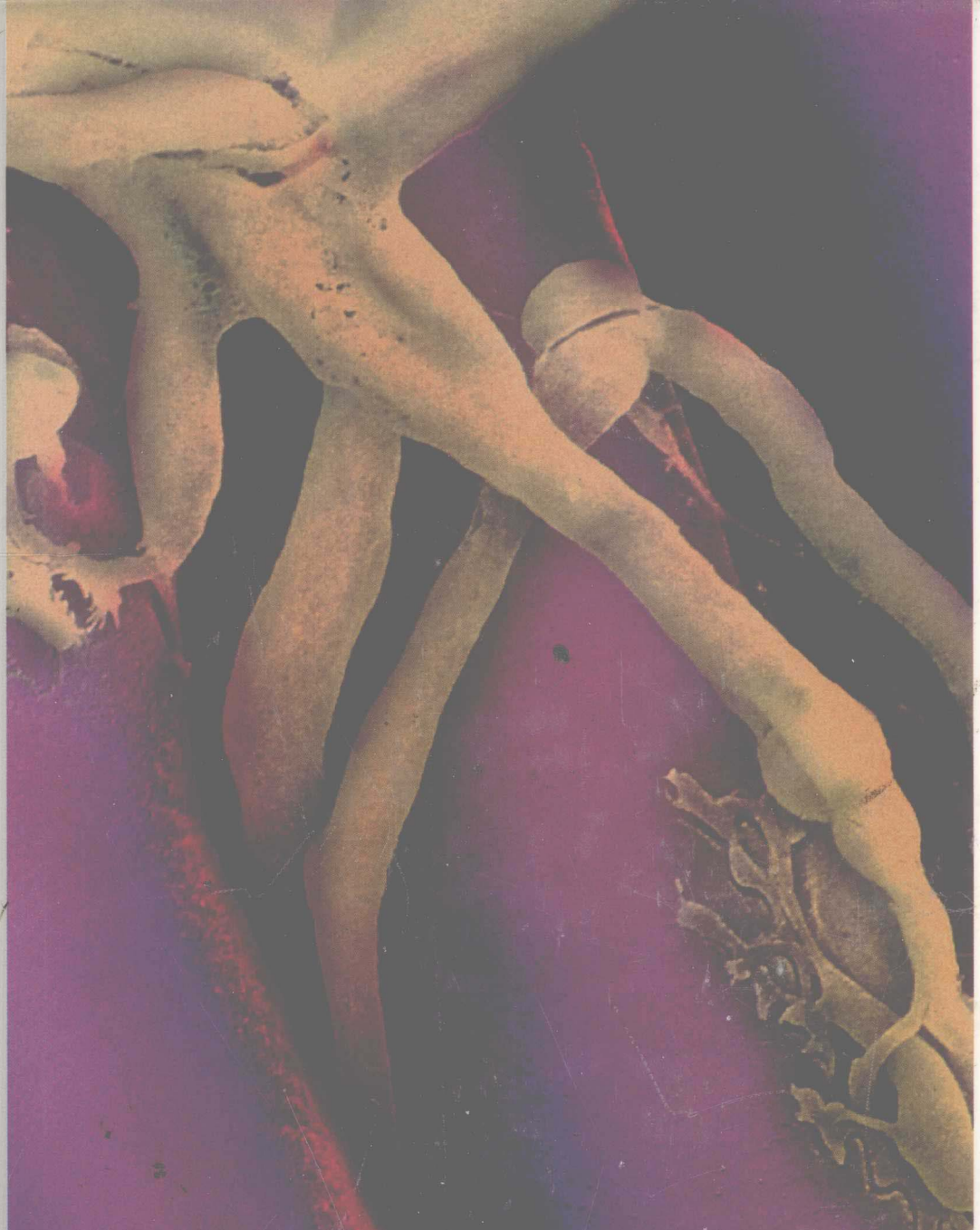

Vander
Sherman
Luciano

eighth edition

# HUMAN Physiology

## The Mechanisms of Body Function

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## The Mechanisms of Body Function

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# Preface

## Goals and Orientation

The purpose of this book remains what it was in the first seven editions: to present the fundamental principles and facts of human physiology in a format that is suitable for undergraduate students, regardless of academic backgrounds or fields of study: liberal arts, biology, nursing, pharmacy, or other allied health professions. The book is also suitable for dental students, and many medical students have also used previous editions to lay the foundation for the more detailed coverage they receive in their courses.

The most significant feature of this book is its clear, up-to-date, accurate explanations of **mechanisms,** rather than the mere description of facts and events. Because there are no limits to what can be covered in an introductory text, it is essential to reinforce over and over, through clear explanations, that physiology can be understood in terms of basic themes and principles. As evidenced by the very large number of flow diagrams employed, the book emphasizes understanding based on the ability to think in **clearly defined chains of causal links.** This approach is particularly evident in our emphasis of the dominant theme of human physiology and of this book—**homeostasis** as achieved through the coordinated function of **homeostatic control systems.**

To repeat, we have attempted to explain, integrate, and synthesize information rather than simply to describe, so that students will achieve a working knowledge of physiology, not just a memory bank of physiological facts. Since our aim has been to tell a coherent story, rather than to write an encyclopedia, we have been willing to devote considerable space to the logical development of difficult but essential concepts; examples are second messengers (Chapter 7), membrane potentials (Chapter 8), and the role of intrapleural pressure in breathing (Chapter 15).

In keeping with our goals, the book progresses from the cell to the body, utilizing information and principles developed previously at each level of complexity. One example of this approach is as follows: the characteristics that account for protein specificity are presented in Part One (Chapter 4), and this concept is used there to explain the "recognition" process exhibited by enzymes. It is then used again in Part Two (Chapter 7) for membrane receptors, and again in Part Three (Chapter 20) for antibodies. In this manner, the student is helped to see the basic foundations upon which more complex functions such as homeostatic neuroendocrine and immune responses are built.

Another example: Rather than presenting, in a single chapter, a gland-by-gland description of all the hormones, we give a description of the basic principles of endocrinology in Chapter 10, but then save the details of individual hormones for later chapters. This permits the student to focus on the functions of the hormones in the context of the homeostatic control systems in which they participate.

## Alternative Sequences

Given the inevitable restrictions of time, our organization permits a variety of sequences and approaches to be adopted. Chapter 1 should definitely be read first as it introduces the basic themes that dominate the book. Depending on the time available, the instructor's goals, and the students' backgrounds in physical science and cellular and molecular biology, the chapters of Part One can be either worked through systematically at the outset or be used more selectively as background reading in the contexts of Parts Two and Three.

In Part Two, the absolutely essential chapters are, in order, Chapters 7, 8, 10, and 11, for they present the basic concepts and facts relevant to homeostasis, intercellular communication, signal transduction, nervous and endocrine systems, and muscle. This material, therefore, is critical for an understanding of Part Three.

We believe it is best to begin the coordinated body functions of Part Three with circulation (Chapter 14), but otherwise the chapters of Part Three, as well as Chapters 9, 12, and 13 of Part Two, can be rearranged and used or not used to suit individual instructor's preferences and time availability.

## Revision Highlights

There were two major goals for this revision: (1) to redo the entire illustration program (and give the

general layout of the book a "face-lift") for greater teaching effectiveness, clarity, consistency, and esthetic appeal; and (2) to update all material and assure the greatest accuracy possible.

### *Illustration Program*

Almost all the figures have been redone to some extent, ranging from a complete redrawing of the figure to simply changing the labeling of graph axes for greater clarity. Figures 20–1 and 20–10 (Figure 20–9 in the previous edition) provide examples of how a more realistic three-dimensional perspective has been added to many of the figures, and Figure 20–13 (Figure 20–12 in the previous edition) shows how the picturing of complex events has been improved. Also, even when a specific part of the text has not required revision, we have added some new figures (for example, Figure 20–7) to illustrate the text, particularly in the case of material we know to be difficult.

