PRACTICE EXAM 15: PATHOPHYSIOLOGY (100 QUESTIONS)

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

1. During a transfer evaluation, in a teaching case, a 84-year-old patient has progressive exertional dyspnea with stiff lungs and reduced diffusion capacity. Which process provides the best explanation?

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

2. During a transfer evaluation, a 23-year-old adult develops anemia, elevated indirect bilirubin, reduced haptoglobin, and hemoglobinuria. Which cellular or physiologic mechanism accounts for these findings?

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

3. During a transfer evaluation, during discussion of Parkinson disease in a 30-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. declining neuromuscular transmission safety during repetitive stimulation
D. cytotoxic edema with expansion of an irreversibly injured core

4. During a transfer evaluation, a clinical scenario involving a 37-year-old patient demonstrates respiratory acidosis. Which additional finding fits the same causal process?

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

5. During a transfer evaluation, a case involves a 44-year-old adult who has jugular venous distention, dependent edema, and hepatic congestion. Which pathophysiologic process is most directly responsible?

A. widespread vasodilation and maldistribution of circulating blood flow
B. elevated left atrial and pulmonary capillary hydrostatic pressure
C. severe pump failure reducing stroke volume and tissue perfusion
D. elevated systemic venous pressure from impaired right ventricular output

6. During a transfer evaluation, a 51-year-old patient is being evaluated for apoptosis. Which additional consequence is most consistent with this disorder?

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

7. During a transfer evaluation, the pathophysiology of primary hyperaldosteronism is reviewed in a 58-year-old patient. Which downstream finding should be expected?

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

8. During a transfer evaluation, a 65-year-old adult has findings compatible with myasthenia gravis. Which associated change best follows from the underlying mechanism?

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

9. During a transfer evaluation, during discussion of iron-deficiency anemia in a 72-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

10. During a transfer evaluation, during clinical review, a 79-year-old patient has bradykinesia, rigidity, and resting tremor from degeneration in the substantia nigra. What is the principal underlying mechanism?

A. fixed cranial volume forcing compensatory displacement before pressure rises steeply
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

11. During a transfer evaluation, a case involves a 18-year-old adult who develops generalized edema after a marked fall in plasma albumin. Which pathophysiologic process is most directly responsible?

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

12. During a transfer evaluation, the clinician evaluates a 25-year-old patient who has isolated damaged cells that shrink without provoking surrounding inflammation. Which mechanism should be identified as primary?

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

13. During a transfer evaluation, in a teaching case, a 32-year-old patient develops sudden focal neurologic deficits after arterial cerebral blood flow is interrupted. Which process provides the best explanation?

A. fixed cranial volume forcing compensatory displacement before pressure rises steeply
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

14. During a transfer evaluation, a 39-year-old adult has findings compatible with right-sided heart failure. Which associated change best follows from the underlying mechanism?

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

15. During a transfer evaluation, a 46-year-old patient has exertional dyspnea, orthopnea, and inspiratory crackles. Which mechanism best explains this pattern?

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

16. During a transfer evaluation, during clinical review, a 53-year-old patient has macrocytic anemia accompanied by paresthesias and impaired vibration sensation. What is the principal underlying mechanism?

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

17. During a transfer evaluation, a case involves a 60-year-old adult who has fluctuating skeletal muscle weakness that worsens with repeated activity. Which pathophysiologic process is most directly responsible?

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

18. During a transfer evaluation, the clinician evaluates a 67-year-old patient who develops acidemia after prolonged high-volume diarrhea. Which mechanism should be identified as primary?

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

19. During a transfer evaluation, the pathophysiology of type 1 diabetes mellitus is reviewed in a 74-year-old patient. Which downstream finding should be expected?

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

20. During a transfer evaluation, a 81-year-old adult has findings compatible with chronic inflammation. Which associated change best follows from the underlying mechanism?

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

21. During a transfer evaluation, a 20-year-old patient develops cellular swelling shortly after a brief episode of tissue hypoxia. Which mechanism best explains this pattern?

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

22. During a transfer evaluation, a clinical scenario involving a 27-year-old patient demonstrates atherosclerotic plaque formation. Which additional finding fits the same causal process?

