

PRACTICE EXAM 24: PATHOPHYSIOLOGY SIMULATION

(100 QUESTIONS)

This independent 100-question pathophysiology simulation emphasizes mechanism-first clinical reasoning across major body systems. Select the single best answer for each question. Every item is answerable from text alone. Complete the questions before consulting the answer key and explanations.

1. A biopsy of an enlarged liver shows triglyceride droplets within hepatocytes, but no primary increase in fatty-acid synthesis. Which mechanism most directly produces this cellular accumulation?

- A. Impaired apoprotein synthesis or lipid export
- B. Excess lysosomal digestion of glycogen
- C. Increased renal filtration of albumin
- D. Accelerated collagen hydroxylation

2. A damaged heart valve develops calcium deposits even though the patient's serum calcium is normal. Which process is present?

- A. Metastatic calcification of normal tissue
- B. Dystrophic calcification of injured tissue
- C. Physiologic mineralization of new bone
- D. Osmotic precipitation within red cells

3. A patient with chronic renal failure develops calcium deposits in gastric mucosa and pulmonary tissue. Which mechanism best explains the deposits?

- A. Dystrophic calcification limited to necrotic tissue
- B. Normal osteoblast activity inside the lung
- C. Direct deposition of bilirubin in connective tissue
- D. Metastatic calcification from systemic phosphate and calcium imbalance

4. A patient with chronic inflammation develops extracellular deposits of insoluble protein fibrils that impair kidney and heart function. Which disorder is most likely?

- A. Hydropic degeneration
- B. Fatty liver change
- C. Amyloidosis
- D. Acute coagulative necrosis

5. A patient taking a medication continuously for months requires a higher dose to achieve the same receptor response. Which cellular adaptation contributes?

- A. Increased receptor density on every target cell
- B. Permanent activation of all intracellular enzymes
- C. Receptor downregulation or desensitization
- D. Conversion of receptors into structural collagen

6. A hormone binds a membrane receptor and rapidly increases intracellular cyclic AMP. Which signaling sequence is most likely?

- A. The receptor directly enters the nucleus and binds DNA
- B. G-protein activation stimulates adenylyl cyclase
- C. The hormone opens every lysosomal membrane
- D. The signal requires antibody deposition in tissue

7. A tumor cell continues dividing because a damaged checkpoint fails to restrain entry into DNA synthesis. Which protein normally helps block this transition?

- A. Surfactant protein A
- B. Intrinsic factor
- C. Thrombomodulin
- D. Retinoblastoma protein

8. A cervical carcinoma contains viral proteins that inactivate p53 and retinoblastoma pathways. Which infectious agent is classically associated with this mechanism?

- A. High-risk human papillomavirus
- B. Hepatitis A virus
- C. Epstein-Barr virus in every epithelial tumor
- D. Respiratory syncytial virus

9. A patient with a bronchial tumor develops muscle weakness and hypokalemia from excess hormone production by the tumor. Which phenomenon is occurring?

- A. A paraneoplastic endocrine syndrome
- B. Direct skeletal muscle invasion only
- C. Primary adrenal cortical destruction
- D. Benign epithelial metaplasia

10. A patient with a myeloid malignancy has a fusion protein that continuously activates tyrosine kinase signaling. Which molecular change is most likely?

- A. A reversible increase in lysosomal water
- B. Complete deletion of mitochondrial membranes
- C. A chromosomal translocation creating an oncogenic fusion
- D. Reduced intestinal villous height

11. A patient with recurrent bacterial infections has reduced complement-mediated inflammation but normal antibody levels. Which fragment normally promotes mast-cell activation and vascular permeability?

- A. C3b
- B. C3a
- C. C5b
- D. Factor I

12. A patient with congestive heart failure has a pleural fluid sample with low protein concentration and few inflammatory cells. Which type of fluid is present?

- A. An exudate caused by intense local inflammation
- B. Purulent fluid from bacterial invasion
- C. Chylous fluid from lymphatic rupture
- D. A transudate caused by altered hydrostatic or oncotic pressure

13. A patient with a parasitic infection has marked eosinophilia and elevated IgE. Which cell is especially important in the host response?

- A. Eosinophils
- B. Neutrophils only
- C. Erythrocytes
- D. Osteoclasts

14. A patient with recurrent respiratory infections has very low secretory antibody in the gastrointestinal and respiratory tracts. Which immunoglobulin is deficient?

- A. Immunoglobulin M only
- B. Immunoglobulin E only
- C. Immunoglobulin D only
- D. Immunoglobulin A

15. A previously unexposed patient produces an early antibody response after a new bacterial antigen is encountered. Which immunoglobulin appears first?

- A. Immunoglobulin G
- B. Immunoglobulin M
- C. Immunoglobulin A
- D. Immunoglobulin E

16. A bone marrow transplant recipient develops a rash, diarrhea, and liver injury caused by donor immune cells attacking recipient tissues. Which complication is present?

- A. Hyperacute graft rejection by recipient antibodies
- B. Primary antibody deficiency
- C. Graft-versus-host disease
- D. Immune-complex vasculitis from food exposure

17. A patient with severe complement C3 deficiency has recurrent infections with encapsulated bacteria. Which host defense is impaired?

- A. Antibody-independent red-cell production
- B. Surfactant synthesis by type II pneumocytes
- C. Opsonization and amplification of complement-mediated inflammation
- D. Neural transmission at the neuromuscular junction

18. A patient without a functioning spleen is especially vulnerable to rapidly invasive infection with encapsulated organisms. Which mechanism explains the risk?

- A. Reduced splenic filtration and impaired opsonized-bacteria clearance
- B. Excess gastric acid destroys all bacterial capsules
- C. Increased renal filtration removes antibodies
- D. Enhanced thymic maturation of neutrophils

19. A patient has photosensitivity, arthritis, nephritis, and antibodies against nuclear material. Which immune mechanism contributes to the tissue injury?

- A. Direct IgE activation of mast cells only
- B. Loss of all neutrophil production
- C. Mechanical obstruction of lymphatic vessels
- D. Deposition of circulating immune complexes

20. A patient loses blood rapidly after trauma and develops tachycardia with reduced urine output, but serum sodium initially remains near normal. Which volume disturbance is present?

- A. Pure free-water loss
- B. Isotonic extracellular-fluid loss
- C. Hypertonic saline excess
- D. Isolated intracellular potassium depletion

21. A patient receives a large osmotic load that remains within the intestinal lumen and develops watery diarrhea without mucosal inflammation. Which mechanism is responsible?

- A. Increased plasma oncotic pressure pulls water into blood
- B. Unabsorbed solute retains water osmotically
- C. Loss of all intestinal peristalsis
- D. Excessive renal erythropoietin secretion

22. A patient with severe hyperlipidemia has a low measured serum sodium without symptoms of cellular swelling. Which laboratory phenomenon may be present?

- A. True sodium loss causing brain edema
- B. Hypernatremia from free-water depletion
- C. Pseudohyponatremia from altered plasma water fraction
- D. Potassium replacement causing dilutional anemia

23. A patient with a solid tumor develops weakness, constipation, and high serum calcium because the tumor secretes a hormone-like peptide. Which mediator is likely?

- A. Antidiuretic hormone only
- B. Erythropoietin
- C. Intrinsic factor
- D. Parathyroid hormone-related peptide

24. A patient with acute pancreatitis develops perioral tingling and carpopedal spasm. Which mechanism can lower serum calcium?

