

PRACTICE EXAM 4: 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 an acute-care case conference, a 77-year-old adult has macrocytic anemia accompanied by paresthesias and impaired vibration sensation. Which cellular or physiologic mechanism accounts for these findings?
 - A. widespread pathologic thrombin generation throughout the microcirculation
 - B. insufficient iron limiting hemoglobin synthesis
 - C. impaired DNA synthesis plus defective myelin maintenance
 - D. premature rupture of erythrocytes within the circulation
2. During an acute-care case conference, a 84-year-old patient has persistent tissue injury with macrophages, lymphocytes, fibrosis, and attempted repair. Which mechanism best explains this pattern?
 - A. sensitized T lymphocytes releasing cytokines and activating macrophages
 - B. ongoing immune activation occurring alongside tissue repair
 - C. immune-complex deposition with complement recruitment
 - D. IgG or IgM binding to cell-surface targets
3. During an acute-care case conference, the care team reviews bicarbonate-loss metabolic acidosis in a 23-year-old adult. Which consequence best reflects its established pathophysiology?
 - A. compensatory hyperventilation that lowers arterial carbon dioxide
 - B. volume expansion with hypokalemia and metabolic alkalosis
 - C. movement of fluid from capillaries into the interstitial compartment
 - D. increased carbonic acid with renal bicarbonate retention if persistent
4. During an acute-care case conference, a 30-year-old patient is being evaluated for multiple sclerosis. Which additional consequence is most consistent with this disorder?
 - 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

5. During an acute-care case conference, the pathophysiology of acute asthma is reviewed in a 37-year-old patient. Which downstream finding should be expected?
- A. protein-rich edema with surfactant dysfunction and intrapulmonary shunting
 - B. increased airway resistance that is greatest during expiration
 - C. premature small-airway collapse with reduced gas-exchange surface area
 - D. increased alveolar dead space with redistribution of perfusion
6. During an acute-care case conference, in a teaching case, a 44-year-old patient experiences reperfusion of previously ischemic tissue followed by additional membrane damage. Which process provides the best explanation?
- A. ischemic protein denaturation preserving tissue outlines temporarily
 - B. caspase-mediated programmed cellular self-destruction
 - C. reactive oxygen species attacking lipids, proteins, and DNA
 - D. reprogramming of local stem cells toward a stress-resistant phenotype
7. During an acute-care case conference, during discussion of myasthenia gravis in a 51-year-old patient, which physiologic consequence is most appropriate to anticipate?
- 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
8. During an acute-care case conference, a clinical scenario involving a 58-year-old patient demonstrates portal hypertension in cirrhosis. Which additional finding fits the same causal process?
- A. fibrosis and penetrating tracts between bowel and adjacent structures
 - B. reflux of water-soluble conjugated bilirubin into blood and urine
 - C. fat necrosis, edema, and systemic inflammatory mediator release
 - D. portosystemic collateral formation and elevated splanchnic hydrostatic pressure
9. During an acute-care case conference, during clinical review, a 65-year-old patient has fatigue, cold intolerance, slowed reflexes, and elevated pituitary stimulation. What is the principal underlying mechanism?
- A. absolute insulin deficiency from autoimmune beta-cell destruction
 - B. insufficient thyroid hormone reducing basal metabolic activity
 - C. excess thyroid hormone increasing metabolic and adrenergic activity
 - D. unrestrained lipolysis generating acidic ketone bodies

10. During an acute-care case conference, a 72-year-old patient is being evaluated for vitamin B12 deficiency. Which additional consequence is most consistent with this disorder?

- A. simultaneous microvascular thrombosis and consumption-related hemorrhage
- B. free hemoglobin binding haptoglobin until circulating stores are depleted
- C. progressively smaller red cells with reduced oxygen-carrying capacity
- D. megaloblastic erythropoiesis with neurologic dysfunction

11. During an acute-care case conference, the clinician evaluates a 79-year-old patient who develops sudden focal neurologic deficits after arterial cerebral blood flow is interrupted. Which mechanism should be identified as primary?

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

12. During an acute-care case conference, in a teaching case, a 18-year-old patient has chronic kidney dysfunction with disturbed calcium and phosphate balance. Which process provides the best explanation?

