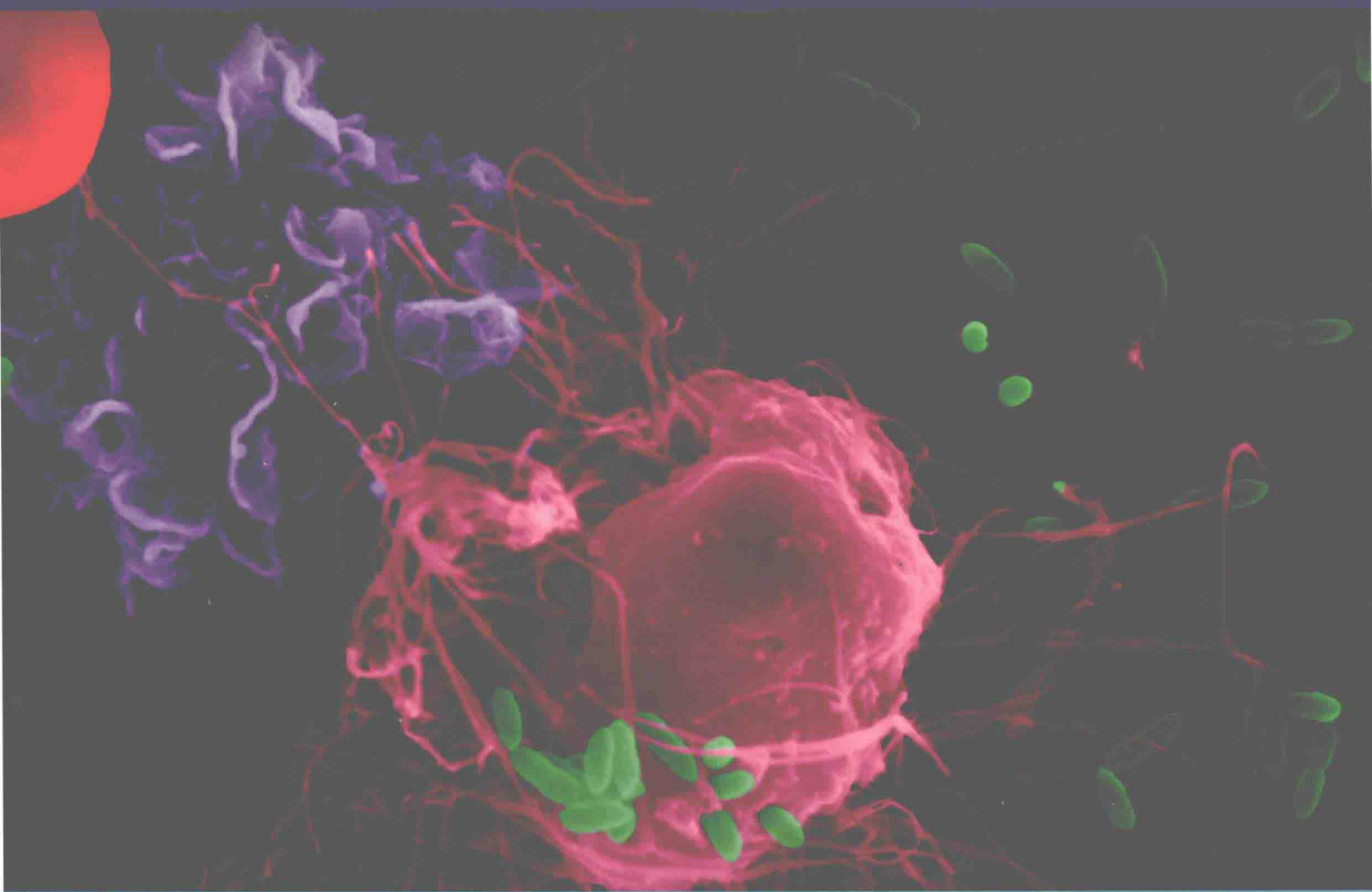
PHARMACOTHERAPY

A PATHOPHYSIOLOGIC APPROACH

NINTH EDITION



Joseph T. DiPiro ▪ Robert L. Talbert ▪ Gary C. Yee
Gary R. Matzke ▪ Barbara G. Wells ▪ L. Michael Posey

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9TH EDITION

PHARMACOTHERAPY

A PATHOPHYSIOLOGIC APPROACH

Joseph T. DiPiro, PharmD, FCCP

Executive Dean and Professor, South Carolina College of Pharmacy, University of South Carolina and Medical University of South Carolina, Charleston and Columbia, South Carolina

Robert L. Talbert, PharmD, FCCP, BCPS, FAHA

Professor, Pharmacotherapy Division, College of Pharmacy, University of Texas at Austin, Professor, Department of Medicine, School of Medicine, University of Texas Health Science Center at San Antonio, San Antonio, Texas

Gary C. Yee, PharmD, FCCP, BCOP

Professor and Associate Dean, Department of Pharmacy Practice, College of Pharmacy, University of Nebraska Medical Center, Omaha, Nebraska

Gary R. Matzke, PharmD, FCP, FCCP, FASN, FNAP

Professor and Director, Pharmacy Practice Transformation Initiatives and Founding Director, ACCP/ASHP/VCU Congressional Health Care Policy Fellow Program, Department of Pharmacotherapy and Outcome Sciences, School of Pharmacy, Virginia Commonwealth University, Richmond, Virginia

Barbara G. Wells, PharmD, FCCP, FASHP

Dean Emeritus and Professor Emeritus, Department of Pharmacy Practice, University of Mississippi, School of Pharmacy, Oxford, Mississippi

L. Michael Posey, BSPharm, MA

Associate Vice President, Periodicals Department, American Pharmacists Association, Washington, District of Columbia

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New York Chicago San Francisco Athens Lisbon Madrid
Mexico City Milan New Delhi Singapore Sydney Toronto

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Dedication

To our patients, who have challenged and inspired us and given meaning to all our endeavors.

To practitioners who continue to improve patient health outcomes and thereby serve as role models for their colleagues and students while clinging tenaciously to the highest standards of practice.

To our mentors, whose vision provided educational and training programs that encouraged our professional growth and challenged us to be innovators in our patient care, research, and education.

To our faculty colleagues for their efforts and support for our mission to provide a comprehensive and challenging educational foundation for the pharmacists of the future.

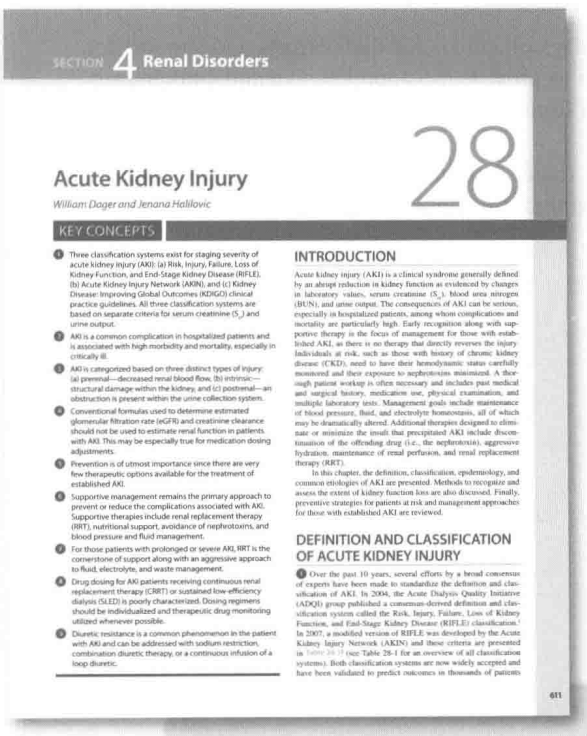
And finally to our families for the time that they have sacrificed so that this ninth edition would become a reality.