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

23. During a transfer evaluation, a case involves a 34-year-old adult who has hyperglycemia after immune-mediated loss of pancreatic beta cells. Which pathophysiologic process is most directly responsible?

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

24. During a transfer evaluation, a 41-year-old patient is being evaluated for type I hypersensitivity. Which additional consequence is most consistent with this disorder?

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

25. During a transfer evaluation, the pathophysiology of hypoalbuminemic edema is reviewed in a 48-year-old patient. Which downstream finding should be expected?

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

26. During a transfer evaluation, a 55-year-old adult has antibodies directed against antigens fixed on red blood cell membranes. Which cellular or physiologic mechanism accounts for these findings?

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

27. During a transfer evaluation, during discussion of SIADH in a 62-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

28. During a transfer evaluation, a clinical scenario involving a 69-year-old patient demonstrates type II hypersensitivity. Which additional finding fits the same causal process?

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

29. During a transfer evaluation, a case involves a 76-year-old adult who experiences reperfusion of previously ischemic tissue followed by additional membrane damage. Which pathophysiologic process is most directly responsible?

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

30. During a transfer evaluation, the clinician evaluates a 83-year-old patient who has heavy protein loss in urine, hypoalbuminemia, edema, and hyperlipidemia. Which mechanism should be identified as primary?

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

31. During a transfer evaluation, in a teaching case, a 22-year-old patient has ascites, splenomegaly, and esophageal varices with advanced hepatic fibrosis. Which process provides the best explanation?

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

32. During a transfer evaluation, a 29-year-old adult has persistent tissue injury with macrophages, lymphocytes, fibrosis, and attempted repair. Which cellular or physiologic mechanism accounts for these findings?

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

33. During a transfer evaluation, a 36-year-old patient has patchy transmural intestinal inflammation with strictures and fistulas. Which mechanism best explains this pattern?

A. failure of conjugated bilirubin and bile salts to reach the intestine
B. discontinuous immune-mediated inflammation extending through the bowel wall
C. transient or chronically reduced lower esophageal sphincter competence
D. intrapancreatic enzyme activation causing autodigestion

34. During a transfer evaluation, during clinical review, a 43-year-old patient has fatigue with microcytic, hypochromic erythrocytes after chronic blood loss. What is the principal underlying mechanism?

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

35. During a transfer evaluation, a case involves a 50-year-old adult who has hyperinflation and reduced elastic recoil after destruction of alveolar walls. Which pathophysiologic process is most directly responsible?

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

36. During a transfer evaluation, the clinician evaluates a 57-year-old patient who has heat intolerance, tremor, weight loss, and suppressed pituitary stimulation. Which mechanism should be identified as primary?

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

37. During a transfer evaluation, the pathophysiology of oxidative cellular injury is reviewed in a 64-year-old patient. Which downstream finding should be expected?

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

38. During a transfer evaluation, a 71-year-old adult develops sudden hypoxemia when a clot blocks perfusion to ventilated alveoli. Which cellular or physiologic mechanism accounts for these findings?

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

39. During a transfer evaluation, during discussion of primary hyperthyroidism in a 78-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

40. During a transfer evaluation, a clinical scenario involving a 85-year-old patient demonstrates ischemic stroke. Which additional finding fits the same causal process?

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

41. During a transfer evaluation, a case involves a 24-year-old adult who has severe epigastric pain after premature activation of pancreatic digestive enzymes. Which pathophysiologic process is most directly responsible?

A. failure of conjugated bilirubin and bile salts to reach the intestine
B. transient or chronically reduced lower esophageal sphincter competence
C. discontinuous immune-mediated inflammation extending through the bowel wall
D. intrapancreatic enzyme activation causing autodigestion

42. During a transfer evaluation, a 31-year-old patient is being evaluated for diabetic ketoacidosis. Which additional consequence is most consistent with this disorder?

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

43. During a transfer evaluation, in a teaching case, a 38-year-old patient develops an indurated skin reaction two days after antigen exposure. Which process provides the best explanation?

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

44. During a transfer evaluation, a 45-year-old adult has findings compatible with type IV hypersensitivity. Which associated change best follows from the underlying mechanism?

