Mc Graw Hill Education

哈里森
胃肠病学与肝病学

HARRISON'S Gastroenterology and Hepatology

DAN L. LONGO
ANTHONY S. FAUCI

北京大学医学出版社

哈里森
胃肠病学与肝病学

HARRISON'S Gastroenterology and Hepatology

Editors

Dan L. Longo, MD
Scientific Director, National Institute on Aging,
National Institutes of Health, Bethesda and Baltimore

Anthony S. Fauci, MD
Chief, Laboratory of Immunoregulation;
Director, National Institute of Allergy and Infectious Diseases,
National Institutes of Health, Bethesda

Associate Editor

Carol A. Langford, MD, MHS
Associate Professor of Medicine
Cleveland Clinic, Cleveland

Mc Graw Hill Medical

北京大学医学出版社

图书在版编目（CIP）数据

哈里森胃肠病学与肝病学：英文/（美）娄格（D. L. Longo），福斯（A. S. Fauci）编写
—双语版. —北京：北京大学医学出版社，2011.4
书名原文：HARRISON'S Gastroenterology and Hepatology
ISBN 978-7-5659-0136-2

Ⅰ.①哈… Ⅱ.①娄… ②福… Ⅲ.①胃肠病学—英文②肝疾病—英文 Ⅳ.①R57

中国版本图书馆 CIP 数据核字（2011）第 039917 号

北京市版权局著作权合同登记号：01-2011-1841

Dan L. Longo，Anthony S. Fauci
Harrison's Gastroenterology and Hepatology
ISBN 978-0-07-166333-5

哈里森胃肠病学与肝病学

编　　写： Dan L. Longo Anthony S. Fauci
出版发行： 北京大学医学出版社（电话：010-82802230）
地　　址：（100191）北京市海淀区学院路 38 号　北京大学医学部院内
网　　址： http://www.pumpress.com.cn
E - mail： booksale@bjmu.edu.cn
印　　刷： 北京画中画印刷有限公司
经　　销： 新华书店
责任编辑： 冯智勇　　**责任校对：** 齐　欣　　**责任印制：** 张京生
开　　本： 889mm×1194mm　1/16　　**印张：** 47　　**字数：** 1876 千字
版　　次： 2011 年 6 月第 1 版　2011 年 6 月第 1 次印刷
书　　号： ISBN 978-7-5659-0136-2
定　　价： 198.00 元

出版说明

《哈里森内科学》（Harrison's Principles of Internal Medicine）是一部内科学经典名著，也是美国及多个国家医学院校的首选内科学教科书。该书1945年由美国权威内科学家哈里森（Tinsley R. Harrison）首先提议并组织编写，第1版于1950年问世，并立即引起广泛的赞誉与好评。自此，随着医学科学的发展以及在市场的热销，该书每4年修订一次，历时半个多世纪，已出版至第17版，成为内科学发展的基石和风向标，享有“内科学著作之父”的美誉。

为了读者阅读和携带方便，更专注于内科学各亚科领域，《哈里森内科学》分册系列书问世了。该分册系列以《哈里森内科学》（第17版）中相关领域的内容为蓝本，并参考了《哈里森内科学》（第17版）出版以来的最新文献，强调基础与临床的整合，汇集了本领域内最新的进展，是内科学各亚科领域的权威教科书。

在医学领域，英文原版经典专著经过几十年甚至上百年的发展，在知识点的架构上形成了科学而完备的体系，不但语言规范、地道，而且更新及时，具有权威性和先进性。无论是临床医生、教师还是医学生，有这样一本经典专著放在案头，经常翻阅，不但可以获取医学知识，对提高专业外语水平也大有裨益。

本次引进出版：

- 哈里森心血管病学
- 哈里森肾脏病学
- 哈里森临床神经病学
- 哈里森胃肠病学与肝病学
- 哈里森呼吸病学与危重症医学

北京大学医学出版社

CONTRIBUTORS

Numbers in brackets refer to the chapter(s) written or co-written by the contributor.

JOHN C. ATHERTON, MD
Professor of Gastroenterology; Director, Wolfson Digestive Diseases Centre, University of Nottingham, United Kingdom [25]

JANE C. ATKINSON, DDS
Program Director, Clinical Trials Program, Center for Clinical Research, National Institute of Dental and Craniofacial Research, National Institutes of Health, Bethesda [3]

BRUCE R. BACON, MD
James F. King Endowed Chair in Gastroenterology; Professor of Internal Medicine, Division of Gastroenterology & Hepatology, St. Louis [41, 42]

MIRIAM J. BARON, MD
Instructor in Medicine, Harvard Medical School, Boston [24]

JEAN BERGOUNIOUX, MD
Medical Doctor of Pediatrics, Unité de Pathogénie Microbienne Moléculaire, Paris [27]

HENRY J. BINDER, MD
Professor of Medicine; Professor of Cellular & Molecular Physiology, Yale University, New Haven [15]

BRUCE R. BISTRIAN, MD, PhD
Chief, Clinical Nutrition, Beth Israel Deaconess Medical Center; Professor of Medicine, Harvard Medical School, Boston [54]