Of course, the extensive use of flow diagrams, which we introduced in our first edition, has been continued. Conventions, which have been expanded in this edition, are used in these diagrams throughout the book to enhance learning. Look, for example, at Figure 16–28. The beginning and ending boxes of the flow diagram are in green, and the beginning is further clarified by the use of a "Begin" logo. Blue three-dimensional boxes are used to denote events that occur inside organs and tissues (identified by bold-faced underlined labels in the upper right of the boxes), so that the reader can easily pick out the anatomic entities that participate in the sequences of events. The participation of hormones in the sequences stand out by the placing of changes in their plasma concentrations in reddish/orange boxes. Similarly, changes in urinary excretion are shown in yellow boxes. All other boxes are purple. Thus, color is used in these diagrams for particular purposes, not just for the sake of decoration.

Other types of color coding are also now used consistently throughout the book. Thus, to take just a few examples, there are specific colors for the extracellular fluid, the intracellular fluid, muscle, particular molecules (the two strands of DNA, for example), and the lumen of the renal tubules and GI tract. Even a quick perusal of Chapter 20 will reveal how consistent use of different colors for the different types of lymphocytes, as well as macrophages, should help learning.

### *Updating of Material*

Once again, we have considerably rewritten material to improve clarity of presentation. In addition, as noted above, most figures have been extensively redone, and new figures have been added (only a few of these are listed below). Finally, as a result of new research or in response to suggestions by our colleagues, many topics have either been significantly altered or added for the first time in this edition; the following is a partial list of these topics.

**Chapter 1** Introductory section: "The Scope of Human Physiology"

**Chapter 2** New figures: Hemoglobin molecule, DNA double helix base pairings, purine-pyrimidine hydrogen bond pairings

**Chapter 3** Cholesterol in membrane function
Procedures for studying cell organelles
Endosomes
Peroxisomes

**Chapter 5** Mitochondrial DNA
Preinitiation complex
Factors altering the activity of specific cell proteins
Protein delivery and entry into mitochondria
Regulation of cell division at checkpoints in mitotic cycle

**Chapter 6** Patch clamping
Primary active-transport mechanisms
Digitalis and inhibition of Na,K-ATPase
Cystic fibrosis chloride channel
Endocytosis
New figures illustrating transporter conformational changes

**Chapter 7** Paracrine/autocrine agents
Melatonin and brain pacemakers
Receptors as tyrosine kinases and guanylyl cyclase
JAK kinases and receptors
Phospholipase, diacylglycerol, and inositol trisphosphate
Calcium-induced calcium release
Receptor inactivation

**Chapter 8** Regeneration of neurons
Comparison of voltage-gated sodium and potassium channels
Information on neurotransmitters
Functional anatomy of the central nervous system

**Chapter 9** Pain
Olfaction

**Chapter 10** Diagnosis of the site of a hormone abnormality

**Chapter 11** Passive elastic properties and role of titan
Factors causing fatigue
Role of nitric oxide in relaxing smooth muscle

**Chapter 12** Cortical control of motor behavior
Parkinson's disease
Effect of the corticospinal pathways on local-level neurons
Walking

**Chapter 13** Electroencephalogram
Sleep
Binding problem
Emotions

Schizophrenia
Serotonin-specific reuptake inhibitors (SSRIs)
Learning and memory, and their neural bases

**Chapter 14** Erythropoietin mechanism of action
Anti-angiogenic factors in treatment of cancer
Capillary filtration coefficient
Shock
Static exercise and blood pressure
Aging and heart rate
Drug therapy for hypertension, heart failure, and coronary artery disease
Dysfunctional endothelium in atherosclerosis
Homocysteine, folate, and vitamin E in atherosclerosis
Coronary stents
Nitric oxide and peripheral veins
Platelet receptors for fibrinogen
Therapy of stroke with t-PA

**Chapter 15** Pulmonary vessels and gravitational/physical forces
Hemoglobin cooperativity
Carbon monoxide and oxygen carriage
Emphysema

**Chapter 16** Mesangial cells and glomerular filtration coefficient
Channels, transporters, and genetic renal diseases
Micturition, including role of sympathetic neurons
Aquaporins
Medullary circulation and urinary concentration
Pressure natriuresis
Calcitonin
Bisphosphonates and osteoporosis

**Chapter 17** Colipase and fat digestion
HCl secretion and inhibitory role of somatostatin
Intestinal fluid secretion and absorption

**Chapter 18** Inhibition of glucagon secretion by insulin
Roles of HDL and LDL
IGF-I and fetal growth
IGF-II
Mechanism of calorigenic effect of thyroid hormones
Leptin effects on hypothalamus and anterior pituitary
Overweight and obesity
Fever and neural pathways from liver
Endogenous cryogens

**Chapter 19** Dehydroepiandrosterone (DHEA)
Viagra (mechanism of action)
Therapy of prostate cancer with blockers of dihydrotestosterone formation
Mechanism of dominant follicle selection and function
Mechanism of corpus luteum regression
Estrogen effect in males
Cause of premenstrual tension, syndrome, and dysphoric disorder
Estrogen, learning, and Alzheimer's disease
Oxytocin and sperm transport
Parturition and placental corticotropin releasing hormone
Postcoital contraception
Lack of crossing-over in X and Y chromosomes
ACTH and onset of puberty
Leptin and onset of puberty
Tamoxifen and selective estrogen receptor modulators (SERMs)

**Chapter 20** Carbohydrates and lipids as nonspecific markers on foreign cells
C-reactive protein and other nonspecific opsonins
Apoptosis of immune cells
Mechanism by which diversity arises in lymphocytes
Tumor necrosis factor and lymphocyte activation
Roles of acute phase proteins
Mechanisms of immune tolerance
Psychological stress and disease

Also, our coverage of pathophysiology, everyday applications of physiology, exercise physiology, and molecular biology have again been expanded.

Despite many additions, a ruthless removal of material no longer deemed essential has permitted us to maintain the text size unchanged from the previous edition.

Finally, *The Dynamic Human* CD-ROM is correlated to several figures. A Dynamic Human (dancing man) icon appears in appropriate figure legends. The *WCB Life Science Animations* Videotape Series is also correlated to several figure legends, and videotape icons appear in relevant figure legends.

## Study Aids

A variety of pedagogical aids are utilized:

1. **Bold-faced key terms** throughout each chapter. Clinical terms are designated by ***bold-faced italics.***
2. The illustration program is described earlier in the preface.
3. Summary tables. We have increased the number of reference and summary tables in this edition. Some summarize small or moderate amounts of information (for example, the summary of the major hormones influencing growth in Table 18–6), whereas others bring together large amounts of information that may be scattered throughout the book (for example, the reference figure of liver functions in Chapter 17). In several places, mini-glossaries are included as reference tables in the text (for example, the list of immune-system cells and chemical mediators in Chapter 20). Because the tables complement the figures, these two learning aids taken

together provide a rapid means of reviewing the most important material in a chapter.

4. End-of-section or chapter study aids
   a. Extensive summaries in outline form
   b. Key-term lists of all bold-faced words in the section/chapter (excluding the clinical terms)
   c. Comprehensive review questions in essay format. These review questions, in essence, constitute a complete list of learning objectives.
   d. Clinical term lists of all bold-face italicized words in the chapter. This serves to remind the student of how the physiology has been applied to clinical examples in the chapter.
   e. Thought questions that challenge the student to go beyond the memorization of facts to solve problems, often presented as case histories or experiments. Complete Answers to Thought Questions are given in Appendix A.

The chapter summaries, key-term definition lists, and review questions appear at the ends of the sections in those chapters that are broken into sections. These aids appear at the ends of nonsectioned chapters. Clinical term lists and thought questions are always at the ends of chapters.

5. A very extensive glossary, with pronunciation guides, is provided in Appendix B.
6. Appendixes C and D present, respectively, English-metric interconversions and Electrophysiology equations. Appendix E is an outline index of exercise physiology.
7. A complete alphabetized list of all abbreviations used in the text is given on the endpapers (the insides of the book's covers).