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

13. During an acute-care case conference, during discussion of primary hyperthyroidism in a 25-year-old patient, which physiologic consequence is most appropriate to anticipate?

- A. high-anion-gap metabolic acidosis with respiratory compensation
- B. increased TSH when the pituitary-thyroid feedback loop is intact
- C. reduced TSH through negative feedback despite increased tissue stimulation
- D. reduced cellular glucose uptake with increased lipolysis and ketogenesis

14. During an acute-care case conference, a 32-year-old patient has hematuria, red-cell casts, hypertension, and reduced filtration. Which mechanism best explains this pattern?

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

15. During an acute-care case conference, the care team reviews prerenal acute kidney injury in a 39-year-old adult. Which consequence best reflects its established pathophysiology?

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

16. During an acute-care case conference, a case involves a 46-year-old adult who has isolated damaged cells that shrink without provoking surrounding inflammation. Which pathophysiologic process is most directly responsible?

- A. ischemic protein denaturation preserving tissue outlines temporarily
- B. reactive oxygen species attacking lipids, proteins, and DNA
- C. ATP depletion causing failure of membrane ion pumps
- D. caspase-mediated programmed cellular self-destruction

17. During an acute-care case conference, the pathophysiology of nephritic syndrome is reviewed in a 53-year-old patient. Which downstream finding should be expected?

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

18. During an acute-care case conference, in a teaching case, a 60-year-old patient has severe epigastric pain after premature activation of pancreatic digestive enzymes. Which process provides the best explanation?

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

19. During an acute-care case conference, during discussion of acute respiratory distress syndrome in a 67-year-old patient, which physiologic consequence is most appropriate to anticipate?

- A. restrictive mechanics with impaired oxygen transfer across the alveolar membrane
- B. protein-rich edema with surfactant dysfunction and intrapulmonary shunting
- C. premature small-airway collapse with reduced gas-exchange surface area
- D. increased airway resistance that is greatest during expiration

20. During an acute-care case conference, a 74-year-old patient develops vasculitic injury where circulating antigen-antibody complexes lodge in small vessels. Which mechanism best explains this pattern?

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

21. During an acute-care case conference, during clinical review, a 81-year-old patient has exertional dyspnea, orthopnea, and inspiratory crackles. What is the principal underlying mechanism?

- 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

22. During an acute-care case conference, a 20-year-old patient is being evaluated for type IV hypersensitivity. Which additional consequence is most consistent with this disorder?

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

23. During an acute-care case conference, the pathophysiology of diabetic ketoacidosis is reviewed in a 27-year-old patient. Which downstream finding should be expected?

- 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. reduced cellular glucose uptake with increased lipolysis and ketogenesis

24. During an acute-care case conference, in a teaching case, a 34-year-old patient develops bilateral pulmonary opacities and severe hypoxemia after a major inflammatory insult. Which process provides the best explanation?

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

25. During an acute-care case conference, during discussion of chronic inflammation in a 41-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

26. During an acute-care case conference, a clinical scenario involving a 48-year-old patient demonstrates emphysema. Which additional finding fits the same causal process?

- A. increased airway resistance that is greatest during expiration
- B. restrictive mechanics with impaired oxygen transfer across the alveolar membrane
- C. premature small-airway collapse with reduced gas-exchange surface area
- D. protein-rich edema with surfactant dysfunction and intrapulmonary shunting

27. During an acute-care case conference, during clinical review, a 55-year-old patient has hypotension with warm extremities early in a severe systemic inflammatory state. What is the principal underlying mechanism?

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

28. During an acute-care case conference, a 62-year-old patient is being evaluated for atherosclerotic plaque formation. Which additional consequence is most consistent with this disorder?

- 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. compensatory vasoconstriction that raises afterload and worsens congestion
- D. transudation of fluid into pulmonary interstitium and alveoli

29. During an acute-care case conference, the clinician evaluates a 69-year-old patient who has progressive arterial narrowing after endothelial injury and lipid accumulation. Which mechanism should be identified as primary?

- A. elevated systemic venous pressure from impaired right ventricular output
- 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

30. During an acute-care case conference, in a teaching case, a 76-year-old patient develops cellular swelling shortly after a brief episode of tissue hypoxia. Which process provides the best explanation?

