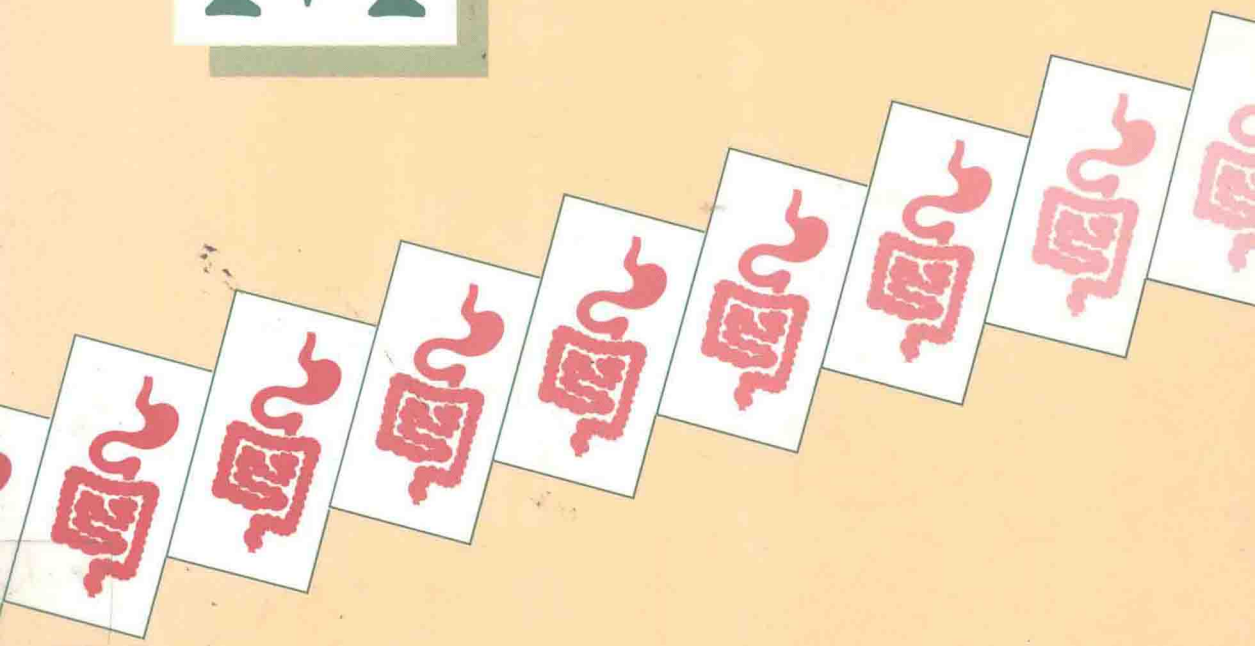
SELECTED GASTROINTESTINAL DISORDERS

Intractability and its Management



edited by
Ward O. Griffen, Jr.
Paul Mandelstam

Selected Gastrointestinal Disorders:

INTRACTABILITY AND ITS MANAGEMENT

EDITED BY

WARD O. GRIFFEN, Jr., M.D., Ph.D.

Executive Director/Secretary-Treasurer
The American Board of Surgery
Professor of Surgery, Temple University School of Medicine, Philadelphia, Pennsylvania

PAUL MANDELSTAM, M.D., Ph.D.

Professor of Medicine
University of Kentucky College of Medicine
Consultant in Gastroenterology
Veterans Administration Hospital
Lexington, Kentucky

WILLIAMS & WILKINS
Baltimore • London • Los Angeles • Sydney

Editor: Kimberly Kist
Associate Editor: Victoria M. Vaughn
Copy Editor: Gail Chalew
Design: JoAnne Janowiak
Illustration Planning: Wayne Hubbel
Production: Anne G. Seitz

Made in the United States of America

Library of Congress Cataloging-in-Publication Data

Selected Gastrointestinal Disorders: Intractability and Its Management.

Includes index.
1. Gastrointestinal system—Diseases. 2. Chronic diseases. I. Griffen, Ward O., Jr. II. Mandelstam, Paul. [DNLM: 1. Gastrointestinal Diseases. 2. Pain, Intractable. WI 100 I649]
RC802.I58 1986 616.3'3 86-4022
ISBN 0-683-03736-6

Composed and printed at the
Waverly Press, Inc.

87 88 89 90 91
10 9 8 7 6 5 4 3 2 1

For Pudge and for Carol for their unstinting forbearance

Preface

Some gastrointestinal diseases fall almost entirely within the province of the physician whereas others, such as bowel obstruction, are surgical from the start.