## Supplements

1. *Essential Study Partner* (007-235897-1). This CD-ROM is an interactive study tool packed with hundreds of animations and learning activities, including quizzes, and interactive diagrams. A self-quizzing feature allows students to check their knowledge of a topic before moving on to a new module. Additional unit exams give students the opportunity to review coverage after completing entire units. A large number of anatomical supplements are also included. The ESP is packaged free with textbooks.
2. *Online Learning Center* (http://www.mhhe.com/biosci/ap/vander8e/). Students and instructors gain access to a world of opportunities through this Web site. Students will find quizzes, activities, links, suggested readings, and much more. Instructors will find all the enhancement tools needed for teaching on-line, or for incorporating technology in the traditional course.
3. *The Student Study Guide* is now available as part of the Online Learning Center. Written by Donna Van Wynsberghe of the University of Wisconsin—Milwaukee, it contains a large variety of study aids, including learning hints and many test questions with answers.
4. *Instructor's Manual and Test Item File* (007-290803-3) by Sharon Russell of the University of California—Berkeley contains suggestions for teaching, as well as a complete test item file.
5. *MicroTest III testing software.* Available in Windows (007-290805-X) and Macintosh (007-290804-1). A computerized test generator for use with the text allows for quick creation of tests based on questions from the test item file and requires no programming experience.
6. *Overhead transparencies* (007-290806-8). A set of 200 full-color transparencies representing the most important figures from the book is available to instructors.
7. *McGraw-Hill Visual Resource Library* (007-290807-6). A CD-ROM containing all of the line art from the text with an easy-to-use interface program enabling the user to quickly move among the images, show or hide labels, and create a multimedia presentation.

## Other Materials Available from McGraw-Hill

8. *The Dynamic Human* CD-ROM (0697-38935-9) illustrates the important relationships between anatomical structures and their functions in the human body. Realistic computer visualization and three-dimensional visualizations are the premier features of this CD-ROM. Various figures throughout this text are correlated to modules of *The Dynamic Human.* See pages xxvi–xxvii for a detailed listing of figures.
9. *The Dynamic Human Videodisc* (0-667-38937-5) contains all the animations (200+) from the CD-ROM. A bar code directory is also available.
10. *Life Science Animations Videotape Series* is a series of five videotapes containing 53 animations that cover many of the key physiological processes. Another videotape containing similar animations is also available, entitled *Physiological Concepts of Life Science.* Various figures throughout this text are correlated to animations from the *Life Science Animations.* See pages xxvii–xxviii for a detailed listing of figures.

Tape 1: Chemistry, The Cell, Energetics (0-697-25068-7)
Tape 2: Cell Division, Heredity, Genetics, Reproduction and Development (0-697-25069-5)
Tape 3: Animal Biology I (0-697-25070-9)
Tape 4: Animal Biology II (0-697-25071-7)
Tape 5: Plant Biology, Evolution, and Ecology (0-697-26600-1)
Tape 6: Physiological Concepts of Life Science (0-697-21512-1)

11. *Life Science Animations 3D* CD-ROM (007-234296-X). More than 120 animations that illustrate key biological processes are available at your fingertips on this exciting CD-ROM. This CD contains all of the animations found on the *Essential Study Partner* and much more. The animations can be imported into presentation programs, such as PowerPoint. Imagine the benefit of showing the animations during lecture.
12. *Life Science Animations 3D Videotape* (007-290652-9). Featuring 42 animations of key biologic processes, this tape contains 3D animations and is fully narrated. Various figures throughout this text are correlated to video animations. See page xxviii for a detailed listing of figures.
13. *Life Science Living Lexicon* CD-ROM (0-697-37993-0 hybrid) contains a comprehensive collection of life science terms, including definitions of their roots, prefixes, and suffixes as well as audio pronunciations and illustrations. The Lexicon is student-interactive, featuring quizzing and notetaking capabilities.
14. *The Virtual Physiology Lab* CD-ROM (0-697-37994-9 hybrid) containing 10 dry labs of the most common and important physiology experiments.
15. *Anatomy and Physiology Videodisc* (0-697-27716-X) is a four-sided videodisc containing more than 30 animations of physiological processes, as well as line art and micrographs. A bar code directory is also available.
16. *Anatomy and Physiology Video Series* consists of the following:
    a. Internal Organs and the Circulatory System of the Cat (0-697-13922-0)
    b. Blood Cell Counting, Identification & Grouping (0-697-11629-8)
    c. Introduction to the Human Cadaver and Prosection (0-697-11177-6)
    d. Introduction to Cat Dissection: Musculature (0-697-11630-1)
17. *Study Cards for Anatomy and Physiology* (007-290818-1) by Van De Graaff, et al., is a boxed set of 300 3-by-5 inch cards. It serves as a well-organized and illustrated synopsis of the structure and function of the human body. The Study Cards offer a quick and effective way for students to review human anatomy and physiology.
18. *Coloring Guide to Anatomy and Physiology* (0-697-17109-4) by Robert and Judith Stone emphasizes learning through the process of color association. The Coloring Guide provides a thorough review of anatomical and physiological concepts.
19. *Atlas of the Skeletal Muscles* (0-697-13790-2) by Robert and Judith Stone is a guide to the structure and function of human skeletal muscles. The illustrations help students locate muscles and understand their actions.
20. *Laboratory Atlas of Anatomy and Physiology* (0-697-39480-8) by Eder, et al., is a full-color atlas containing histology, human skeletal anatomy, human muscular anatomy, dissections, and reference tables.
21. *Case Histories in Human Physiology*, third edition, by Donna Van Wynesberghe and Gregory Cooley is a web-based workbook that stimulates analytical thinking through case studies and problem solving; includes an instructor's answer key. (www.mhhe.com/biosci/ap/vanwyn/).
22. *Survey of Infectious and Parasitic Diseases* (0-697-27535-3) by Kent M. Van De Graaff is a black-and-white booklet that presents the essential information on 100 of the most common and clinically significant diseases.

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**To our parents, and to Judy, Peggy, and Joe without whose understanding it would have been impossible**

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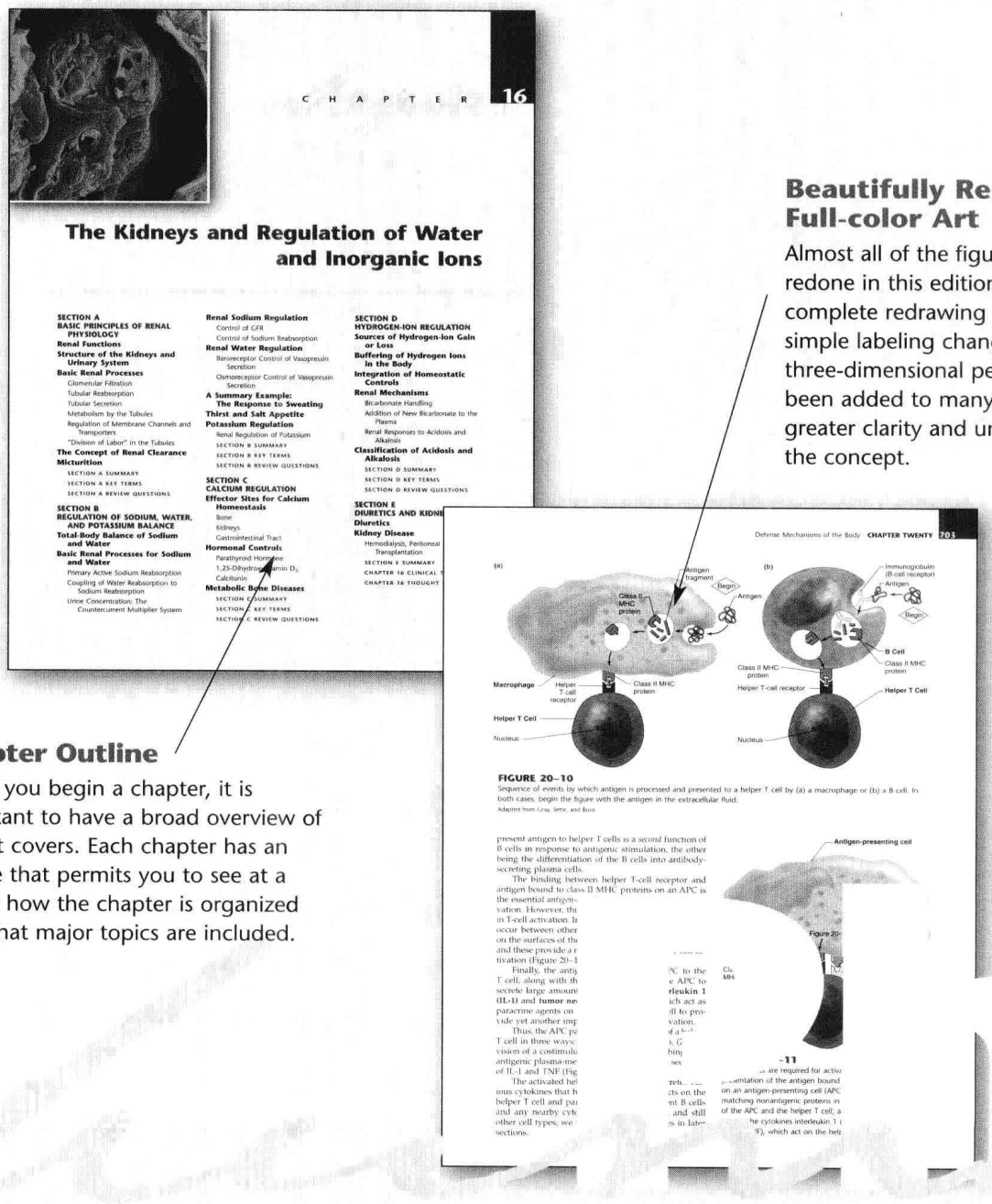
# Visual Tour


