PRACTICE EXAM 21: 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. During a teaching-round evaluation, the pathophysiology of type I hypersensitivity is reviewed in a 26-year-old patient. Which downstream finding should be expected?

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

2. During a teaching-round evaluation, a 33-year-old adult has hyperglycemia after immune-mediated loss of pancreatic beta cells. Which cellular or physiologic mechanism accounts for these findings?

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

3. During a teaching-round evaluation, during discussion of pulmonary embolic VQ mismatch in a 40-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. increased airway resistance that is greatest during expiration
B. increased alveolar dead space with redistribution of perfusion
C. premature small-airway collapse with reduced gas-exchange surface area
D. restrictive mechanics with impaired oxygen transfer across the alveolar membrane

4. During a teaching-round evaluation, a clinical scenario involving a 47-year-old patient demonstrates pulmonary fibrosis. Which additional finding fits the same causal process?

A. premature small-airway collapse with reduced gas-exchange surface area
B. increased airway resistance that is greatest during expiration
C. increased alveolar dead space with redistribution of perfusion
D. restrictive mechanics with impaired oxygen transfer across the alveolar membrane

5. During a teaching-round evaluation, the care team reviews primary hyperthyroidism in a 54-year-old adult. Which consequence best reflects its established pathophysiology?

A. reduced TSH through negative feedback despite increased tissue stimulation
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

6. During a teaching-round evaluation, the clinician evaluates a 61-year-old patient who develops urticaria and wheezing within minutes of allergen exposure. Which mechanism should be identified as primary?

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

7. During a teaching-round evaluation, in a teaching case, a 68-year-old patient develops oliguria after substantial volume depletion while tubular function remains initially intact. Which process provides the best explanation?

A. phosphate retention and reduced renal activation of vitamin D
B. tubular epithelial damage causing obstruction and filtrate backleak
C. increased glomerular permeability to plasma proteins
D. reduced renal perfusion without primary parenchymal damage

8. During a teaching-round evaluation, a 75-year-old adult has findings compatible with nephrotic syndrome. Which associated change best follows from the underlying mechanism?

A. erythrocyte leakage into tubules with sodium and water retention
B. impaired concentrating ability despite reduced glomerular filtration
C. reduced oncotic pressure with compensatory hepatic lipoprotein synthesis
D. avid renal sodium and water conservation with concentrated urine

9. During a teaching-round evaluation, during discussion of atherosclerotic plaque formation in a 82-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

10. During a teaching-round evaluation, during clinical review, a 21-year-old patient develops fever with minimal pus formation during a bacterial infection. What is the principal underlying mechanism?

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

11. During a teaching-round evaluation, a case involves a 28-year-old adult who has burning retrosternal discomfort after meals and when lying flat. Which pathophysiologic process is most directly responsible?

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

12. During a teaching-round evaluation, a 35-year-old patient is being evaluated for right-sided heart failure. Which additional consequence is most consistent with this disorder?

A. foam-cell formation followed by a fibrous cap over a lipid core
B. compensatory vasoconstriction that raises afterload and worsens congestion
C. reduced systemic vascular resistance despite normal or increased cardiac output
D. peripheral fluid filtration with congestion of abdominal organs

13. During a teaching-round evaluation, in a teaching case, a 42-year-old patient has antibodies directed against antigens fixed on red blood cell membranes. Which process provides the best explanation?

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

14. During a teaching-round evaluation, a 49-year-old adult has progressive arterial narrowing after endothelial injury and lipid accumulation. Which cellular or physiologic mechanism accounts for these findings?

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

15. During a teaching-round evaluation, during discussion of Parkinson disease in a 56-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

16. During a teaching-round evaluation, a clinical scenario involving a 63-year-old patient demonstrates disseminated intravascular coagulation. Which additional finding fits the same causal process?

A. blunted local inflammation despite risk of rapidly progressive infection
B. simultaneous microvascular thrombosis and consumption-related hemorrhage
C. free hemoglobin binding haptoglobin until circulating stores are depleted
D. megaloblastic erythropoiesis with neurologic dysfunction

17. During a teaching-round evaluation, a case involves a 70-year-old adult who has fluctuating skeletal muscle weakness that worsens with repeated activity. Which pathophysiologic process is most directly responsible?