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

45. During a transfer evaluation, during discussion of coagulative necrosis in a 52-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

46. During a transfer evaluation, during clinical review, a 59-year-old patient develops fever with minimal pus formation during a bacterial infection. What is the principal underlying mechanism?

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

47. During a transfer evaluation, a case involves a 66-year-old adult who has hypotension, cool clammy skin, pulmonary congestion, and low cardiac output after myocardial injury. Which pathophysiologic process is most directly responsible?

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

48. During a transfer evaluation, a 73-year-old patient is being evaluated for prerenal acute kidney injury. Which additional consequence is most consistent with this disorder?

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

49. During a transfer evaluation, in a teaching case, a 80-year-old patient has diffuse bleeding while laboratory studies show consumption of platelets and clotting factors. Which process provides the best explanation?

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

50. During a transfer evaluation, a 19-year-old adult has findings compatible with acute pancreatitis. Which associated change best follows from the underlying mechanism?

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

51. During a transfer evaluation, during discussion of type III hypersensitivity in a 26-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

52. During a transfer evaluation, during clinical review, a 33-year-old patient has a pale wedge-shaped renal infarct after arterial occlusion. What is the principal underlying mechanism?

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

53. During a transfer evaluation, a case involves a 40-year-old adult who has hypotension with warm extremities early in a severe systemic inflammatory state. Which pathophysiologic process is most directly responsible?

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

54. During a transfer evaluation, a 47-year-old patient is being evaluated for nephritic syndrome. Which additional consequence is most consistent with this disorder?

A. erythrocyte leakage into tubules with sodium and water retention
B. 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

55. During a transfer evaluation, in a teaching case, a 54-year-old patient develops bilateral pulmonary opacities and severe hypoxemia after a major inflammatory insult. Which process provides the best explanation?

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

56. During a transfer evaluation, a 61-year-old adult has findings compatible with epithelial metaplasia. Which associated change best follows from the underlying mechanism?

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

57. During a transfer evaluation, a 68-year-old patient has hypertension, potassium depletion, and increased renal hydrogen ion secretion. Which mechanism best explains this pattern?

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

58. During a transfer evaluation, a clinical scenario involving a 75-year-old patient demonstrates acute asthma. Which additional finding fits the same causal process?

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

59. During a transfer evaluation, a case involves a 82-year-old adult who develops declining consciousness as an expanding intracranial lesion reduces cerebral perfusion. Which pathophysiologic process is most directly responsible?

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

60. During a transfer evaluation, a 21-year-old patient is being evaluated for left-sided heart failure. Which additional consequence is most consistent with this disorder?

A. reduced systemic vascular resistance despite normal or increased cardiac output
B. transudation of fluid into pulmonary interstitium and alveoli
C. peripheral fluid filtration with congestion of abdominal organs
D. foam-cell formation followed by a fibrous cap over a lipid core

61. During a transfer evaluation, the pathophysiology of disseminated intravascular coagulation is reviewed in a 28-year-old patient. Which downstream finding should be expected?

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

62. During a transfer evaluation, a 35-year-old adult has jaundice, dark urine, pale stools, and pruritus from blocked bile flow. Which cellular or physiologic mechanism accounts for these findings?

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

63. During a transfer evaluation, during discussion of multiple sclerosis in a 42-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

64. During a transfer evaluation, a clinical scenario involving a 49-year-old patient demonstrates severe neutropenia. Which additional finding fits the same causal process?

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

65. During a transfer evaluation, a case involves a 56-year-old adult who has progressive arterial narrowing after endothelial injury and lipid accumulation. Which pathophysiologic process is most directly responsible?

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

66. During a transfer evaluation, a 63-year-old patient is being evaluated for extrahepatic biliary obstruction. Which additional consequence is most consistent with this disorder?

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

67. During a transfer evaluation, in a teaching case, a 70-year-old patient has chronic kidney dysfunction with disturbed calcium and phosphate balance. Which process provides the best explanation?

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

68. During a transfer evaluation, a 77-year-old adult has findings compatible with portal hypertension in cirrhosis. Which associated change best follows from the underlying mechanism?

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

69. During a transfer evaluation, a 84-year-old patient has proximal muscle weakness, hyperglycemia, thin skin, and central fat accumulation. Which mechanism best explains this pattern?