No other text helps you achieve optimal patient outcomes through evidence-based medication therapy like DiPiro's

Pharmacotherapy: A Pathophysiologic Approach, Ninth Edition

KEY FEATURES

- Goes beyond drug indications and doses to include drug selection, administration, and monitoring
- Enriched by more than 300 expert contributors
- Revised and updated to reflect the latest evidence-based information and recommendations
- Includes valuable learning aids such Key Concepts at the beginning of each chapter, Clinical Presentation tables that summarize disease signs and symptoms, and Clinical Controversies boxes that examine the complicated issues faced by students and clinicians in providing drug therapy



SECTION 4 Renal Disorders

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Acute Kidney Injury

William Dager and Jenana Halilovic

KEY CONCEPTS

1. Three classification systems exist for staging severity of acute kidney injury (AKI): (a) Risk, Injury, Failure, Loss of Kidney Function, and End-Stage Kidney Disease (RIFLE), (b) Acute Kidney Injury Network (AKIN), and (c) Kidney Disease: Improving Global Outcomes (KDIGO) clinical practice guidelines. All three classification systems are based on separate criteria for serum creatinine (S_{cr}) and urine output.
2. AKI is a common complication in hospitalized patients and is associated with high morbidity and mortality, especially in critically ill.
3. AKI is categorized based on three distinct types of injury: (a) prerenal—decreased renal blood flow, (b) intrinsic—structural damage within the kidney, and (c) postrenal—an obstruction is present within the urine collection system.
4. Conventional formulas used to determine estimated glomerular filtration rate (eGFR) and creatinine clearance should not be used to estimate renal function in patients with AKI. This may be especially true for medication dosing adjustments.
5. Prevention is of utmost importance since there are very few therapeutic options available for the treatment of established AKI.
6. Supportive management remains the primary approach to prevent or reduce the complications associated with AKI. Supportive therapies include renal replacement therapy (RRT), nutritional support, avoidance of nephrotoxins, and blood pressure and fluid management.
7. For those patients with prolonged or severe AKI, RRT is the cornerstone of support along with an aggressive approach to fluid, electrolyte, and waste management.
8. Drug dosing for AKI patients receiving continuous renal replacement therapy (CRRT) or sustained low-efficiency dialysis (SLED) is poorly characterized. Dosing regimens should be individualized and therapeutic drug monitoring utilized whenever possible.
9. Diuretic resistance is a common phenomenon in the patient with AKI and can be addressed with sodium restriction, combination diuretic therapy, or a continuous infusion of a loop diuretic.

INTRODUCTION

Acute kidney injury (AKI) is a clinical syndrome generally defined by an abrupt reduction in kidney function as evidenced by changes in laboratory values, serum creatinine (S_{cr}), blood urea nitrogen (BUN), and urine output. The consequences of AKI can be serious, especially in hospitalized patients, among whom complications and mortality are particularly high. Early recognition along with supportive therapy is the focus of management for those with established AKI, as there is no therapy that directly reverses the injury. Individuals at risk, such as those with history of chronic kidney disease (CKD), need to have their hemodynamic status carefully monitored and their exposure to nephrotoxins minimized. A thorough patient workup is often necessary and includes past medical and surgical history, medication use, physical examination, and multiple laboratory tests. Management goals include maintenance of blood pressure, fluid, and electrolyte homeostasis, all of which may be dramatically altered. Additional therapies designed to eliminate or minimize the insult that precipitated AKI include discontinuation of the offending drug (i.e., the nephrotoxin), aggressive hydration, maintenance of renal perfusion, and renal replacement therapy (RRT).

In this chapter, the definition, classification, epidemiology, and common etiologies of AKI are presented. Methods to recognize and assess the extent of kidney function loss are also discussed. Finally, preventive strategies for patients at risk and management approaches for those with established AKI are reviewed.

DEFINITION AND CLASSIFICATION OF ACUTE KIDNEY INJURY

Over the past 10 years, several efforts by a broad consensus of experts have been made to standardize the definition and classification of AKI. In 2004, the Acute Dialysis Quality Initiative (ADQI) group published a consensus-derived definition and classification system called the Risk, Injury, Failure, Loss of Kidney Function, and End-Stage Kidney Disease (RIFLE) classification.[1] In 2007, a modified version of RIFLE was developed by the Acute Kidney Injury Network (AKIN) and these criteria are presented in Table 28-1 (see Table 28-1 for an overview of all classification systems). Both classification systems are now widely accepted and have been validated to predict outcomes in thousands of patients

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Key Concepts summarize must-know information in each chapter

NEW TO THIS EDITION

- A section on personalized pharmacotherapy appears in most sections
- All diagnostic flow diagrams, treatment algorithms, dosing guideline recommendations, and monitoring approaches have been updated in full color to clearly distinguish treatment pathways
- New drug monitoring tables have been added
- Most of the disease-oriented chapters have incorporated evidence-based treatment guidelines when available, include ratings of the level of evidence to support the key therapeutic approaches
- Twenty-four online-only chapters are available at www.pharmacotherapyonline.com