In a number of gastrointestinal diseases, however, the responsibility often comes to be shared. In these, the initial assessment and management are usually nonsurgical. With the all-too-frequent development of intractability, whether of the disease, the patient or even the physician,[1] an "escalation in treatment"[2] is called for. This escalation usually requires surgery. What constitutes intractability and what further measures are most appropriate are the concerns of this book.

For each of nine major "shared" gastrointestinal ailments, we have solicited two chapters, one from a physician(s) and one from a surgeon(s), and to each pair of chapters we have, separately, contributed Editorial Commentary. Differences in opinion concerning optimal management are not infrequent between the chapter authors and, indeed, between the two editors. It is noteworthy and heartening that these differences are not entirely along disciplinary lines.

We hope that this book, in its focus on areas of care that are so very often gray, will prove interesting and rewarding to readers, and will contribute to the more effective care of even one patient.

W.O.G.
P.M.

[1] See Edwards' Chapter 1: "Even the physician may be 'perverse' and 'stubbornly' believe in the wrong concept of the disorder so that his treatment is inappropriate and so cause a degree of intractability."
[2] See Klein and Spiro's Chapter 19.

Acknowledgment

One of us (P.M.) would like to acknowledge with great appreciation the contributions that the late Ruth M. Jackson has made not only to the preparation of this book but to his overall professional activities for a period of almost 25 years.

Contributors

David R. Antonow, M.D.
Assistant Professor of Medicine, University of Kentucky College of Medicine, Lexington, Kentucky

Walter F. Ballinger, M.D.
Professor of Surgery, Washington University School of Medicine, St. Louis, Missouri

Arthur E. Baue, M.D.
Vice President for the Medical Center, St. Louis University, and Professor of Surgery St. Louis University School of Medicine, St. Louis, Missouri

P. C. Bornman, M.B., F.R.C.S.
Consultant Surgeon and Head of Surgical Gastroenterology, Gastrointestinal Clinic and The Department of Surgery, University of Cape Town, Cape Town, South Africa

Clarence Dennis, M.D., Ph.D.
Professor of Surgery, State University of New York at Stony Brook, Associate Chief of Staff for Research and Development, Veterans Administration Medical Center, Northport, New York. Formerly Professor and Chairman, Department of Surgery, State University of New York Downstate Medical Center, Brooklyn, New York

Frederic E. Eckhauser, M.D.
Associate Professor of Surgery, University of Michigan Medical Center, Chief of Surgery, Ann Arbor Veterans Administration Hospital, Ann Arbor, Michigan

D. A. W. Edwards, M.H., M.D., F.R.C.P.
Reader in Gastroenterology (Emeritus), Faculty of Clinical Sciences, University College, London, England

John T. Galambos, M.D.
Professor of Medicine and Director, Division of Digestive Diseases, Emory University School of Medicine, Atlanta, Georgia

Alexander S. Geha, M.D.
Professor and Chief, Cardiac Surgery, Department of Surgery, Yale University School of Medicine, New Haven, Connecticut

A. H. Girdwood, M.B., F.R.C.P.
Consultant Physician, Gastrointestinal Clinic, Groote Schuur Hospital, and Department of Medicine, University of Cape Town, Cape Town, South Africa

Myron D. Goldberg, M.D.
Associate Attending Physician, Section of Gastroenterology, Department of Medicine, Lenox Hill Hospital, New York, New York; Clinical Instructor of Medicine, New York Medical College, Valhalla, New York

David Y. Graham, M.D.
Professor of Medicine and Virology, Baylor College of Medicine, and Chief, Digestive Disease Section, Veterans Administration Medical Center, Houston, Texas

Ward O. Griffen, Jr., M.D., Ph.D.
Executive Director/Secretary-Treasurer, The American Board of Surgery, Professor of Surgery, Temple University School of Medicine, Philadelphia, Pennsylvania

Carl Robert Grosz, M.D.
Attending Surgeon, St. Alphonsus Regional Medical Center, St. Luke's Regional Medical Center, Boise, Idaho. Formerly Associate Professor of Surgery, State University of New York Downstate Medical Center, Brooklyn, New York

Donald H. Hanscom, M.D.
Consulting Physician, Veterans Administration Hospital, Hines, Illinois; Director, Gastroenterology, Hinsdale Hospital, Hinsdale, Illinois

Gordon L. Hyde, M.D.
Professor of Surgery, University of Kentucky College of Medicine, Lexington, Kentucky

R. Scott Jones, M.D.
Stephen H. Watts Professor and Chairman, Department of Surgery, University of Virginia Medical Center, Charlottesville, Virginia

Kenneth B. Klein, M.D.
Clinical Assistant Professor of Medicine, University of North Carolina School of Medicine, Chapel Hill, North Carolina

Burton I. Korelitz, M.D.
Chief, Section of Gastroenterology, Department of Medicine, Lenox Hill Hospital, New York, New York; Clinical Professor of Medicine, New York Medical College, Valhalla, New York

David H. Law, M.D.
Director, Medical Service, Veterans Administration Central Office, Washington, D.C.