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

18. During a teaching-round evaluation, a 77-year-old patient is being evaluated for acute asthma. Which additional consequence is most consistent with this disorder?

A. restrictive mechanics with impaired oxygen transfer across the alveolar membrane
B. increased alveolar dead space with redistribution of perfusion
C. increased airway resistance that is greatest during expiration
D. premature small-airway collapse with reduced gas-exchange surface area

19. During a teaching-round evaluation, the pathophysiology of acute pancreatitis is reviewed in a 84-year-old patient. Which downstream finding should be expected?

A. portosystemic collateral formation and elevated splanchnic hydrostatic pressure
B. fibrosis and penetrating tracts between bowel and adjacent structures
C. repeated acid exposure causing distal esophageal mucosal injury
D. fat necrosis, edema, and systemic inflammatory mediator release

20. During a teaching-round evaluation, a 23-year-old adult has findings compatible with acute tubular injury. Which associated change best follows from the underlying mechanism?

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

21. During a teaching-round evaluation, during discussion of extrahepatic biliary obstruction in a 30-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

22. During a teaching-round evaluation, during clinical review, a 37-year-old patient has concentrated urine despite low plasma osmolality and dilutional hyponatremia. What is the principal underlying mechanism?

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

23. During a teaching-round evaluation, a case involves a 44-year-old adult who develops sudden focal neurologic deficits after arterial cerebral blood flow is interrupted. Which pathophysiologic process is most directly responsible?

A. loss of dopaminergic input to basal ganglia motor circuits
B. immune-mediated destruction of central nervous system myelin
C. fixed cranial volume forcing compensatory displacement before pressure rises steeply
D. energy failure causing loss of ion gradients and excitotoxic injury

24. During a teaching-round evaluation, a 51-year-old patient is being evaluated for cortisol excess. Which additional consequence is most consistent with this disorder?

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

25. During a teaching-round evaluation, the pathophysiology of apoptosis is reviewed in a 58-year-old patient. Which downstream finding should be expected?

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

26. During a teaching-round evaluation, a 65-year-old adult has isolated damaged cells that shrink without provoking surrounding inflammation. Which cellular or physiologic mechanism accounts for these findings?

A. reactive oxygen species attacking lipids, proteins, and DNA
B. caspase-mediated programmed cellular self-destruction
C. ATP depletion causing failure of membrane ion pumps
D. reprogramming of local stem cells toward a stress-resistant phenotype

27. During a teaching-round evaluation, a 72-year-old patient has jugular venous distention, dependent edema, and hepatic congestion. Which mechanism best explains this pattern?

A. elevated systemic venous pressure from impaired right ventricular output
B. severe pump failure reducing stroke volume and tissue perfusion
C. elevated left atrial and pulmonary capillary hydrostatic pressure
D. intimal inflammation with oxidized lipoprotein uptake by macrophages

28. During a teaching-round evaluation, during clinical review, a 79-year-old patient has fatigue with microcytic, hypochromic erythrocytes after chronic blood loss. What is the principal underlying mechanism?

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

29. During a teaching-round evaluation, a case involves a 18-year-old adult who has jaundice, dark urine, pale stools, and pruritus from blocked bile flow. Which pathophysiologic process is most directly responsible?

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

30. During a teaching-round evaluation, the clinician evaluates a 25-year-old patient who has hypotension, cool clammy skin, pulmonary congestion, and low cardiac output after myocardial injury. Which mechanism should be identified as primary?

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

31. During a teaching-round evaluation, in a teaching case, a 32-year-old patient has hypotension with warm extremities early in a severe systemic inflammatory state. Which process provides the best explanation?

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

32. During a teaching-round evaluation, a 39-year-old adult has findings compatible with distributive shock. Which associated change best follows from the underlying mechanism?

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

33. During a teaching-round evaluation, a 46-year-old patient develops declining consciousness as an expanding intracranial lesion reduces cerebral perfusion. Which mechanism best explains this pattern?

A. loss of dopaminergic input to basal ganglia motor circuits
B. energy failure causing loss of ion gradients and excitotoxic injury
C. fixed cranial volume forcing compensatory displacement before pressure rises steeply
D. immune-mediated destruction of central nervous system myelin

34. During a teaching-round evaluation, during clinical review, a 53-year-old patient has a pale wedge-shaped renal infarct after arterial occlusion. What is the principal underlying mechanism?