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

70. During a transfer evaluation, a clinical scenario involving a 23-year-old patient demonstrates secondary hyperparathyroidism in chronic kidney disease. 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. avid renal sodium and water conservation with concentrated urine

71. During a transfer evaluation, the care team reviews distributive shock in a 30-year-old adult. Which consequence best reflects its established pathophysiology?

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

72. During a transfer evaluation, the clinician evaluates a 37-year-old patient who has neurologic deficits separated in time and location within the central nervous system. 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

73. During a transfer evaluation, in a teaching case, a 44-year-old patient has episodic wheezing with reversible expiratory airflow limitation. Which process provides the best explanation?

A. diffuse alveolar-capillary barrier injury increasing permeability
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

74. During a transfer evaluation, a 51-year-old adult has hyperglycemia, dehydration, ketones, and deep rapid respirations. Which cellular or physiologic mechanism accounts for these findings?

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

75. During a transfer evaluation, during discussion of reversible hypoxic cell injury in a 58-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

76. During a transfer evaluation, during clinical review, a 65-year-old patient develops vasculitic injury where circulating antigen-antibody complexes lodge in small vessels. What is the principal underlying mechanism?

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

77. During a transfer evaluation, the care team reviews acute tubular injury in a 72-year-old adult. Which consequence best reflects its established pathophysiology?

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

78. During a transfer evaluation, a 79-year-old patient is being evaluated for vitamin B12 deficiency. Which additional consequence is most consistent with this disorder?

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

79. During a transfer evaluation, in a teaching case, a 18-year-old patient has hematuria, red-cell casts, hypertension, and reduced filtration. Which process provides the best explanation?

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

80. During a transfer evaluation, a 25-year-old adult has fatigue, cold intolerance, slowed reflexes, and elevated pituitary stimulation. Which cellular or physiologic mechanism accounts for these findings?

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

81. During a transfer evaluation, during discussion of Crohn disease in a 32-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

82. During a transfer evaluation, a clinical scenario involving a 39-year-old patient demonstrates nephrotic syndrome. Which additional finding fits the same causal process?

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

83. During a transfer evaluation, the care team reviews pulmonary fibrosis in a 46-year-old adult. Which consequence best reflects its established pathophysiology?

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

84. During a transfer evaluation, the clinician evaluates a 53-year-old patient who has hypoventilation with carbon dioxide retention during a severe obstructive exacerbation. Which mechanism should be identified as primary?

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

85. During a transfer evaluation, in a teaching case, a 60-year-old patient has chronic airway irritation and replacement of ciliated columnar cells by squamous cells. Which process provides the best explanation?

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

86. During a transfer evaluation, a 67-year-old adult has findings compatible with intravascular hemolysis. Which associated change best follows from the underlying mechanism?

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

87. During a transfer evaluation, a 74-year-old patient has persistent kidney dysfunction after prolonged ischemia with granular casts in urine. Which mechanism best explains this pattern?

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

88. During a transfer evaluation, during clinical review, a 81-year-old patient develops oliguria after substantial volume depletion while tubular function remains initially intact. What is the principal underlying mechanism?

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

89. During a transfer evaluation, the care team reviews primary hypothyroidism in a 20-year-old adult. Which consequence best reflects its established pathophysiology?

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

90. During a transfer evaluation, the clinician evaluates a 27-year-old patient who has concentrated urine despite low plasma osmolality and dilutional hyponatremia. Which mechanism should be identified as primary?

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

91. During a transfer evaluation, the pathophysiology of emphysema is reviewed in a 34-year-old patient. Which downstream finding should be expected?

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

92. During a transfer evaluation, a 41-year-old adult has findings compatible with bicarbonate-loss metabolic acidosis. Which associated change best follows from the underlying mechanism?

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

93. During a transfer evaluation, during discussion of cortisol excess in a 48-year-old patient, which physiologic consequence is most appropriate to anticipate?

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

94. During a transfer evaluation, a clinical scenario involving a 55-year-old patient demonstrates acute respiratory distress syndrome. Which additional finding fits the same causal process?