- A. Fat necrosis binds calcium in insoluble soaps
- B. Increased parathyroid hormone secretion raises calcium immediately
- C. Excess albumin releases calcium into plasma
- D. Accelerated intestinal calcium absorption

25. A patient receiving chemotherapy develops hyperkalemia, hyperphosphatemia, hypocalcemia, and rising uric acid after rapid tumor destruction. Which syndrome is present?

- A. Adrenal insufficiency
- B. Nephrotic syndrome
- C. Respiratory alkalosis
- D. Tumor lysis syndrome

26. A patient with repeated vomiting has elevated bicarbonate and low urine chloride. Which acid-base pattern is most likely?

- A. High-anion-gap metabolic acidosis
- B. Chloride-unresponsive metabolic alkalosis from mineralocorticoid excess
- C. Chloride-responsive metabolic alkalosis
- D. Primary respiratory acidosis

27. A patient with severe chronic obstructive lung disease has elevated carbon dioxide and increased serum bicarbonate. Which compensatory process is occurring?

- A. Immediate respiratory hyperventilation that lowers carbon dioxide
- B. Renal hydrogen secretion and bicarbonate retention
- C. Renal bicarbonate wasting that worsens acidity
- D. Complete suppression of ammonium production

28. A patient overdoses on salicylates and develops both rapid breathing and an increased anion gap. Which acid-base combination is characteristic?

- A. Respiratory alkalosis with metabolic acidosis
- B. Respiratory acidosis with metabolic alkalosis
- C. Two simultaneous alkaloses
- D. Two simultaneous respiratory acidoses

29. A patient with sick sinus syndrome cannot appropriately increase heart rate during exercise. Which physiologic impairment is present?

- A. Chronotropic incompetence
- B. Excessive ventricular preload
- C. Increased coronary vasospasm
- D. Accelerated atrioventricular conduction

30. A patient with a ruptured coronary plaque develops an acute myocardial infarction. Which event most directly converts the plaque into an occlusive lesion?

- A. Gradual venous valve failure
- B. Immediate dilation of every coronary branch
- C. Reduced fibrin formation at the plaque surface
- D. Platelet activation and thrombus formation over exposed tissue factor

31. A patient has episodic chest pain at rest with transient ST-segment elevation and no fixed severe coronary obstruction. Which mechanism is most likely?

- A. Permanent aortic outflow obstruction
- B. Transient coronary vasospasm
- C. Systemic venous valve failure
- D. Reduced platelet production in marrow

32. A young adult develops chest pain, arrhythmias, and reduced ventricular function after a viral illness. Which process is most likely?

- A. Isolated pulmonary embolism without myocardial inflammation
- B. Chronic venous insufficiency
- C. Myocarditis with inflammatory injury to cardiac muscle
- D. Primary gastric smooth-muscle spasm

33. A patient has a markedly enlarged heart, reduced ejection fraction, and global systolic dysfunction without a localized infarct. Which cardiomyopathy is most likely?

- A. Dilated cardiomyopathy
- B. Hypertrophic obstructive cardiomyopathy
- C. Restrictive pericarditis
- D. Isolated mitral stenosis

34. A patient with asymmetric septal hypertrophy has exertional syncope caused by dynamic obstruction that worsens when ventricular volume falls. Which condition is present?

- A. Dilated cardiomyopathy with thin ventricular walls
- B. Hypertrophic obstructive cardiomyopathy
- C. Acute pericardial tamponade
- D. Uncomplicated sinus bradycardia

35. A patient has edema, ascites, and prominent ventricular interdependence after prior pericardial inflammation. Which mechanism is most likely?

- A. Excess pericardial compliance increases filling
- B. Isolated mitral valve prolapse
- C. A rigid pericardium restricts diastolic filling
- D. Complete loss of systemic venous pressure

36. A child with an atrial septal defect has a long-standing left-to-right shunt and increased pulmonary blood flow. Which consequence is expected initially?

- A. Immediate systemic desaturation from right-to-left flow
- B. Reduced pulmonary perfusion from complete obstruction
- C. Isolated left ventricular pressure overload
- D. Right-sided volume overload

37. A patient with an unrepaired congenital shunt develops cyanosis and clubbing years later as pulmonary vascular resistance becomes very high. Which process has occurred?

- A. Spontaneous closure of the septal defect
- B. Reduced pulmonary resistance with increased oxygenation
- C. Eisenmenger reversal to right-to-left flow
- D. Pure left-sided volume depletion

38. A patient with worsening heart failure has elevated plasma B-type natriuretic peptide. Which physiologic response does this hormone promote?

- A. Natriuresis and vasodilation that reduce ventricular load
- B. Sodium retention and intense vasoconstriction
- C. Increased aldosterone synthesis from the adrenal cortex
- D. Bronchial smooth-muscle contraction

39. A patient with sudden pleuritic pain and severe dyspnea has absent breath sounds on one side, hypotension, and tracheal deviation. Which process is occurring?

- A. Simple bronchitis without pleural pressure change
- B. Bilateral pulmonary edema from low albumin
- C. Chronic restrictive fibrosis of both lungs
- D. Tension pneumothorax compresses the mediastinum and venous return

40. A postoperative patient has shallow breathing and a mucus plug blocking a segmental bronchus. Which type of atelectasis can result?

- A. Compression atelectasis from pleural fluid only
- B. Resorption atelectasis from trapped, absorbed alveolar gas
- C. Cicatrization from long-term fibrosis only
- D. Congenital absence of all alveoli

41. A patient with severe pneumonia develops rising carbon dioxide and fatigue after prolonged rapid breathing. Which failure has occurred?

- A. Respiratory muscle fatigue reduces effective alveolar ventilation
- B. Increased alveolar ventilation clears all carbon dioxide
- C. Complete restoration of respiratory reserve
- D. Isolated renal bicarbonate loss

42. A patient with emphysema has enlarged air spaces, flattened diaphragms, and increased total lung capacity after destruction of alveolar walls. Which mechanical change is central?

- A. Increased surface tension from excess surfactant
- B. Reduced airway compliance from pleural fluid
- C. Loss of elastic recoil with air trapping
- D. Complete closure of all conducting bronchi

43. A smoker has shoulder pain, hand weakness, ptosis, and miosis from an apical lung mass. Which structure is being affected?

- A. The phrenic nerve alone
- B. The lower brachial plexus and cervical sympathetic chain
- C. The vagus nerve at the diaphragm only
- D. The optic nerve within the orbit

44. A patient with bacterial pleuritis has cloudy pleural fluid with high protein and many leukocytes. Which type of fluid is present?

- A. A low-protein transudate
- B. Pure chyle from thoracic duct disruption
- C. Normal ultrafiltrate without inflammation
- D. An inflammatory exudate

45. A patient with bilateral hilar lymphadenopathy and noncaseating granulomas has cough and fatigue. Which disease process is most likely?

- A. Acute bacterial lobar consolidation
- B. Sarcoidosis with immune-mediated granuloma formation
- C. Pulmonary edema from left ventricular failure
- D. Isolated bronchial smooth-muscle spasm

46. A patient with progressive dyspnea has pleural thickening after decades of occupational fiber exposure. Which disease is most strongly associated with this history?

- A. Acute viral bronchiolitis
- B. Simple allergic rhinitis
- C. Isolated pulmonary artery spasm
- D. Malignant pleural mesothelioma after asbestos exposure

47. A patient with heavy proteinuria has selective loss of albumin and diffuse podocyte foot-process effacement on electron microscopy. Which lesion is most likely?