Valuable tables encapsulate important information

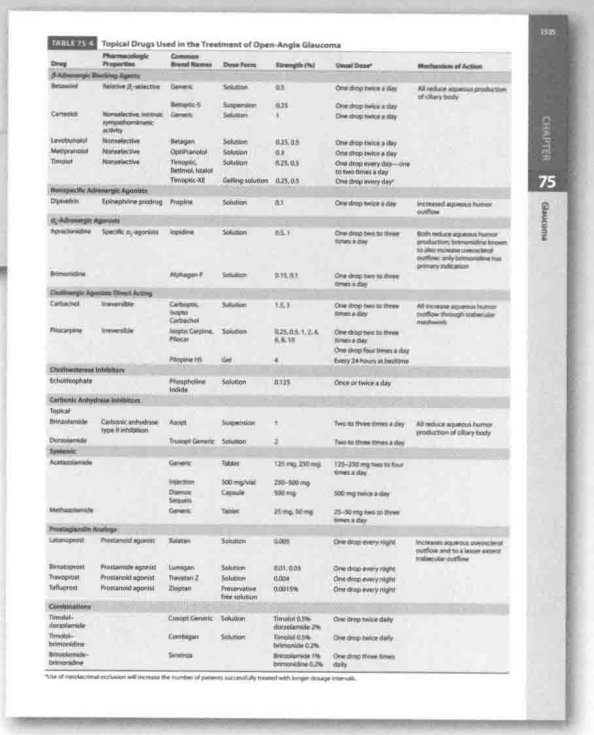

TABLE 75-4 Topical Drugs Used in the Treatment of Open-Angle Glaucoma

Drug	Pharmacologic Properties	Common Brand Names	Dose Form	Strength (%)	Usual Dose*	Mechanism of Action
β-Adrenergic Blocking Agents						
Betaxolol	Relative β_1-selective	Generic	Solution	0.5	One drop twice a day	All reduce aqueous production of ciliary body
		Betoptic-S	Suspension	0.25	One drop twice a day	
Carteolol	Nonselective, Intrinsic sympathomimetic activity	Generic	Solution	1	One drop twice a day	
Levobunolol	Nonselective	Betagan	Solution	0.25, 0.5	One drop twice a day	
Metipranolol	Nonselective	OptiPranolol	Solution	0.3	One drop twice a day	
Timolol	Nonselective	Timoptic, Betimol, Istalol	Solution	0.25, 0.5	One drop every day—one to two times a day	
		Timoptic-XE	Gelling solution	0.25, 0.5	One drop every day†	
Nonspecific Adrenergic Agonists						
Dipivefrin	Epinephrine prodrug	Propine	Solution	0.1	One drop twice a day	Increased aqueous humor outflow
α_2-Adrenergic Agonists						
Apraclonidine	Specific α_2-agonists	Iopidine	Solution	0.5, 1	One drop two to three times a day	Both reduce aqueous humor production; brimonidine known to also increase uveoscleral outflow; only brimonidine has primary indication
Brimonidine		Alphagan P	Solution	0.15, 0.1	One drop two to three times a day	
Cholinergic Agonists: Direct Acting						
Carbachol	Irreversible	Carboptic, Isopto Carbachol	Solution	1.5, 3	One drop two to three times a day	All increase aqueous humor outflow through trabecular meshwork
Pilocarpine	Irreversible	Isopto Carpine, Pilocar	Solution	0.25, 0.5, 1, 2, 4, 6, 8, 10	One drop two to three times a day	
					One drop four times a day	
		Pilopine HS	Gel	4	Every 24 hours at bedtime	
Cholinesterase Inhibitors						
Echothiophate		Phospholine Iodide	Solution	0.125	Once or twice a day	
Carbonic Anhydrase Inhibitors						
Topical						
Brinzolamide	Carbonic anhydrase type II inhibition	Azopt	Suspension	1	Two to three times a day	All reduce aqueous humor production of ciliary body
Dorzolamide		Trusopt Generic	Solution	2	Two to three times a day	
Systemic						
Acetazolamide		Generic	Tablet	125 mg, 250 mg	125–250 mg two to four times a day	
		Injection		500 mg/vial	250–500 mg	
		Diamox Sequels	Capsule	500 mg	500 mg twice a day	
Methazolamide		Generic	Tablet	25 mg, 50 mg	25–50 mg two to three times a day	
Prostaglandin Analogs						
Latanoprost	Prostanoid agonist	Xalatan	Solution	0.005	One drop every night	Increases aqueous uveoscleral outflow and to a lesser extent trabecular outflow
Bimatoprost	Prostamide agonist	Lumigan	Solution	0.01, 0.03	One drop every night	
Travoprost	Prostanoid agonist	Travatan Z	Solution	0.004	One drop every night	
Tafluprost	Prostanoid agonist	Zioptan	Preservative free solution	0.0015%	One drop every night	
Combinations						
Timolol–dorzolamide		Cosopt Generic	Solution	Timolol 0.5% dorzolamide 2%	One drop twice daily	
Timolol–brimonidine		Combigan	Solution	Timolol 0.5% brimonide 0.2%	One drop twice daily	
Brinzolamide–brimonidine		Simbrinza		Brinzolamide 1% brimonidine 0.2%	One drop three times daily	

*Use of nasolacrimal occlusion will increase the number of patients successfully treated with longer dosage intervals.

Full-color illustrations enhance and clarify the text

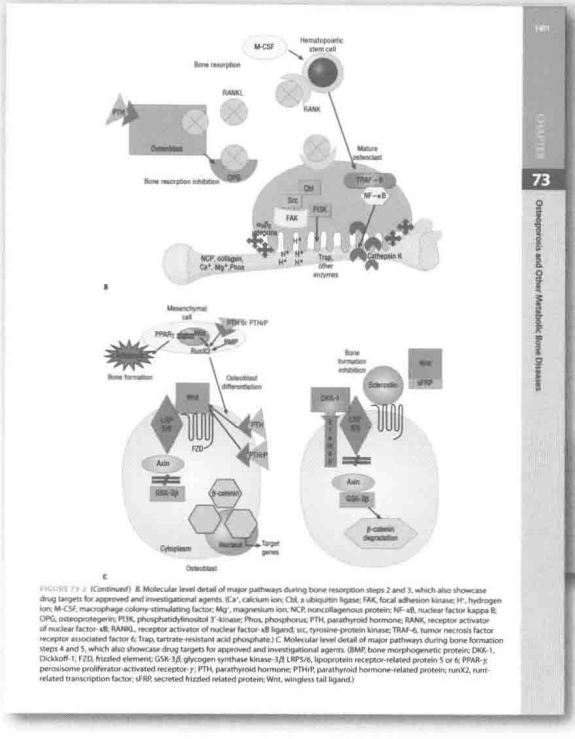

FIGURE 73-2 (Continued) B. Molecular level detail of major pathways during bone resorption steps 2 and 3, which also showcase drug targets for approved and investigational agents. (Ca^+, calcium ion; Cbl, a ubiquitin ligase; FAK, focal adhesion kinase; H^+, hydrogen ion; M-CSF, macrophage colony-stimulating factor; Mg^+, magnesium ion; NCP, noncollagenous protein; NF-κB, nuclear factor kappa B; OPG, osteoprotegerin; PI3K, phosphatidylinositol 3'-kinase; Phos, phosphorus; PTH, parathyroid hormone; RANK, receptor activator of nuclear factor- κB; RANKL, receptor activator of nuclear factor- κB ligand; src, tyrosine-protein kinase; TRAF-6, tumor necrosis factor receptor associated factor 6; Trap, tartrate-resistant acid phosphate.) C. Molecular level detail of major pathways during bone formation steps 4 and 5, which also showcase drug targets for approved and investigational agents. (BMP, bone morphogenetic protein; DKK-1, Dickkoff-1; FZD, frizzled element; GSK-3β, glycogen synthase kinase-3β; LRP5/6, lipoprotein receptor-related protein 5 or 6; PPAR-γ, peroxisome proliferator-activated receptor-γ; PTH, parathyroid hormone; PTHrP, parathyroid hormone-related protein; runX2, runt-related transcription factor; sFRP, secreted frizzled related protein; Wnt, wingless tail ligand.)