MARTIN J. BLASER, MD
Frederick H. King Professor of Internal Medicine; Chair, Department of Medicine; Professor of Microbiology, New York University School of Medicine, New York [25, 28]

GERALD BLOOMFIELD, MD, MPH
Department of Internal Medicine, The Johns Hopkins University School of Medicine, Baltimore [Review and Self-Assessment]

RICHARD S. BLUMBERG, MD
Professor of Medicine, Harvard Medical School; Chief, Division of Gastroenterology, Hepatology and Endoscopy, Brigham and Women's Hospital, Boston [16]

CYNTHIA D. BROWN, MD
Department of Internal Medicine, The Johns Hopkins University School of Medicine, Baltimore [Review and Self-Assessment]

JOAN R. BUTTERTON, MD
Assistant Clinical Professor of Medicine, Harvard Medical School; Clinical Associate in Medicine, Massachusetts General Hospital, Boston [22]

STEPHEN B. CALDERWOOD, MD
Morton N. Swartz, MD Academy Professor of Medicine (Microbiology and Molecular Genetics), Harvard Medical School; Chief, Division of Infectious Diseases, Massachusetts General Hospital, Boston [22]

MICHAEL CAMILLERI, MD
Atherton and Winifred W. Bean Professor; Professor of Medicine and Physiology, Mayo Clinic College of Medicine, Rochester [6]

BRIAN I. CARR, MD, PhD
Professor of Medicine, Thomas Jefferson University; Director of the Liver Tumor Program, Kimmel Cancer Center, Philadelphia [48]

YU JO CHUA, MBBS
Research Fellow (Medical Oncology), Royal Marsden Hospital, London [49]

RAYMOND T. CHUNG, MD
Associate Professor of Medicine, Harvard Medical School; Director of Hepatology, Massachusetts General Hospital; Medical Director, Liver Transplant Program, Massachusetts General Hospital, Boston [44]

DAVID CUNNINGHAM, MD
Professor of Cancer Medicine, Institute of Cancer Research; Consultant Medical Oncologist, Head of Gastrointestinal Unit, Royal Marsden Hospital, London [49]

JOHN DEL VALLE, MD
Professor and Senior Associate Chair of Graduate Medical Education, Department of Internal Medicine, Division of Gastroenterology, University of Michigan Health System, Ann Arbor [14]

JULES L. DIENSTAG, MD
Carl W. Walter Professor of Medicine and Dean for Medical Education, Harvard Medical School; Physician, Gastrointestinal Unit, Massachusetts General Hospital, Boston [37–39, 44]

DAVID F. DRISCOLL, PhD
Assistant Professor of Medicine, Harvard Medical School, Boston [54]

SAMUEL C. DURSO, MD, MBA
Associate Professor of Medicine, Clinical Director, Division of Geriatric Medicine and Gerontology, The Johns Hopkins University School of Medicine, Baltimore [2, 3]

JOHANNA DWYER, DSc, RD
Professor of Medicine and Community Health, Tufts University School of Medicine and Friedman School of Nutrition Science and Policy; Senior Scientist Jean Mayer Human Nutrition Research Center on Aging at Tufts; Director of the Frances Stern Nutrition Center, Tufts-New England Medical Center Hospital, Boston [51]

ROBERT H. ECKEL, MD
Professor of Medicine, Division of Endocrinology, Metabolism and Diabetes, Division of Cardiology; Professor of Physiology and Biophysics; Charles A. Boettcher II Chair in Atherosclerosis; Program Director, Adult General Clinical Research Center, University of Colorado at Denver and Health Sciences Center; Director Lipid Clinic, University Hospital, Aurora [58]

DANIEL J. FINK,† MD, MPH
Associate Professor of Clinical Pathology, College of Physicians and Surgeons, Columbia University, New York [Appendix]

JEFFREY S. FLIER, MD
Caroline Shields Walker Professor of Medicine, Harvard Medical School; Dean of the Faculty of Medicine, Harvard School of Medicine, Boston [55]

†Deceased.

SONIA FRIEDMAN, MD
Assistant Professor of Medicine, Harvard Medical School; Associate Physician, Brigham and Women's Hospital, Boston [16]

SUSAN L. GEARHART, MD
Assistant Professor of Colorectal Surgery and Oncology, The Johns Hopkins University School of Medicine, Baltimore [18–21]

DALE N. GERDING, MD
Assistant Chief of Staff for Research, Hines VA Hospital, Hines; Professor, Stritch School of Medicine, Loyola University, Maywood [23]

MARC GHANY, MD
Staff Physician, Liver Diseases Branch, National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda [34]

ROGER I. GLASS, MD, PhD
Director, Fogarty International Center; Associate Director for International Research, National Institutes of Health, Bethesda [30]

ROBERT M. GLICKMAN, MD
Professor of Medicine, New York University School of Medicine, New York [10]

RAJ K. GOYAL, MD
Mallinckrodt Professor of Medicine, Harvard Medical School, Boston; Physician, VA Boston Healthcare and Beth Israel Deaconess Medical Center, West Roxbury [4, 13]