- A. ATP depletion causing failure of membrane ion pumps
- B. ischemic protein denaturation preserving tissue outlines temporarily
- C. reactive oxygen species attacking lipids, proteins, and DNA
- D. caspase-mediated programmed cellular self-destruction

31. During an acute-care case conference, during discussion of primary hypothyroidism in a 83-year-old patient, which physiologic consequence is most appropriate to anticipate?

- A. reduced cellular glucose uptake with increased lipolysis and ketogenesis
- B. increased TSH when the pituitary-thyroid feedback loop is intact
- C. reduced TSH through negative feedback despite increased tissue stimulation
- D. high-anion-gap metabolic acidosis with respiratory compensation

32. During an acute-care case conference, a clinical scenario involving a 22-year-old patient demonstrates nephrotic syndrome. Which additional finding fits the same causal process?

- A. avid renal sodium and water conservation with concentrated urine
- B. persistent parathyroid stimulation with increased bone resorption
- C. reduced oncotic pressure with compensatory hepatic lipoprotein synthesis
- D. impaired concentrating ability despite reduced glomerular filtration

33. During an acute-care case conference, during clinical review, a 29-year-old patient develops sudden hypoxemia when a clot blocks perfusion to ventilated alveoli. What is the principal underlying mechanism?

- A. bronchial smooth-muscle constriction plus mucosal inflammation
- B. diffuse alveolar-capillary barrier injury increasing permeability
- C. excess collagen deposition thickening the pulmonary interstitium
- D. abrupt loss of regional pulmonary blood flow

34. During an acute-care case conference, a 36-year-old patient is being evaluated for apoptosis. Which additional consequence is most consistent with this disorder?

- 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. lipid peroxidation with increased membrane permeability

35. During an acute-care case conference, the pathophysiology of reversible hypoxic cell injury is reviewed in a 43-year-old patient. Which downstream finding should be expected?
- A. loss of nuclei while the basic tissue architecture remains visible
 - B. intracellular sodium and water accumulation with cell swelling
 - C. formation of membrane-bound fragments cleared by phagocytes
 - D. lipid peroxidation with increased membrane permeability
36. During an acute-care case conference, a 50-year-old adult has findings compatible with cortisol excess. Which associated change best follows from the underlying mechanism?
- A. reduced cellular glucose uptake with increased lipolysis and ketogenesis
 - B. muscle wasting with impaired connective tissue maintenance
 - C. increased TSH when the pituitary-thyroid feedback loop is intact
 - D. reduced TSH through negative feedback despite increased tissue stimulation
37. During an acute-care case conference, during discussion of gastroesophageal reflux in a 57-year-old patient, which physiologic consequence is most appropriate to anticipate?
- A. fat necrosis, edema, and systemic inflammatory mediator release
 - B. fibrosis and penetrating tracts between bowel and adjacent structures
 - C. repeated acid exposure causing distal esophageal mucosal injury
 - D. reflux of water-soluble conjugated bilirubin into blood and urine
38. During an acute-care case conference, a clinical scenario involving a 64-year-old patient demonstrates left-sided heart failure. Which additional finding fits the same causal process?
- A. peripheral fluid filtration with congestion of abdominal organs
 - B. compensatory vasoconstriction that raises afterload and worsens congestion
 - C. transudation of fluid into pulmonary interstitium and alveoli
 - D. foam-cell formation followed by a fibrous cap over a lipid core
39. During an acute-care case conference, during clinical review, a 71-year-old patient has antibodies directed against antigens fixed on red blood cell membranes. What is the principal underlying mechanism?
- A. IgG or IgM binding to cell-surface targets
 - B. allergen cross-linking of IgE on sensitized mast cells
 - C. sensitized T lymphocytes releasing cytokines and activating macrophages
 - D. ongoing immune activation occurring alongside tissue repair
40. During an acute-care case conference, a 78-year-old patient is being evaluated for type II hypersensitivity. Which additional consequence is most consistent with this disorder?
- A. delayed mononuclear inflammation without an antibody requirement
 - 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

41. During an acute-care case conference, the clinician evaluates a 85-year-old patient who has hyperglycemia, dehydration, ketones, and deep rapid respirations. Which mechanism should be identified as primary?

