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Pathology

PreTest® Self-Assessment and Review

Tenth Edition

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Pathology

PreTest® Self-Assessment and Review

Notice

Medicine is an ever-changing science. As new research and clinical experience broaden our knowledge, changes in treatment and drug therapy are required. The authors and the publisher of this work have checked with sources believed to be reliable in their efforts to provide information that is complete and generally in accord with the standards accepted at the time of publication. However, in view of the possibility of human error or changes in medical sciences, neither the authors nor the publisher nor any other party who has been involved in the preparation or publication of this work warrants that the information contained herein is in every respect accurate or complete, and they disclaim all responsibility for any errors or omissions or for the results obtained from use of the information contained in this work. Readers are encouraged to confirm the information contained herein with other sources. For example and in particular, readers are advised to check the product information sheet included in the package of each drug they plan to administer to be certain that the information contained in this work is accurate and that changes have not been made in the recommended dose or in the contraindications for administration. This recommendation is of particular importance in connection with new or infrequently used drugs.

Preface

The study of pathology, a science so basic to clinical medicine, has been abbreviated sadly in many medical schools in recent years, and at a time when explosive growth is occurring in the science. Recent advances in immunopathology, diagnosis of bacterial and viral diseases including AIDS, and detection of infectious agents such as papillomavirus in cervical dysplasia are proceeding at a tremendous rate. The tenth edition of *Pathology: PreTest® Self-Assessment and Review* includes such new subject areas as predictive values in the interpretation of laboratory data, the importance of cytokines, the molecular basis of genetic and other disease processes, and molecular biology techniques as these apply to lymphoproliferative disorders and other tumors.

The medical student must feel submerged at times in the flood of information—occasionally instructors may have similar feelings. This edition is not intended to cover all new knowledge in addition to including older anatomic and clinical pathology. It is, rather, a serious attempt to present important facts about many disease processes in hopes that the student will read much further in major textbooks and journals and will receive some assistance in passing medical school, licensure, or board examinations.

Introduction

Each *PreTest® Self-Assessment and Review* allows medical students to comprehensively and conveniently assess and review their knowledge of a particular basic science, in this instance pathology. The 500 questions parallel the format and degree of difficulty of the questions found in the United States Medical Licensing Examination (USMLE) Step 1. Practicing physicians who want to hone their skills before USMLE Step 3 or recertification may find this to be a good beginning in their review process.

Each question is accompanied by an answer, a paragraph explanation, and a specific page reference to an appropriate textbook or journal article. A bibliography listing sources can be found following the last chapter of this text.

An effective way to use this PreTest® is to allow yourself one minute to answer each question in a given chapter. As you proceed, indicate your answer beside each question. By following this suggestion, you approximate the time limits imposed by the Step 1 exam.

After you finish going through the questions in the section, spend as much time as you need verifying your answers and carefully reading the explanations provided. Pay special attention to the explanations for the questions you answered incorrectly—but read *every* explanation. The author of this material has designed the explanations to reinforce and supplement the information tested by the questions. If you feel you need further information about the material covered, consult and study the references indicated.

The High-Yield Facts added for this edition are provided to facilitate rapid review of pathology topics. It is anticipated that the reader will use the High-Yield Facts as a "memory jog" before proceeding through the questions.

