The Right Questions for the Right Answers

Pathophysiology

THIRD EDITION

- 500 USMLE-type questions, answers, and explanations
- Rationales for correct and incorrect answers
- High-yield facts reinforce key concepts
- Student-tested and reviewed

Maurice A. Mufson

Pathophysiology

PreTest™ Self-Assessment and Review

Third Edition

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Notice

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Introduction

In this Third Edition of *Pathophysiology PreTest™ Self-Assessment and Review,* many questions are new and more of them begin with a brief clinical vignette to frame the question and place the teaching point in a clinical context. The chapter on "High-Yield Facts" has been revised and expanded and represents a synopsis of significant points in the format of condensed summaries. These "High-Yield Facts" highlight key points in pathophysiology for rapid review. They provide a "memory jog" when reviewing the questions, and it is important to read the source reference citations accompanying them.

Testing your knowledge by answering the pathophysiology questions in this book serves as a competition in which you compete against yourself for the satisfaction of doing well. It's a great feeling when you know the answers to difficult medical questions. It reflects well on your ability to learn the material in medical school, and it's a signal that you're prepared for the certifying examinations. This competition also can increase your knowledge base, as any competition sharpens your skills. That is an important part of testing ourselves. When we don't know an answer, it's an opportunity to look it up in a "big book" of internal medicine and improve our understanding of the topic. Each answer includes a reference to that answer, as a starting point for reading more about the topic. Although knowing the answer to any individual question provides some measure of satisfaction, it does not, and should not, represent a stopping point. Importantly, it should encourage you to read further so that you can easily answer questions from any point of view on that topic.

Consider using this book in the following manner:

- Read the question and then record your answer before you read the correct answer.
- Look at the correct answer and the explanation.
- Read the source reference citation.

The process of studying remains paramount, not necessarily whether you know the correct answer to one question or many questions. Don't fail to consult the source reference citation listed for each question, especially the questions for which you do not readily know the answer. In this manner, you will increase the depth and breadth of your knowledge, which after all is the goal of testing yourself on these questions.

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High-Yield Facts

1. Many diseases have an immunologic basis. Example: Graft-versus-host disease (GVHD) can develop in an immunosuppressed individual who receives immunocompetent donor cells. The donor cells respond to histocompatibility antigens present on the recipient's cells that are NOT found on the donor cells. Bone marrow contains immunocompetent T cells. *(Levinson, 7/e, p 798. Braunwald, 15/e, pp 741–742.)*
2. The normal fetus and newborn produce only the IgM class of immunoglobulins in response to an antigen; any IgG that is present likely crossed the placenta as it is the only immunoglobulin that does so. *(McPhee, 4/e, p 392.)*
3. Persons with the autosomal dominant gene of type I osteogenesis imperfecta characteristically have blue scleras. *(McPhee, 4/e, p 7. Braunwald, 15/e, pp 388–389.)*
4. The most common cause of trisomy 21 is maternal nondisjunction. *(McPhee, 4/e, pp 23–28.)*
5. The following chart compares bacterial meningitis and viral meningitis. *(McPhee, 4/e, pp 71–74.)*

	Bacterial Meningitis	**Viral Meningitis**
Disease state	Acute: significant mortality without antibiotic therapy	Acute: usually self-limited
Symptoms	Fever	Fever
	Meningismus	Meningismus
	Mental status changes	Mental status changes
Physical exam findings	Photophobia	Photophobia
	Nausea	Nausea
	Vomiting	Vomiting
	Fever	Fever
	Kernig's sign—positive	Kernig's sign—positive
	Brudzinski's sign—positive	Brudzinski's sign—positive
Etiology	Neonates	Coxsackie A and B viruses
	Escherichia coli	Poliovirus
	Group B streptococcus	Mumps virus
	Listeria monocytogenes	Epstein-Barr virus

(Continued)

	Bacterial Meningitis	Viral Meningitis
	Children	Adenovirus
	Neisseria meningitidis	Cytomegalovirus
	Streptococcus pneumoniae	
	Haemophilus influenzae, nonimmunized	
	Adults (>18 years old)	
	N. meningitidis, especially among freshman and sophomore college students who reside in the dorms	Enteroviruses
		Arboviruses
		HIV
		Herpesvirus Simplex
	S. pneumoniae	LCMV
	L. monocytogenes	
	Gram-negative bacilli	
Cerebrospinal fluid results	Decreased glucose	Normal glucose
	Increased protein	Slightly increased protein
	Increased neutrophils	Increased monocytes
	Increased pressure	Normal or slightly increased pressure
	Gram stain shows bacteria	Gram stain shows no bacteria
Treatment	IV antibiotics	Supportive therapy
	Supportive therapy	
Complications	Cerebral edema	Deafness
	Deafness	Weakness
	Death	

6. Carcinomas undergo phenotypic transition from **normal → hyperplasia → carcinoma in situ → invasive carcinoma → metastasis.** Carcinomas occur as a result of a constellation of physiologic and genetic changes (e.g., APC, hMLH1, and hMSH2—colon carcinoma/BRCA1 and BRCA2—breast carcinoma). *(McPhee, 4/e, pp 96–101.)*
7. Colon carcinoma begins when cell cycle regulation loses control over growth, and a collection of rapidly multiplying cells (hyperplasia) form an adenoma. The adenoma can continue to develop into carcinoma in situ. The first evidence of disease may be occult rectal bleeding indicating the appearance of new friable vessels supplying the tumor. Next,

the cancer cells invade the basement membrane of the colon (invasive carcinoma), gaining access to the body's transport systems (lymphatic and hematogenous). Metastasis to lymph nodes and distant body regions can occur. *(McPhee, 4/e, pp 99–100.)*