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

42. During an acute-care case conference, in a teaching case, a 24-year-old patient has heavy protein loss in urine, hypoalbuminemia, edema, and hyperlipidemia. Which process provides the best explanation?

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

43. During an acute-care case conference, a 31-year-old adult has hypotension, cool clammy skin, pulmonary congestion, and low cardiac output after myocardial injury. Which cellular or physiologic mechanism accounts for these findings?

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

44. During an acute-care case conference, a 38-year-old patient has a pale wedge-shaped renal infarct after arterial occlusion. Which mechanism best explains this pattern?

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

45. During an acute-care case conference, during clinical review, a 45-year-old patient develops urticaria and wheezing within minutes of allergen exposure. What is the principal underlying mechanism?

- A. allergen cross-linking of IgE on sensitized mast cells
- B. IgG or IgM binding to cell-surface targets
- C. ongoing immune activation occurring alongside tissue repair
- D. sensitized T lymphocytes releasing cytokines and activating macrophages

46. During an acute-care case conference, a case involves a 52-year-old adult who develops acidemia after prolonged high-volume diarrhea. Which pathophysiologic process is most directly responsible?

- A. continued antidiuretic hormone effect despite hypo-osmolality
- B. gastrointestinal loss of bicarbonate-rich fluid
- C. alveolar ventilation inadequate for carbon dioxide production
- D. reduced plasma colloid oncotic pressure favoring filtration

47. During an acute-care case conference, the clinician evaluates a 59-year-old patient who has burning retrosternal discomfort after meals and when lying flat. Which mechanism should be identified as primary?

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

48. During an acute-care case conference, in a teaching case, a 66-year-old patient has bradykinesia, rigidity, and resting tremor from degeneration in the substantia nigra. Which process provides the best explanation?

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

49. During an acute-care case conference, during discussion of increased intracranial pressure in a 73-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

50. During an acute-care case conference, a clinical scenario involving a 80-year-old patient demonstrates acute tubular injury. Which additional finding fits the same causal process?

- 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. reduced oncotic pressure with compensatory hepatic lipoprotein synthesis

51. During an acute-care case conference, during clinical review, a 19-year-old patient has proximal muscle weakness, hyperglycemia, thin skin, and central fat accumulation. What is the principal underlying mechanism?

- A. excess thyroid hormone increasing metabolic and adrenergic activity
- B. absolute insulin deficiency from autoimmune beta-cell destruction
- C. glucocorticoid excess promoting protein catabolism and gluconeogenesis
- D. insufficient thyroid hormone reducing basal metabolic activity

52. During an acute-care case conference, a 26-year-old patient is being evaluated for Crohn disease. Which additional consequence is most consistent with this disorder?

- A. fat necrosis, edema, and systemic inflammatory mediator release
- 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

53. During an acute-care case conference, the pathophysiology of acute pancreatitis is reviewed in a 33-year-old patient. Which downstream finding should be expected?

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

54. During an acute-care case conference, a 40-year-old adult has findings compatible with secondary hyperparathyroidism in chronic kidney disease. Which associated change best follows from the underlying mechanism?

- A. avid renal sodium and water conservation with concentrated urine
- B. persistent parathyroid stimulation with increased bone resorption
- C. reduced oncotic pressure with compensatory hepatic lipoprotein synthesis
- D. impaired concentrating ability despite reduced glomerular filtration

55. During an acute-care case conference, during discussion of distributive shock in a 47-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. compensatory vasoconstriction that raises afterload and worsens congestion
- C. peripheral fluid filtration with congestion of abdominal organs
- D. reduced systemic vascular resistance despite normal or increased cardiac output