Armand Littman, M.D., Ph.D.
Chief, Medical Service, Veterans Administration Hospital, Hines, Illinois; Professor of Medicine, Loyola Stritch School of Medicine, Maywood, Illinois

Paul Mandelstam, M.D., Ph.D.
Professor of Medicine, University of Kentucky College of Medicine; Consultant in Gastroenterology, Veterans Administration Hospital, Lexington, Kentucky

I. N. Marks, M.B., F.R.C.P.
Head, Gastrointestinal Clinic, Groote Schuur Hospital, Professor, Department of Medicine, University of Cape Town, Cape Town, South Africa

Frank G. Moody, M.D.
Professor and Chairman, Department of Surgery, The University of Texas Medical School, Houston, Texas

Howard A. Reber, M.D.
Chief, Surgical Service, Harry S. Truman Memorial Veterans Hospital and Professor of Surgery, University of Missouri Health Sciences Center, Columbia, Missouri

Grant V. Rodkey, M.D.
Associate Clinical Professor of Surgery, Harvard Medical School, Visiting Surgeon, Massachusetts General Hospital, Boston, Massachusetts

Richard W. Schwartz, M.D.
University of Kentucky Clinical Fellow-Postdoctoral Scholar, Instructor in General Surgery and Transplant Fellow, University of Michigan Medical Center, Ann Arbor, Michigan

Brian F. Smale, M.D.
Formerly Staff Surgeon, Harry S. Truman Memorial Veterans Hospital and Assistant Professor of Surgery, University of Missouri Health Sciences Center, Columbia, Missouri

J. Lacey Smith, M.D.
Associate Professor of Medicine, Baylor College of Medicine, Digestive Disease Section, Veterans Administration Medical Center, Houston, Texas

Howard M. Spiro, M.D.
Professor of Medicine, Yale University School of Medicine, New Haven, Connecticut

Richard J. Strauss, M.D.
Associate Professor of Clinical Surgery, State University of New York, Stony Brook, New York

William E. Strodel, M.D.
Associate Professor of Surgery, University of Michigan Medical Center, Ann Arbor, Michigan

Jeremiah G. Turcotte, M.D.
F. A. Coller Professor and Chairman, Department of Surgery, University of Michigan Medical Center, Ann Arbor, Michigan

Claude E. Welch, M.D.
Clinical Professor of Surgery (Emeritus), Harvard Medical School, Senior Surgeon, Massachusetts General Hospital, Boston, Massachusetts

Leslie Wise, M.D.
Chairman, Department of Surgery, Long Island Jewish Medical Center, Professor of Surgery, State University of New York, Stony Brook, New York

Contents

SECTION 8
Ascites in Cirrhosis

SECTION 9
Esophageal Varices

SECTION

1

HIATAL HERNIA AND GASTROESOPHAGEAL REFLUX

CHAPTER

1

Hiatal Hernia and Gastroesophageal Reflux

D. A. W. EDWARDS, M.A., M.D., F.R.C.P.

Of the dictionary meanings given for the term "intractable," in the context of gastrointestinal disease this author includes "unchangeable," "inexorable," "refractory," "rebellious," and "not yielding to treatment." Within these meanings one must consider whether it is the anatomy, physiology, symptom pattern, histologic or other pathologic consequence, or the patient that is intractable. Even the physician may be "perverse," and stubbornly believe in the wrong concept of the disorder so that his or her treatment is inappropriate and so causes a degree of intractability. The author's own concept of the disorder necessarily colors the view presented in this chapter of what is intractable, what might be amenable to treatment, and where we should be looking for improvements in therapy. Briefly this concept is as follows (1, 2).