NORTON J. GREENBERGER, MD
Clinical Professor of Medicine, Harvard Medical School; Senior Physician, Brigham and Women's Hospital, Boston [43, 45, 46]

WILLIAM L. HASLER, MD
Professor of Medicine, Division of Gastroenterology, University of Michigan Health System, Ann Arbor [5, 11]

DOUGLAS C. HEIMBURGER, MD, MS
Professor of Nutrition Sciences; Professor of Medicine; Director, Clinical Nutrition Fellowship Program, University of Alabama at Birmingham, Birmingham [53]

JAY H. HOOFNAGLE, MD
Director, Liver Diseases Research Branch, Division of Digestive Diseases and Nutrition, National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda [34]

ROBERT T. JENSEN, MD
Chief, Digestive Diseases Branch, National Institute of Diabetes, Digestive and Kidney Diseases, National Institutes of Health, Bethesda [50]

STUART JOHNSON, MD
Associate Professor, Stritch School of Medicine, Loyola University, Maywood; Staff Physician, Hines VA Hospital, Hines [23]

MARSHALL M. KAPLAN, MD
Professor of Medicine, Tufts University School of Medicine; Chief Emeritus, Division of Gastroenterology, Tufts-New England Medical Center, Boston [9, 35]

DENNIS L. KASPER, MD, MA (Hon)
William Ellery Channing Professor of Medicine, Professor of Microbiology and Molecular Genetics, Harvard Medical School; Director, Channing Laboratory, Department of Medicine, Brigham and Women's Hospital, Boston [24]

GERALD T. KEUSCH, MD
Associate Provost and Associate Dean for Global Health, Boston University School of Medicine, Boston [29]

ALEXANDER KRATZ, MD, PhD, MPH
Assistant Professor of Clinical Pathology, Columbia University College of Physicians and Surgeons; Associate Director, Core Laboratory, Columbia University Medical Center, New York-Presbyterian Hospital; Director, Allen Pavilion Laboratory, New York [Appendix]

ROBERT F. KUSHNER, MD
Professor of Medicine, Northwestern University Feinberg School of Medicine, Chicago [56]

LOREN LAINE, MD
Professor of Medicine, Keck School of Medicine, University of Southern California, Los Angeles [8]

MARK E. MALLIARD, MD
Associate Professor and Chief, Division of Gastroenterology and Hepatology, Omaha [40]

ELEFTHERIA MARATOS-FLIER, MD
Associate Professor of Medicine, Harvard Medical School; Chief, Obesity Section, Joslin Diabetes Center, Boston [55]

ROBERT J. MAYER, MD
Stephen B. Kay Family Professor of Medicine, Harvard Medical School, Dana- Farber Cancer Institute, Boston [47]

SAMUEL I. MILLER, MD
Professor of Genome Sciences, Medicine, and Microbiology, University of Washington, Seattle [26]

JOSEPH A. MURRAY, MD
Professor of Medicine, Division of Gastroenterology and Hepatology, The Mayo Clinic, Rochester [6]

THOMAS B. NUTMAN, MD
Head, Helminth Immunology Section; Head, Clinical Parasitology Unit; Laboratory of Parasitic Diseases, National Institute of Allergy and Infectious Diseases, National Insitutes of Health, Bethesda [33]

CHUNG OWYANG, MD
Professor of Internal Medicine, H. Marvin Pollard Collegiate Professor; Chief, Division of Gastroenterology, University of Michigan Health System, Ann Arbor [11, 17]

UMESH D. PARASHAR, MBBS, MPH
Lead, Enteric and Respiratory Viruses Team, Epidemiology Branch, Division of Viral Diseases, National Center for Immunization and Respiratory Diseases, Centers for Disease Control and Prevention, Atlanta [30]

GUSTAV PAUMGARTNER, MD
Professor of Medicine, University of Munich, Munich, Germany [43]

DAVID A. PEGUES, MD
Professor of Medicine, Division of Infectious Diseases, David Geffen School of Medicine at UCLA, Los Angeles [26]

MICHAEL A. PESCE, PhD
Clinical Professor of Pathology, Columbia University College of Physicians and Surgeons; Director of Specialty Laboratory, New York Presbyterian Hospital, Columbia University Medical Center, New York [Appendix]

DANIEL S. PRATT, MD
Assistant Professor of Medicine, Harvard Medical School; Director, Liver- Billary-Pancreas Center, Massachusetts General Hospital, Boston [9, 35]

ROSHINI RAJAPAKSA, MD, BA
Assistant Professor, Department of Medicine, Gastroenterology, New York University Medical Center School of Medicine and Hospitals Center, New York [10]

SHARON L. REED, MD
Professor of Pathology and Medicine; Director, Microbiology and Virology Laboratories, University of California, San Diego Medical Center, San Diego [31]

CAROL M. REIFE, MD
Clinical Associate Professor of Medicine, Jefferson Medical College, Philadelphia [7]

ROBERT M. RUSSELL, MD
Director, Jean Mayer USDA Human Nutrition Research Center on Aging at Tufts University; Professor of Medicine and Nutrition, Tufts University, Boston [52]