Clinical Presentation tables summarize disease signs and symptoms

CLINICAL PRESENTATION **Erectile Dysfunction**

General
- Men are affected emotionally in many different ways
- Depression
- Performance anxiety
- Marital difficulties and avoidance of sexual intimacy (patients are often brought to a physician by their partners)
- Nonadherence to medications patient believes are causing erectile dysfunction

Symptoms
- Erectile dysfunction or inability to have sexual intercourse

Signs
- If completing an International Index of Erectile Dysfunction survey, results are consistent with low satisfaction with the quality of erectile function
- Medical history may identify concurrent medical illnesses, past surgical procedures that interfere with good vascular flow to the penis or damage nerve function to the corpora, or mental disorders associated with decreased reception of sexual stimuli
- Medication history may reveal prescription or nonprescription medications that could cause erectile dysfunction
- Physical examination may reveal signs of hypogonadism (e.g., gynecomastia, small testicles, decreased body hair or beard, and decreased muscle mass), which may contribute to erectile dysfunction. The patient may have an abnormally curved penis when erect, decreased pulses in the pelvic region (suggesting impaired vascular flow to the penis), or decreased anal sphincter tone (suggesting impaired nerve function to the corpora). Men older than 50 years should undergo a digital rectal examination to determine whether an enlarged prostate is contributing to the patient's erectile dysfunction

Laboratory Tests
- If the patient has signs of hypogonadism and complains of decreased libido, a serum testosterone concentration may be below the normal range, which would be consistent with a hormonal cause of erectile dysfunction
- If the patient has an enlarged prostate noted on digital rectal examination, a blood sample for prostate-specific antigen should be obtained. If elevated, the patient should be evaluated for a prostate disorder, which could contribute to erectile dysfunction

A complete listing of the patient's prescription and nonprescription medications and dietary supplements should be reviewed by the clinician, who should identify drugs that may be contributing to erectile dysfunction. If possible, causative agents should be discontinued or the dose should be reduced.

A physical examination of the patient should include a check for hypogonadism (i.e., signs of gynecomastia, small testicles, and decreased beard or body hair). The penis should be evaluated for diseases associated with abnormal penile curvature (e.g., Peyronie's disease), which are associated with erectile dysfunction. Femoral and lower extremity pulses should be assessed to provide an indication of vascular supply to the genitals. Anal sphincter tone and other genital reflexes should be checked for the integrity of the nerve supply to the penis. A digital rectal examination in patients 50 years or older is needed to rule out benign prostatic hyperplasia, which may contribute to erectile dysfunction.

Selected laboratory tests should be obtained to identify the presence of underlying diseases that could cause erectile dysfunction. They include a fasting serum blood glucose and lipid profile. Serum testosterone levels should be checked in patients older than 50 years and in younger patients who complain of decreased libido and erectile dysfunction. At least two early morning serum testosterone levels on different days are needed to confirm the presence of hypogonadism.[7]

Finally, erectile dysfunction is a potential marker for arteriosclerosis. Therefore, older patients and those at intermediate and high risk for cardiovascular disease should undergo a cardiovascular risk assessment before starting on drug treatment for erectile dysfunction. By doing so, patients will be categorized into low-, intermediate-, or high-risk groups for cardiovascular morbidity related to sexual intercourse. Patients in the intermediate-risk group should undergo additional testing to reclassify them into the low- or high-risk group. The high-risk group should defer sexual activity. Patients in the low-risk group may start specific treatment for erectile dysfunction.[8,9,10]

TREATMENT **Erectile Dysfunction**

Desired Outcomes

The goal of treatment is improvement in the quantity and quality of penile erections suitable for intercourse and considered satisfactory by the patient and his partner. Simple as this may sound, healthcare providers must ensure that patients and their partners have reasonable expectations for any therapies that are initiated. Furthermore, only patients with erectile dysfunction should be treated. Patients who have normal sexual function should not seek—or be encouraged to seek—treatment in an effort to enhance sexual function or enable increased activity. In addition, treatment should be well tolerated and be of reasonable cost.

General Approach to Treatment

1 The Third Princeton Consensus Conference is a widely accepted multidisciplinary approach to managing erectile dysfunction that maps out a stepwise treatment plan.[10–12] The first step in clinical management of erectile dysfunction is to identify and, if possible, reverse underlying causes. Risk factors for erectile dysfunction, including hypertension, coronary artery disease, dyslipidemia, diabetes mellitus, smoking, or chronic ethanol abuse, should be

- *Pharmacotherapy Casebook* provides the case studies students need to learn how to identify and resolve the drug therapy problems most likely encountered in real-world practice. This new edition is packed with patient cases and makes the ideal study companion to the 9th edition of DiPiro's *Pharmacotherapy: A Pathophysiologic Approach*.
- Online Learning Center is designed to benefit the student and faculty. Both learning objectives and self-assessment questions for each chapter are available online at www.accesspharmacy.com

Contents

e|CHAPTERS

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Contributors

Val R. Adams, PharmD, FCCP, BCOP
Associate Professor
Department of Pharmacy Practice and Science
College of Pharmacy
University of Kentucky
Lexington, Kentucky
Chapter 106

Jeffrey R. Aeschlimann, PharmD
Associate Professor
Department of Pharmacy Practice
School of Pharmacy
University of Connecticut
Storrs, Connecticut
eChapter 25

Ahmed Alhusban, PharmD, PhD
Assistant Professor
Department of Clinical Pharmacy
Jordan University of Science and Technology
Irbid, Jordan
eChapter 19

Rondall E. Allen, PharmD
Clinical Associate Professor
Associate Dean for Student Affairs
Division of Clinical and Administrative Sciences
Xavier University of Louisiana College of Pharmacy
New Orleans, Louisiana
eChapter 17

Carlos A. Alvarez, PharmD, MSc, MSCS, BCPS
Assistant Professor
Department of Pharmacy Practice
Texas Tech University Health Sciences Center
Dallas, Texas
Chapter 57

JV Anandan, PharmD
Adjunct Associate Professor
Eugene Applebaum College of Pharmacy
Wayne State University, Detroit
Pharmacy Specialist
Center for Drug Use Analysis and Information
Henry Ford Hospital
Department of Pharmacy Services
Detroit, Michigan
Chapter 93

Peter L. Anderson, PharmD
Associate Professor
Department of Pharmaceutical Sciences
Skaggs School of Pharmacy and Pharmaceutical Sciences
University of Colorado Anschutz Medical Campus
Aurora, Colorado
Chapter 103

Tami R. Argo, PharmD, MS
Clinical Pharmacy Specialist-Psychiatry
Department of Pharmacy
Iowa City Veterans Affairs Health Care System
Iowa City, Iowa
Chapter 50

Edward P. Armstrong, PharmD
Professor
Department of Pharmacy Practice and Science
College of Pharmacy
University of Arizona
Tucson, Arizona
Chapter 96

Susanne M. Arnold, MD
Professor
Department of Internal Medicine
Division of Medical Oncology
Markey Cancer Center
University of Kentucky
Lexington, Kentucky
Chapter 106

Jill Astolfi, PharmD
Philadelphia, Pennsylvania
eChapter 9

Rebecca L. Attridge, PharmD, MSc, BCPS
Assistant Professor
Department of Pharmacy Practice
University Incarnate Word Feik School of Pharmacy
Adjunct Assistant Professor
The University of Texas Health Science Center at San Antonio
Division of Pulmonary Diseases and Critical Care Medicine
San Antonio, Texas
Chapter 17

Jacquelyn L. Bainbridge, PharmD, FCCP
Professor
Department of Clinical Pharmacy and Neurology
University of Colorado
Anschutz Medical Campus
Skaggs School of Pharmacy and Pharmaceutical Sciences
Aurora, Colorado
Chapter 39