56. During an acute-care case conference, a 54-year-old patient develops fever with minimal pus formation during a bacterial infection. Which mechanism best explains this pattern?
- A. insufficient iron limiting hemoglobin synthesis
 - B. premature rupture of erythrocytes within the circulation
 - C. insufficient neutrophil availability for an effective acute response
 - D. widespread pathologic thrombin generation throughout the microcirculation
57. During an acute-care case conference, during clinical review, a 61-year-old patient has jugular venous distention, dependent edema, and hepatic congestion. What is the principal underlying mechanism?
- A. severe pump failure reducing stroke volume and tissue perfusion
 - B. widespread vasodilation and maldistribution of circulating blood flow
 - C. elevated systemic venous pressure from impaired right ventricular output
 - D. intimal inflammation with oxidized lipoprotein uptake by macrophages
58. During an acute-care case conference, a 68-year-old patient is being evaluated for right-sided heart failure. Which additional consequence is most consistent with this disorder?
- A. peripheral fluid filtration with congestion of abdominal organs
 - B. transudation of fluid into pulmonary interstitium and alveoli
 - C. compensatory vasoconstriction that raises afterload and worsens congestion
 - D. foam-cell formation followed by a fibrous cap over a lipid core
59. During an acute-care case conference, the pathophysiology of pulmonary fibrosis is reviewed in a 75-year-old patient. Which downstream finding should be expected?
- A. increased airway resistance that is greatest during expiration
 - B. restrictive mechanics with impaired oxygen transfer across the alveolar membrane
 - C. protein-rich edema with surfactant dysfunction and intrapulmonary shunting
 - D. increased alveolar dead space with redistribution of perfusion
60. During an acute-care case conference, in a teaching case, a 82-year-old patient has neurologic deficits separated in time and location within the central nervous system. Which process provides the best explanation?
- A. antibody-mediated loss or dysfunction of postsynaptic acetylcholine receptors
 - B. loss of dopaminergic input to basal ganglia motor circuits
 - C. fixed cranial volume forcing compensatory displacement before pressure rises steeply
 - D. immune-mediated destruction of central nervous system myelin

61. During an acute-care case conference, a 21-year-old adult has chronic airway irritation and replacement of ciliated columnar cells by squamous cells. Which cellular or physiologic mechanism accounts for these findings?
- A. reactive oxygen species attacking lipids, proteins, and DNA
 - B. reprogramming of local stem cells toward a stress-resistant phenotype
 - C. ATP depletion causing failure of membrane ion pumps
 - D. ischemic protein denaturation preserving tissue outlines temporarily
62. During an acute-care case conference, a clinical scenario involving a 28-year-old patient demonstrates epithelial metaplasia. Which additional finding fits the same causal process?
- A. loss of nuclei while the basic tissue architecture remains visible
 - B. intracellular sodium and water accumulation with cell swelling
 - C. greater injury resistance accompanied by reduced mucociliary clearance
 - D. lipid peroxidation with increased membrane permeability
63. During an acute-care case conference, during clinical review, a 35-year-old patient develops declining consciousness as an expanding intracranial lesion reduces cerebral perfusion. What is the principal underlying mechanism?
- A. loss of dopaminergic input to basal ganglia motor circuits
 - B. immune-mediated destruction of central nervous system myelin
 - C. energy failure causing loss of ion gradients and excitotoxic injury
 - D. fixed cranial volume forcing compensatory displacement before pressure rises steeply
64. During an acute-care case conference, a 42-year-old patient is being evaluated for severe neutropenia. Which additional consequence is most consistent with this disorder?
- A. blunted local inflammation despite risk of rapidly progressive infection
 - B. progressively smaller red cells with reduced oxygen-carrying capacity
 - C. megaloblastic erythropoiesis with neurologic dysfunction
 - D. simultaneous microvascular thrombosis and consumption-related hemorrhage
65. During an acute-care case conference, the clinician evaluates a 49-year-old patient who has fatigue with microcytic, hypochromic erythrocytes after chronic blood loss. Which mechanism should be identified as primary?
- A. insufficient iron limiting hemoglobin synthesis
 - B. impaired DNA synthesis plus defective myelin maintenance
 - C. insufficient neutrophil availability for an effective acute response
 - D. widespread pathologic thrombin generation throughout the microcirculation

66. During an acute-care case conference, a 56-year-old adult has findings compatible with type 1 diabetes mellitus. Which associated change best follows from the underlying mechanism?

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

67. During an acute-care case conference, a 63-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. ongoing immune activation occurring alongside tissue repair
- C. sensitized T lymphocytes releasing cytokines and activating macrophages
- D. allergen cross-linking of IgE on sensitized mast cells

68. During an acute-care case conference, a 70-year-old patient has fluctuating skeletal muscle weakness that worsens with repeated activity. Which mechanism best explains this pattern?

