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Maurice A. Mufson

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Pathophysiology

PreTest® Self-Assessment and Review

Second Edition

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Notice

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Introduction

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.

Also, read the "High-Yield Facts," a synopsis of significant points presented as condensed summaries. These "High-Yield Facts" highlight key points in pathophysiology for rapid review. They serve also as a "memory jog" when reviewing the questions, and it is important to read the source reference citations accompanying them.

The process of studying remains paramount, not necessarily whether you know the correct answer to one question or many questions. Don't fail to read 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 in Pathophysiology

1. Many diseases have an immunologic basis. Example: **Graft versus host (GVH) disease** 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. *(Murray, 5/e, p 123.)*
2. The following chart compares **bacterial meningitis** and **viral meningitis.** *(McPhee, 2/e, pp 61–63.)*

	Bacterial Meningitis	Viral Meningitis
Disease state	Acute: significant mortality without antibiotic therapy	Acute: usually self-limited
Symptoms	Fever	Fever
	Worst headache of life	Worst headache of life
	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
	Brudinski's sign—positive	Brudinski's sign—positive
Etiology	Neonates	Cocksackie A and B viruses
	Escherichia coli	Poliovirus
	Group B *Streptococcus*	Mumps virus
	Listeria monocytogenes	Epstein-Barr virus
	Children	Adenovirus
	Neisseria meningitidis	Cytomegalovirus
	Streptococcus pneumoniae	
	Haeophilus influenzae, nonimmunized	

	Bacterial Meningitis	Viral Meningitis
Etiology (*cont'd*)	Adults (more than 18 years old) *N. meningitidis* *S. pneumoniae* *L. monocytogenes* Gram-negative bacilli	
Cerebrospinal fluid results	Decreased glucose Increased protein Increased neutrophils Increased pressure	Normal glucose Slightly increased protein Increased monocytes Normal or slightlly increased pressure Gram stain shows no bacteria
Treatment	IV antibiotics Supportive therapy	Supportive therapy
Complications	Cerebral edema Deafness Death	Deafness Weakness

3. 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, 2/e, pp 83–84.)*
4. **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, 2/e, pp 85–87.)*
5. Many malignancies have characteristic indirect systemic effects via multiple mechanisms. In lung malignancies, excess adrenocorticotropic hormone (ACTH) production results in a Cushing-like syn-

drome and excess antidiuretic hormone (ADH) production results in a syndrome of inappropriate antidiuretic hormone secretion (SIADH). Malignancies (e.g., squamous cell carcinoma) can produce peptides related to PTH, causing hypercalcemia. Carcinoid syndromes produce serotonin or prostaglandins that can cause flushing, restrictive lung symptoms, ascites, and hypotension. *(McPhee, 2/e, p 96.)*

6. **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. *(Fauci, 14/e, pp 655–656; McPhee, 2/e, p 111.)*
7. Pathophysiology of hearing loss *(McPhee, 2/e, pp 145–146.)*

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
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

8. **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, 2/e, pp 152–153.)*

9. **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. *(McPhee, 2/e, pp 169–170.)*
10. **Asthma** is an obstructive pulmonary disease characterized by airway narrowing as a result of smooth muscle spasms, inflammation, edema, and thick mucus production. 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, 2/e, pp 200–201.)*
11. Pulmonary function tests: obstructive lung disease vs. restrictive lung disease.

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

12. **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. *(McPhee, 2/e, pp 214–216.)*
13. 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. *(Lilly, p 150.)*

14. **Stable angina** is caused by a fixed partial atherosclerotic plaque in one or more of coronary arteries. 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. 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, 2/e, pp 246–248.)*
15. Chronic esophageal reflux (as a result of a transient weakened lower esophageal sphincter, alcohol use, and tobacco abuse) can result in **Barrett's esophagus.** In the disease, columnar epithelium replaces normal squamous epithelium. Individuals with Barrett's esophagus have an increased risk of developing adenocarcinoma of the esophagus. *(McPhee, 2/e, p 306.)*
16. **Helicobacter pylori** is a common bacteria that infects the gastric mucosa, providing an increased propensity for peptic ulcer disease through inflammatory mechanisms. Other risk factors for peptic ulcer disease are use of a nonsteroidal anti-inflammatory drug (NSAID), family history, smoking, and Zollinger-Ellison syndrome (gastrinoma). *(McPhee, 2/e, p 307.)*
17. **Crohn's disease** is a chronic inflammatory bowel disease that affects the whole gastrointestinal tract (from mouth to anus) and is distinguished by **alternating regions** ("skip lesions") of normal bowel and full-thickness ulcerations and granuloma formation of the bowel wall. Common manifestations are bloody diarrhea, fistula, iritis, arthritis, abscess formation, and small bowel obstruction. *(McPhee, 2/e, p 315.)*
18. **Ulcerative colitis** is an inflammatory bowel disease that causes **continuous**, partial-thickness (mucosa only) ulcerations of all or part of the colon and is manifested by bloody diarrhea and abdominal pain. *(McPhee, 2/e, pp 315–316.)*
19. **Type 1 diabetes mellitus** (previously called insulin-dependent diabetes mellitus) and **type 2 diabetes mellitus** (previously called non-insulin-