Jeffrey F. Barletta, PharmD, FCCM
Associate Professor and Vice Chair
Department of Pharmacy Practice
Midwestern University
College of Pharmacy
Glendale, Arizona
Chapter 2

Chad M. Barnett, PharmD, BCOP
Clinical Pharmacy Specialist-Breast Oncology
Division of Pharmacy
Clinical Pharmacy Services
University of Texas MD Anderson Cancer Center
Houston, Texas
Chapter 105

Larry A. Bauer, PharmD, FCP, FCCP
Professor
Department of Pharmacy
School of Pharmacy
Adjunct Professor
Department of Laboratory Medicine
School of Medicine
University of Washington
Seattle, Washington
eChapter 5

Jerry L. Bauman, PharmD, FCCP, FACC
Dean
College of Pharmacy, University of Illinois at Chicago
Professor
Departments of Pharmacy Practice and Medicine, Section of Cardiology
Colleges of Pharmacy and Medicine, University of Illinois at Chicago
Chicago, Illinois
Chapter 8

Terry J. Baumann, PharmD, BCPS
Clinical Manager, Pain Practitioner
Department of Pharmacy
Munson Medical Center
Traverse City, Michigan
Chapter 44

Oralia V. Bazaldua, PharmD, FCCP, BCPS
Associate Professor
Department of Family and Community Medicine
The University of Texas Health Science Center at San Antonio
San Antonio, Texas
eChapter 1

Martha G. Blackford, PharmD
Clinical Pharmacologist & Toxicologist
Clinical Pharmacology and Toxicology
Department of Pediatrics
Akron Children's Hospital
Akron, Ohio
Chapter 85

Scott Bolesta, PharmD, BCPS
Associate Professor
Department of Pharmacy Practice
Wilkes University
Wilkes-Bare, Pennsylvania
Clinical Pharmacist
Regional Hospital of Scranton
Scranton, Pennsylvania
Chapter 25

Jill S. Borchert, PharmD, BCPS, FCCP
Professor and Vice Chair
Department of Pharmacy Practice
Midwestern University Chicago College of Pharmacy
Downers Grove, Illinois
Chapter 73

Bradley A. Boucher, PharmD, FCCP, FCCM
Professor of Clinical Pharmacy and Associate Professor of Neurosurgery
Department of Clinical Pharmacy
University of Tennessee Health Science Center
Memphis, Tennessee
Chapter 42

Sharya V. Bourdet, PharmD, BCPS
Critical Care Pharmacist/Clinical Inpatient Program Manager
Pharmacy Service
Veterans Affairs Medical Center
Health Sciences Clinical Associate Professor
Department of Clinical Pharmacy
School of Pharmacy, University of California
San Francisco, California
Chapter 16

Nancy C. Brahm, PharmD, MS, BCPP, CGP
Clinical Professor
The University of Oklahoma, College of Pharmacy
Tulsa, Oklahoma
Chapter 56

Donald F. Brophy, PharmD, MSc, FCCP, FASN, BCPS
McFarlane Professor and Chairman
Department of Pharmacotherapy and Outcomes Sciences
Virginia Commonwealth University School of Pharmacy
Richmond, Virginia
Chapter 36

Jason E. Brouillard, PharmD, MBA
Clinical Pharmacy Advisor
TheraDoc
Hospira, Inc.
Spokane, Washington
eChapter 11

Thomas E. R. Brown, PharmD
Associate Professor
Leslie Dan Faculty of Pharmacy
University of Toronto and Women's College Hospital
Toronto, Ontario, Canada
Chapter 98

Peter F. Buckley, MD
Dean
Medical College of Georgia
Georgia Regents University
Augusta, Georgia
Chapter 50

David S. Burgess, PharmD, FCCP
Professor and Chair
Department of Pharmacy Practice and Science
College of Pharmacy, University of Kentucky
Lexington, Kentucky
Chapter 83

Lucinda M. Buys, PharmD, BCPS
Associate Professor
Department of Pharmacy Practice and Science
University of Iowa
College of Pharmacy and The Siouxland Medical Education Foundation
Sioux City, Iowa
Chapter 71

Karim Anton Calis, PharmD, MPH, FASHP, FCCP
Clinical Investigator
Office of the Clinical Director
Eunice Kennedy Shriver National Institute of Child Health and Human Development
National Institutes of Health
Bethesda, Maryland
Clinical Professor
Department of Pharmacy Practice and Science
School of Pharmacy, University of Maryland
Baltimore, Maryland
Clinical Professor
Department of Pharmacotherapy and Outcomes Science
School of Pharmacy
Virginia Commonwealth University
Richmond, Virginia
Chapters 60, 65, and 121

Peggy L. Carver, PharmD, FCCP
Associate Professor of Pharmacy
Clinical Pharmacist, Infectious Diseases
Department of Clinical, Social, and Administrative Sciences
University of Michigan College of Pharmacy and University of Michigan Health System
Ann Arbor, Michigan
Chapter 99

Larisa H. Cavallari, PharmD
Associate Professor
Department of Pharmacy Practice
University of Illinois at Chicago
Chicago, Illinois
eChapter 6
Chapter 4

Jose E. Cavazos, MD, PhD
Professor
Departments of Neurology, Pharmacology and Physiology
Assistant Dean for MD/PhD Program
The University of Texas Health Science Center at San Antonio
San Antonio, Texas
Chapter 40

Alexandre Chan, PharmD, MPH, BCPS, BCOP
Associate Professor
Department of Pharmacy
Faculty of Science
National University of Singapore
Associate Consultant Clinical Pharmacist
Department of Pharmacy
National Cancer Centre Singapore
Singapore, Singapore
Chapter 109

C. Y. Jennifer Chan, PharmD
Clinical Assistant Professor of Pharmacy
Pharmacotherapy Education and Research Center
University of Texas at Austin College of Pharmacy
Adjunct Associate Professor of Pediatrics
Department of Pediatrics
University of Texas Health Science Center
San Antonio, Texas
Chapter 82

Jack J. Chen, PharmD, BCPS
Associate Professor
Department of Neurology
Movement Disorders Clinic
Schools of Medicine and Pharmacy
Loma Linda University
Loma Linda, California
Chapter 43

Judy T. Chen, PharmD, BCPS
Clinical Associate Professor
Department of Pharmacy Practice
Purdue University College of Pharmacy
Clinical Pharmacy Specialist
The Jane Pauley Community Health Center, Inc.
Indianapolis, Indiana
Chapter 121

Katherine Hammond Chessman, BSPharm, PharmD
Professor
Clinical Pharmacy and Outcomes Sciences
South Carolina College of Pharmacy, MUSC Campus
Clinical Pharmacy Specialist, Pediatrics/Pediatric Surgery
Department of Pharmacy Services
The Children's Hospital of South Carolina
Charleston, South Carolina
Chapters 34, 118, and 120

Elaine Chiquette
San Antonio, Texas
eChapter 4

Mariann D. Churchwell, PharmD, BCPS
Associate Professor
University of Toledo College of Pharmacy
Toledo, Ohio
Chapter 30

Peter A. Chyka, PharmD, FAACT, DPNAP, DABAT
Professor and Executive Associate Dean
Department of Clinical Pharmacy
University of Tennessee Health Science Center
College of Pharmacy
Knoxville, Tennessee
eChapter 10