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

35. During a teaching-round evaluation, the care team reviews secondary hyperparathyroidism in chronic kidney disease in a 60-year-old adult. Which consequence best reflects its established pathophysiology?

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. reduced oncotic pressure with compensatory hepatic lipoprotein synthesis

36. During a teaching-round evaluation, the clinician evaluates a 67-year-old patient who has proximal muscle weakness, hyperglycemia, thin skin, and central fat accumulation. Which mechanism should be identified as primary?

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

37. During a teaching-round evaluation, in a teaching case, a 74-year-old patient has chronic airway irritation and replacement of ciliated columnar cells by squamous cells. Which process provides the best explanation?

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

38. During a teaching-round evaluation, a 81-year-old adult develops an indurated skin reaction two days after antigen exposure. Which cellular or physiologic mechanism accounts for these findings?

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

39. During a teaching-round evaluation, a 20-year-old patient has macrocytic anemia accompanied by paresthesias and impaired vibration sensation. Which mechanism best explains this pattern?

A. premature rupture of erythrocytes within the circulation
B. widespread pathologic thrombin generation throughout the microcirculation
C. impaired DNA synthesis plus defective myelin maintenance
D. insufficient neutrophil availability for an effective acute response

40. During a teaching-round evaluation, a clinical scenario involving a 27-year-old patient demonstrates iron-deficiency anemia. Which additional finding fits the same causal process?

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

41. During a teaching-round evaluation, a case involves a 34-year-old adult who develops vasculitic injury where circulating antigen-antibody complexes lodge in small vessels. Which pathophysiologic process is most directly responsible?

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

42. During a teaching-round evaluation, the clinician evaluates a 41-year-old patient who has episodic wheezing with reversible expiratory airflow limitation. Which mechanism should be identified as primary?

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

43. During a teaching-round evaluation, in a teaching case, a 48-year-old patient has ascites, splenomegaly, and esophageal varices with advanced hepatic fibrosis. Which process provides the best explanation?

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

44. During a teaching-round evaluation, a 55-year-old adult has findings compatible with oxidative cellular injury. Which associated change best follows from the underlying mechanism?

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

45. During a teaching-round evaluation, a 62-year-old patient has heavy protein loss in urine, hypoalbuminemia, edema, and hyperlipidemia. Which mechanism best explains this pattern?

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

46. During a teaching-round evaluation, during clinical review, a 69-year-old patient has patchy transmural intestinal inflammation with strictures and fistulas. What is the principal underlying mechanism?

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

47. During a teaching-round evaluation, a case involves a 76-year-old adult who has diffuse bleeding while laboratory studies show consumption of platelets and clotting factors. Which pathophysiologic process is most directly responsible?

A. impaired DNA synthesis plus defective myelin maintenance
B. insufficient neutrophil availability for an effective acute response
C. widespread pathologic thrombin generation throughout the microcirculation
D. premature rupture of erythrocytes within the circulation

48. During a teaching-round evaluation, the clinician evaluates a 83-year-old patient who has persistent tissue injury with macrophages, lymphocytes, fibrosis, and attempted repair. Which mechanism should be identified as primary?

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

49. During a teaching-round evaluation, the pathophysiology of primary hyperaldosteronism is reviewed in a 22-year-old patient. Which downstream finding should be expected?

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

50. During a teaching-round evaluation, a 29-year-old adult has chronic kidney dysfunction with disturbed calcium and phosphate balance. Which cellular or physiologic mechanism accounts for these findings?

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

51. During a teaching-round evaluation, during discussion of portal hypertension in cirrhosis in a 36-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. repeated acid exposure causing distal esophageal mucosal injury
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

52. During a teaching-round evaluation, a clinical scenario involving a 43-year-old patient demonstrates emphysema. Which additional finding fits the same causal process?

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

53. During a teaching-round evaluation, a case involves a 50-year-old adult who develops anemia, elevated indirect bilirubin, reduced haptoglobin, and hemoglobinuria. Which pathophysiologic process is most directly responsible?

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

54. During a teaching-round evaluation, a 57-year-old patient is being evaluated for diabetic ketoacidosis. Which additional consequence is most consistent with this disorder?

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

55. During a teaching-round evaluation, the pathophysiology of cardiogenic shock is reviewed in a 64-year-old patient. Which downstream finding should be expected?