8. Many malignancies have characteristic indirect systemic effects via multiple mechanisms. Carcinoid syndromes produce serotonin or prostaglandins that can cause flushing, restrictive lung symptoms, ascites, and hypotension. Excess adrenocorticotropic hormone (ACTH) production in small-cell bronchogenic carcinoma results in a Cushing-like syndrome, and excess antidiuretic hormone (ADH) production in these malignancies results in a syndrome of inappropriate antidiuretic hormone secretion (SIADH). Malignancies (e.g., squamous cell carcinoma) can produce peptides related to PTH, causing hypercalcemia. *(McPhee, 4/e, pp 101–103. Braunwald, 15/e, p 2091.)*
9. Pernicious anemia occurs when antibodies to intrinsic factor and parietal cells attack the gastric mucosa, causing gastric atrophy. The disruption of the normal function of the gastric mucosa affects vitamin B_{12} absorption on two levels: stomach acid deficiency (achlorhydria) prevents the release of vitamin B_{12} from food digestion, and intrinsic factor is necessary for vitamin B_{12} absorption in the terminal ileum. The chronic loss of vitamin B_{12} results in abnormal RBC maturation without changes in hemoglobin synthesis leading to macrocytic anemia. The Schilling test is abnormal and confirms the diagnosis. Patients with pernicious anemia require treatment with cobalamin, which reverses the anemia and the neurologic abnormalities. Folate treatment alone fails to reverse neurologic abnormaties of cobalamin deficiency. *(McPhee, 4/e, pp 128–132; Braunwald, 15/e, pp 677–680.)*
10. Pathophysiology of hearing loss *(McPhee, 4/e, pp 166–167.)*

Type of Hearing Loss	Etiology	Testing
Conductive deafness	Disruption of conduction and amplification of sound from the external auditory canal to the inner ear	Negative Rinne test Weber test: heard best in the affected ear Audiometry

(Continued)

Type of Hearing Loss	Etiology	Testing
Sensorineural	Impaired function of inner ear or cranial nerve VIII	Positive Rinne test Weber test: heard best in the unaffected ear Audiometry
Central deafness	Damaged CNS auditory pathways	Audiometry

11. Myasthenia gravis is an autoimmune disease characterized by antibodies to acetylcholine receptors, causing a deficiency in the number of acetylcholine receptors on the postsynaptic (muscle) terminal, resulting in reduced efficiency of neuromuscular activity. The disease commonly presents in small muscle groups, accompanied by intermittent fatigue and weakness relieved by rest. *(McPhee, 4/e, pp 174–175.)*
12. Psoriasis is an inflammatory parakeratotic accumulation of skin cells that features erythematous, demarcated lesions with scaly patches commonly found on scalp, extensor surfaces of extremities, and fingernails. The lesions appear pink/silver described as "salmon-like." *(McPhee, 4/e, pp 193–196.)*
13. Asthma is an obstructive pulmonary disease characterized by airway narrowing as a result of smooth muscle spasms, inflammation, edema, and thick mucus production. The lung function tests (LFTs) show an obstructive picture with a decreased FEV_1/FVC ($FEV_1\%$). The pathophysiologic response is mediated by local cellular injury, lymphocyte activation (antigen exposure, B cell activation, and cytokine activity), IgE-mediated mast cell (producing histamine, leukotrienes, and platelet-activating factor), and eosinophil activation. *(McPhee, 4/e, pp 236–240.)*
14. Pulmonary function tests: obstructive lung disease vs. restrictive lung disease. *(McPhee, 4/e, pp 240–246.)*

Pulmonary Function Test	Obstructive Lung Disease (e.g., Chronic Obstructive Pulmonary Disease)	Restrictive Lung Disease (e.g., Pulmonary Fibrosis)
FVC	↓	↓
FEV_1	↓	↓
FEV_1% (FEV_1/FVC)	↓	Normal/↑
TLC	↑	↓
RV	↑	Normal/↓

15. Pulmonary embolism occurs when a venous thrombi (usually from a deep vein thrombosis) lodges in the pulmonary circulation. The pathophysiology includes hemodynamic changes, increased alveolar dead space with increased ventilation/perfusion ratios, and decreased oxygen perfusion to body tissues. Common acute presentations include tachypnea, hemoptysis, tachycardia, fever, cough, and pleuritic pain. Pulmonary embolism occurs in persons postoperatively and in persons who must sit for long periods, such as airplane travelers. *(McPhee, 4/e, pp 251–257.)*

16. In normal individuals, as left ventricular end-diastolic pressure or preload increases, stroke volume will increase proportionately. In patients who suffer heart failure, increased left ventricular end-diastolic pressure is not met with increased stroke volume, because the contractility is depressed and is unable to function; thus, the patient ultimately experiences heart failure. Frank-Starling curves or ventricular function curves are diagrams that show the relationship between stroke volume or cardiac output and preload or left ventricular end-diastolic volume. *(Braunwald, 15/e, pp 1313–1318.)*

17. Stable angina is caused by a fixed partial atherosclerotic plaque in one or more of coronary arteries that involves a 75% blockage. When at rest, blood flow is able to provide adequate oxygenation to the heart muscle. On exertion, oxygen demand increases. The partial occlusion prevents adequate oxygenation to the heart, resulting in chest discomfort that is relieved by rest. Unstable angina is caused by thrombus formation on a fissuring atherosclerotic plaque, which transiently prevents adequate oxygenation to the heart. The resulting ischemia causes chest discomfort whether at rest or during exertion. *(McPhee, 4/e, pp 292–294.)*