Elizabeth C. Clark, MD, MPH
Assistant Professor
Family Medicine and Community Health
Rutgers University, Robert Wood Johnson Medical School
New Brunswick, New Jersey
Chapter 74

Nathan P. Clark, PharmD, BCPS
Clinical Pharmacy Supervisor
Clinical Pharmacy
Anticoagulation and Anemia Service
Kaiser Permanente
Aurora, Colorado
Chapter 9

Kristen Cook, PharmD, BCPS
Assistant Professor
Department of Pharmacy Practice
College of Pharmacy
University of Nebraska Medical Center
Omaha, Nebraska
Chapter 80

John R. Corboy, MD
Professor
Department of Neurology
University of Colorado School of Medicine
Co-Director
Rocky Mountain MS Center
University of Colorado
Anschutz Medical Campus
Aurora, Colorado
Chapter 39

Lisa T. Costanigro, PharmD, BCPS
Staff Pharmacist
Poudre Valley Health System
Fort Collins, Colorado
eChapter 11

Elizabeth A. Coyle, PharmD, FCCM, BCPS
Assistant Dean of Assessment
Clinical Professor
University of Houston
College of Pharmacy
Houston, Texas
Chapter 94

Catherine M. Crill, PharmD, BCPS, BCNSP, FCCP
Associate Professor
Departments of Clinical Pharmacy and Pediatrics
The University of Tennessee Health Science Center
Memphis, Tennessee
Chapter 119

M. Lynn Crismon, PharmD
Dean
James T. Doluisio Regents Chair and Behrens Centennial Professor
College of Pharmacy
The University of Texas at Austin
Austin, Texas
Chapter 50

Michael A. Crouch, PharmD, FASHP, BCPS
Executive Associate Dean and Professor
Department of Pharmacy Practice
Gatton College of Pharmacy
East Tennessee State University
Johnson City, Tennessee
Chapter 89

William Dager, PharmD, BCPS (AQ Cardiology)
Pharmacist Specialist
UC Davis Medical Center
Clinical Professor of Pharmacy
UC San Francisco School of Pharmacy
Clinical Professor of Medicine
UC Davis School of Medicine
Clinical Professor of Pharmacy
Touro School of Pharmacy
Sacramento, California
Chapter 28

Devra K. Dang, PharmD, BCPS, CDE
Associate Clinical Professor
University of Connecticut
School of Pharmacy
Storrs, Connecticut
Chapter 65

Joseph F. Dasta, MSc, FCCM, FCCP
Adjunct Professor
The University of Texas
Professor Emeritus
The Ohio State University
Austin, Texas
Chapter 13

Dewayne A. Davidson, PharmD
Assistant Professor
Department of Family and Community Medicine
The University of Texas Health Science Center at San Antonio
San Antonio, Texas
eChapter 1

Lisa E. Davis, BS, PharmD
Professor of Clinical Pharmacy
Philadelphia College of Pharmacy
University of the Sciences in Philadelphia
Philadelphia, Pennsylvania
Chapter 107

Brian S. Decker, MD, PharmD, MS
Assistant Professor of Clinical Medicine
Department of Medicine, Division of Nephrology
Indiana University School of Medicine
Indianapolis, Indiana
Chapter 30

Timothy Dellenbaugh, MD
Associate Professor
Department of Psychiatry
University of Missouri-Kansas City
Center for Behavioral Medicine
Kansas City, Missouri
eChapter 20

Paulina Deming, PharmD
Associate Professor
Department of Pharmacy Practice
College of Pharmacy
University of New Mexico Health Sciences Center
Albuquerque, New Mexico
Chapter 26

Simon de Denus, B.Pharm, MSc, PhD
Research Pharmacist
Montreal Heart Institute
Associate Professor
Faculty of Pharmacy Université de Montréal
Université de Montréal Beaulieu-Saucier
Chair in Pharmacogenomics
Montreal, Quebec
Chapter 7

John W. Devlin, PharmD, FCCP, FCCM
Associate Professor
Department of Pharmacy Practice
Bouve College of Health Professions
Northeastern University
Boston, Massachusetts
Chapter 37

Eric Dietrich, PharmD
Post Doctoral Fellow
Family Medicine College of Pharmacy and Medicine
University of Florida
Gainesville, Florida
Chapter 59

Cecily V. DiPiro, PharmD
Consultant Pharmacist
Mount Pleasant, South Carolina
Chapter 22

Joseph T. DiPiro, PharmD, FCCP
Executive Dean and Professor
South Carolina College of Pharmacy
University of South Carolina and Medical University
of South Carolina
Charleston and Columbia, South Carolina
Chapter 92

Paul L. Doering, MS
Distinguished Service Professor of Pharmacy Practice Emeritus
Department of Pharmacotherapy and Translational Research
College of Pharmacy
University of Florida
Gainesville, Florida
Chapters 48 and 49

Julie A. Dopheide, PharmD, BCPP
Associate Professor of Clinical Pharmacy, Psychiatry
and the Behavioral Sciences
University of Southern California School of Pharmacy
and Keck School of Medicine
Los Angeles, California
Chapter 46

John M. Dopp, PharmD
Associate Professor
School of Pharmacy
University of Wisconsin
Madison, Wisconsin
Chapter 55

Thomas C. Dowling, PharmD, PhD, FCP
Associate Professor and Vice Chair
Department of Pharmacy Practice and Science
University of Maryland School of Pharmacy
Baltimore, Maryland
eChapter 18

Shannon J. Drayton, PharmD, BCPP
Associate Professor
Clinical Pharmacy and Outcomes Sciences
South Carolina College of Pharmacy
Medical University of South Carolina
Charleston, South Carolina
Chapter 52

Linda D. Dresser, PharmD, FCSHP
Assistant Professor
Leslie Dan Faculty of Pharmacy
University of Toronto
University Health Network, Toronto
Ontario, Canada
Chapter 98

Deepak P. Edward, MD
Jonas S Friedenwald Professor of Ophthalmology and Pathology
Department of Ophthalmology
Wilmer Eye Institute
Johns Hopkins University
Baltimore, Maryland
Chapter 75

Mary Elizabeth Elliott, PharmD, PhD
Associate Professor
Pharmacy Practice Division
School of Pharmacy
University of Wisconsin
Madison, Wisconsin
Chapter 71

Ramy H. Elshaboury, PharmD
Pharmacy Resident in Infectious Diseases
Department of Pharmacy
Abbott Northwestern Hospital, Part of Allina Health
Minneapolis, Minnesota
Chapter 84

Michael E. Ernst, PharmD
Professor (Clinical)
Department of Pharmacy Practice and Science
College of Pharmacy and Department of Family Medicine
Carver College of Medicine
The University of Iowa
Iowa City, Iowa
Chapter 74

Brian L. Erstad, PharmD, FCCM, FCCP, FASHP
Professor
Department of Pharmacy Practice and Science
University of Arizona College of Pharmacy
Tucson, Arizona
Chapter 14

Francisco J. Esteva, MD, PhD
Director
Breast Medical Oncology Program
Division of Hematology-Oncology
Department of Medicine
Associate Director of Clinical Investigation
New York University Cancer Institute
New York University Langone Medical Center
New York, New York
Chapter 105