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

69. During an acute-care case conference, during clinical review, a 77-year-old patient has patchy transmural intestinal inflammation with strictures and fistulas. What is the principal underlying mechanism?

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

70. During an acute-care case conference, a 84-year-old patient is being evaluated for Parkinson disease. Which additional consequence is most consistent with this disorder?

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

71. During an acute-care case conference, the clinician evaluates a 23-year-old patient who has episodic wheezing with reversible expiratory airflow limitation. Which mechanism should be identified as primary?

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

72. During an acute-care case conference, a 30-year-old adult has findings compatible with oxidative cellular injury. Which associated change best follows from the underlying mechanism?

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

73. During an acute-care case conference, a 37-year-old adult develops oliguria after substantial volume depletion while tubular function remains initially intact. Which cellular or physiologic mechanism accounts for these findings?

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

74. During an acute-care case conference, a clinical scenario involving a 44-year-old patient demonstrates intravascular hemolysis. Which additional finding fits the same causal process?

- A. simultaneous microvascular thrombosis and consumption-related hemorrhage
- 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

75. During an acute-care case conference, the care team reviews primary hyperaldosteronism in a 51-year-old adult. Which consequence best reflects its established pathophysiology?

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

76. During an acute-care case conference, a case involves a 58-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. insufficient neutrophil availability for an effective acute response
- B. widespread pathologic thrombin generation throughout the microcirculation
- C. insufficient iron limiting hemoglobin synthesis
- D. impaired DNA synthesis plus defective myelin maintenance

77. During an acute-care case conference, the clinician evaluates a 65-year-old patient who has jaundice, dark urine, pale stools, and pruritus from blocked bile flow. Which mechanism should be identified as primary?

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

78. During an acute-care case conference, in a teaching case, a 72-year-old patient has heat intolerance, tremor, weight loss, and suppressed pituitary stimulation. Which process provides the best explanation?

- 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. glucocorticoid excess promoting protein catabolism and gluconeogenesis

79. During an acute-care case conference, during discussion of respiratory acidosis in a 79-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

80. During an acute-care case conference, a clinical scenario involving a 18-year-old patient demonstrates iron-deficiency anemia. Which additional finding fits the same causal process?

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

- 81.** During an acute-care case conference, the care team reviews ischemic stroke in a 25-year-old adult. Which consequence best reflects its established pathophysiology?
- A. declining neuromuscular transmission safety during repetitive stimulation
 - B. cytotoxic edema with expansion of an irreversibly injured core
 - C. slowed or blocked saltatory conduction along affected axons
 - D. falling cerebral perfusion pressure as intracranial pressure approaches arterial pressure
- 82.** During an acute-care case conference, a 32-year-old patient is being evaluated for type I hypersensitivity. Which additional consequence is most consistent with this disorder?
- A. complement activation and antibody-mediated cellular destruction
 - B. simultaneous parenchymal destruction and collagen deposition
 - C. delayed mononuclear inflammation without an antibody requirement
 - D. rapid histamine release causing vasodilation and bronchoconstriction
- 83.** During an acute-care case conference, the pathophysiology of hypoalbuminemic edema is reviewed in a 39-year-old patient. Which downstream finding should be expected?
- A. water retention that lowers serum sodium without proportional sodium loss
 - B. compensatory hyperventilation that lowers arterial carbon dioxide
 - C. movement of fluid from capillaries into the interstitial compartment
 - D. increased carbonic acid with renal bicarbonate retention if persistent
- 84.** During an acute-care case conference, in a teaching case, a 46-year-old patient has ascites, splenomegaly, and esophageal varices with advanced hepatic fibrosis. Which process provides the best explanation?
- A. increased resistance to portal flow through a scarred nodular liver
 - B. discontinuous immune-mediated inflammation extending through the bowel wall
 - C. failure of conjugated bilirubin and bile salts to reach the intestine
 - D. transient or chronically reduced lower esophageal sphincter competence
- 85.** During an acute-care case conference, during discussion of SIADH in a 53-year-old patient, which physiologic consequence is most appropriate to anticipate?
- A. volume expansion with hypokalemia and metabolic alkalosis
 - B. water retention that lowers serum sodium without proportional sodium loss
 - C. compensatory hyperventilation that lowers arterial carbon dioxide
 - D. increased carbonic acid with renal bicarbonate retention if persistent
- 86.** During an acute-care case conference, a 60-year-old patient develops generalized edema after a marked fall in plasma albumin. Which mechanism best explains this pattern?
- A. gastrointestinal loss of bicarbonate-rich fluid
 - B. excess mineralocorticoid-driven sodium reabsorption in the distal nephron
 - C. reduced plasma colloid oncotic pressure favoring filtration
 - D. alveolar ventilation inadequate for carbon dioxide production