PHILIPPE SANSONETTI
Professeur á l'Institut Pasteur, Paris, France [27]

JOSHUA SCHIFFER, MD
Department of Internal Medicine, The Johns Hopkins University School of Medicine, Baltimore [Review and Self-Assessment]

WILLIAM SILEN, MD
Johnson and Johnson Distinguished Professor of Surgery, Emeritus, Harvard Medical School, Boston [1, 20, 21]

MICHAEL F. SORRELL, MD
Robert L. Grissom Professor of Medicine, University of Nebraska Medical Center, Omaha [40]

ADAM SPIVAK, MD
Department of Internal Medicine, The Johns Hopkins University School of Medicine, Baltimore [Review and Self-Assessment]

PAOLO M. SUTER, MD, MS
Professor of Medicine, Medical Policlinic, Zurich, Switzerland [52]

MARK TOPAZIAN, MD
Associate Professor of Medicine, Mayo College of Medicine, Rochester [12]

PHILLIP P. TOSKES, MD
Professor of Medicine, Division of Gastroenterology, Hepatology and Nutrition, University of Florida College of Medicine, Gainesville [45, 46]

MATTHEW K. WALDOR, MD, PhD
Professor of Medicine (Microbiology and Molecular Genetics), Channing Laboratory, Brigham and Women's Hospital, Harvard Medical School, Boston [29]

B. TIMOTHY WALSH, MD
Professor of Psychiatry, College of Physicians & Surgeons, Columbia University; Director, Eating Disorders Research Unit, New York Psychiatric Institute, New York [57]

PETER F. WELLER, MD
Professor of Medicine, Harvard Medical School; Co-Chief, Infectious Diseases Division; Chief, Allergy and Inflammation Division; Vice-Chair for Research, Department of Medicine, Beth Israel Deaconess Medical Center, Boston [32, 33]

CHARLES WIENER, MD
Professor of Medicine and Physiology; Vice Chair, Department of Medicine; Director, Osler Medical Training Program, The Johns Hopkins University School of Medicine, Baltimore [Review and Self-Assessment]

ALLAN W. WOLKOFF, MD
Professor of Medicine and Anatomy and Structural Biology; Director, Belfer Institute for Advanced Biomedical Studies; Associate Chair of Medicine for Research; Chief, Division of Hepatology, Albert Einstein College of Medicine, Bronx [36]

LOUIS MICHEL WONG-KEE-SONG, MD
Assistant Professor of Medicine, Division of Gastroenterology and Hepatology, Mayo College of Medicine, Rochester [12]

JANET A. YELLOWITZ, DMD, MPH
Associate Professor; Director, Geriatric Dentistry, The Johns Hopkins University School of Medicine, Baltimore [3]

PREFACE

Harrison's Principles of Internal Medicine (HPIM) has long been a major source of information related to the practice of medicine for many practitioners and trainees. Yet in its aim to cover the broad spectrum of medicine, the book has become nearly 3000 pages in length and is pushing the envelope of "portability." *HPIM* has spawned several offspring tailored to diverse uses for sources of medical information. The entire book plus a large cache of supplemental visual and textual information is available as *Harrison's Online*, a component of McGraw-Hill's *Access Medicine* offering. A condensed version of *HPIM*, called *Harrison's Manual of Medicine,* has been published in print format suitable for carrying in a white coat pocket and in several electronic formats (PDA, Blackberry, iPhone). A companion to *HPIM* that serves as a study guide for standardized tests in medicine, *HPIM Self-Assessment and Board Review*, is an effective teaching tool that highlights important areas of medicine discussed in *HPIM. Harrison's Practice* is another electronic information source, organized by medical topic or diagnosis with information presented in a consistent structured format for ease of finding specific information to facilitate clinical care and decision-making at the bedside. All of these products retain the broad spectrum of topics presented in the *HPIM* "mother book" in variable degrees of depth.

In 2006, for the first time, the Editors of *HPIM* experimented with extracting portions of *HPIM* that were focused on a specific subspecialty of internal medicine. The products of that effort, *Harrison's Endocrinology, Harrison's Rheumatology,* and *Harrison's Neurology in Clinical Medicine*, were very well-received by audiences keenly interested in the respective subspecialities of internal medicine. Accordingly, we are expanding the effort to include books focused on other specialties.

According to a report from the National Institute of Diabetes and Digestive and Kidney Diseases, for every 100 residents of the United States, there were 35 ambulatory care contacts and 5 overnight hospital stays at which a digestive disease diagnosis was noted. In 2004, digestive diseases accounted for more than 236,000 deaths. Thus, training in the disciplines of gastroenterology and hepatology is essential to any primary care physician or general internist and even to practitioners of other internal medicine subspecialties.

This book is aimed at bringing together the chapters of *HPIM* related to gastroenterology and hepatology in a conveniently sized book for a focused study of this medical subspecialty. The book is organized into 58 chapters and 11 sections: (I) Cardinal Manifestations of Gastrointestinal Disease; (II) Evaluation of the Patient with Alimentary Tract Symptoms; (III) Disorders of the Alimentary Tract; (IV) Infections of the Alimentary Tract; (V) Evaluation of the Patient with Liver Disease; (VI) Disorders of the Liver and Biliary Tree; (VII) Liver Transplantation; (VIII) Disorders of the Pancreas; (IX) Neoplastic Diseases of the Gastrointestinal System; (X) Nutrition; and (XI) Obesity and Eating Disorders.