- A. Minimal-change disease
- B. Focal segmental glomerulosclerosis
- C. Membranous nephropathy
- D. Postrenal obstruction

48. A patient with obesity has nephrotic-range proteinuria and scarring involving portions of some glomeruli. Which lesion is most likely?

- A. Minimal-change disease with uniform normal glomeruli
- B. Distal ureteral obstruction
- C. Focal segmental glomerulosclerosis
- D. Acute interstitial nephritis with eosinophils

49. A patient has nephrotic syndrome with subepithelial immune deposits and diffuse thickening of glomerular capillary walls. Which condition is present?

- A. IgA nephropathy with mesangial deposits only
- B. Acute tubular necrosis
- C. Papillary necrosis from analgesics
- D. Membranous nephropathy

50. A patient rapidly loses kidney function and develops hematuria with crescent formation in glomeruli. Which syndrome is present?

- A. Uncomplicated nephrotic syndrome
- B. Rapidly progressive glomerulonephritis
- C. Chronic cystitis
- D. Prerenal azotemia from mild thirst

51. A patient develops visible hematuria shortly after an upper-respiratory infection. Which renal disorder is most likely?

- A. Membranous nephropathy
- B. Acute pyelonephritis from obstruction
- C. IgA nephropathy
- D. Minimal-change disease

52. A patient with diabetes and long-term analgesic use develops papillary sloughing and flank pain. Which lesion is most likely?

- A. Renal papillary necrosis
- B. Renal cortical hypertrophy
- C. Minimal-change podocyte injury
- D. Uncomplicated cystitis

53. A patient with recurrent urinary infections has branching renal stones, alkaline urine, and a urease-producing organism. Which stone type is most likely?

- A. Cystine stones from inherited transport failure
- B. Uric acid stones from acidic urine
- C. Cholesterol stones from bile supersaturation
- D. Struvite stones

54. A patient has repeated urinary infections and imaging shows thin-walled cystic spaces within the renal medulla. Which condition is suggested?

- A. Medullary sponge kidney
- B. Acute cortical necrosis
- C. Membranous nephropathy
- D. Renal artery stenosis

55. A postpartum patient develops severe hypotension, disseminated coagulation, and abrupt loss of renal function with cortical ischemia. Which lesion is most likely?

- A. Simple prerenal azotemia corrected by oral water
- B. Bilateral renal cortical necrosis
- C. Minimal-change disease
- D. Uncomplicated renal colic

56. A patient with volume depletion has intact tubules that avidly conserve sodium and water. Which urine pattern is expected?

- A. Dilute urine with high sodium excretion
- B. Persistent glycosuria from proximal injury
- C. Concentrated urine with low sodium excretion
- D. Alkaline urine from complete acidification failure

57. A patient with an adrenal adenoma has hypertension, proximal weakness, hyperglycemia, and low plasma ACTH. Which disorder is most likely?

- A. Pituitary ACTH excess
- B. Primary adrenal insufficiency
- C. ACTH-independent Cushing syndrome
- D. Central diabetes insipidus

58. A patient with pituitary disease has low cortisol, low ACTH, fatigue, and no hyperpigmentation. Which endocrine disorder is present?

- A. Primary adrenal insufficiency with high ACTH
- B. Cortisol excess from an adrenal tumor
- C. Primary hyperparathyroidism
- D. Secondary adrenal insufficiency

59. A patient with Graves disease develops eye prominence and restricted extraocular movement. Which mechanism contributes?

- A. Autoimmune activation of orbital fibroblasts and extraocular tissues
- B. Direct bacterial invasion of the optic nerve
- C. Loss of all thyroid hormone receptors
- D. Reduced orbital glycosaminoglycan production

60. A patient has fatigue, weight gain, and high TSH with antibodies against thyroid peroxidase. Which disorder is most likely?

- A. Graves hyperthyroidism
- B. Hashimoto autoimmune thyroiditis
- C. Pheochromocytoma
- D. Primary hyperaldosteronism

61. A pregnant patient develops hyperglycemia because placental hormones reduce maternal insulin sensitivity. Which mechanism is involved?

- A. Complete absence of placental hormones
- B. Permanent destruction of all maternal beta cells
- C. Excess renal erythropoietin secretion
- D. Gestational insulin resistance with inadequate beta-cell compensation

62. A patient has recurrent kidney stones, peptic ulcers, and a pituitary tumor as part of an inherited endocrine syndrome. Which syndrome is most likely?

- A. Multiple endocrine neoplasia type 2 only
- B. Isolated autoimmune thyroiditis
- C. Multiple endocrine neoplasia type 1
- D. Congenital adrenal hyperplasia

63. A patient has hypocalcemia and elevated parathyroid hormone, but target tissues fail to respond to the hormone. Which disorder is suggested?

- A. Pseudohypoparathyroidism
- B. Primary hyperparathyroidism
- C. Vitamin D intoxication
- D. Thyroid storm

64. A patient with fasting hypoglycemia first becomes sweaty and tremulous, then confused and unresponsive. Which sequence is occurring?

- A. Neuroglycopenia precedes every autonomic response
- B. Adrenergic warning signs precede neuroglycopenia
- C. Hyperglycemia causes the early symptoms
- D. Aldosterone directly supplies glucose to neurons

65. A patient with a transient episode of unilateral weakness has no lasting neurologic deficit and no evidence of infarction. Which event is most likely?

- A. Completed cerebral infarction with permanent necrosis
- B. A transient ischemic attack from temporary focal hypoperfusion
- C. Diffuse meningitis
- D. Progressive peripheral neuropathy

66. An ischemic stroke contains a central infarct surrounded by potentially salvageable tissue with reduced but not absent blood flow. What is this peripheral zone called?

- A. The ventricular ependyma
- B. The subarachnoid cistern
- C. The medullary pyramid only
- D. The ischemic penumbra

67. A patient with traumatic brain injury develops headache and vomiting from water movement into injured brain cells. Which type of edema is present?

- A. Vasogenic edema from blood-brain barrier leakage only
- B. Peripheral dependent edema
- C. Cytotoxic edema from cellular ion-pump failure
- D. Lymphatic edema of the lower extremities

68. A patient has enlarged ventricles after obstruction of the cerebral aqueduct. Which type of hydrocephalus is present?

- A. Noncommunicating hydrocephalus
- B. Communicating hydrocephalus from impaired arachnoid absorption
- C. Hydrostatic pulmonary edema
- D. Renal obstructive uropathy

69. A patient has distal muscle wasting, foot deformity, and slowed peripheral nerve conduction caused by an inherited myelin abnormality. Which disorder is most likely?

- A. Multiple sclerosis
- B. Myasthenia gravis
- C. Charcot-Marie-Tooth disease
- D. Parkinson disease

70. An older hospitalized patient develops abrupt inattention, fluctuating awareness, and disorganized thinking during an infection. Which syndrome is most likely?

- A. Delirium from acute brain dysfunction
- B. Alzheimer dementia with gradual progression
- C. Chronic peripheral neuropathy
- D. Isolated cerebellar ataxia

71. A patient with alcoholism has confusion, ataxia, and abnormal eye movements. Which deficiency is most likely responsible?

- A. Vitamin K excess
- B. Thiamine deficiency causing Wernicke encephalopathy
- C. Iron overload
- D. Calcium excess

72. A patient has a prolonged generalized seizure with ongoing neuronal firing and rising lactic acid. Which mechanism contributes to neuronal injury?

- A. Complete suppression of glutamate release
- B. Increased cerebral oxygen reserve without demand
- C. Immediate restoration of ATP stores
- D. Excitotoxic calcium influx and metabolic exhaustion

73. After a cervical spinal cord injury, a patient initially has flaccid paralysis and absent reflexes below the lesion. Which phase is present?