87. During an acute-care case conference, the care team reviews cardiogenic shock in a 67-year-old adult. Which consequence best reflects its established pathophysiology?

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

88. During an acute-care case conference, a 74-year-old patient is being evaluated for pulmonary embolic V/Q mismatch. Which additional consequence is most consistent with this disorder?

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

89. During an acute-care case conference, the clinician evaluates a 81-year-old patient who has concentrated urine despite low plasma osmolality and dilutional hyponatremia. Which mechanism should be identified as primary?

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

90. During an acute-care case conference, in a teaching case, a 20-year-old patient has progressive exertional dyspnea with stiff lungs and reduced diffusion capacity. Which process provides the best explanation?

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

91. During an acute-care case conference, a 27-year-old adult has hypertension, potassium depletion, and increased renal hydrogen ion secretion. Which cellular or physiologic mechanism accounts for these findings?

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

92. During an acute-care case conference, a 34-year-old patient has persistent kidney dysfunction after prolonged ischemia with granular casts in urine. Which mechanism best explains this pattern?

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

93. During an acute-care case conference, during clinical review, a 41-year-old patient has hypoventilation with carbon dioxide retention during a severe obstructive exacerbation. What is the principal underlying mechanism?

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

94. During an acute-care case conference, a case involves a 48-year-old adult who has hyperinflation and reduced elastic recoil after destruction of alveolar walls. Which pathophysiologic process is most directly responsible?

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

95. During an acute-care case conference, the pathophysiology of coagulative necrosis is reviewed in a 55-year-old patient. Which downstream finding should be expected?

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

96. During an acute-care case conference, in a teaching case, a 62-year-old patient has hyperglycemia after immune-mediated loss of pancreatic beta cells. Which process provides the best explanation?

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

97. During an acute-care case conference, during discussion of disseminated intravascular coagulation in a 69-year-old patient, which physiologic consequence is most appropriate to anticipate?

- A. simultaneous microvascular thrombosis and consumption-related hemorrhage
- B. progressively smaller red cells with reduced oxygen-carrying capacity
- C. megaloblastic erythropoiesis with neurologic dysfunction
- D. free hemoglobin binding haptoglobin until circulating stores are depleted

98. During an acute-care case conference, a clinical scenario involving a 76-year-old patient demonstrates extrahepatic biliary obstruction. Which additional finding fits the same causal process?

- A. portosystemic collateral formation and elevated splanchnic hydrostatic pressure
- B. repeated acid exposure causing distal esophageal mucosal injury
- C. reflux of water-soluble conjugated bilirubin into blood and urine
- D. fat necrosis, edema, and systemic inflammatory mediator release

99. During an acute-care case conference, during clinical review, a 83-year-old patient develops anemia, elevated indirect bilirubin, reduced haptoglobin, and hemoglobinuria. What is the principal underlying mechanism?

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

100. During an acute-care case conference, a 22-year-old patient is being evaluated for type III hypersensitivity. Which additional consequence is most consistent with this disorder?

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

Practice Exam 4: Answer Key And Explanations

1. 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.

2. B — Chronic inflammation combines prolonged injurious signaling with repair. Macrophages and lymphocytes sustain cytokine activity while growth factors stimulate angiogenesis and fibrosis, producing tissue destruction and scarring at the same time. Therefore, 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.

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

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

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

6. C — Reintroduced oxygen can generate reactive oxygen species faster than antioxidant systems neutralize them. Free radicals initiate lipid peroxidation, alter proteins, and damage DNA, thereby worsening membrane and organelle injury after perfusion returns. Therefore, 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.