The information presented here is contributed by physician/authors who have personally made notable advances in the fields of their expertise. The chapters reflect authoritative analyses by individuals who have been active participants in the amazing surge of new information on genetics, cell biology, pathophysiology, and treatment that has characterized all of medicine in the last 20 years. In addition to the didactic value of the chapters, a section of test questions, answers, and an explanation of the correct answers is provided to facilitate learning and assist the reader in preparing for standardized examinations.

Gastroenterology and hepatology, like many other areas of medicine, are changing rapidly. Novel technologies of imaging, development of new drugs, and the application of molecular pathogenesis information to detect disease early and prevent disease in people at risk are just a few of the advances that have made an impact on the practice of gastroenterology. Physicians are now applying endoscopic techniques in ways that were once unimaginable including performing operations successfully without an incision; operations that once required major surgery with attendant morbidity and expense. The pace of discovery demands that physicians undertake nearly continuous self-education. It is our hope that this book will help physicians in this process.

We are grateful to Kim Davis and James Shanahan at McGraw-Hill for their help in producing this book.

Dan L. Longo, MD
Anthony S. Fauci, MD

NOTICE

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

Review and self-assessment questions and answers were taken from Wiener C, Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, Loscalzo J (editors) Bloomfield G, Brown CD, Schiffer J, Spivak A (contributing editors). *Harrison's Principles of Internal Medicine Self-Assessment and Board Review,* 17th ed. New York, McGraw-Hill, 2008, ISBN 978-0-07-149619-3.

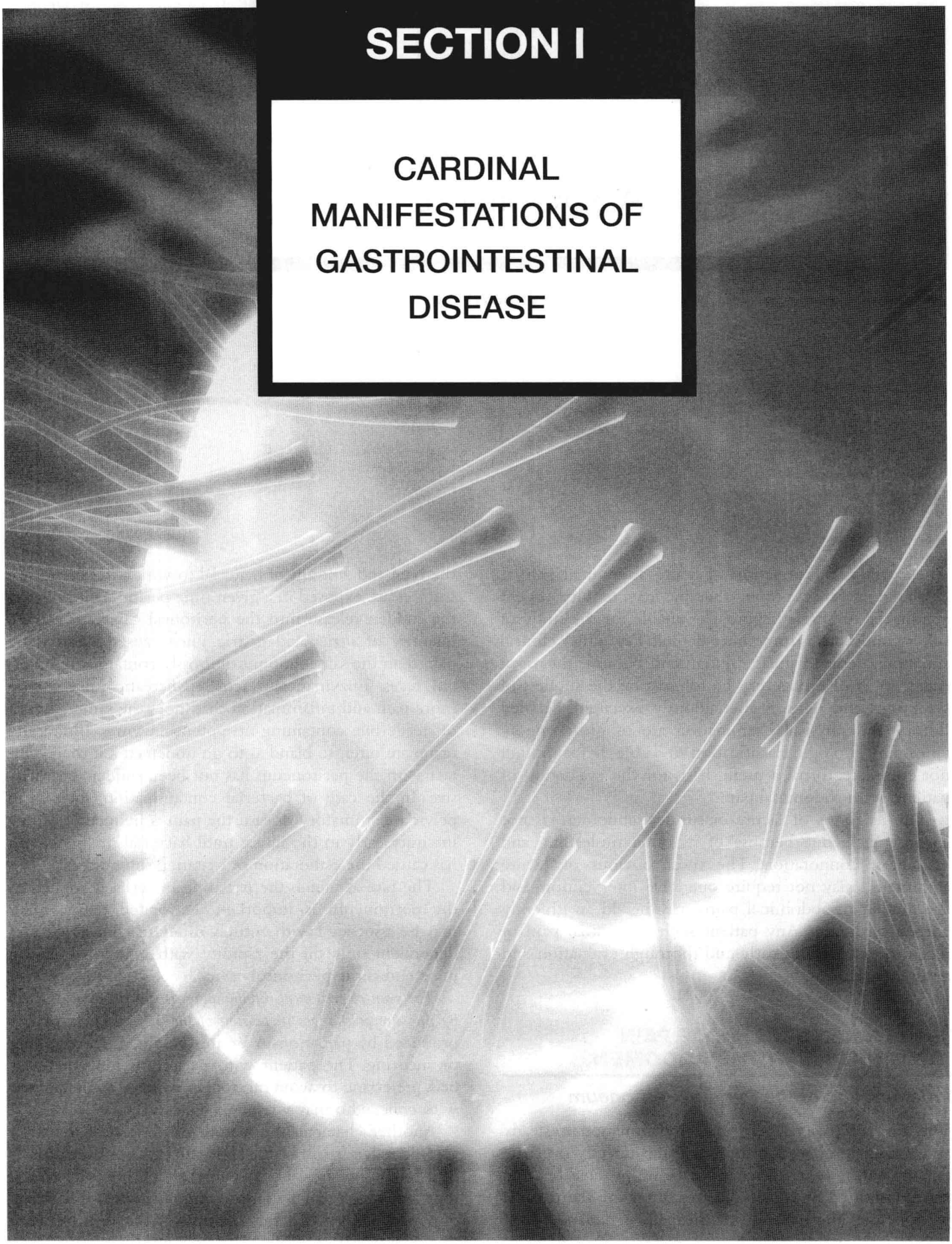
The global icons call greater attention to key epidemiologic and clinical differences in the practice of medicine throughout the world.