Patricia H. Fabel, PharmD
Clinical Assistant Professor
Clinical Pharmacy and Outcomes Sciences
South Carolina College of Pharmacy, University of South Carolina Campus
Columbia, South Carolina
Chapter 23

Susan C. Fagan, PharmD, BCPS, FCCP
Jowdy Professor, Assistant Dean
Department of Clinical and Administrative Pharmacy
University of Georgia College of Pharmacy
Augusta, Georgia
eChapter 19
Chapter 10

Christopher A. Fausel, PharmD, MHA, BCOP
Clinical Manager, Oncology Pharmacy
Department of Pharmacy
Indiana University Simon Cancer Center
Indianapolis, Indiana
Chapter 112

Richard G. Fiscella, PharmD, MPH
Clinical Professor Emeritus
Department of Pharmacy Practice
University of Illinois at Chicago
Chicago, Illinois
Chapter 75

Douglas N. Fish, PharmD
Professor and Chair
Department of Clinical Pharmacy
Skaggs School of Pharmacy and Pharmaceutical Sciences
University of Colorado Anschutz Medical Campus
Clinical Specialist in Infectious Diseases/Critical Care
University of Colorado Hospital
Aurora, Colorado
Chapters 88 and 100

Courtney V. Fletcher, PharmD
Dean and Professor
Departments of Pharmacy Practice and Medicine
College of Pharmacy
University of Nebraska Medical Center
Omaha, Nebraska
Chapter 103

Michelle A. Fravel, PharmD
Assistant Professor (Clinical)
Department of Pharmacy Practice and Science and Department of Pharmaceutical Care
University of Iowa College of Pharmacy and University of Iowa Hospitals and Clinics
Iowa City, Iowa
Chapter 74

Bradi Frei, PharmD, MS, BCPS, BCOP
Associate Professor
Department of Pharmacy Practice
Feik School of Pharmacy
University of the Incarnate Word
San Antonio, Texas
Chapter 86

Christopher Frei, PharmD, MS, BCPS
Associate Professor
Division of Pharmacotherapy
College of Pharmacy
The University of Texas at Austin
Austin, Texas
Chapter 86

Melissa Frei-Jones, MD, MSCI
Assistant Professor
Department of Pediatrics
Division of Hematology/Oncology
School of Medicine
University of Texas Health Science Center
San Antonio, Texas
Chapter 82

Mark L. Glover, PharmD
Medical Writer
Global Medical Writing
PPD
Wilmington, North Carolina
Chapter 85

Shelly L. Gray, PharmD, MS
Professor and Vice Chair for Curriculum and Instruction
Department of Pharmacy
Director
Geriatric Pharmacy Program and Plein Certificate
School of Pharmacy
University of Washington
Seattle, Washington
eChapter 8

Alan E. Gross, PharmD, BCPS
Clinical Assistant Professor
Department of Pharmacy Practice
College of Pharmacy
University of Nebraska Medical Center
Adjunct Assistant Professor
Department of Medicine
College of Medicine
Section of Infectious Diseases
University of Nebraska Medical Center
Omaha, Nebraska
Chapter 92

Wayne P. Gulliver, MD, FRCPC
Professor of Dermatology and Medicine
Memorial University of Newfoundland
St. John's Newfoundland and Labrador
Canada
Chapter 78

John G. Gums, PharmD, FCCP
Professor of Pharmacy and Translational Research
Department of Family Medicine
College of Pharmacy and Medicine
University of Florida
Gainesville, Florida
Chapter 59

Emily R. Hajjar, PharmD, BCPS, BCAP, CGP
Associate Professor
Department of Pharmacy Practice
Jefferson School of Pharmacy
Thomas Jefferson University
Philadelphia, Pennsylvania
eChapter 8

Jenana Halilovic, PharmD, BCPS, AAHIVP
Assistant Professor
Department of Pharmacy Practice
Thomas J Long School of Pharmacy and Health Sciences
Stockton, California
Chapter 28

Philip D. Hall, PharmD, FCCP, BCPS, BCOP
Campus Dean and Professor
South Carolina College of Pharmacy
Medical University of South Carolina
Charleston, South Carolina
eChapter 21

Joseph T. Hanlon, PharmD, MS
Professor
Department of Medicine, Pharmacy and Therapeutics, and Epidemiology
University of Pittsburgh
Pittsburgh, Pennsylvania
eChapter 8

Michelle Harkins, MD
Associate Professor of Medicine
Department of Internal Medicine
University of New Mexico Health Sciences Center
Albuquerque, New Mexico
eChapter 15

Mary S. Hayney, PharmD, MPH, FCCP, BCPS
Professor of Pharmacy
University of Wisconsin School of Pharmacy
Madison, Wisconsin
Chapter 102

Brian A. Hemstreet, PharmD, FCCP, BCPS
Associate Professor
Department of Clinical Pharmacy
University of Colorado Skaggs School of Pharmacy and Pharmaceutical Sciences
Aurora, Colorado
Chapter 21

Elizabeth D. Hermsen, PharmD, MBA, BCPS-ID
Clinical Scientific Director
Cubist Pharmaceuticals, Inc.
Lexington, Massachusetts
Adjunct Associate Professor
Department of Pharmacy Practice
College of Pharmacy, University of Nebraska Medical Center
Omaha, Nebraska
Chapters 84 and 87

Chris M. Herndon, PharmD, BCPS, CPE
Associate Professor
Department of Pharmacy Practice
Southern Illinois University Edwardsville
Edwardsville, Illinois
Chapter 44

Lauren R. Hersh, MD
Instructor
Department of Family and Community Medicine
Thomas Jefferson University
Philadelphia, Pennsylvania
eChapter 8

David C. Hess, MD
Professor and Chairman
Department of Neurology
Medical College of Georgia
Augusta, Georgia
Chapter 10

Angela Massey Hill, PharmD, BCPP, CPH
Professor and Chair
Department of Pharmacotherapeutics
University of South Florida College of Pharmacy
Tampa, Florida
Chapter 38

L. David Hillis, MD
Professor and Chair
Department of Medicine
University of Texas Health Science Center
San Antonio, Texas
eChapter 13
Chapter 1

Barbara J. Hoeben, PharmD, MSPharm, BCPS
Pharmacy Flight Commander
Davis-Monthan Air Force Base
Tucson, Arizona
Chapter 12

Jessica S. Holt, PharmD, BCPS (AQ-ID)
Infectious Diseases Pharmacy Coordinator
Department of Pharmacy
Abbott Northwestern Hospital, Part of Allina Health
Minneapolis, Minnesota
Chapter 84

Joanna Q. Hudson, PharmD, BCPS, FASN, FCCP
Associate Professor
Departments of Clinical Pharmacy and Medicine (Nephrology)
The University of Tennessee Health Science Center
Memphis, Tennessee
Chapter 29

Grant F. Hutchins, MD
Assistant Professor, Internal Medicine
Division of Gastroenterology-Hepatology
University of Nebraska Medical Center
Omaha, Nebraska
eChapter 16

Robert J. Ignoffo, PharmD, FASHP, FCSHP
Professor of Pharmacy
Touro University California
Clinical Professor Emeritus
University of California
San Francisco, California
Chapter 22