CHAPTER 16

The Kidneys and Regulation of Water and Inorganic Ions

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Tubular Reabsorption
Tubular Secretion
Metabolism by the Tubules
Regulation of Membrane Channels and Transporters
"Division of Labor" in the Tubules
The Concept of Renal Clearance
Micturition
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SECTION A KEY TERMS
SECTION A REVIEW QUESTIONS

SECTION B
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Control of Sodium Reabsorption
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SECTION D
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Buffering of Hydrogen Ions in the Body
Integration of Homeostatic Controls
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Bicarbonate Handling
Addition of New Bicarbonate to the Plasma
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Classification of Acidosis and Alkalosis
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SECTION E
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Diuretics
Kidney Disease
Hemodialysis, Peritoneal Transplantation
SECTION E SUMMARY
CHAPTER 16 CLINICAL T
CHAPTER 16 THOUGHT


Defense Mechanisms of the Body CHAPTER TWENTY 703

FIGURE 20–10
Sequence of events by which antigen is processed and presented to a helper T cell by (a) a macrophage or (b) a B cell. In both cases, begin the figure with the antigen in the extracellular fluid.
Adapted from Gray, Sette, and Buus

present antigen to helper T cells is a *second* function of B cells in response to antigenic stimulation, the other being the differentiation of the B cells into antibody-secreting plasma cells.

The binding between helper T-cell receptor and antigen bound to class II MHC proteins on an APC is the essential *antigen-*

Antigen-presenting cell

## Beautifully Rendered Full-color Art

Almost all of the figures have been redone in this edition, ranging from a complete redrawing of the figure to simple labeling changes. A realistic three-dimensional perspective has been added to many of the figures for greater clarity and understanding of the concept.

## Chapter Outline

Before you begin a chapter, it is important to have a broad overview of what it covers. Each chapter has an outline that permits you to see at a glance how the chapter is organized and what major topics are included.

## Color-coded Illustrations

Color-coding is effectively used to promote learning. For example, there are specific colors for the extracellular fluid, the intracellular fluid, muscle, and the lumen of the renal tubules and GI tract.

called **parathyroid hormone**, produced by the parathyroid glands. These glands are in the neck, embedded in the surface of the thyroid gland, but are distinct from it. Parathyroid hormone production is controlled by the extracellular calcium concentration acting directly on the secretory cells (via a plasma-membrane calcium receptor). *Decreased* plasma calcium concentration *stimulates* parathyroid hormone

compensating for the decreased concentration that originally stimulated secretion of this hormone (Figure 16–28).

1. It directly increases the resorption of bone by osteoclasts, which results in the movement of calcium (and phosphate) from bone into extracellular fluid.

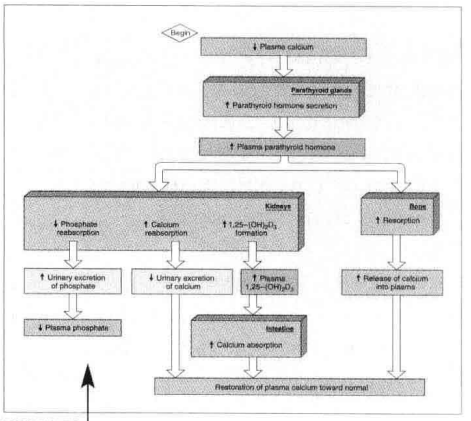



**FIGURE 16–28**
Reflexes by which a reduction in plasma calcium concentration is restored toward normal via the actions of parathyroid hormone. See Figure 16–29 for a more complete description of $1,25\text{-}(OH)_2D_3$.

Movement of Molecules Across Cell Membranes **CHAPTER SIX** 125

The net movement from lower to higher concentration and the maintenance of a higher steady-state concentration on one side of a membrane can be achieved only by the continuous input of energy into the active-transport process. This energy can (1) alter the affinity of the binding site on the transporter such that it has a higher affinity when facing one side of the membrane than when facing the other side; or (2) alter the rates at which the binding site on the transporter is shifted from one surface to the other.

To repeat, in order to move molecules from a lower concentration (lower energy state) to a higher concentration (higher energy state), energy must be added. Therefore, active transport must be coupled to the simultaneous flow of some energy source from a higher energy level to a lower energy level. Two means of coupling an energy flow to transporters are known: (1) the direct use of ATP in **primary active transport**, and (2) the use of an ion concentration difference across a membrane to drive the process in **secondary active transport**.

**Primary Active Transport** The hydrolysis of ATP by a transporter provides the energy for primary active transport. The transporter is an enzyme (an ATPase) that catalyzes the breakdown of ATP and, in the process, phosphorylates itself. Phosphorylation of the transporter protein (covalent modulation) changes the affinity of the transporter's solute binding site. Figure 6–11 illustrates the sequence of events leading to the active transport (that is, transport from low to higher concentration) of a solute into a cell. (1) Initially, the binding site for the transported solute is exposed to the extracellular fluid and has a high affinity because the protein has been phosphorylated on its intracellular surface by ATP. This phosphorylation occurs only when the transporter is in the conformation shown on the left side of the figure. (2) The transported solute in the extracellular fluid binds to the high-affinity binding site. Random thermal oscillations repeatedly expose the binding site to one side of the membrane, then to the other, independent of the protein's phosphorylation. (3) Removal of the phosphate group from the transporter decreases the affinity of the binding site, leading to (4) the release of the transported solute into the intracellular fluid. When the low-affinity site is returned to the extracellular face of the membrane by the random oscillation of the transporter (5), it is in a conformation which again permits phosphorylation, and the cycle can be repeated.

To see why this will lead to movement from low to higher concentration (that is, uphill movement), consider the flow of solute through the transporter at a point in time when the concentration is equal on the two sides of the membrane. More solute will be bound to the high-affinity site at the extracellular surface of the membrane than to the low-affinity site on the intracellular surface. Thus more solute will move in than out when the transporter oscillates between sides.

The major primary active-transport proteins found in most cells are (1) $Na,K$-ATPase; (2) $Ca$-ATPase; (3) $H$-ATPase; and (4) $H,K$-ATPase.

$Na,K$-ATPase is present in all plasma membranes. The pumping activity of this primary active-transport protein leads to the characteristic distribution of high intracellular potassium and low intracellular sodium

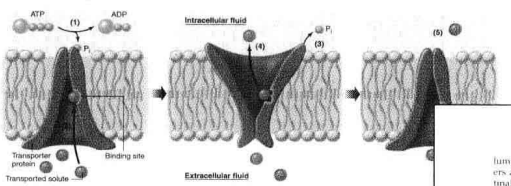



**FIGURE 6–11**
Primary active-transport model. Changes in the binding site affinity for a transported solute are produced by phosphorylation and dephosphorylation of the transporter (covalent modulation) as it oscillates between two conformations. See numbered sequence of events occurring during transport.