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

95. During a transfer evaluation, the care team reviews cardiogenic shock in a 62-year-old adult. Which consequence best reflects its established pathophysiology?

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

96. During a transfer evaluation, a 69-year-old patient is being evaluated for pulmonary embolic VQ mismatch. Which additional consequence is most consistent with this disorder?

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

97. During a transfer evaluation, in a teaching case, a 76-year-old patient has burning retrosternal discomfort after meals and when lying flat. Which process provides the best explanation?

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

98. During a transfer evaluation, a 83-year-old adult has findings compatible with increased intracranial pressure. Which associated change best follows from the underlying mechanism?

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

99. During a transfer evaluation, a 22-year-old patient develops urticaria and wheezing within minutes of allergen exposure. Which mechanism best explains this pattern?

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

100. During a transfer evaluation, a clinical scenario involving a 29-year-old patient demonstrates gastroesophageal reflux. Which additional finding fits the same causal process?

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

Practice Exam 15: Answer Key And Explanations

1. 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, 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.

2. 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.

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

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

5. D — Right ventricular pump failure raises pressure in the systemic venous circulation. The resulting hydrostatic force promotes dependent edema and distends neck veins while venous backup congests the liver and other organs. Therefore, 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.

6. B — Apoptosis activates regulated caspase pathways. The cell condenses and fragments into membrane-bound apoptotic bodies, allowing phagocytosis without leakage of intracellular contents or a prominent inflammatory response. Therefore, 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.

7. A — Aldosterone increases distal sodium reabsorption while promoting potassium and hydrogen secretion. Excess activity expands extracellular volume and raises blood pressure while creating potassium depletion and an alkalotic tendency. Therefore, 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.

8. C — 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.

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

10. 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.

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

12. 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.

13. 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, 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.

14. 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, 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.

15. D — 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.

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

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

18. D — 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.

19. 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.

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

21. 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, 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.

22. C — 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.

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

24. 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.

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

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

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

28. C — Type II reactions involve antibodies against antigens on cells or extracellular structures. Complement, opsonization, or receptor alteration then causes tissue-specific injury rather than deposition of circulating antigen-antibody complexes. Therefore, 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.

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

30. 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.

31. 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, 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.

32. A — 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.

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

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

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

36. 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.

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

38. A — 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.

39. 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.

40. 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.

41. 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.

42. 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.

43. B — Delayed hypersensitivity is mediated by antigen-specific T cells rather than antibodies. Cytokine release recruits and activates macrophages, so induration and tissue injury evolve over many hours instead of appearing immediately. Therefore, 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.

44. 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, 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.

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

46. 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, 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.

47. C — 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.

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

49. 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, 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.

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

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

52. B — Ischemia in most solid organs denatures structural proteins and enzymes. Proteolysis is delayed, so ghostlike cell outlines persist even though nuclei disappear and the affected cells are irreversibly injured. Therefore, 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.

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

54. 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.

55. 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.

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

57. 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, 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.

58. A — Asthma combines airway inflammation, hyperresponsiveness, bronchoconstriction, and mucus production. Narrowed intrathoracic airways impede expiratory flow particularly strongly, producing wheezing and air trapping that can improve when the airway obstruction reverses. Therefore, 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..

59. 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.

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

61. 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.

62. 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, 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.

63. B — 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.

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

65. C — 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.

66. A — Obstruction prevents bile from entering the intestine. Conjugated bilirubin returns to the circulation and is filtered into urine, while less pigment reaches stool; retained bile salts contribute to itching. Therefore, 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..

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

68. 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.

69. A — 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.

70. 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.

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

72. 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.

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

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

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

76. 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..

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

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

79. C — 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..

80. 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.

81. 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, 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.

82. 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..

83. 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.

84. 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.

85. A — 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.

86. C — 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.

87. 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.

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

89. D — 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.

90. 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.

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

92. 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.

93. A — 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..

94. D — Inflammatory injury disrupts pulmonary endothelial and epithelial barriers, allowing protein-rich fluid into alveoli. Surfactant impairment promotes collapse, and perfusion of poorly ventilated units creates shunt physiology that can resist supplemental oxygen. Therefore, 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.

95. C — 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.

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

97. 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.

98. 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.

99. 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.

100. 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.