Laboratory Values

Substance	Source	Normal
Albumin	Serum	3.2–4.5 g/dL
Alkaline phosphatase	Serum	20–130 IU/L
Bicarbonate	Plasma	21–28 mM
Bilirubin, direct (conjugated)	Serum	<0.3 mg/dL
Bilirubin, indirect (unconjugated)	Serum	0.1–1.0 mg/dL
Bilirubin, total	Serum	0.1–1.2 mg/dL
BUN	Serum	8–23 mg/dL
Calcium	Serum	9.2–11.0 mg/dL (4.6–5.5 meq/L)
Chloride	Serum	95–103 meq/L
Cholesterol	Serum	150–250 mg/dL
Creatinine	Serum	0.6–1.2 mg/dL
GGT (γ-glutamyltransferase)	Serum	5–40 IU/L
Glucose (fasting)	Serum	70–110 mg/dL
Insulin	Plasma	4–24 μIU/mL
Iron	Serum	60–150 μg/dL
Iron saturation	Serum	20–55%
Osmolality	Serum	280–295 mosm/L
Phosphorus	Serum	2.3–4.7 mg/dL
Potassium	Plasma	3.8–5.0 meq/L
Protein	Serum	6.0–7.8 g/dL
Sodium	Plasma	136–142 meq/L
T_3 resin uptake	Serum	25–38 relative % uptake
Thyrotropin (TSH)	Serum	0.5–5 μIU/mL
Thyroxine, free (FT_4)	Serum	0.9–2.3 ng/dL
Thyroxine, total (T_4)	Serum	5.5–12.5 μg/dL
Triiodothyronine (T_3)	Serum	80–200 mg/dL
Hematology		
Platelet count		150,000–450,000/μL
White cell count		4,440–11,000/μL
Lymphocyte count		1,000–4,800/μL (about 34%)
Mean corpuscular volume (MCV)		80–96 μm^3
Mean corpuscular hemoglobin (MCH)		27.5–33.2 pg
Mean corpuscular hemoglobin concentration (MCHC)		33.4–35.5%
Hemoglobin	Whole blood	Female 12–16 g/dL Male 13.5–18 g/dL

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High-Yield Facts in Pathology

1. CELL INJURY

Reversible Cell Injury

- swelling of cell organelles and entire cell
- dissociation of ribosomes from endoplasmic reticulum
- decreased energy production by mitochondria
- increased glycolysis → decreased pH → nuclear chromatin clumping

Irreversible Cell Injury

- dense bodies within mitochondria (flocculent densities in heart)
- release of cellular enzymes (e.g., SGOT, LDH, and CPK after MI)
- nuclear degeneration (pyknosis, karyolysis, karyorrhexis)
- cell death

2. FATTY CHANGE OF THE LIVER

Mechanisms

1. Increased delivery of free fatty acids to liver
   - starvation
   - corticosteroids
   - diabetes mellitus
2. Increased formation of triglycerides
   - alcohol (note: NADH > NAD)
3. Decreased formation of apoproteins
   - carbon tetrachloride
   - protein malnutrition (kwashiorkor)

3. CELL DEATH

Apoptosis

- "programmed" cell death
- single cells (not large groups of cells)
- cells shrink → form apoptotic bodies
- gene activation → forms endonucleases
- peripheral condensation of chromatin with DNA ladder
- no inflammatory response

Examples of apoptosis:

1. Physiologic
   - involution of thymus
   - cell death within germinal centers of lymph nodes
   - fragmentation of endometrium during menses
   - lactating breast during weaning
2. Pathologic
   - viral hepatitis
   - cytotoxic T cell–mediated immune destruction (type IV hypersensitivity)

Necrosis

- cause → hypoxia or toxins (irreversible injury)
- many cells or clusters of cells
- cells swell
- inflammation present

Examples of necrosis:

- coagulative necrosis → ischemia (except the brain)
- liquefactive necrosis → bacterial infection (and brain infarction)
- fat necrosis → pancreatitis and trauma to the breast
- caseous necrosis → tuberculosis
- fibrinoid necrosis → autoimmune disease (type III hypersensitivity reaction)
- gangrene → ischemia to extremities → dry (mainly coagulative necrosis) or wet (mainly liquefactive necrosis due to bacterial infection)

4. TERMS

Adaptation

- hypertrophy → increase in the size of cells
- hyperplasia → increase in the number of cells
- atrophy → decrease in the size of an organ
- aplasia → failure of cell production
- hypoplasia → decrease in the number of cells
- metaplasia → replacement of one cell type by another
- dysplasia → abnormal cell growth

Abnormal Organ Development

- anlage → primitive mass of cells
- aplasia → complete failure of an organ to develop (anlage present)

- agenesis → complete failure of an organ to develop (no anlage present)
- hypoplasia → reduction in the size of an organ due to a decrease in the number of cells
- atrophy → decrease in the size of an organ due to a decrease in the number of preexisting cells

5. CARDINAL SIGNS OF INFLAMMATION

- rubor → red
- calor → hot
- tumor → swollen
- dolor → pain

6. COMPLEMENT CASCADE

Products

- C3b → opsonin
- C5a → chemotaxis and leukocyte activation
- C3a, C4a, C5a → anaphylatoxins
- C5–9 → membrane attack complex