The genetic icons identify a clinical issue with an explicit genetic relationship.

CONTENTS

SECTION XI

OBESITY AND EATING DISORDERS



SECTION I

CARDINAL MANIFESTATIONS OF GASTROINTESTINAL DISEASE

CHAPTER 1
ABDOMINAL PAIN

William Silen

The correct interpretation of acute abdominal pain is challenging. Since proper therapy may require urgent action, the unhurried approach suitable for the study of other conditions is sometimes denied. Few other clinical situations demand greater judgment, because the most catastrophic of events may be forecast by the subtlest of symptoms and signs. A meticulously executed, detailed history and physical examination are of great importance. The etiologic classification in **Table 1-1**, although not complete, forms a useful basis for the evaluation of patients with abdominal pain.

The diagnosis of "acute or surgical abdomen" is not an acceptable one because of its often misleading and erroneous connotation. The most obvious of "acute abdomens" may not require operative intervention, and the mildest of abdominal pains may herald an urgently correctable lesion. Any patient with abdominal pain of recent onset requires early and thorough evaluation and accurate diagnosis.

SOME MECHANISMS OF PAIN ORIGINATING IN THE ABDOMEN

Inflammation of the Parietal Peritoneum

The pain of parietal peritoneal inflammation is steady and aching in character and is located directly over the inflamed area, its exact reference being possible because it is transmitted by somatic nerves supplying the parietal peritoneum. The intensity of the pain is dependent on the type and amount of material to which the peritoneal surfaces are exposed in a given time period. For example, the sudden release into the peritoneal cavity of a small quantity of *sterile* acid gastric juice causes much more pain than the same amount of grossly contaminated neutral feces. Enzymatically active pancreatic juice incites more pain and inflammation than does the same amount of sterile bile containing no potent enzymes. Blood and urine are often so bland as to go undetected if their contact with the peritoneum has not been sudden and massive. In the case of bacterial contamination, such as in pelvic inflammatory disease, the pain is frequently of low intensity early in the illness until bacterial multiplication has caused the elaboration of irritating substances.

The rate at which the irritating material is applied to the peritoneum is important. Perforated peptic ulcer may be associated with entirely different clinical pictures dependent only on the rapidity with which the gastric juice enters the peritoneal cavity.

The pain of peritoneal inflammation is invariably accentuated by pressure or changes in tension of the peritoneum, whether produced by palpation or by movement, as in coughing or sneezing. The patient with peritonitis lies quietly in bed, preferring to avoid motion, in contrast to the patient with colic, who may writhe incessantly.

Another characteristic feature of peritoneal irritation is tonic reflex spasm of the abdominal musculature, localized to the involved body segment. The intensity of the tonic muscle spasm accompanying peritoneal inflammation is dependent on the location of the inflammatory

TABLE 1-1

SOME IMPORTANT CAUSES OF ABDOMINAL PAIN

PAIN ORIGINATING IN THE ABDOMEN	
Parietal peritoneal inflammation	Vascular disturbances
Bacterial contamination	Embolism or thrombosis
Perforated appendix or other perforated viscus	Vascular rupture
Pelvic inflammatory disease	Pressure or torsional occlusion
Chemical irritation	Sickle cell anemia
Perforated ulcer	Abdominal wall
Pancreatitis	Distortion or traction of mesentery
Mittelschmerz	Trauma or infection of muscles
Mechanical obstruction of hollow viscera	Distension of visceral surfaces, e.g., by hemorrhage
Obstruction of the small or large intestine	Hepatic or renal capsules
Obstruction of the biliary tree	Inflammation of a viscus
Obstruction of the ureter	Appendicitis
	Typhoid fever
	Typhlitis
PAIN REFERRED FROM EXTRAABDOMINAL SOURCE	
Cardiothoracic	Pneumothorax
Acute myocardial infarction	Empyema
Myocarditis, endocarditis, pericarditis	Esophageal disease, spasm, rupture, inflammation
Congestive heart failure	Genitalia
Pneumonia	Torsion of the testis
Pulmonary embolus	
Pleurodynia	
METABOLIC CAUSES	
Diabetes	Familial Mediterranean fever
Uremia	Porphyria
Hyperlipidemia	C1-esterase inhibitor deficiency (angioneurotic edema)
Hyperparathyroidism	
Acute adrenal insufficiency	
NEUROLOGIC/PSYCHIATRIC CAUSES	
Herpes zoster	Spinal cord or nerve root compression
Tabes dorsalis	Functional disorders
Causalgia	Psychiatric disorders
Radiculitis from infection or arthritis	
TOXIC CAUSES	
Lead poisoning	Black widow spiders
Insect or animal envenomations	Snake bites
UNCERTAIN MECHANISMS	
Narcotic withdrawal	Heat stroke

process, the rate at which it develops, and the integrity of the nervous system. Spasm over a perforated retrocecal appendix or perforated ulcer into the lesser peritoneal sac may be minimal or absent because of the protective effect of overlying viscera. A slowly developing process often greatly attenuates the degree of muscle spasm. Catastrophic abdominal emergencies such as a perforated ulcer may be associated with minimal or no detectable pain or muscle spasm in obtunded, seriously ill, debilitated elderly patients or in psychotic patients.