## Flow Diagrams

Long a hallmark of this book, extensive use of flow diagrams have been continued and expanded in this edition. A bookmark has been included with your book to give a further explanation.

The Digestion and Absorption of Food **CHAPTER SEVENTEEN** 557

luminal surface of the intestinal lining cells, while others are secreted by the pancreas and enter the intestinal lumen. The products of digestion are absorbed across the epithelial cells and enter the blood and/or lymph. Vitamins, minerals, and water, which do not require enzymatic digestion, are also absorbed in the small intestine.

The small intestine is divided into three segments. An initial short segment, the **duodenum**, is followed by the **jejunum** and then by the longest segment, the **ileum**. Normally, most of the chyme entering from the stomach is digested and absorbed in the first quarter of the small intestine, in the duodenum and jejunum.

Two major glands—the pancreas and liver—secrete substances that flow via ducts into the duodenum. The **pancreas**, an elongated gland located behind the stomach, has both endocrine (Chapter 18) and exocrine functions, but only the latter are directly involved in gastrointestinal function and are described in this chapter. The exocrine portion of the pancreas secretes (1) digestive enzymes and (2) a fluid rich in bicarbonate ions. The high acidity of the chyme coming from the stomach would inactivate the pancreatic enzymes in the small intestine if the acid were not neutralized by the bicarbonate ions in the pancreatic fluid.

The **liver**, a large gland located in the upper right portion of the abdomen, has a variety of functions, which are described in various chapters. This is a convenient place to provide, in Table 17–1, a comprehensive reference list of these **hepatic** (the term means "pertaining to the liver") functions and the chapters in which they are described. We will be concerned in this

**TABLE 17–1 Summary of Liver Functions**

**A. Exocrine (digestive) functions (Chapter 17)**
1. Synthesizes and secretes bile salts, which are necessary for adequate digestion and absorption of fats.
2. Secretes into the bile a bicarbonate-rich solution, which helps neutralize acid in the duodenum.

**B. Endocrine functions**
1. In response to growth hormone, secretes insulin-like growth factor I (IGF-I), which promotes growth by stimulating cell division in various tissues, including bone (Chapter 18).
2. Contributes to the activation of vitamin D (Chapter 16).
3. Forms triiodothyronine ($T_3$) from thyroxine ($T_4$) (Chapter 10).
4. Secretes angiotensinogen, which is acted upon by renin to form angiotensin I (Chapter 16).
5. Metabolizes hormones (Chapter 10).
6. Secretes cytokines involved in immune defenses (Chapter 20).

**C. Clotting functions**
1. Produces many of the plasma clotting factors, including prothrombin and fibrinogen (Chapter 14).
2. Produces bile salts, which are essential for the gastrointestinal absorption of vitamin K, which is, in turn, needed for production of the clotting factors (Chapter 14).

**D. Plasma proteins**
1. Synthesizes and secretes plasma albumin (Chapter 14), acute phase proteins (Chapter 20), binding proteins for various hormones (Chapter 10) and trace elements (Chapter 14), lipoproteins (Chapter 18), and other proteins mentioned elsewhere in this table.

**E. Organic metabolism (Chapter 18)**
1. Converts plasma glucose into glycogen and triacylglycerols during absorptive period.
2. Converts plasma amino acids to fatty acids, which can be incorporated into triacylglycerols during absorptive period.
3. Synthesizes triacylglycerols and secretes them as lipoproteins during absorptive period.
4. Produces glucose from glycogen (glycogenolysis) and other sources (gluconeogenesis) during postabsorptive period and releases the glucose into the blood.
5. Converts fatty acids into ketones during fasting.
6. Produces urea, the major end product of amino acid (protein) catabolism, and releases it into the blood.

**F. Cholesterol metabolism (Chapter 18)**
1. Synthesizes cholesterol and releases it into the blood.
2. Secretes plasma cholesterol into the bile.
3. Converts plasma cholesterol into bile salts.

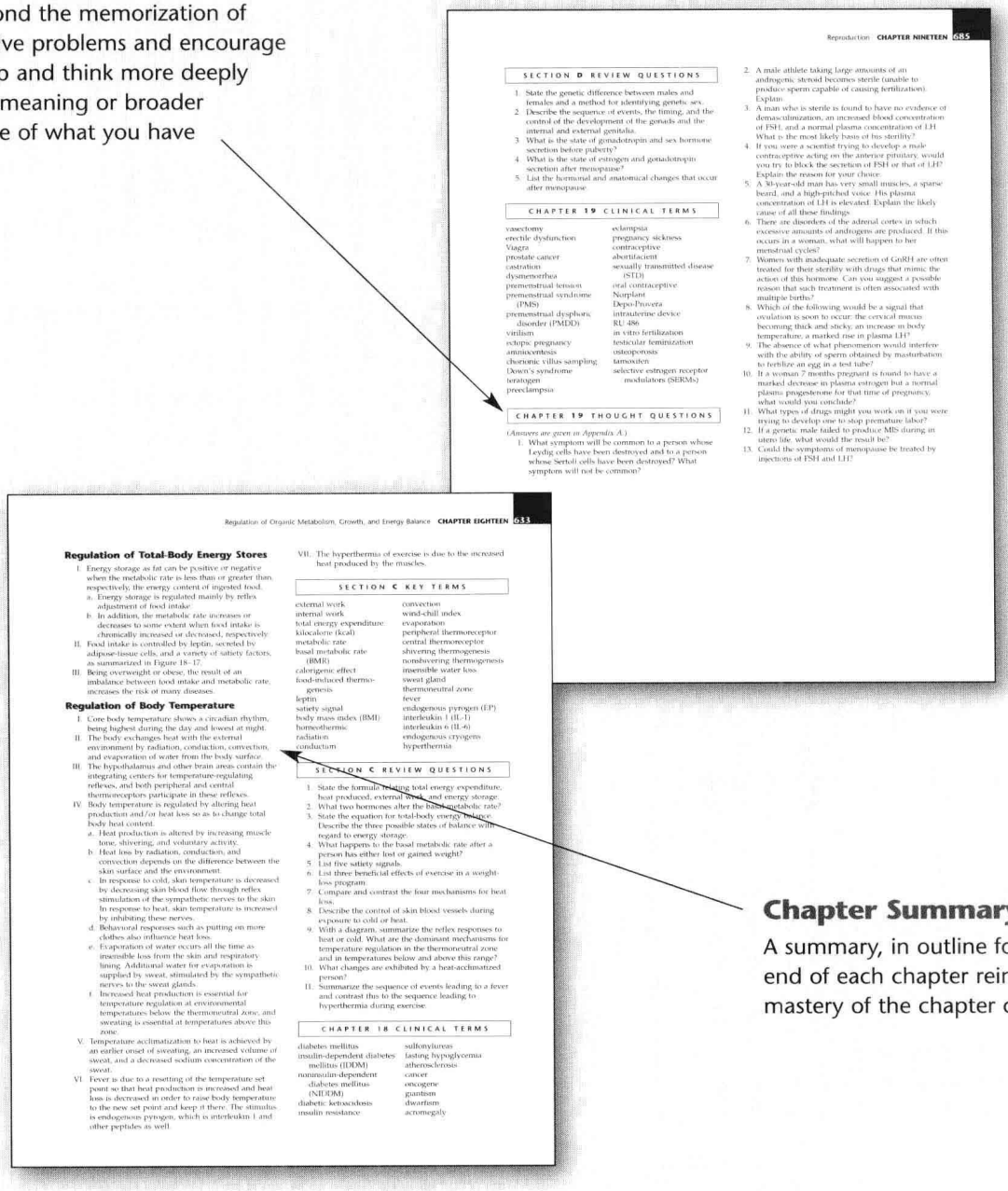
**G. Excretory and degradative functions**
1. Secretes bilirubin and other bile pigments into the bile (Chapter 17).
2. Excretes, via the bile, many endogenous and foreign organic molecules as well as trace metals (Chapter 20).
3. Biotransforms many endogenous and foreign organic molecules (Chapter 20).
4. Destroys old erythrocytes (Chapter 14).

## Summary Tables

Some summary tables summarize small or moderate amounts of information whereas others bring together large amounts of information that may be scattered throughout the book. The tables complement the accompanying figures to provide a rapid means of reviewing the most important material in a chapter.