- A. Spinal shock
- B. Autonomic dysreflexia
- C. Brown-Sequard syndrome
- D. Chronic peripheral neuropathy

74. A comatose patient has impaired arousal after injury to the network that maintains wakefulness through the brainstem and thalamus. Which structure is affected?

- A. The renal medulla
- B. The neuromuscular junction
- C. The intestinal villus
- D. The ascending reticular activating system

75. A patient with chronic reflux develops replacement of distal esophageal squamous epithelium by intestinal-type columnar cells. Which process is present?

- A. Acute epithelial necrosis only
- B. Gastric volvulus
- C. Barrett metaplasia
- D. Small-bowel obstruction

76. An infant has projectile nonbilious vomiting and a palpable olive-shaped mass near the pylorus. Which mechanism is most likely?

- A. Relaxation of the lower esophageal sphincter
- B. Hypertrophic thickening of the pyloric muscle obstructs gastric emptying
- C. Inflammation of the terminal ileum
- D. Loss of colonic haustral contractions

77. A patient with atherosclerosis has severe postprandial abdominal pain and weight loss because eating increases intestinal oxygen demand. Which disorder is most likely?

- A. Chronic mesenteric ischemia
- B. Uncomplicated lactose intolerance
- C. Achalasia
- D. Nephrotic syndrome

78. A patient develops severe abdominal pain out of proportion to examination findings and later has bloody stool after an embolus lodges in the superior mesenteric artery. Which process is present?

- A. Functional irritable bowel syndrome
- B. Acute mesenteric ischemia with bowel infarction
- C. Painless hemorrhoidal bleeding
- D. Uncomplicated viral gastroenteritis

79. A patient with recurrent ulcers and diarrhea has markedly elevated gastrin from a pancreatic neuroendocrine tumor. Which syndrome is most likely?

- A. Achalasia
- B. Acute cholecystitis
- C. Zollinger-Ellison syndrome
- D. Celiac disease

80. A patient has bronze skin, diabetes, cirrhosis, and iron deposition in hepatocytes and pancreatic tissue. Which disorder is most likely?

- A. Wilson disease from copper accumulation only
- B. Gilbert syndrome from mild bilirubin-conjugation impairment
- C. Acute viral pharyngitis
- D. Hereditary hemochromatosis with excessive iron absorption

81. A patient with chronic hepatitis B has liver injury caused partly by immune destruction of infected hepatocytes. Which mechanism is central?

- A. The virus directly replaces every hepatocyte with collagen
- B. IgE degranulation causes isolated liver necrosis
- C. Reduced bile flow alone destroys viral DNA
- D. Cytotoxic T cells recognize viral antigens on hepatocytes

82. A patient with jaundice has an inherited mild defect in bilirubin conjugation but no hemolysis or liver inflammation. Which disorder is most likely?

- A. Acute cholangitis
- B. Hemochromatosis
- C. Gilbert syndrome
- D. Wilson disease

83. A patient has schistocytes, thrombocytopenia, neurologic changes, renal dysfunction, and hemolytic anemia after endothelial injury. Which process is present?

- A. Iron-deficiency anemia
- B. Microangiopathic hemolytic anemia
- C. Hereditary spherocytosis
- D. Simple leukopenia

84. A patient has severe microcytosis with target cells and a family history of anemia despite normal iron stores. Which disorder is most likely?

- A. Thalassemia from impaired globin-chain synthesis
- B. Acute blood loss
- C. Vitamin B12 deficiency
- D. Autoimmune thrombocytopenia

85. A patient develops jaundice and bite cells after taking an oxidizing medication. Which inherited defect is most likely?

- A. Factor VIII deficiency
- B. Fibrinogen excess
- C. Glucose-6-phosphate dehydrogenase deficiency
- D. Vitamin B12 excess

86. A patient has chronic hemolytic anemia, jaundice, and splenomegaly from spherical red cells that are removed in the spleen. Which disorder is most likely?

- A. Thalassemia major
- B. Aplastic anemia
- C. Disseminated intravascular coagulation
- D. Hereditary spherocytosis

87. Several days after starting heparin, a patient has a major platelet fall and a new venous thrombosis. Which mechanism is most likely?

- A. IgG antibodies against platelet factor 4-heparin complexes
- B. Direct destruction of erythrocytes by bile salts
- C. Inherited absence of von Willebrand factor
- D. Suppressed thrombin generation from excess antithrombin

88. A child has repeated fractures, blue sclerae, and hearing impairment from a structural collagen defect. Which disorder is most likely?

- A. Osteomalacia from vitamin D deficiency
- B. Osteogenesis imperfecta from abnormal type I collagen
- C. Osteoarthritis from cartilage wear
- D. Acute osteomyelitis

89. A patient has marked joint hypermobility, fragile skin, and easy bruising from an inherited extracellular-matrix disorder. Which mechanism is most likely?

- A. Excessive osteoclast inhibition
- B. Loss of pulmonary surfactant only
- C. Increased platelet production
- D. Defective collagen synthesis or processing

90. A teenager has progressive pain and a rapidly enlarging metaphyseal bone mass that produces osteoid. Which tumor is most likely?

- A. Chondromalacia
- B. Osteosarcoma
- C. Osteoporosis
- D. Simple bone cyst

91. A pearly papule on sun-exposed skin has local tissue invasion but rarely spreads to distant organs. Which tumor is most likely?

- A. Basal cell carcinoma
- B. Metastatic melanoma
- C. Osteosarcoma
- D. Kaposi sarcoma

92. A patient develops a sharply demarcated, bright-red, raised facial skin infection with fever and tender regional lymph nodes. Which condition is most likely?

- A. Cellulitis limited to deep muscle
- B. A chronic keloid scar
- C. Erysipelas involving superficial dermis and lymphatics
- D. A noninfectious urticarial eruption

93. A critically ill patient has oozing from venipuncture sites, prolonged clotting times, low platelets, and schistocytes after widespread tissue-factor activation. Which process is present?

- A. Isolated hemophilia A
- B. Disseminated intravascular coagulation with consumption of clotting factors
- C. Uncomplicated iron deficiency
- D. Localized allergic inflammation

94. A patient develops episodic severe bone pain after dehydration because deoxygenated hemoglobin forms rigid polymers inside erythrocytes. Which disorder is most likely?

- A. Sickle cell disease with hemoglobin S polymerization
- B. Hereditary spherocytosis from membrane loss
- C. Autoimmune neutropenia
- D. Folate-deficiency macrocytosis

95. A patient has fatigable ptosis and diplopia that worsen late in the day because antibodies impair neuromuscular transmission. Which target is most likely affected?

- A. Presynaptic dopamine transporters in the substantia nigra
- B. Myelin in the dorsal columns only
- C. Voltage-gated calcium channels in renal tubules
- D. Postsynaptic nicotinic acetylcholine receptors at the neuromuscular junction

96. A patient has ipsilateral limb dysmetria, intention tremor, and a wide-based gait after a posterior-fossa lesion. Which structure is primarily involved?

- A. The substantia nigra pars compacta
- B. The anterior pituitary
- C. The cerebellar hemisphere
- D. The renal glomerulus

97. A patient has bloody diarrhea and urgency from continuous superficial inflammation that begins in the rectum and extends proximally. Which disorder is most likely?