Obstruction of Hollow Viscera

The pain of obstruction of hollow abdominal viscera is classically described as intermittent, or colicky. Yet the lack of a truly cramping character should not be misleading, because distention of a hollow viscus may produce steady pain with only very occasional exacerbations. It is not nearly as well localized as the pain of parietal peritoneal inflammation.

The colicky pain of obstruction of the small intestine is usually periumbilical or supraumbilical and is poorly

localized. As the intestine becomes progressively dilated with loss of muscular tone, the colicky nature of the pain may diminish. With superimposed strangulating obstruction, pain may spread to the lower lumbar region if there is traction on the root of the mesentery. The colicky pain of colonic obstruction is of lesser intensity than that of the small intestine and is often located in the infraumbilical area. Lumbar radiation of pain is common in colonic obstruction.

Sudden distention of the biliary tree produces a steady rather than colicky type of pain; hence the term *biliary colic* is misleading. Acute distention of the gallbladder usually causes pain in the right upper quadrant with radiation to the right posterior region of the thorax or to the tip of the right scapula, and distention of the common bile duct is often associated with pain in the epigastrium radiating to the upper part of the lumbar region. Considerable variation is common, however, so that differentiation between these may be impossible. The typical subscapular pain or lumbar radiation is frequently absent. Gradual dilatation of the biliary tree, as in carcinoma of the head of the pancreas, may cause no pain or only a mild aching sensation in the epigastrium or right upper quadrant. The pain of distention of the pancreatic ducts is similar to that described for distention of the common bile duct but, in addition, is very frequently accentuated by recumbency and relieved by the upright position.

Obstruction of the urinary bladder results in dull suprapubic pain, usually low in intensity. Restlessness without specific complaint of pain may be the only sign of a distended bladder in an obtunded patient. In contrast, acute obstruction of the intravesicular portion of the ureter is characterized by severe suprapubic and flank pain that radiates to the penis, scrotum, or inner aspect of the upper thigh. Obstruction of the ureteropelvic junction is felt as pain in the costovertebral angle, whereas obstruction of the remainder of the ureter is associated with flank pain that often extends into the same side of the abdomen.

Vascular Disturbances

A frequent misconception, despite abundant experience to the contrary, is that pain associated with intraabdominal vascular disturbances is sudden and catastrophic in nature. The pain of embolism or thrombosis of the superior mesenteric artery, or that of impending rupture of an abdominal aortic aneurysm, certainly may be severe and diffuse. Yet just as frequently, the patient with occlusion of the superior mesenteric artery has only mild continuous diffuse pain for 2 or 3 days before vascular collapse or findings of peritoneal inflammation appear. The early, seemingly insignificant discomfort is caused by hyperperistalsis rather than peritoneal inflammation. Indeed, absence of tenderness and rigidity in the presence of continuous, diffuse pain in a patient likely to have vascular disease is quite characteristic of occlusion of the superior mesenteric artery. Abdominal pain with radiation to the sacral region, flank, or genitalia should always signal the possible presence of a rupturing abdominal aortic aneurysm. This pain may persist over a period of several days before rupture and collapse occur.

Abdominal Wall

Pain arising from the abdominal wall is usually constant and aching. Movement, prolonged standing, and pressure accentuate the discomfort and muscle spasm. In the case of hematoma of the rectus sheath, now most frequently encountered in association with anticoagulant therapy, a mass may be present in the lower quadrants of the abdomen. Simultaneous involvement of muscles in other parts of the body usually serves to differentiate myositis of the abdominal wall from an intraabdominal process that might cause pain in the same region.

REFERRED PAIN IN ABDOMINAL DISEASES

Pain referred to the abdomen from the thorax, spine, or genitalia may prove a vexing diagnostic problem, because diseases of the upper part of the abdominal cavity such as acute cholecystitis or perforated ulcer are frequently associated with intrathoracic complications. A most important, yet often forgotten, dictum is that the possibility of intrathoracic disease must be considered in every patient with abdominal pain, especially if the pain is in the upper part of the abdomen. Systematic questioning and examination directed toward detecting myocardial or pulmonary infarction, pneumonia, pericarditis, or esophageal disease (the intrathoracic diseases that most often masquerade as abdominal emergencies) will often provide sufficient clues to establish the proper diagnosis. Diaphragmatic pleuritis resulting from pneumonia or pulmonary infarction may cause pain in the right upper quadrant and pain in the supraclavicular area, the latter radiation to be distinguished from the referred subscapular pain caused by acute distention of the extrahepatic biliary tree. The ultimate decision as to the origin of abdominal pain may require deliberate and planned observation over a period of several hours, during which repeated questioning and examination will provide the diagnosis or suggest the appropriate studies.

