HESI Pathophysiology — Practice Study Guide (Sampler)

Inflammation & immunity • Acid–base • Shock & sepsis • Cardiac & respiratory disorders • Endocrine & renal • Neuro & heme • Fluid/electrolytes • Oncology basics • MCQs with explanations

- - 20 scenario-style prompts with concise model answers
- - 20 multiple-choice questions with step-by-step rationales
- - Covers: innate/adaptive immunity, hypersensitivity I–IV, inflammation mediators, wound healing, ABGs, shock stages, HF patho, COPD/asthma, DKA vs HHS, SIADH vs DI, AKI vs CKD, stroke/ICP, anemia types, coagulation, electrolyte crises, oncologic emergencies, sepsis bundles
- - One-time purchase • Lifetime updates • Instant download

Disclaimer: Original practice material aligned to common HESI pathophysiology domains. Always correlate with current textbooks/protocols.

Part I — Scenario-Style Drills & Model Answers (20)

Essay 1: Acid–Base: COPD Exacerbation

ABG: pH 7.31, $PaCO_2$ 60, HCO_3^- 29. Interpret and priority action.

Model answer: Partially compensated respiratory acidosis; improve ventilation (bronchodilators, treat trigger, NIV/vent changes) while maintaining oxygenation.

Essay 2: DKA vs HHS

Type 1 DM with Kussmaul respirations and fruity breath. Contrast with HHS.

Model answer: DKA: absolute insulin deficiency → ketogenesis and AG metabolic acidosis; HHS: relative deficiency → hyperosmolar state with minimal ketosis.

Essay 3: Shock: Early Sepsis

Fever, tachycardia, MAP 58, lactate 4.2.

Model answer: Distributive shock with vasodilation/capillary leak. Priorities: cultures, antibiotics in 1 hr, 30 mL/kg fluids if hypoperfused, norepinephrine for MAP ≥65.

Essay 4: Heart Failure Patho

Why LV systolic failure causes pulmonary edema.

Model answer: ↑ LVEDP → ↑ LA pressure → pulmonary venous congestion and alveolar fluid; RAAS activation worsens retention.

Essay 5: Asthma vs COPD

Key differences.

Model answer: Asthma: reversible hyperresponsiveness, eosinophils, mucus; COPD: progressive airflow limitation, neutrophils, emphysema/chronic bronchitis.

Essay 6: SIADH vs DI

Post-op Na^+ 122, low serum Osm.

Model answer: SIADH: excess ADH → dilutional hyponatremia with high urine Osm/Na; DI shows polyuria and hypernatremia.

Essay 7: AKI Types

BUN/Cr 35:1, FeNa <1%, dehydration.

Model answer: Prerenal azotemia with intact tubules conserving Na^+/water; intrinsic ATN shows FeNa >2% with muddy casts.

Essay 8: Stroke: Ischemic Cascade

Events after MCA occlusion.

Model answer: Energy failure → Na^+/K^+ pump failure → cytotoxic edema, glutamate excitotoxicity, Ca^{2+} influx, free radicals, apoptosis; penumbra salvageable with reperfusion.

Essay 9: Anemia Types

MCV 72 fL. Mechanism.

Model answer: Microcytic anemia from impaired Hb synthesis (iron deficiency, thalassemia, chronic disease).

Essay 10: Hyperkalemia

K^+ 6.7 with peaked T waves. Why?

Model answer: High extracellular K^+ reduces resting membrane potential → peaked T, then widened QRS and arrest if severe.

Essay 11: Type I Hypersensitivity

Anaphylaxis after peanut exposure.

Model answer: IgE-mediated mast cell degranulation causing vasodilation/bronchospasm; treat with IM epinephrine first.

Essay 12: Autoimmunity

Molecular mimicry concept.

Model answer: Cross-reactive lymphocytes target self after pathogen exposure; failure of tolerance perpetuates damage.

Essay 13: Coagulopathy in Liver Failure

Bleeding tendency patho.

Model answer: Reduced synthesis of clotting factors and platelets; vitamin K issues in cholestasis add to coagulopathy.

Essay 14: Oncologic Emergency: TLS

After chemo: hyperkalemia, hyperuricemia, AKI.

Model answer: Cell lysis releases K^+, phosphate, nucleic acids → uric acid and electrolyte derangements; fluids and rasburicase/allopurinol.

Essay 15: ARDS

Refractory hypoxemia with bilateral infiltrates.

Model answer: Diffuse alveolar damage, loss of surfactant, low compliance, shunt/VQ mismatch; cytokine-driven injury.

Essay 16: Cirrhosis & Ascites

Mechanism of ascites.

Model answer: Portal HTN + hypoalbuminemia + RAAS/NO changes → Na^+/water retention and third spacing.

Essay 17: Thyroid States

Hypo vs hyper metabolism.

Model answer: Hypo: ↓ BMR, bradycardia, cold intolerance. Hyper: ↑ BMR, tachycardia, heat intolerance, catabolism.

Essay 18: Multiple Sclerosis

Why symptoms fluctuate.

Model answer: Demyelination slows conduction; remyelination/compensation partial; heat exacerbates block (Uhthoff).

Essay 19: Pancreatitis

Patho of organ damage.

Model answer: Premature trypsin activation → autodigestion, fat necrosis, cytokines; third spacing → hypovolemia/SIRS.

Essay 20: Sepsis-Induced Coagulopathy

High D-dimer, low platelets.

Model answer: Inflammation activates coagulation → microthrombi consume platelets/fibrinogen; fibrinolysis raises D-dimer.