Deficiencies

- deficiency of C3 and C5 → recurrent pyogenic bacterial infections
- deficiency of C6, C7, and C8 → recurrent infections with *Neisseria* species
- deficiency of C1 esterase inhibitor → hereditary angioedema
- deficiency of decay-accelerating factor → paroxysmal nocturnal hemoglobinuria

7. THROMBOXANE VS. PROSTACYCLIN

Thromboxane

- produced by platelets
- causes vasoconstriction
- stimulates platelet aggregation

Prostacyclin

- produced by endothelial cells
- causes vasodilation
- inhibits platelet aggregation

8. GRANULOMATOUS INFLAMMATION

Caseating Granulomas

- aggregates of activated macrophages (epitheloid cells)
- tuberculosis

Noncaseating Granulomas

- sarcoidosis
- fungal infections
- foreign-body reaction

9. COLLAGEN TYPES

Fibrillar Collagens

- type I → skin, bones, tendons, mature scars
- type II → cartilage
- type III → embryonic tissue, blood vessels, pliable organs, immature scars

Amorphous Collagens

- type IV → basement membranes
- type VI → connective tissue

10. EDEMA

Exudates

1. Composition
   - increased protein
   - increased cells
   - specific gravity greater than 1.020
2. Cause
   - inflammation
   - increased blood vessel permeability

Transudates

1. Composition
   - no increased protein
   - no increased cells
   - specific gravity less than 1.012
2. Cause → abnormality of Starling forces
   a. increased hydrostatic (venous) pressure
      - congestive heart failure
      - portal hypertension
   b. decreased oncotic pressure → due to decreased albumin
      - liver disease
      - renal disease (nephrotic syndrome)

13. ONCOGENE EXPRESSION

Growth Factors

1. *c-sis*
   - β chain of platelet-derived growth factor
   - astrocytomas and osteogenic sarcomas

Growth Factor Receptors

1. *c-erb B1*
   - receptor for epidermal growth factor
   - breast cancer and squamous cell carcinoma of the lung
2. *c-neu*
   - receptor for epidermal growth factor
   - breast cancer
3. *c-fms*
   - receptor for colony-stimulating factor (CSF)
   - leukemia

Abnormal Membrane Protein Kinase

1. *c-abl*
   - membrane tyrosine kinase
   - chronic myelocytic leukemia (CML)

GTP-Binding Proteins

1. *c-ras*
   - product is p21 (protein)
   - adenocarcinomas

Nuclear Regulatory Proteins

1. *c-myc* → Burkitt's lymphoma
2. *N-myc* → neuroblastoma
3. *L-myc* → small cell carcinoma of the lung
4. *c-jun*
5. *c-fos*

14. CHROMOSOMES AND CANCER

Point Mutations

- *c-ras* → adenocarcinomas

Translocations

- *c-abl* on chromosome 9 → CML
- *c-myc* on chromosome 8 → Burkitt's lymphoma
- *bcl-2* on chromosome 18 → nodular lymphoma

Gene Amplification

- *N-myc* → neuroblastoma
- *c-neu* → breast cancer
- *c-erb B2* → breast cancer

15. ANTIONCOGENES

Tumor Suppressor Genes

- Rb → retinoblastoma and osteogenic sarcoma
- p53 → many tumors and the Li-Fraumeni syndrome
- WT1 → Wilms' tumor and aniridia
- NF1 → neurofibromatosis type 1

16. CHEMICAL CARCINOGENS

Initiators

- tobacco smoke → many tumors
- benzene → leukemias
- vinyl chloride → angiosarcomas of the liver
- β-naphthylamine → cancer of the urinary bladder
- azo dyes → tumors of the liver
- aflatoxin → hepatoma
- asbestos → mesotheliomas and lung tumors
- arsenic → skin cancer

Promoters

- saccharin → bladder cancer in rats
- hormones (estrogen)

17. VIRUSES AND CANCER

RNA Viruses

- acute-transforming viruses
- slow-transforming viruses
- HTLV-1 → adult T cell leukemia/lymphoma