7. A — Autoantibodies impair the postsynaptic neuromuscular membrane and reduce functional acetylcholine receptors. Repeated stimulation exhausts the available transmission margin, so weakness becomes more apparent with activity and improves with rest. Therefore, 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.

8. D — 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.

9. 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.

10. D — 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.

11. C — Interrupted oxygen and glucose delivery rapidly depletes neuronal ATP. Membrane depolarization, calcium influx, and excitatory neurotransmitter release amplify injury, producing cytotoxic edema and cell death in the most severely underperfused tissue. Therefore, 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.

12. A — Failing kidneys retain phosphate and produce less active vitamin D, decreasing intestinal calcium absorption. The resulting signals stimulate parathyroid hormone release, which promotes skeletal mineral mobilization and contributes to renal bone disease. Therefore, 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.

13. 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, 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.

14. 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..

15. 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..

16. D — 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.

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

18. 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, 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.

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

20. C — In type III hypersensitivity, soluble immune complexes deposit in tissues and activate complement. Recruited neutrophils release enzymes and oxidants, producing inflammation at sites such as vessels, glomeruli, or joints. Therefore, 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..

21. 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, 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.

22. 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, 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.

23. 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, 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.

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

25. 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, 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.

26. 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, 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.

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

28. 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.

29. D — Endothelial dysfunction permits lipoprotein entry and inflammatory-cell recruitment. Macrophages ingest modified lipids to become foam cells, while smooth-muscle migration and matrix deposition create a fibrous plaque that can narrow or rupture. Therefore, 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.

30. 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.

31. 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.

32. 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..

33. 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.

34. 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.

35. B — 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.

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

37. 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.

38. 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..

39. 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, ige 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.

40. 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, 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.

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

42. B — 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.

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

44. D — 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.

45. A — 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.

46. B — Lower intestinal fluid contains bicarbonate, so sustained diarrhea reduces the body's base reserve and produces metabolic acidosis. Chemoreceptor stimulation increases ventilation, lowering carbon dioxide as respiratory compensation rather than correcting the original loss. Therefore, 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.

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

48. 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, loss of dopaminergic input to basal ganglia motor circuits is the best answer. The competing choices represent mechanisms from related disorders, but they do.

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

50. 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.

51. C — 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.

52. 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.

53. A — 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..

54. 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.

55. 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.

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

57. C — 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.

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

59. B — 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.

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

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

62. C — 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.

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

64. A — 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.

65. 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, 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..

66. B — Loss of beta cells removes insulin's anabolic signals. Glucose uptake falls in insulin-dependent tissues while hepatic glucose output, fat breakdown, and ketone production rise, creating hyperglycemia and a risk of ketoacidosis. Therefore, 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.

67. C — 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.

68. 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, 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.

69. A — 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.

70. 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.

71. D — 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.

72. B — Reintroduced oxygen can generate reactive oxygen species faster than antioxidant systems neutralize them. Free radicals initiate lipid peroxidation, alter proteins, and damage DNA, thereby worsening membrane and organelle injury after perfusion returns. Therefore, 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..

73. A — 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.

74. D — 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.

75. C — 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.

76. 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, 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.

77. 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, 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.

78. 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.

79. A — 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. D — Iron is required for heme production. Depleted stores restrict hemoglobin synthesis, causing developing erythrocytes to undergo additional divisions and emerge smaller and paler, with diminished capacity to deliver oxygen. Therefore, 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.

81. B — 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.

82. D — 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.

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

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

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

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

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

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

89. 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.

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

91. 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, 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.

92. 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.

93. D — When effective alveolar ventilation falls, carbon dioxide accumulates and shifts the carbonic acid equilibrium toward hydrogen ions. With persistence, the kidneys compensate by conserving bicarbonate and increasing acid excretion. Therefore, 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.

94. B — Destruction of alveolar septa removes both capillary surface and elastic support for small airways. Reduced recoil favors expiratory collapse and air trapping, while the lost interface decreases diffusing capacity. Therefore, 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.

95. 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, 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.

96. C — Loss of beta cells removes insulin's anabolic signals. Glucose uptake falls in insulin-dependent tissues while hepatic glucose output, fat breakdown, and ketone production rise, creating hyperglycemia and a risk of ketoacidosis. Therefore, 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.

97. A — 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.

98. C — 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..

99. D — 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.

100. 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.