Referred pain of thoracic origin is often accompanied by splinting of the involved hemithorax with respiratory lag and decrease in excursion more marked than that seen in the presence of intraabdominal disease. In addition, apparent abdominal muscle spasm caused by referred pain will diminish during the inspiratory phase of respiration, whereas it is persistent throughout both

respiratory phases if it is of abdominal origin. Palpation over the area of referred pain in the abdomen also does not usually accentuate the pain and in many instances actually seems to relieve it. Thoracic disease and abdominal disease frequently coexist and may be difficult or impossible to differentiate. For example, the patient with known biliary tract disease often has epigastric pain during myocardial infarction, or biliary colic may be referred to the precordium or left shoulder in a patient who has suffered previously from angina pectoris.

Referred pain from the spine, which usually involves compression or irritation of nerve roots, is characteristically intensified by certain motions such as cough, sneeze, or strain, and is associated with hyperesthesia over the involved dermatomes. Pain referred to the abdomen from the testes or seminal vesicles is generally accentuated by the slightest pressure on either of these organs. The abdominal discomfort is of dull aching character and is poorly localized.

METABOLIC ABDOMINAL CRISES

Pain of metabolic origin may simulate almost any other type of intraabdominal disease. Several mechanisms may be at work. In certain instances, such as hyperlipidemia, the metabolic disease itself may be accompanied by an intraabdominal process such as pancreatitis, which can lead to unnecessary laparotomy unless recognized. C1-esterase deficiency associated with angioneurotic edema is often associated with episodes of severe abdominal pain. Whenever the cause of abdominal pain is obscure, a metabolic origin always must be considered. Abdominal pain is also the hallmark of familial Mediterranean fever.

The problem of differential diagnosis is often not readily resolved. The pain of porphyria and of lead colic is usually difficult to distinguish from that of intestinal obstruction, because severe hyperperistalsis is a prominent feature of both. The pain of uremia or diabetes is nonspecific, and the pain and tenderness frequently shift in location and intensity. Diabetic acidosis may be precipitated by acute appendicitis or intestinal obstruction, so if prompt resolution of the abdominal pain does not result from correction of the metabolic abnormalities, an underlying organic problem should be suspected. Black widow spider bites produce intense pain and rigidity of the abdominal muscles and back, an area infrequently involved in intraabdominal disease.

NEUROGENIC CAUSES

Causalgic pain may occur in diseases that injure sensory nerves. It has a burning character and is usually limited to the distribution of a given peripheral nerve. Normal stimuli such as touch or change in temperature may be transformed into this type of pain, which is frequently present in a patient at rest. The demonstration of irregularly spaced cutaneous pain spots may be the only indication of an old nerve lesion underlying causalgic pain. Even though the pain may be precipitated by gentle palpation, rigidity of the abdominal muscles is absent, and the respirations are not disturbed. Distention of the abdomen is uncommon, and the pain has no relationship to the intake of food.

Pain arising from spinal nerves or roots comes and goes suddenly and is of a lancinating type. It may be caused by herpes zoster, impingement by arthritis, tumors, herniated nucleus pulposus, diabetes, or syphilis. It is not associated with food intake, abdominal distention, or changes in respiration. Severe muscle spasm, as in the gastric crises of tabes dorsalis, is common but is either relieved or is not accentuated by abdominal palpation. The pain is made worse by movement of the spine and is usually confined to a few dermatomes. Hyperesthesia is very common.

Pain due to functional causes conforms to none of the aforementioned patterns. The mechanism is hard to define. Irritable bowel syndrome (IBS) is a functional gastrointestinal disorder characterized by abdominal pain and altered bowel habits. The diagnosis is made on the basis of clinical criteria (Chap. 17) and after exclusion of demonstrable structural abnormalities. The episodes of abdominal pain are often brought on by stress, and the pain varies considerably in type and location. Nausea and vomiting are rare. Localized tenderness and muscle spasm are inconsistent or absent. The causes of IBS or related functional disorders are not known.

Approach to the Patient: ABDOMINAL PAIN

Few abdominal conditions require such urgent operative intervention that an orderly approach need be abandoned, no matter how ill the patient. Only those patients with exsanguinating intraabdominal hemorrhage (e.g., ruptured aneurysm) must be rushed to the operating room immediately, but in such instances only a few minutes are required to assess the critical nature of the problem. Under these circumstances, all obstacles must be swept aside, adequate venous access for fluid replacement obtained, and the operation begun. Many patients of this type have died in the radiology department or the emergency room while awaiting such unnecessary examinations as electrocardiograms or abdominal films. *There are no contraindications to operation when massive intraabdominal hemorrhage is present.* Fortunately, this situation is relatively rare. These comments do not pertain to gastrointestinal hemorrhage, which can often be managed by other means (Chap. 8).