Part II — Multiple-Choice Questions with Explanations (20)

MCQ 1. Primary mediator of Type I hypersensitivity is:

A) IgG
B) IgE
C) IgM
D) TGF■β

Explanation: Type I reactions (anaphylaxis) are IgE-mediated.

MCQ 2. Which ABG suggests metabolic acidosis with respiratory compensation?

A) pH 7.52, PaCO■ 28, HCO■■ 23
B) pH 7.28, PaCO■ 30, HCO■■ 14
C) pH 7.45, PaCO■ 40, HCO■■ 26
D) pH 7.34, PaCO■ 55, HCO■■ 29

Explanation: Low pH with low HCO_3^- and low $PaCO_2$ indicates compensation.

MCQ 3. Hallmark of ARDS on pathophysiology is:

A) Cardiogenic edema
B) Hyaline membranes and diffuse alveolar damage
C) Bronchial hyperplasia
D) Pulmonary embolus

Explanation: Diffuse alveolar damage with hyaline membranes defines ARDS.

MCQ 4. DKA typically presents with:

A) Severe hyperosmolarity without ketosis
B) Kussmaul respirations and ketonemia
C) Bradycardia and alkalosis
D) Hypoglycemia

Explanation: DKA causes metabolic acidosis with compensatory hyperventilation.

MCQ 5. Electrolyte change that risks torsades by prolonging QT is:

A) Hyperkalemia
B) Hypocalcemia

C) Hypernatremia

D) Hypomagnesemia

Explanation: Hypomagnesemia (and often hypocalcemia) prolongs QT; Mg is key in torsades.

MCQ 6. SIADH lab pattern includes:

A) Hypernatremia, low urine osmolality

B) Hyponatremia, high urine osmolality

C) Hyperkalemia, metabolic acidosis

D) Hypocalcemia, low phosphate

Explanation: Water retention dilutes Na^+; urine remains concentrated.

MCQ 7. Prerenal AKI typically shows:

A) FeNa >2%

B) Muddy brown casts

C) FeNa <1% and high BUN/Cr

D) RBC casts

Explanation: Kidneys conserve Na^+/water → FeNa <1%.

MCQ 8. Earliest event in atherosclerosis is:

A) Plaque rupture

B) Foam cell formation from LDL oxidation

C) Complete occlusion

D) Calcification only

Explanation: LDL oxidation and macrophage uptake create foam cells/fatty streaks.

MCQ 9. Which anemia is macrocytic?

A) Iron deficiency

B) Thalassemia

C) B12 deficiency

D) Anemia of chronic disease

Explanation: B12/folate deficiency → macrocytosis (↑MCV).

MCQ 10. Mechanism of beta-blockers post-MI benefit:

A) Increase myocardial O■ demand

B) Reduce heart rate and contractility

C) Increase arrhythmias

D) Increase renin release

Explanation: They reduce O_2 demand and arrhythmic risk.

MCQ 11. Patho of emphysema is:

A) Bronchial smooth muscle hyperreactivity only

B) Destruction of alveolar walls and loss of elastic recoil

C) Pulmonary fibrosis exclusively

D) Large airway edema only

Explanation: Protease-antiprotease imbalance damages alveoli.

MCQ 12. Raised ICP classic response is:

A) Bradycardia, hypertension, irregular respirations

B) Hypotension only

C) Tachycardia only

D) Bradycardia only

Explanation: Cushing triad indicates raised ICP.

MCQ 13. Spider angiomas in cirrhosis result from:

A) Hypoestrogenemia

B) Hyperestrogenemia due to impaired metabolism

C) Vitamin C deficiency

D) Hyperandrogenism

Explanation: Impaired hepatic estrogen clearance leads to vascular changes.

MCQ 14. Tumor lysis syndrome typically causes:

A) Hypokalemia and hypouricemia

B) Hyperkalemia and hyperuricemia

C) Hypernatremia only

D) Hypophosphatemia

Explanation: Cell lysis releases K^+ and nucleic acids → uric acid.

MCQ 15. Septic shock vascular change is:

A) Peripheral vasoconstriction and low CO

B) Vasodilation with low SVR and maldistribution

C) No change in SVR

D) Primary hypovolemia only

Explanation: Cytokine-mediated vasodilation lowers SVR.

MCQ 16. Graves disease mechanism is:

A) TSH■secreting pituitary tumor

B) TSH receptor–stimulating antibodies

C) Iodine deficiency

D) Thyroiditis only

Explanation: TSI antibodies stimulate the TSH receptor → hyperthyroidism.

MCQ 17. MS symptoms worsen with heat because:

A) Improved conduction velocity

B) Saltatory conduction restored

C) Conduction block in demyelinated axons increases

D) Immune suppression

Explanation: Heat further impairs conduction in demyelinated fibers.

MCQ 18. Hyperkalemia ECG hallmark is:

A) U waves

B) Peaked T waves

C) Delta waves

D) Osborn waves

Explanation: Tall peaked T waves are classic early change.

MCQ 19. ARDS ventilation strategy includes:

A) High tidal volume to normalize CO■
B) Low tidal volume with plateau <30
C) Zero PEEP
D) Prolonged hyperventilation

Explanation: Lung-protective ventilation is standard.

MCQ 20. Which hypersensitivity is T-cell mediated (delayed)?

A) Type I
B) Type II
C) Type III
D) Type IV

Explanation: Type IV involves T-cell–mediated delayed response (e.g., contact dermatitis).

About This Study Guide

This sampler mirrors the full HESI Pathophysiology pack: scenario drills and MCQs with clear rationales across inflammation/immunity, acid–base, shock, major organ systems, electrolytes, and oncologic emergencies. Use it to self-check decision-making before exams.