- A. Crohn disease with skip lesions
- B. Acute appendicitis
- C. Ulcerative colitis
- D. Irritable bowel syndrome without mucosal inflammation

98. A patient has abdominal pain, weight loss, and a draining perianal fistula from patchy full-thickness intestinal inflammation. Which disorder is most likely?

- A. Ulcerative colitis limited to the mucosa
- B. Acute viral pharyngitis
- C. Isolated gastric reflux
- D. Crohn disease with transmural skip lesions

99. A patient develops fever and right-upper-quadrant pain after a gallstone obstructs the cystic duct. Which condition is most likely?

- A. Acute cholecystitis from gallbladder inflammation
- B. Acute appendicitis from cecal obstruction
- C. Pancreatic insufficiency from absent lipase
- D. Nephrolithiasis from urinary stasis

100. A patient has chronic diarrhea, iron deficiency, and weight loss after gluten exposure, with small-intestinal villous atrophy. Which mechanism is most likely?

- A. Cystic-duct obstruction
- B. Immune-mediated celiac injury to small-intestinal mucosa
- C. Loss of gastric parietal cells only
- D. Excess colonic water absorption

Practice Exam 24: Answer Key and Explanations

- 1. A** — Fatty change occurs when hepatocytes cannot adequately oxidize fatty acids or package them into lipoproteins for export. Triglycerides then accumulate within the cytoplasm. Glycogen digestion, albumin filtration, and collagen hydroxylation do not explain intracellular lipid storage in the liver. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.
- 2. B** — Dystrophic calcification occurs in necrotic or chronically damaged tissue despite normal serum calcium. Injured membranes bind phosphate and promote local mineral deposition. Metastatic calcification affects otherwise normal tissues during systemic calcium or phosphate imbalance, while bone mineralization and red-cell osmosis are unrelated. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.
- 3. D** — Metastatic calcification occurs when elevated calcium-phosphate product causes mineral deposition in otherwise viable tissues. Chronic renal failure promotes phosphate retention and secondary hormonal changes. Dystrophic calcification is restricted to damaged tissue, and osteoblasts or bilirubin do not explain pulmonary and gastric deposits. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.
- 4. C** — Amyloidosis results from extracellular deposition of misfolded protein fibrils in organs. Deposits distort tissue architecture and impair filtration, contractility, or other functions depending on location. Hydropic change is intracellular water accumulation, fatty change involves lipid droplets, and coagulative necrosis is an ischemic pattern of cell death. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.
- 5. C** — Chronic stimulation can reduce receptor number or uncouple receptors from downstream signaling, producing desensitization and tolerance. Increased receptor density would amplify the response. Enzyme activation and collagen formation are not the usual adaptive mechanisms for persistent agonist exposure. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.
- 6. B** — Many membrane receptors activate heterotrimeric G proteins. The alpha subunit can stimulate adenylyl cyclase, increasing cyclic AMP and activating protein kinase A. Nuclear receptors act directly on gene transcription, while lysosomal injury and antibody deposition are unrelated mechanisms. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.
- 7. D** — Retinoblastoma protein restrains the G1-to-S transition by binding transcription factors that promote DNA synthesis. Loss of this checkpoint permits inappropriate proliferation. Surfactant supports alveolar stability, intrinsic factor enables vitamin B12 absorption, and thrombomodulin regulates coagulation rather than the cell cycle. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

8. A — High-risk human papillomavirus produces E6 and E7 proteins that interfere with p53 and retinoblastoma tumor-suppressor pathways. This promotes genomic instability and uncontrolled cervical epithelial proliferation. Hepatitis A, Epstein-Barr virus, and respiratory syncytial virus do not explain this classic cervical carcinogenesis mechanism. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

9. A — Paraneoplastic syndromes arise when tumors release hormones, cytokines, or other mediators that produce effects distant from the primary mass. Ectopic hormone production can cause weakness and electrolyte abnormalities. Direct invasion, adrenal destruction, and metaplasia do not explain systemic findings caused by secreted tumor products. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

10. C — Chromosomal translocations can join regulatory and signaling regions to create constitutively active fusion proteins, as in BCR-ABL signaling. Continuous kinase activity promotes proliferation and survival. Lysosomal water, mitochondrial membranes, and intestinal villi do not create an oncogenic fusion signal. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

11. B — C3a is an anaphylatoxin that contributes to mast-cell degranulation, vasodilation, and increased vascular permeability. C3b functions mainly as an opsonin, C5b begins membrane attack complex assembly, and factor I helps regulate complement cleavage. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern. This is the key distinction here.

12. D — Transudates form when systemic hydrostatic or oncotic forces change without primary local inflammation. Heart failure commonly raises hydrostatic pressure and produces low-protein fluid. Exudates are protein-rich inflammatory fluids, purulent fluid contains infection, and chyle contains lymph and triglycerides. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

13. A — Eosinophils participate in defense against helminths and in IgE-associated allergic inflammation. Their granules contain proteins that can damage large parasites. Neutrophils are important for many bacterial infections, erythrocytes transport gases, and osteoclasts remodel bone. The distinction matters because assessment findings follow the disease process rather than one isolated symptom. That explains the clinical pattern.

14. D — Secretory IgA protects mucosal surfaces by neutralizing organisms and preventing adherence to epithelial cells. Deficiency predisposes to respiratory and gastrointestinal infection. IgM is prominent in early systemic responses, IgE mediates allergy and parasite defense, and IgD is mainly a B-cell surface receptor. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

15. B — Naive B cells typically produce IgM first during a primary immune response. With helper signals and class switching, later antibodies may include IgG, IgA, or IgE. IgM therefore best represents the initial systemic antibody response. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

16. C — Graft-versus-host disease occurs when immunocompetent donor T cells recognize recipient antigens as foreign and attack skin, gastrointestinal tract, and liver. Hyperacute rejection involves recipient antibodies against a graft. Antibody deficiency and food-related immune complexes do not explain this transplant complication. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

17. C — C3 is central to complement activation and generates C3b, an important opsonin. Loss of C3 weakens microbial coating, phagocytosis, and downstream inflammatory activation, increasing susceptibility to pyogenic organisms. Red-cell production, surfactant, and neuromuscular transmission do not depend on C3. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

18. A — The spleen filters blood and helps clear opsonized encapsulated bacteria through macrophages and antibody-assisted phagocytosis. Asplenia therefore increases risk from organisms such as pneumococcus. Gastric acid, renal filtration, and thymic neutrophil maturation do not provide this splenic function. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

19. D — Systemic lupus erythematosus can produce antibodies against nuclear antigens. Circulating immune complexes deposit in tissues, activate complement, and recruit inflammatory cells, causing nephritis, skin disease, and arthritis. IgE allergy, neutrophil production, and lymphatic obstruction do not explain this multisystem pattern. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

20. B — Acute hemorrhage removes plasma and cells in approximately isotonic proportions, so the primary early abnormality is reduced extracellular volume rather than a major sodium shift. Free-water loss raises sodium, saline excess expands volume, and isolated potassium depletion does not explain hemorrhagic shock. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

21. B — Osmotic diarrhea occurs when poorly absorbed solutes remain in the intestinal lumen and retain water. Symptoms improve when the offending substance is removed. Plasma oncotic pressure, erythropoietin, and complete loss of peristalsis do not explain this luminal water retention. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

22. C — Marked lipids or proteins can reduce the plasma water fraction measured by some laboratory methods, creating pseudohyponatremia without a true fall in plasma tonicity. True sodium loss causes hypotonic symptoms, while hyponatremia and anemia do not explain the laboratory artifact. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