A. foam-cell formation followed by a fibrous cap over a lipid core
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

56. During a teaching-round evaluation, a 71-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. premature small-airway collapse with reduced gas-exchange surface area
C. protein-rich edema with surfactant dysfunction and intrapulmonary shunting
D. restrictive mechanics with impaired oxygen transfer across the alveolar membrane

57. During a teaching-round evaluation, during discussion of nephritic syndrome in a 78-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. avid renal sodium and water conservation with concentrated urine
B. reduced oncotic pressure with compensatory hepatic lipoprotein synthesis
C. impaired concentrating ability despite reduced glomerular filtration
D. erythrocyte leakage into tubules with sodium and water retention

58. During a teaching-round evaluation, a clinical scenario involving a 85-year-old patient demonstrates intravascular hemolysis. Which additional finding fits the same causal process?

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

59. During a teaching-round evaluation, a case involves a 24-year-old adult who has persistent kidney dysfunction after prolonged ischemia with granular casts in urine. Which pathophysiologic process is most directly responsible?

A. reduced renal perfusion without primary parenchymal damage
B. tubular epithelial damage causing obstruction and filtrate backleak
C. phosphate retention and reduced renal activation of vitamin D
D. increased glomerular permeability to plasma proteins

60. During a teaching-round evaluation, a 31-year-old patient is being evaluated for left-sided heart failure. Which additional consequence is most consistent with this disorder?

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

61. During a teaching-round evaluation, in a teaching case, a 38-year-old patient has hyperinflation and reduced elastic recoil after destruction of alveolar walls. Which process provides the best explanation?

A. diffuse alveolar-capillary barrier injury increasing permeability
B. bronchial smooth-muscle constriction plus mucosal inflammation
C. protease-mediated loss of alveolar septa and supporting elastic tissue
D. excess collagen deposition thickening the pulmonary interstitium

62. During a teaching-round evaluation, a 45-year-old adult has findings compatible with increased intracranial pressure. Which associated change best follows from the underlying mechanism?

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

63. During a teaching-round evaluation, during discussion of type IV hypersensitivity in a 52-year-old patient, which physiologic consequence is most appropriate to anticipate?

A. rapid histamine release causing vasodilation and bronchoconstriction
B. delayed mononuclear inflammation without an antibody requirement
C. complement activation and antibody-mediated cellular destruction
D. simultaneous parenchymal destruction and collagen deposition

64. During a teaching-round evaluation, during clinical review, a 59-year-old patient has neurologic deficits separated in time and location within the central nervous system. What is the principal underlying mechanism?

A. immune-mediated destruction of central nervous system myelin
B. fixed cranial volume forcing compensatory displacement before pressure rises steeply
C. loss of dopaminergic input to basal ganglia motor circuits
D. energy failure causing loss of ion gradients and excitotoxic injury

65. During a teaching-round evaluation, a case involves a 66-year-old adult who experiences reperfusion of previously ischemic tissue followed by additional membrane damage. Which pathophysiologic process is most directly responsible?

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

66. During a teaching-round evaluation, the clinician evaluates a 73-year-old patient who has hypertension, potassium depletion, and increased renal hydrogen ion secretion. Which mechanism should be identified as primary?

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

67. During a teaching-round evaluation, the pathophysiology of prerenal acute kidney injury is reviewed in a 80-year-old patient. Which downstream finding should be expected?

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

68. During a teaching-round evaluation, a 19-year-old adult has findings compatible with bicarbonate-loss metabolic acidosis. Which associated change best follows from the underlying mechanism?

A. increased carbonic acid with renal bicarbonate retention if persistent
B. water retention that lowers serum sodium without proportional sodium loss
C. compensatory hyperventilation that lowers arterial carbon dioxide
D. volume expansion with hypokalemia and metabolic alkalosis

69. During a teaching-round evaluation, a 26-year-old patient develops bilateral pulmonary opacities and severe hypoxemia after a major inflammatory insult. Which mechanism best explains this pattern?

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

70. During a teaching-round evaluation, during clinical review, a 33-year-old patient has hematuria, red-cell casts, hypertension, and reduced filtration. What is the principal underlying mechanism?

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

71. During a teaching-round evaluation, the care team reviews type III hypersensitivity in a 40-year-old adult. Which consequence best reflects its established pathophysiology?