## Thought Questions

At the end of each chapter are Thought Questions that challenge you to go beyond the memorization of facts to solve problems and encourage you to stop and think more deeply about the meaning or broader significance of what you have just read.



Reproduction CHAPTER NINETEEN 685

SECTION D REVIEW QUESTIONS

1. State the genetic difference between males and females and a method for identifying genetic sex.
2. Describe the sequence of events, the timing, and the control of the development of the gonads and the internal and external genitalia.
3. What is the state of gonadotropin and sex hormone secretion before puberty?
4. What is the state of estrogen and gonadotropin secretion after menopause?
5. List the hormonal and anatomical changes that occur after menopause.

CHAPTER 19 CLINICAL TERMS

vasectomy
erectile dysfunction
Viagra
prostate cancer
castration
dysmenorrhea
premenstrual tension
premenstrual syndrome (PMS)
premenstrual dysphoric disorder (PMDD)
virilism
ectopic pregnancy
amniocentesis
chorionic villus sampling
Down's syndrome
teratogen
preeclampsia
eclampsia
pregnancy sickness
contraceptive
abortifacient
sexually transmitted disease (STD)
oral contraceptive
Norplant
Depo-Provera
intrauterine device
RU 486
in vitro fertilization
testicular feminization
osteoporosis
tamoxifen
selective estrogen receptor modulators (SERMs)

CHAPTER 19 THOUGHT QUESTIONS

*(Answers are given in Appendix A.)*

1. What symptom will be common to a person whose Leydig cells have been destroyed and to a person whose Sertoli cells have been destroyed? What symptom will not be common?
2. A male athlete taking large amounts of an androgenic steroid becomes sterile (unable to produce sperm capable of causing fertilization). Explain.
3. A man who is sterile is found to have no evidence of demasculinization, an increased blood concentration of FSH, and a normal plasma concentration of LH. What is the most likely basis of his sterility?
4. If you were a scientist trying to develop a male contraceptive acting on the anterior pituitary, would you try to block the secretion of FSH or that of LH? Explain the reason for your choice.
5. A 30-year-old man has very small muscles, a sparse beard, and a high-pitched voice. His plasma concentration of LH is elevated. Explain the likely cause of all these findings.
6. There are disorders of the adrenal cortex in which excessive amounts of androgens are produced. If this occurs in a woman, what will happen to her menstrual cycles?
7. Women with inadequate secretion of GnRH are often treated for their sterility with drugs that mimic the action of this hormone. Can you suggest a possible reason that such treatment is often associated with multiple births?
8. Which of the following would be a signal that ovulation is soon to occur: the cervical mucus becoming thick and sticky, an increase in body temperature, a marked rise in plasma LH?
9. The absence of what phenomenon would interfere with the ability of sperm obtained by masturbation to fertilize an egg in a test tube?
10. If a woman 7 months pregnant is found to have a marked decrease in plasma estrogen but a normal plasma progesterone for that time of pregnancy, what would you conclude?
11. What types of drugs might you work on if you were trying to develop one to stop premature labor?
12. If a genetic male failed to produce MIS during in utero life, what would the result be?
13. Could the symptoms of menopause be treated by injections of FSH and LH?

Regulation of Organic Metabolism, Growth, and Energy Balance CHAPTER EIGHTEEN 633

**Regulation of Total-Body Energy Stores**

I. Energy storage as fat can be positive or negative when the metabolic rate is less than or greater than, respectively, the energy content of ingested food.
   a. Energy storage is regulated mainly by reflex adjustment of food intake.
   b. In addition, the metabolic rate increases or decreases to some extent when food intake is chronically increased or decreased, respectively.

II. Food intake is controlled by leptin, secreted by adipose-tissue cells, and a variety of satiety factors, as summarized in Figure 18–17.

III. Being overweight or obese, the result of an imbalance between food intake and metabolic rate, increases the risk of many diseases.

**Regulation of Body Temperature**

I. Core body temperature shows a circadian rhythm, being highest during the day and lowest at night.

II. The body exchanges heat with the external environment by radiation, conduction, convection, and evaporation of water from the body surface.

III. The hypothalamus and other brain areas contain the integrating centers for temperature-regulating reflexes, and both peripheral and central thermoreceptors participate in these reflexes.

IV. Body temperature is regulated by altering heat production and/or heat loss so as to change total body heat content.
   a. Heat production is altered by increasing muscle tone, shivering, and voluntary activity.
   b. Heat loss by radiation, conduction, and convection depends on the difference between the skin surface and the environment.
   c. In response to cold, skin temperature is decreased by decreasing skin blood flow through reflex stimulation of the sympathetic nerves to the skin. In response to heat, skin temperature is increased by inhibiting these nerves.
   d. Behavioral responses such as putting on more clothes also influence heat loss.
   e. Evaporation of water occurs all the time as insensible loss from the skin and respiratory lining. Additional water for evaporation is supplied by sweat, stimulated by the sympathetic nerves to the sweat glands.
   f. Increased heat production is essential for temperature regulation at environmental temperatures below the thermoneutral zone, and sweating is essential at temperatures above this zone.

V. Temperature acclimatization to heat is achieved by an earlier onset of sweating, an increased volume of sweat, and a decreased sodium concentration of the sweat.

VI. Fever is due to a resetting of the temperature set point so that heat production is increased and heat loss is decreased in order to raise body temperature to the new set point and keep it there. The stimulus is endogenous pyrogen, which is interleukin 1 and other peptides as well.

VII. The hyperthermia of exercise is due to the increased heat produced by the muscles.

SECTION C KEY TERMS

external work
internal work
total energy expenditure
kilocalorie (kcal)
metabolic rate
basal metabolic rate (BMR)
calorigenic effect
food-induced thermogenesis
leptin
satiety signal
body mass index (BMI)
homeothermic
radiation
conduction
convection
wind-chill index
evaporation
peripheral thermoreceptor
central thermoreceptor
shivering thermogenesis
nonshivering thermogenesis
insensible water loss
sweat gland
thermoneutral zone
fever
endogenous pyrogen (EP)
interleukin 1 (IL-1)
interleukin 6 (IL-6)
endogenous cryogens
hyperthermia

SECTION C REVIEW QUESTIONS

1. State the formula relating total energy expenditure, heat produced, external work, and energy storage.
2. What two hormones alter the basal metabolic rate?
3. State the equation for total-body energy balance. Describe the three possible states of balance with regard to energy storage.
4. What happens to the basal metabolic rate after a person has either lost or gained weight?
5. List five satiety signals.
6. List three beneficial effects of exercise in a weight-loss program.
7. Compare and contrast the four mechanisms for heat loss.
8. Describe the control of skin blood vessels during exposure to cold or heat.
9. With a diagram, summarize the reflex responses to heat or cold. What are the dominant mechanisms for temperature regulation in the thermoneutral zone and in temperatures below and above this range?
10. What changes are exhibited by a heat-acclimatized person?
11. Summarize the sequence of events leading to a fever and contrast this to the sequence leading to hyperthermia during exercise.

CHAPTER 18 CLINICAL TERMS

diabetes mellitus
insulin-dependent diabetes mellitus (IDDM)
noninsulin-dependent diabetes mellitus (NIDDM)
diabetic ketoacidosis
insulin resistance
sulfonylureas
fasting hypoglycemia
atherosclerosis
cancer
oncogene
giantism
dwarfism
acromegaly

## Chapter Summary

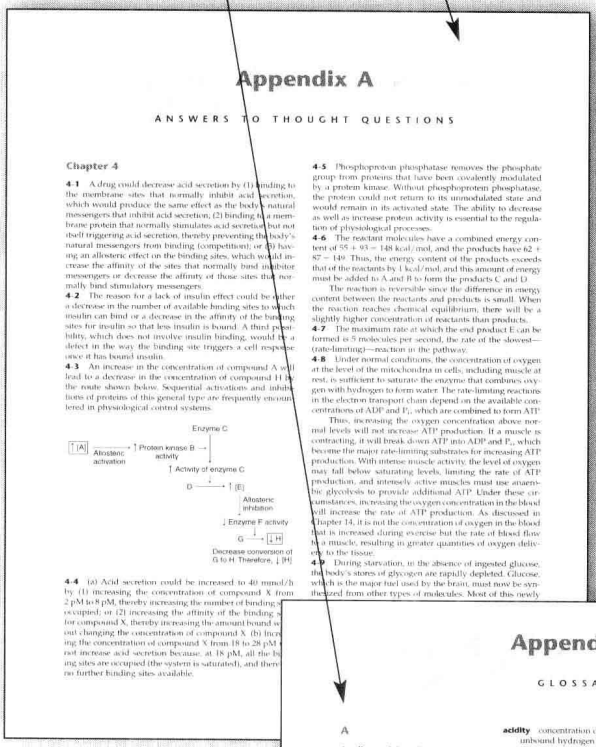
A summary, in outline form, at the end of each chapter reinforces your mastery of the chapter content.