23. D — Some malignancies secrete parathyroid hormone-related peptide, which increases bone resorption and renal calcium retention, producing humoral hypercalcemia. ADH causes water retention, erythropoietin stimulates red-cell production, and intrinsic factor supports B12 absorption. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

24. A — Pancreatic lipase can cause fat necrosis, and released fatty acids bind calcium to form insoluble soaps. Serum calcium falls, contributing to neuromuscular excitability. Increased parathyroid hormone and intestinal absorption would raise calcium, while albumin does not release calcium in this manner. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

25. D — Tumor lysis syndrome releases intracellular potassium and phosphate and nucleic-acid breakdown products that become uric acid. Phosphate binds calcium, and acute kidney injury can worsen all abnormalities. Adrenal insufficiency, nephrotic syndrome, and respiratory alkalosis do not produce this post-treatment constellation. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

26. C — Vomiting removes hydrochloric acid and chloride, producing metabolic alkalosis with low urine chloride. Volume and chloride replacement can help restore renal bicarbonate excretion. Mineralocorticoid excess is usually chloride resistant, while respiratory acidosis and high-anion-gap acidosis begin with different primary abnormalities. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

27. B — Chronic respiratory acidosis stimulates the kidneys to excrete more hydrogen ion and generate or retain bicarbonate. This compensation raises serum bicarbonate but may not fully normalize pH. Bicarbonate wasting and suppressed ammonium production would worsen the acidosis. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

28. A — Salicylates stimulate the respiratory center, producing early respiratory alkalosis, and later impair cellular metabolism, producing an anion-gap metabolic acidosis. The combination is important because one isolated disorder does not explain the complete pattern. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

29. A — Chronotropic incompetence is the inability to increase heart rate adequately in response to activity or demand. It can occur with sinus-node dysfunction or medication effects and limits cardiac output during exertion. Preload, coronary spasm, and AV conduction are different determinants of cardiovascular performance. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

30. D — Plaque rupture exposes thrombogenic material and tissue factor, activating platelets and the coagulation cascade. A rapidly enlarging thrombus can obstruct coronary flow and cause infarction. Venous valve failure, diffuse coronary dilation, and reduced fibrin formation do not create the acute occlusion. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

31. B — Prinzmetal angina results from transient coronary smooth-muscle constriction, reducing myocardial blood flow at rest. Fixed plaque, aortic stenosis, venous disease, and platelet production abnormalities do not explain brief rest pain with reversible ST elevation. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

32. C — Myocarditis involves inflammatory injury to cardiomyocytes, often after infection. Edema, necrosis, and conduction-system involvement can reduce contractility and cause arrhythmias. Pulmonary embolism, venous insufficiency, and gastric spasm do not directly explain new myocardial dysfunction after a viral illness. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

33. A — Dilated cardiomyopathy enlarges ventricular chambers and impairs contractile force, reducing ejection fraction. Hypertrophic cardiomyopathy thickens myocardium and may obstruct outflow, restrictive pericarditis limits filling externally, and mitral stenosis primarily obstructs left atrial emptying. The distinction matters because assessment findings follow the disease process rather than one isolated symptom. That explains the clinical pattern.

34. B — Hypertrophic obstructive cardiomyopathy produces a thick septum and dynamic left ventricular outflow obstruction. Reduced preload can worsen the obstruction, lowering forward output and causing syncope. Dilated cardiomyopathy, tamponade, and sinus bradycardia involve different structural or electrical problems. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

35. C — Constrictive pericarditis produces a stiff pericardial shell that limits diastolic expansion. Ventricular interdependence increases, and systemic venous congestion causes edema and ascites. Increased compliance, mitral prolapse, and absent venous pressure do not explain the filling restriction. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

36. D — An atrial septal defect initially allows oxygenated left-atrial blood to flow into the right atrium. Right-sided chambers and pulmonary circulation receive excess volume, while systemic oxygenation remains relatively preserved. Cyanosis develops later only if pulmonary vascular disease reverses the shunt. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

37. C — Long-standing left-to-right flow can remodel pulmonary arterioles and raise pulmonary vascular resistance. Once right-sided pressure exceeds left-sided pressure, the shunt reverses, producing cyanosis and clubbing. Closure, reduced resistance, and left-sided depletion do not explain late cyanosis. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

38. A — B-type natriuretic peptide is released by stretched cardiac chambers. It promotes renal sodium excretion, vasodilation, and suppression of maladaptive RAAS activity, reducing preload and afterload. Sodium retention, aldosterone stimulation, and bronchoconstriction oppose its principal actions. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

39. D — A tension pneumothorax creates a one-way air leak that raises intrapleural pressure, collapses the affected lung, shifts the mediastinum, and reduces venous return. Bronchitis, bilateral edema, and chronic fibrosis do not produce this acute obstructive shock pattern. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

40. B — When an airway is obstructed, trapped alveolar gas is absorbed into blood and the affected air spaces collapse. This is resorption atelectasis. Pleural fluid causes compression, fibrosis causes cicatrization, and congenital absence of alveoli is not an acquired postoperative mechanism. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

41. A — Sustained high work of breathing can fatigue respiratory muscles. Tidal volume and effective alveolar ventilation then fall, causing carbon dioxide retention and worsening respiratory failure. Increased ventilation would lower carbon dioxide, and renal bicarbonate loss does not initiate this ventilatory decline. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

42. C — Emphysema destroys alveolar septa and elastic fibers, reducing elastic recoil and allowing air trapping. Hyperinflation flattens the diaphragm and increases total lung capacity. Excess surfactant, pleural fluid, and total bronchial closure do not explain the obstructive mechanics. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

43. B — A Pancoast tumor at the lung apex can invade the lower brachial plexus, causing shoulder and hand symptoms, and the cervical sympathetic chain, producing Horner syndrome with ptosis and miosis. The phrenic, vagus, and optic nerves do not explain the combined pattern. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

44. D — Inflammatory exudates contain increased protein and cells because vascular permeability rises at the affected site. Transudates result from systemic hydrostatic or oncotic changes and are low in protein. Chyle and normal ultrafiltrate do not explain purulent pleural inflammation. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

45. B — Sarcoidosis is a systemic inflammatory disorder characterized by noncaseating granulomas in affected organs, commonly the lungs and lymph nodes. Bacterial pneumonia produces neutrophilic exudate, heart failure causes hydrostatic edema, and bronchospasm does not form granulomas. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

46. D — Asbestos exposure is associated with pleural plaques, interstitial fibrosis, and malignant mesothelioma. A slowly progressive pleural process fits this exposure history. Viral bronchiolitis, allergic rhinitis, and pulmonary artery spasm do not produce diffuse pleural thickening after occupational exposure. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

47. A — Minimal-change disease causes nephrotic syndrome through podocyte foot-process effacement with little visible light-microscope change. Focal segmental glomerulosclerosis produces segmental scars, membranous disease produces immune deposits and basement-membrane thickening, and obstruction does not directly injure podocytes. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

48. C — Focal segmental glomerulosclerosis affects only portions of some glomeruli and commonly produces substantial proteinuria. Obesity, HIV, and adaptive hyperfiltration can contribute. Minimal-change disease lacks segmental scarring, while obstruction and interstitial nephritis affect different renal compartments. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors. This clarifies the mechanism.