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

72. During a teaching-round evaluation, the clinician evaluates a 47-year-old patient who develops generalized edema after a marked fall in plasma albumin. Which mechanism should be identified as primary?

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

73. During a teaching-round evaluation, in a teaching case, a 54-year-old patient has hypoventilation with carbon dioxide retention during a severe obstructive exacerbation. Which process provides the best explanation?

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

74. During a teaching-round evaluation, a 61-year-old adult develops cellular swelling shortly after a brief episode of tissue hypoxia. Which cellular or physiologic mechanism accounts for these findings?

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

75. During a teaching-round evaluation, during discussion of Crohn disease in a 68-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

76. During a teaching-round evaluation, during clinical review, a 75-year-old patient develops acidemia after prolonged high-volume diarrhea. What is the principal underlying mechanism?

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

77. During a teaching-round evaluation, the care team reviews myasthenia gravis in a 82-year-old adult. Which consequence best reflects its established pathophysiology?

A. falling cerebral perfusion pressure as intracranial pressure approaches arterial pressure
B. slowed or blocked saltatory conduction along affected axons
C. reduced facilitation of intended movement with excessive inhibitory output
D. declining neuromuscular transmission safety during repetitive stimulation

78. During a teaching-round evaluation, the clinician evaluates a 21-year-old patient who has severe epigastric pain after premature activation of pancreatic digestive enzymes. Which mechanism should be identified as primary?

A. failure of conjugated bilirubin and bile salts to reach the intestine
B. discontinuous immune-mediated inflammation extending through the bowel wall
C. intrapancreatic enzyme activation causing autodigestion
D. increased resistance to portal flow through a scarred nodular liver

79. During a teaching-round evaluation, the pathophysiology of respiratory acidosis is reviewed in a 28-year-old patient. Which downstream finding should be expected?

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

80. During a teaching-round evaluation, a 35-year-old adult has findings compatible with SIADH. Which associated change best follows from the underlying mechanism?

A. water retention that lowers serum sodium without proportional sodium loss
B. movement of fluid from capillaries into the interstitial compartment
C. volume expansion with hypokalemia and metabolic alkalosis
D. compensatory hyperventilation that lowers arterial carbon dioxide

81. During a teaching-round evaluation, during discussion of chronic inflammation in a 42-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

82. During a teaching-round evaluation, a clinical scenario involving a 49-year-old patient demonstrates type 1 diabetes mellitus. Which additional finding fits the same causal process?

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. reduced cellular glucose uptake with increased lipolysis and ketogenesis

83. During a teaching-round evaluation, the care team reviews epithelial metaplasia in a 56-year-old adult. Which consequence best reflects its established pathophysiology?

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

84. During a teaching-round evaluation, a 63-year-old patient is being evaluated for hypoalbuminemic edema. Which additional consequence is most consistent with this disorder?

A. movement of fluid from capillaries into the interstitial compartment
B. compensatory hyperventilation that lowers arterial carbon dioxide
C. water retention that lowers serum sodium without proportional sodium loss
D. volume expansion with hypokalemia and metabolic alkalosis

85. During a teaching-round evaluation, the pathophysiology of severe neutropenia is reviewed in a 70-year-old patient. Which downstream finding should be expected?

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

86. During a teaching-round evaluation, a 77-year-old adult has findings compatible with ischemic stroke. Which associated change best follows from the underlying mechanism?

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

87. During a teaching-round evaluation, a 84-year-old patient develops sudden hypoxemia when a clot blocks perfusion to ventilated alveoli. Which mechanism best explains this pattern?

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

88. During a teaching-round evaluation, a clinical scenario involving a 23-year-old patient demonstrates primary hypothyroidism. Which additional finding fits the same causal process?

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

89. During a teaching-round evaluation, the care team reviews multiple sclerosis in a 30-year-old adult. Which consequence best reflects its established pathophysiology?

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

90. During a teaching-round evaluation, the clinician evaluates a 37-year-old patient who has progressive exertional dyspnea with stiff lungs and reduced diffusion capacity. Which mechanism should be identified as primary?

A. protease-mediated loss of alveolar septa and supporting elastic tissue
B. diffuse alveolar-capillary barrier injury increasing permeability
C. excess collagen deposition thickening the pulmonary interstitium
D. bronchial smooth-muscle constriction plus mucosal inflammation

91. During a teaching-round evaluation, the pathophysiology of type II hypersensitivity is reviewed in a 44-year-old patient. Which downstream finding should be expected?