## Answers to Thought Questions

Complete answers to Thought Questions are given in Appendix A.

## Glossary

A very extensive Glossary, with pronunciation guides, is provided in Appendix B.



# Appendix A

ANSWERS TO THOUGHT QUESTIONS

**Chapter 4**

**4-1** A drug could decrease acid secretion by (1) binding to the membrane sites that normally inhibit acid secretion, which would produce the same effect as the body's natural messengers that inhibit acid secretion, (2) binding to a membrane protein that normally stimulates acid secretion but not itself triggering acid secretion, thereby preventing the body's natural messengers from binding (competition); or (3) having an allosteric effect on the binding sites, which would increase the affinity of the sites that normally bind inhibitor messengers or decrease the affinity of those sites that normally bind stimulatory messengers.

**4-2** The reason for a lack of insulin effect could be either a decrease in the number of available binding sites to which insulin can bind or a decrease in the affinity of the binding sites for insulin so that less insulin is bound. A third possibility, which does not involve insulin binding, would be a defect in the way the binding site triggers a cell response once it has bound insulin.

**4-3** An increase in the concentration of compound A will lead to a decrease in the concentration of compound H via the route shown below. Sequential activations and inhibitions of proteins of this general type are frequently encountered in physiological control systems.




**4-4** (a) Acid secretion could be increased to 40 mmol/h by (1) increasing the concentration of compound X from 2 pM to 8 pM, thereby increasing the number of binding sites occupied; or (2) increasing the affinity of the binding sites for compound X, thereby increasing the amount bound without changing the concentration of compound X. (b) Increasing the concentration of compound X from 18 to 28 pM would not increase acid secretion because, at 18 pM, all the binding sites are occupied (the system is saturated), and there are no further binding sites available.

**4-5** Phosphoprotein phosphatase removes the phosphate group from proteins that have been covalently modulated by a protein kinase. Without phosphoprotein phosphatase, the protein could not return to its unmodulated state and would remain in its activated state. The ability to decrease as well as increase protein activity is essential to the regulation of physiological processes.

**4-6** The reactant molecules have a combined energy content of 95 + 93 = 148 kcal/mol, and the products have 62 + 87 = 149. Thus, the energy content of the products exceeds that of the reactants by 1 kcal/mol, and this amount of energy must be added to A and B to form the products C and D.

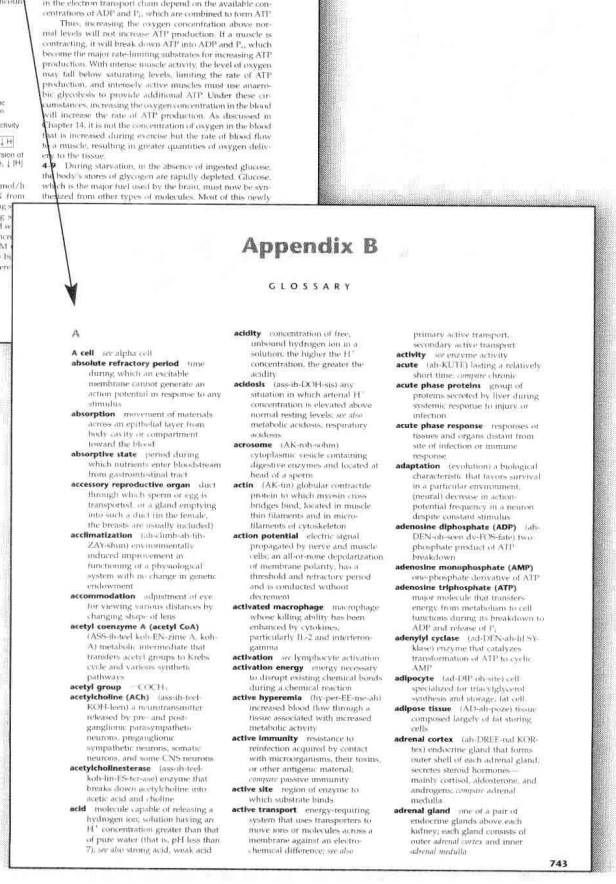
The reaction is reversible since the difference in energy content between the reactants and products is small. When the reaction reaches chemical equilibrium, there will be a slightly higher concentration of reactants than products.

**4-7** The maximum rate at which the end product E can be formed is 5 molecules per second, the rate of the slowest—(rate-limiting)—reaction in the pathway.

**4-8** Under normal conditions, the concentration of oxygen at the level of the mitochondria in cells, including muscle at rest, is sufficient to saturate the enzyme that combines oxygen with hydrogen to form water. The rate-limiting reactions in the electron transport chain depend on the available concentrations of ADP and $P_i$, which are combined to form ATP.

Thus, increasing the oxygen concentration above normal levels will not increase ATP production. If a muscle is contracting, it will break down ATP into ADP and $P_i$, which become the major rate-limiting substrates for increasing ATP production. With intense muscle activity, the level of oxygen may fall below saturating levels, limiting the rate of ATP production, and intensely active muscles must use anaerobic glycolysis to provide additional ATP. Under these circumstances, increasing the oxygen concentration in the blood will increase the rate of ATP production. As discussed in Chapter 14, it is not the concentration of oxygen in the blood that is increased during exercise but the rate of blood flow to a muscle, resulting in greater quantities of oxygen delivery to the tissue.

**4-9** During starvation, in the absence of ingested glucose, the body's stores of glycogen are rapidly depleted. Glucose, which is the major fuel used by the brain, must now be synthesized from other types of molecules. Most of this newly



# Appendix B

GLOSSARY

A

**A cell** see alpha cell

**absolute refractory period** time during which an excitable membrane cannot generate an action potential in response to any stimulus

**absorption** movement of materials across an epithelial layer from body cavity or compartment toward the blood

**absorptive state** period during which nutrients enter bloodstream from gastrointestinal tract

**accessory reproductive organ** duct through which sperm or egg is transported, or a gland emptying into such a duct (in the female, the breasts are usually included)

**acclimatization** (ah-climb-ah-tih-ZAY-shun) environmentally induced improvement in functioning of a physiological system with no change in genetic endowment

**accommodation** adjustment of eye for viewing various distances by changing shape of lens

**acetyl coenzyme A (acetyl CoA)** (ASS-ih-tuhl koh-EN-zime A, koh-A) metabolic intermediate that transfers acetyl groups to Krebs cycle and various synthetic pathways

**acetyl group** $-COCH_3$

**acetylcholine (ACh)** (ass-ih-teel-KOH-leen) a neurotransmitter released by pre- and post-ganglionic parasympathetic neurons, preganglionic sympathetic neurons, somatic neurons, and some CNS neurons

**acetylcholinesterase** (ass-ih-teel-koh-lin-ES-ter-aze) enzyme that breaks down acetylcholine into acetic acid and choline

**acid** molecule capable of releasing a hydrogen ion; solution having an $H^+$ concentration greater than that of pure water (that is, pH less than 7); *see also* strong acid, weak acid

**acidity** concentration of free, unbound hydrogen ion in a solution; the higher the $H^+$ concentration, the greater the acidity

**acidosis** (ass-ih-DOH-sis) any situation in which arterial $H^+$ concentration is elevated above normal resting levels; *see also* metabolic acidosis, respiratory acidosis