49. D — Membranous nephropathy causes immune-complex deposition along the outer glomerular basement membrane, producing diffuse capillary-wall thickening and proteinuria. IgA nephropathy primarily affects the mesangium, tubular necrosis affects epithelial cells, and analgesic injury can cause papillary necrosis. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

50. B — Rapidly progressive glomerulonephritis causes a rapid decline in filtration, hematuria, and crescent formation from severe glomerular injury. Nephrotic syndrome emphasizes protein loss, cystitis affects the bladder, and mild thirst does not create crescentic glomerular damage. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

51. C — IgA nephropathy commonly causes episodic hematuria occurring soon after mucosal infection because IgA-containing immune complexes deposit in the mesangium. Membranous and minimal-change disease usually present with nephrotic protein loss, while pyelonephritis causes tubulointerstitial infection and fever. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

52. A — Renal papillary necrosis reflects ischemic injury to the renal papillae. Diabetes, analgesic nephropathy, sickle cell disease, and severe pyelonephritis can contribute. Podocyte injury causes proteinuria, cystitis affects the bladder, and cortical hypertrophy does not produce papillary sloughing. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

53. D — Urease-producing bacteria split urea into ammonia, alkalinizing urine and promoting magnesium-ammonium-phosphate, or struvite, stones. Cystine stones reflect amino-acid transport defects, uric acid stones favor acidic urine, and cholesterol stones form in the gallbladder. The distinction matters because assessment findings follow the disease process rather than one isolated symptom. That explains the clinical pattern.

54. A — Medullary sponge kidney involves cystic dilation of collecting ducts in the renal medulla. Urinary stasis can promote hematuria, infection, and calcium stones. Cortical necrosis, membranous glomerular disease, and renal artery stenosis do not create medullary cystic spaces. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

55. B — Severe obstetric complications, shock, and DIC can thrombose small renal vessels and cause bilateral cortical necrosis. Filtration may be permanently lost. Mild prerenal dehydration, minimal-change disease, and renal colic do not produce diffuse cortical ischemia. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

56. C — During prerenal volume depletion, intact nephrons activate RAAS and sympathetic responses, increasing sodium and water reabsorption. Urine becomes concentrated and urinary sodium falls. Glycosuria and failure to acidify urine suggest tubular dysfunction rather than an uncomplicated perfusion deficit. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

57. C — An adrenal cortisol-producing adenoma causes high cortisol with suppressed pituitary ACTH, producing ACTH-independent Cushing syndrome. Excess cortisol promotes protein catabolism, glucose elevation, and vascular sensitivity. Pituitary ACTH excess has a different source, while adrenal insufficiency and diabetes insipidus cause different hormonal patterns. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

58. D — Secondary adrenal insufficiency results from inadequate ACTH stimulation, so cortisol production falls without the high ACTH that causes hyperpigmentation in primary disease. Cortisol excess and hyperparathyroidism produce different findings and laboratory patterns. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture. These findings follow the disease process.

59. A — Graves orbitopathy involves autoimmune stimulation of orbital fibroblasts and extraocular muscles, with glycosaminoglycan deposition, edema, and tissue expansion. Bacterial invasion, loss of thyroid receptors, and reduced matrix production do not explain the characteristic eye findings. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

60. B — Hashimoto thyroiditis is an autoimmune destruction of thyroid tissue associated with thyroid peroxidase antibodies. Progressive hormone loss raises TSH and causes hypothyroid symptoms. Graves disease produces hormone excess, while pheochromocytoma and hyperaldosteronism affect different endocrine systems. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors. This clarifies the mechanism.

61. D — Placental hormones can increase insulin resistance during pregnancy so more glucose remains available to the fetus. Gestational diabetes develops when maternal beta cells cannot compensate with sufficient insulin secretion. Placental hormone absence, total beta-cell destruction, and erythropoietin do not explain this pattern. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

62. C — MEN1 commonly involves parathyroid, pituitary, and pancreatic neuroendocrine tumors. Hyperparathyroidism can cause kidney stones, while gastrin or other pancreatic tumors may cause ulcers. MEN2, thyroiditis, and congenital adrenal hyperplasia have different tumor or enzyme patterns. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

63. A — Pseudohypoparathyroidism reflects end-organ resistance to parathyroid hormone. Calcium remains low and PTH rises because the kidney and bone do not respond appropriately. Primary hyperparathyroidism raises calcium, vitamin D intoxication causes hypercalcemia, and thyroid storm is unrelated. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

64. B — Falling glucose activates sympathetic and adrenal responses first, causing sweating, tremor, palpitations, and anxiety. Continued deprivation then impairs cerebral function, producing confusion, seizures, and coma. Hyperglycemia and aldosterone do not explain this ordered progression. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

65. B — A transient ischemic attack causes temporary focal neurologic dysfunction without persistent tissue infarction. A short-lived embolus, thrombosis, or perfusion drop can produce the episode. Completed infarction leaves tissue injury, while meningitis and peripheral neuropathy create different patterns. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

66. D — The ischemic penumbra surrounds the infarct core and contains dysfunctional but potentially viable neurons. Restoring perfusion promptly may prevent progression to irreversible necrosis. Ventricular, subarachnoid, and medullary structures do not describe this zone around an ischemic core. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

67. C — Cytotoxic edema occurs when ATP depletion disables cellular ion pumps, causing sodium and water to enter neurons and glia. Vasogenic edema reflects extracellular leakage through a disrupted blood-brain barrier. Peripheral and lymphatic edema do not describe intracellular brain swelling. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

68. A — Aqueductal obstruction prevents CSF from moving between ventricular compartments, producing noncommunicating hydrocephalus. Communicating hydrocephalus results from impaired absorption after the ventricular pathways remain open. Pulmonary edema and urinary obstruction do not involve CSF flow. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

69. C — Charcot-Marie-Tooth disease is an inherited peripheral neuropathy involving demyelination or axonal degeneration. Distal weakness, sensory loss, and foot deformities develop gradually. Multiple sclerosis affects central myelin, myasthenia affects the neuromuscular junction, and Parkinson disease affects basal-ganglia motor circuits. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

70. A — Delirium is an acute, fluctuating disturbance of attention and awareness caused by illness, medications, metabolic changes, or environmental stress. Dementia usually progresses gradually, while peripheral neuropathy and cerebellar disease produce different neurologic deficits. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture. These findings follow the disease process.

71. B — Thiamine deficiency impairs carbohydrate metabolism in vulnerable brain regions and causes Wernicke encephalopathy, characterized by confusion, ataxia, and ocular abnormalities. Vitamin K, iron, and calcium abnormalities do not produce this classic neurologic triad. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern. This is the key distinction here.

72. D — Prolonged seizure activity increases glutamate signaling, calcium entry, metabolic demand, and oxygen consumption. Energy stores become depleted, producing acidosis and neuronal injury. Glutamate suppression, increased oxygen reserve, and ATP restoration would be protective rather than injurious. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors. This clarifies the mechanism.