Arthur A. Islas, MD, MPH
Associate Professor and Director of Sports Medicine
Department of Family and Community Medicine
Paul L. Foster School of Medicine
Texas Tech University Health Sciences Center-El Paso
El Paso, Texas
eChapter 2

Heather J. Johnson, PharmD, BCPS
Assistant Professor
Department of Pharmacy and Therapeutics
University of Pittsburgh School of Pharmacy
Pittsburgh, Pennsylvania
Chapter 70

Shawn E. Johnson, PharmD, MPH
Clinical Generalist Pharmacist
Department of Pharmacy
The Ohio State University Wexner Medical Center
Columbus, Ohio
eChapter 3

Jacqueline Jonklaas, MD, PhD
Associate Professor
Division of Endocrinology
Department of Medicine
Georgetown University
Washington, District of Columbia
Chapter 58

Joseph K. Jordan, PharmD
Associate Professor
Drug Information Specialist
Department of Pharmacy Practice
Butler University College of Pharmacy and Health Sciences
Indiana University Health
Indianapolis, Indiana
Chapter 60

Rose Jung, PharmD, MPH, BCPS
Clinical Associate Professor
Department of Pharmacy Practice
College of Pharmacy and Pharmaceutical Sciences
University of Toledo
Toledo, Ohio
Chapter 91

Thomas N. Kakuda, PharmD
Scientific Director
Department of Clinical Pharmacology
Janssen Research & Development, LLC
Titusville, New Jersey
Chapter 103

Sophia N. Kalantaridou, MD, PhD
Professor of Obstetrics and Gynecology
Department of Obstetrics and Gynecology
University of Ioannina Medical School
Ioannina, Greece
Chapter 65

Judith C. Kando, PharmD, BCPP
Senior Ana Medical Specialist
Sunovion Pharmaceuticals Inc.
Marlborough, Massachusetts
Chapter 51

S. Lena Kang-Birken, PharmD, FCCP
Associate Professor
Department of Pharmacy Practice
School of Pharmacy and Health Sciences
University of the Pacific
Stockton, California
Chapter 97

Salmaan Kanji, PharmD, BSc, Pharm, ACPR
Clinical Pharmacy Specialist
The Ottawa Hospital
Associate Scientist
Ottawa Hospital Research Institute
Adjunct Professor of Pharmacy
University of Montreal
Assistant Professor of Medicine
University of Ottawa
Pharmacy, Clinical Epidemiology
Ottawa, Ontario, Canada
Chapter 101

H. William Kelly, PharmD
Professor Emeritus Pediatrics
Department of Pediatrics
University of New Mexico Health Sciences Center
Albuquerque, New Mexico
Chapter 15

Patrick J. Kiel, PharmD, BCPS, BCOP
Clinical Pharmacy Specialist, Hematology/Stem Cell Transplant
Department of Pharmacy
Indiana University Simon Cancer Center
Indianapolis, Indiana
Chapter 112

William R. Kirchain, PharmD, CDE
Wilbur and Mildred Robichaux Endowed Professorship
Division of Clinical and Administrative Sciences
Xavier University of Louisiana
College of Pharmacy
New Orleans, Louisiana
eChapter 17

Cynthia K. Kirkwood, PharmD, BCPP
Professor
Vice Chair for Education
Department of Pharmacotherapy and Outcomes Science
School of Pharmacy
Virginia Commonwealth University
Richmond, Virginia
Chapters 53 and 54

Jacqueline Klootwyk, PharmD, BCPS
Assistant Professor of Pharmacy Practice
Jefferson School of Pharmacy
Thomas Jefferson University
Philadelphia, Pennsylvania
Chapter 63

Leroy C. Knodel, PharmD
Associate Professor
Department of Surgery
The University of Texas Health Science Center
San Antonio, Texas
Clinical Professor
College of Pharmacy
The University of Texas at Austin
Austin, Texas
Chapter 95

Jill M. Kolesar, PharmD, BCPS, FCCP
Professor
School of Pharmacy
University of Wisconsin
Madison, Wisconsin
Chapter 108

Sunil Kripalani, MD, MSc
Associate Professor
Section of Hospital Medicine
Division of General Internal Medicine and Public Health
Department of Medicine
Vanderbilt University
Nashville, Tennessee
eChapter 1

Vanessa J. Kumpf, PharmD, BCNSP
Clinical Specialist, Nutrition Support
Center for Human Nutrition
Vanderbilt Medical Center
Nashville, Tennessee
Chapters 118 and 120

Po Gin Kwa, MD, FRCPC
Clinical Assistant, Professor of Pediatrics
Faculty of Medicine
Memorial University of Newfoundland and Pediatrician
Eastern Health
St. John's, Newfoundland, Canada
Chapter 79

Sum Lam, PharmD, CGP, BCPS, FASCP
Associate Clinical Professor
Department of Clinical Pharmacy Practice
St. John's University
Queens, New York
Chapter 68

Y. W. Francis Lam, PharmD, FCCP
Professor of Pharmacology and Medicine
Department of Pharmacology
The University of Texas Health Science Center at San Antonio
San Antonio, Texas
Clinical Associate Professor of Pharmacy
University of Texas at Austin
Austin, Texas
eChapter 6

Richard A. Lange, MD, MBA
Professor and Executive Vice Chairman
Department of Medicine
The University of Texas Health Science Center
San Antonio, Texas
eChapter 13
Chapter 1

Kerry L. LaPlante, PharmD, BS
Associate Professor of Pharmacy
Department of Pharmacy Practice
University of Rhode Island College of Pharmacy
Infectious Diseases Pharmacotherapy Specialist
Pharmacy Services
Providence Veterans Affairs Medical Center
Adjunct Associate Professor of Medicine
Division of Infectious Diseases
Warren Alpert School of Medicine
Brown University
Kingston, Rhode Island
eChapter 25

Alan H. Lau, PharmD, FCCP
Professor
Department of Pharmacy Practice
Director, International Clinical Pharmacy Education
University of Illinois at Chicago, College of Pharmacy
Chicago, Illinois
Chapter 32

Michael Lauzardo, MD, MSc
Chief
Division of Infectious Diseases and Global Medicine
Director
Southeastern National Tuberculosis Center
College of Medicine
University of Florida
Gainesville, Florida
Chapter 90

David T.S. Law, BSc, MD, PhD, CCFP
Assistant Professor
Department of Family and Community Medicine
Faculty of Medicine
University of Toronto
Staff, Department of Family Practice
The Scarborough Hospital and Rouge Valley Health System
Scarborough, Ontario, Canada
eChapter 23

Rebecca M. Law, PharmD
School of Pharmacy and Discipline of Family Medicine
Faculty of Medicine
Memorial University of Newfoundland
St. John's Newfoundland, Canada
eChapter 23
Chapters 78 and 79

Grace C. Lee, PharmD, BCPS
Clinical Instructor
Division of Pharmacotherapy
College of Pharmacy
University of Texas at Austin
Austin, Texas
Chapter 83

Mary Lee, PharmD, BCPS, FCCP
Professor of Pharmacy Practice
Chicago College of Pharmacy
Midwestern University
Downers Grove, Illinois
Chapters 66 and 67