**acrosome** (AK-roh-sohm) cytoplasmic vesicle containing digestive enzymes and located at head of a sperm

**actin** (AK-tin) globular contractile protein to which myosin cross bridges bind, located in muscle thin filaments and in microfilaments of cytoskeleton

**action potential** electric signal propagated by nerve and muscle cells; an all-or-none depolarization of membrane polarity, has a threshold and refractory period and is conducted without decrement

**activated macrophage** macrophage whose killing ability has been enhanced by cytokines, particularly IL-2 and interferon-gamma

**activation** *see* lymphocyte activation

**activation energy** energy necessary to disrupt existing chemical bonds during a chemical reaction

**active hyperemia** (hy-per-EE-me-ah) increased blood flow through a tissue associated with increased metabolic activity

**active immunity** resistance to reinfection acquired by contact with microorganisms, their toxins, or other antigenic material; *compare* passive immunity

**active site** region of enzyme to which substrate binds

**active transport** energy-requiring system that uses transporters to move ions or molecules across a membrane against an electrochemical difference; *see also* primary active transport, secondary active transport

**activity** *see* enzyme activity

**acute** (ah-KUTE) lasting a relatively short time; *compare* chronic

**acute phase proteins** group of proteins secreted by liver during systemic response to injury or infection

**acute phase response** responses of tissues and organs distant from site of infection or immune response

**adaptation** (evolution) a biological characteristic that favors survival in a particular environment, (neural) decrease in action-potential frequency in a neuron despite constant stimulus

**adenosine diphosphate (ADP)** (ah-DEN-oh-seen dy-FOS-fate) two-phosphate product of ATP breakdown

**adenosine monophosphate (AMP)** one-phosphate derivative of ATP

**adenosine triphosphate (ATP)** major molecule that transfers energy from metabolism to cell functions during its breakdown to ADP and release of $P_i$

**adenylyl cyclase** (ad-DEN-uh-lil SY-klase) enzyme that catalyzes transformation of ATP to cyclic AMP

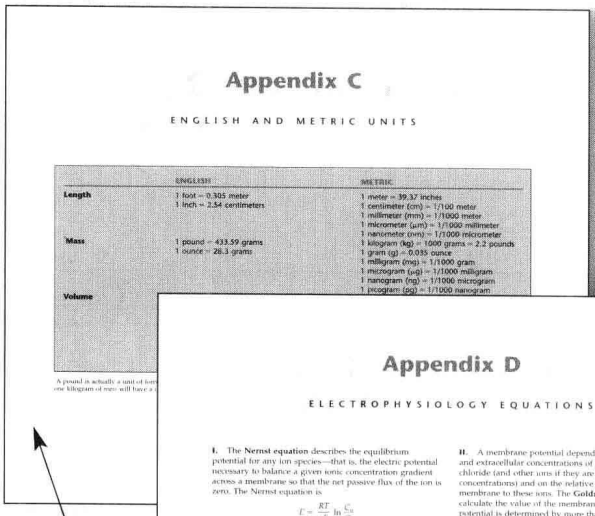
**adipocyte** (ad-DIP-oh-site) cell specialized for triacylglycerol synthesis and storage, fat cell

**adipose tissue** (AD-uh-pose) tissue composed largely of fat storing cells

**adrenal cortex** (ah-DREE-nal KOR-tex) endocrine gland that forms outer shell of each adrenal gland; secretes steroid hormones—mainly cortisol, aldosterone, and androgens; *compare* adrenal medulla

**adrenal gland** one of a pair of endocrine glands above each kidney; each gland consists of outer *adrenal cortex* and inner *adrenal medulla*





# Appendix C

ENGLISH AND METRIC UNITS

| | ENGLISH | METRIC |
|---|---|---|
| **Length** | 1 foot = 0.305 meter<br>1 inch = 2.54 centimeters | 1 meter = 39.37 inches<br>1 centimeter (cm) = 1/100 meter<br>1 millimeter (mm) = 1/1000 meter<br>1 micrometer (μm) = 1/1000 millimeter<br>1 nanometer (nm) = 1/1000 micrometer |
| **Mass** | 1 pound = 453.59 grams<br>1 ounce = 28.3 grams | 1 kilogram (kg) = 1000 grams = 2.2 pounds<br>1 gram (g) = 0.035 ounce<br>1 milligram (mg) = 1/1000 gram<br>1 microgram (μg) = 1/1000 milligram<br>1 nanogram (ng) = 1/1000 microgram<br>1 picogram (pg) = 1/1000 nanogram |
| **Volume** | | |

A pound is actually a unit of force; one kilogram of mass will have a



# Appendix D

ELECTROPHYSIOLOGY EQUATIONS

**I.** The **Nernst equation** describes the equilibrium potential for any ion species—that is, the electric potential necessary to balance a given ionic concentration gradient across a membrane so that the net passive flux of the ion is zero. The Nernst equation is

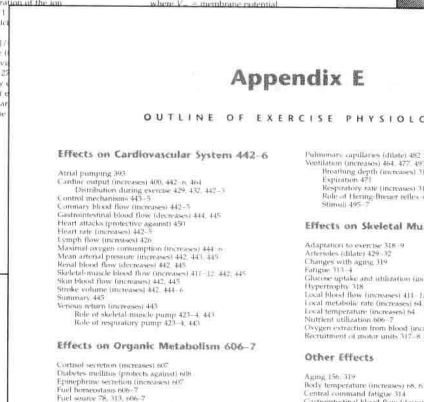
$$E = \frac{RT}{zF} \ln \frac{C_o}{C_i}$$

where $E$ = equilibrium potential for the particular ion in question
$C_i$ = intracellular concentration of the ion
$C_o$ = extracellular concentration of the ion
$z$ = valence of the ion (+1 for potassium, +2 for calcium, −1 for chloride)
$R$ = gas constant [8314.9 J/
$T$ = absolute temperature (
measured on the Kelvin
degrees centigrade + 27
$F$ = Faraday (the quantity
contained in 1 mol of e
96,484 C/mol of char
ln = logarithm taken to the

**II.** A membrane potential depends on the intracellular and extracellular concentrations of potassium, sodium, and chloride (and other ions if they are in sufficient concentrations) and on the relative permeabilities of the membrane to these ions. The **Goldman equation** is used to calculate the value of the membrane potential when the potential is determined by more than one ion species. The Goldman equation is

$$V_m = \frac{RT}{F} \frac{\ln P_K \times K_o + P_{Na} \times Na_o + P_{Cl} \times Cl_i}{P_K \times K_i + P_{Na} \times Na_i + P_{Cl} \times Cl_o}$$

where $V_m$ = membrane potential



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OUTLINE OF EXERCISE PHYSIOLO

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Appendix C presents English-metric interconversions, Appendix D features Electrophysiology equations, and Appendix E is an outline index of Exercise Physiology.

# Correlations

## Dynamic Human 2.0 Correlation Guide

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9-43 Nervous/Explorations/Taste
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## Life Science Animations Correlation Guide

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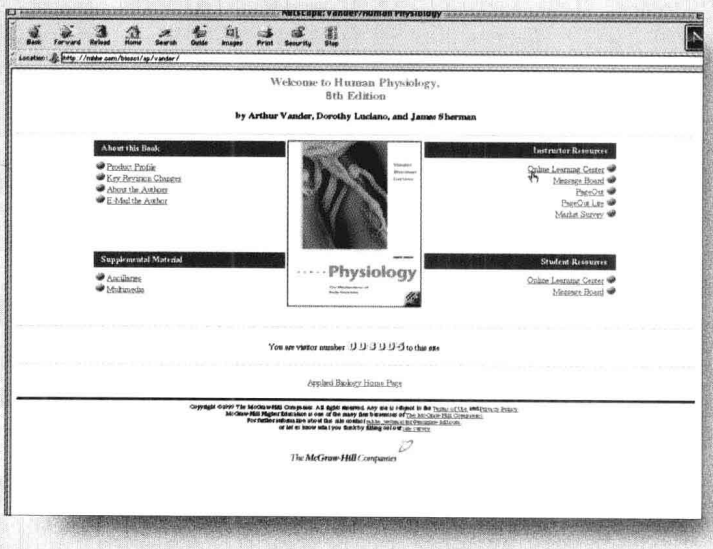


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