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

92. During a teaching-round evaluation, a 51-year-old adult has fatigue, cold intolerance, slowed reflexes, and elevated pituitary stimulation. Which cellular or physiologic mechanism accounts for these findings?

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

93. During a teaching-round evaluation, a 58-year-old patient has heat intolerance, tremor, weight loss, and suppressed pituitary stimulation. Which mechanism best explains this pattern?

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

94. During a teaching-round evaluation, a clinical scenario involving a 65-year-old patient demonstrates reversible hypoxic cell injury. Which additional finding fits the same causal process?

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

95. During a teaching-round evaluation, a case involves a 72-year-old adult who has hyperglycemia, dehydration, ketones, and deep rapid respirations. Which pathophysiologic process is most directly responsible?

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

96. During a teaching-round evaluation, the clinician evaluates a 79-year-old patient who has exertional dyspnea, orthopnea, and inspiratory crackles. Which mechanism should be identified as primary?

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

97. During a teaching-round evaluation, the pathophysiology of gastroesophageal reflux is reviewed in a 18-year-old patient. Which downstream finding should be expected?

A. fat necrosis, edema, and systemic inflammatory mediator release
B. portosystemic collateral formation and elevated splanchnic hydrostatic pressure
C. repeated acid exposure causing distal esophageal mucosal injury
D. fibrosis and penetrating tracts between bowel and adjacent structures

98. During a teaching-round evaluation, a 25-year-old adult has findings compatible with coagulative necrosis. Which associated change best follows from the underlying mechanism?

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

99. During a teaching-round evaluation, a 32-year-old patient has bradykinesia, rigidity, and resting tremor from degeneration in the substantia nigra. Which mechanism best explains this pattern?

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

100. During a teaching-round evaluation, a clinical scenario involving a 39-year-old patient demonstrates vitamin B12 deficiency. Which additional finding fits the same causal process?

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

Practice Exam 21: Answer Key And Explanations

1. 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.

2. A — 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.

3. 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, 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.

4. D — 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.

5. 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, 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.

6. B — 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.

7. 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, 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.

8. 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..

9. A — 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.

10. B — 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.

11. C — 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.

12. D — 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.

13. D — 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.

14. A — 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.

15. C — 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.

16. 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.

17. 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, 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.

18. C — 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..

19. D — 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..

20. A — 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.

21. 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..

22. C — 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.

23. D — 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.

24. 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, 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..

25. A — 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.

26. B — 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.

27. 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.

28. C — 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..

29. A — 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.

30. B — 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.

31. 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, 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.

32. D — 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.

33. 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, fixed cranial volume forcing compensatory displacement before pressure rises steeply is the best answer. The competing choices represent mechanisms from related disorders, but.

34. 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.

35. B — 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.

36. 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.

37. 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.

38. 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.

39. 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, 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.

40. A — 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.

41. D — 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..

42. 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, 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.

43. 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, 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.

44. A — 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..

45. A — 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.

46. B — 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.

47. C — 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.

48. 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.

49. 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.

50. B — 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.

51. 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.

52. A — 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.

53. 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.

54. 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.

55. D — 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.

56. 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, 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.

57. 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, 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.

58. 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.

59. B — 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.

60. C — 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..

61. C — 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.

62. D — 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.

63. B — 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. A — 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.

65. D — 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.

66. A — 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.

67. 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, 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..

68. C — 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.

69. D — 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.

70. 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, 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..

71. B — 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.

72. 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.

73. 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.

74. 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.

75. D — 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.

76. C — 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.

77. D — 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.

78. 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.

79. B — 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..

80. A — 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.

81. C — 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.

82. D — 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.

83. 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, greater injury resistance accompanied by reduced mucociliary clearance is the expected consequence. The other choices follow different disease processes and do not arise from.

84. A — 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.

85. 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, 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.

86. 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, 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.

87. 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, 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.

88. B — 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.

89. 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.

90. C — 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.

91. A — 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.

92. B — 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.

93. C — 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.

94. 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.

95. A — 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.

96. 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, 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.

97. C — 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.

98. B — 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.

99. 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.

100. 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, 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.