state. Therefore, water retention that lowers serum sodium without proportional sodium loss is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting.

79. C — Apoptosis activates regulated caspase pathways. The cell condenses and fragments into membrane-bound apoptotic bodies, allowing phagocytosis without leakage of intracellular contents or a prominent inflammatory response. Therefore, formation of membrane-bound fragments cleared by phagocytes is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting the initiating abnormality to.

80. D — Delayed hypersensitivity is mediated by antigen-specific T cells rather than antibodies. Cytokine release recruits and activates macrophages, so induration and tissue injury evolve over many hours instead of appearing immediately. Therefore, sensitized t lymphocytes releasing cytokines and activating macrophages is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire.

81. C — Low thyroid hormone slows cellular metabolic processes and removes negative feedback from the hypothalamus and pituitary. An intact pituitary therefore increases thyroid-stimulating hormone in an unsuccessful effort to restore hormone production. Therefore, increased tsh when the pituitary-thyroid feedback loop is intact is the expected consequence. The other choices follow different disease processes and do not arise from the.

82. A — Low thyroid hormone slows cellular metabolic processes and removes negative feedback from the hypothalamus and pituitary. An intact pituitary therefore increases thyroid-stimulating hormone in an unsuccessful effort to restore hormone production. Therefore, insufficient thyroid hormone reducing basal metabolic activity is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire.

83. D — Degeneration of nigrostriatal dopamine neurons disrupts the balance of basal ganglia pathways. Reduced activation of movement-facilitating circuits and relative dominance of inhibitory signaling make movement initiation slow and produce characteristic rigidity and tremor. Therefore, loss of dopaminergic input to basal ganglia motor circuits is the best answer. The competing choices represent mechanisms from related disorders, but they do.

84. B — Obstruction prevents bile from entering the intestine. Conjugated bilirubin returns to the circulation and is filtered into urine, while less pigment reaches stool; retained bile salts contribute to itching. Therefore, reflux of water-soluble conjugated bilirubin into blood and urine is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described..

85. B — Asthma combines airway inflammation, hyperresponsiveness, bronchoconstriction, and mucus production. Narrowed intrathoracic airways impede expiratory flow particularly strongly, producing wheezing and air trapping that can improve when the airway obstruction reverses. Therefore, increased airway resistance that is greatest during expiration is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described..

86. D — Glomerular inflammation damages capillary walls, allowing red cells to enter filtrate and form casts. Reduced filtration and activation of sodium-retaining pathways expand volume, contributing to edema and hypertension. Therefore, inflammatory disruption of glomerular capillary walls is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern in the stem..

87. C — Glomerular barrier injury permits substantial albumin loss. Falling plasma oncotic pressure promotes edema, while the liver increases protein and lipoprotein synthesis; urinary loss of anticoagulant proteins can also favor thrombosis. Therefore, reduced oncotic pressure with compensatory hepatic lipoprotein synthesis is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described..

88. A — Autoantibodies impair the postsynaptic neuromuscular membrane and reduce functional acetylcholine receptors. Repeated stimulation exhausts the available transmission margin, so weakness becomes more apparent with activity and improves with rest. Therefore, antibody-mediated loss or dysfunction of postsynaptic acetylcholine receptors is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern.

89. A — Right ventricular pump failure raises pressure in the systemic venous circulation. The resulting hydrostatic force promotes dependent edema and distends neck veins while venous backup congests the liver and other organs. Therefore, peripheral fluid filtration with congestion of abdominal organs is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism.

90. D — Reduced effective circulating volume lowers glomerular perfusion. Intact tubules respond by reabsorbing sodium and water under sympathetic and hormonal influence, producing concentrated urine while attempting to restore intravascular volume. Therefore, avid renal sodium and water conservation with concentrated urine is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described..

91. C — Crohn disease produces skip lesions and transmural inflammation rather than continuous mucosal disease. Deep injury can penetrate to create fistulas, while repeated inflammation and repair narrow the lumen through fibrosis. Therefore, fibrosis and penetrating tracts between bowel and adjacent structures is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism.

92. B — Albumin supplies much of plasma oncotic pressure. When albumin falls substantially, capillary reabsorption is reduced and net filtration into tissues increases, producing interstitial edema even when total body water is not initially excessive. Therefore, reduced plasma colloid oncotic pressure favoring filtration is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain.

93. B — Reintroduced oxygen can generate reactive oxygen species faster than antioxidant systems neutralize them. Free radicals initiate lipid peroxidation, alter proteins, and damage DNA, thereby worsening membrane and organelle injury after perfusion returns. Therefore, reactive oxygen species attacking lipids, proteins, and dna is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain.

94. C — Neutrophils are essential early phagocytes in bacterial defense and major contributors to pus. Profound deficiency can mute redness or purulence even while organisms spread, making systemic signs such as fever especially important. Therefore, insufficient neutrophil availability for an effective acute response is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain.

95. D — Limited oxygen reduces oxidative phosphorylation and ATP. Sodium-potassium pump failure then permits sodium and water to enter the cell; restoration of oxygen before membrane disruption can reverse the swelling. Therefore, intracellular sodium and water accumulation with cell swelling is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described. Connecting.

96. A — Limited oxygen reduces oxidative phosphorylation and ATP. Sodium-potassium pump failure then permits sodium and water to enter the cell; restoration of oxygen before membrane disruption can reverse the swelling. Therefore, atp depletion causing failure of membrane ion pumps is the best answer. The competing choices represent mechanisms from related disorders, but they do not explain the entire pattern.

97. A — Failing kidneys retain phosphate and produce less active vitamin D, decreasing intestinal calcium absorption. The resulting signals stimulate parathyroid hormone release, which promotes skeletal mineral mobilization and contributes to renal bone disease. Therefore, persistent parathyroid stimulation with increased bone resorption is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism.

98. D — When effective alveolar ventilation falls, carbon dioxide accumulates and shifts the carbonic acid equilibrium toward hydrogen ions. With persistence, the kidneys compensate by conserving bicarbonate and increasing acid excretion. Therefore, increased carbonic acid with renal bicarbonate retention if persistent is the expected consequence. The other choices follow different disease processes and do not arise from the mechanism described..

99. B — The rigid skull contains brain, blood, and cerebrospinal fluid. Early displacement can buffer added volume, but exhausted compensation causes a steep pressure rise; increasing intracranial pressure then reduces the gradient driving cerebral blood flow. Therefore, fixed cranial volume forcing compensatory displacement before pressure rises steeply is the best answer. The competing choices represent mechanisms from related disorders, but.

100. C — The rigid skull contains brain, blood, and cerebrospinal fluid. Early displacement can buffer added volume, but exhausted compensation causes a steep pressure rise; increasing intracranial pressure then reduces the gradient driving cerebral blood flow. Therefore, falling cerebral perfusion pressure as intracranial pressure approaches arterial pressure is the expected consequence. The other choices follow different disease processes and do.