73. A — Spinal shock is the early period after severe spinal cord injury in which reflexes and muscle tone below the lesion are temporarily depressed. Autonomic dysreflexia is a later stimulus-triggered hypertensive crisis, Brown-Sequard follows hemisection, and peripheral neuropathy is not an acute cord phase. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

74. D — The ascending reticular activating system supports arousal and consciousness through interconnected brainstem and thalamic pathways. Injury can produce impaired wakefulness or coma. The renal medulla, neuromuscular junction, and intestinal villus do not maintain cortical arousal. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

75. C — Barrett esophagus is metaplastic replacement of normal distal esophageal squamous epithelium by intestinal-type columnar epithelium after chronic reflux injury. It increases risk for dysplasia and adenocarcinoma. Necrosis, volvulus, and obstruction do not describe this epithelial adaptation. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

76. B — Infantile hypertrophic pyloric stenosis narrows the gastric outlet through thickened pyloric smooth muscle. Vomiting is typically projectile and nonbilious because the obstruction is proximal to the bile duct entry. Esophageal, ileal, and colonic disorders do not produce this pattern. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

77. A — Chronic mesenteric ischemia occurs when arterial narrowing limits intestinal blood flow during increased demand after meals. Patients may develop food fear and weight loss. Lactose intolerance causes osmotic symptoms, achalasia affects the esophagus, and nephrotic syndrome affects glomerular permeability. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

78. B — Acute mesenteric ischemia produces severe pain from intestinal hypoperfusion, often before peritoneal signs appear. Prolonged ischemia causes mucosal breakdown, bleeding, necrosis, and infarction. Irritable bowel syndrome, hemorrhoids, and viral gastroenteritis do not cause arterial bowel infarction. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

79. C — Zollinger-Ellison syndrome results from a gastrin-secreting gastrinoma. Excess gastrin stimulates parietal cells, producing severe acid hypersecretion, recurrent ulcers, and diarrhea from impaired intestinal enzyme activity. Achalasia, cholecystitis, and celiac disease do not produce this gastrin-driven pattern. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

80. D — Hereditary hemochromatosis causes excessive intestinal iron absorption and progressive deposition in organs. Iron injury can produce skin pigmentation, diabetes, hepatic fibrosis, and cirrhosis. Wilson disease involves copper, Gilbert syndrome causes mild unconjugated hyperbilirubinemia, and pharyngitis does not cause iron overload. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

81. D — Much of chronic hepatitis B liver injury results from host immune responses against infected hepatocytes, especially cytotoxic T-cell activity. The virus does not simply replace cells with collagen, and IgE or bile-flow changes do not explain the immune-mediated hepatocyte destruction. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

82. C — Gilbert syndrome is a benign inherited reduction in bilirubin conjugation that produces intermittent mild unconjugated hyperbilirubinemia. Hemolysis, obstruction, and hepatocellular disease are absent. Cholangitis, iron overload, and copper accumulation cause different laboratory and clinical patterns. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

83. B — Microangiopathic hemolytic anemia occurs when red cells fragment as they pass through fibrin strands or damaged small vessels. Schistocytes, thrombocytopenia, organ ischemia, and elevated hemolysis markers may occur. Iron deficiency, spherocytosis, and leukopenia do not produce mechanical red-cell fragmentation. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

84. A — Thalassemia results from inherited reduction in alpha- or beta-globin production. Microcytosis can be marked despite normal iron stores, and target cells may appear. Acute blood loss, B12 deficiency, and thrombocytopenia produce different laboratory patterns. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

85. C — G6PD deficiency limits production of reduced glutathione, leaving erythrocytes vulnerable to oxidative stress. Hemoglobin precipitates form Heinz bodies, and splenic removal creates bite cells. Factor VIII, fibrinogen, and B12 do not protect red cells from oxidant injury. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

86. D — Hereditary spherocytosis results from defects in red-cell membrane proteins, producing rigid spherical cells with reduced deformability. The spleen removes them, causing extravascular hemolysis, jaundice, and splenomegaly. Thalassemia affects globin synthesis, aplasia reduces production, and DIC consumes clotting factors. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

87. A — Heparin-induced thrombocytopenia is an immune reaction to platelet factor 4-heparin complexes. IgG-mediated platelet activation causes thrombocytopenia together with paradoxical thrombosis. Hemolysis, von Willebrand disease, and excess antithrombin do not produce this timing and prothrombotic pattern. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

88. B — Osteogenesis imperfecta results from abnormal type I collagen, weakening bone and connective tissues. Fractures, blue sclerae, and hearing loss are characteristic manifestations. Osteomalacia reflects defective mineralization, osteoarthritis affects articular cartilage, and osteomyelitis is infectious bone inflammation. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors. This clarifies the mechanism.

89. D — Ehlers-Danlos syndromes involve abnormalities of collagen structure, synthesis, or processing. Tissues become unusually extensible and fragile, causing joint hypermobility, skin findings, and bruising. Osteoclast activity, surfactant, and platelet production do not explain the systemic connective-tissue weakness. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

90. B — Osteosarcoma is a malignant bone-forming tumor that produces osteoid, often arising in the metaphysis of long bones during adolescence. Chondromalacia affects cartilage, osteoporosis reduces bone mass, and a simple cyst does not produce malignant osteoid. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

91. A — Basal cell carcinoma commonly appears as a pearly or ulcerated lesion on sun-exposed skin. It is locally invasive but usually has low metastatic potential. Melanoma, osteosarcoma, and Kaposi sarcoma have different cell origins and metastatic behaviors. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

92. C — Erysipelas is a superficial bacterial infection involving the upper dermis and lymphatic vessels. It produces a raised, sharply demarcated erythematous plaque with systemic symptoms. Cellulitis spreads more diffusely through deeper tissue, while keloids and urticaria are not acute bacterial infections. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

93. B — Disseminated intravascular coagulation generates thrombin throughout the circulation, producing microvascular clots while consuming platelets and clotting factors. Fibrinolysis then worsens bleeding. Hemophilia, iron deficiency, and localized allergy do not create simultaneous thrombosis, factor consumption, and fragmented erythrocytes. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

94. A — In sickle cell disease, deoxygenated hemoglobin S polymerizes, distorting red cells and obstructing small vessels. Dehydration and hypoxia promote vaso-occlusion, ischemia, and pain. Spherocytosis reflects membrane defects, while neutropenia and folate deficiency do not cause hemoglobin polymerization. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

95. D — Myasthenia gravis commonly involves antibodies against postsynaptic nicotinic acetylcholine receptors, reducing the safety margin for neuromuscular transmission. Weakness worsens with repeated use. Dopamine pathways, spinal myelin, and renal calcium channels do not explain fatigable ocular weakness. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

96. C — The cerebellum coordinates movement, balance, and error correction. A cerebellar hemisphere lesion produces ipsilateral limb dysmetria, intention tremor, and gait ataxia. The substantia nigra, pituitary, and glomerulus do not organize the timing and precision of voluntary movement. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.

97. C — Ulcerative colitis produces continuous mucosal inflammation beginning in the rectum and extending proximally through the colon. Ulceration causes bloody diarrhea and urgency. Crohn disease is typically patchy and transmural, while appendicitis and irritable bowel syndrome do not create this continuous colitis pattern. The distinction matters because assessment findings follow the disease process rather than one isolated symptom.

98. D — Crohn disease can affect discontinuous segments of the gastrointestinal tract and involves the bowel wall transmurally. Deep inflammation promotes strictures, abscesses, and fistulas. Ulcerative colitis is usually continuous and mucosal, while pharyngitis and reflux do not create transmural intestinal disease. Recognizing this sequence helps the nurse connect the abnormal finding with the patient's overall clinical picture.

99. A — Cystic-duct obstruction causes gallbladder distention, chemical irritation, ischemia, and secondary inflammation in acute cholecystitis. The location and mechanism distinguish it from appendicitis, pancreatic enzyme deficiency, and urinary stones. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern. The other choices describe different mechanisms, timing, or compensatory responses and do not fit the pattern.

100. B — Celiac disease is an immune-mediated response to gluten that damages small-intestinal villi and reduces absorptive surface area. Malabsorption can cause diarrhea, weight loss, and nutrient deficiencies. Biliary obstruction, isolated parietal-cell loss, and increased colonic absorption do not explain villous atrophy. This mechanism explains the patient's findings and distinguishes the condition from nearby distractors.