Antireflux Mechanism

There are two parts to the antireflux mechanism in the normal person. The first part, the *lower esophageal sphincter* (LES) protects the esophagus from increases in intragastric pressure caused by contraction of stomach wall muscle. Intragastric or stomach squeeze has to be greater than sphincter squeeze for reflux to occur, and because stomach squeeze is at zero most of the time, one does not reflux every time the LES is relaxed to zero by swallowing. Moreover, stomach squeeze is rarely greater than 10 to 15 cm water, so the LES does not need to contract very hard. This is the normal physiologic part of the antireflux mechanism that regularly breaks down after meals, probably because stomach squeeze rises rather than the LES squeeze falls, so that all persons reflux small amounts frequently at this time. Most of the time people are unaware of this frequent reflux, partly because the volume and force behind it are not sufficient for refluxate to reach the mouth. In some people, however, refluxate does reach the mouth; these individuals are called ruminators, or included among "upright refluxers" and, incorrectly, among "habit vomiters." As is discussed later in the chapter, increase in intra-abdominal pressure does not increase stomach squeeze relative to sphincter squeeze when the sphincter is in its normal place and the hiatal mechanism is functioning.

The second part of the antireflux mechanism, *the hiatal mechanism*, protects the LES from being challenged by the difference in pressure between the abdominal and thoracic cavities. This mechanism has three components.

1. When normally placed, the lower part of the sphincter is subjected to intra-abdominal pressure, so that if this pressure increases, the sphincter is squeezed to the same degree as the stomach.
2. When intra-abdominal pressure increases, the diaphragm contracts and the hiatal walls contract upon the tube of gut going through them so that hiatal squeeze is greater than the abdominal wall squeeze on the stomach (3).
3. The mechanical conditions associated with a soft tube passing through a narrow slit in a diaphragm separating cavities at different pressures creates a *flutter valve*. This prevents the flow of contents between the two cavities as long as intragastric pressure (stomach squeeze) is less than sphincter squeeze or as long as esophageal squeeze (peristalsis) is less than sphincter squeeze plus the difference in pressure between abdomen and thorax. These hiatal mechanisms work at the speed of striated muscle or faster; that is, at least 10 times faster than the speed of contraction of the smooth muscle sphincter. Physiologic reflux is not prevented by the flutter valve. When in its normal position, the sphincter may itself help prime the flutter valve. The hiatus does not squeeze the tube going through it during expiration, but a normal hiatus is narrow enough to oppose lightly the walls of the gut tube passing through it. If the sphincter herniates into the chest, but the hiatus remains small, the hiatus may still squeeze on the neck of stomach passing through it with a force greater than abdominal wall squeeze. In the same way the flutter valve may be formed with the tube of stomach passing through the hiatal slit. Despite the herniation of the sphincter, both these mechanisms may be effective most of the time in protecting the sphincter from being challenged by abdominal wall squeeze or hydrostatic pressure of viscera on the stomach.

Breakdown of the Antireflux Mechanisms

These two mechanisms may break down in several ways and are variably responsive to treatment. The *physiologic mechanism*, dependent on the difference between stomach squeeze and sphincter squeeze, might be expected to fail if the sphincter fails, for example, from loss of smooth muscle in systemic sclerosis or after a cardiomyotomy for achalasia. Failure of the antireflux mechanism sometimes develops in systemic sclerosis, but almost always is then associated with loss of the hiatal mechanism from concurrent loss of striated muscle. Radiologically demonstrable failure of the antireflux mechanism after cardiomyotomy only occurs if the hiatal mechanism has been damaged. In a consecutive series of 170 cardiomyotomies by two surgeons who were careful not to disturb the hiatal mechanism and did not carry out any antireflux procedure, abnormal reflux could not be demonstrated in 159 (3). Loss of the sphincter by itself does not necessarily mean symptoms of an intractable problem and does not necessarily result in a great increase in physiologic reflux.

The mechanism of physiologic reflux seems more likely to be a short-lived rise in stomach squeeze rather than a short-lived fall in sphincter squeeze, which happens every time one swallows. Most of the time stomach squeeze is zero because its motor activity changes the shape of the wall and mixes the contents, but does not propel them by an increase in intraviscus pressure. Physiologic reflux cannot be demonstrated or produced at will by barium examination in a normal subject because stomach squeeze cannot be increased by increasing intra-abdominal pressure. Nor is it provoked by bending or lying or wearing tight garments.

This physiologic mechanism may be breached more frequently than normal, however, if stomach squeeze is increased by, for example, an increase in amplitude

of gastric contractions or by an obstruction to outflow from the stomach or duodenum. Just as removal of the outflow obstruction will relieve the abnormal reflux in pyloric stenosis for example, gastroenterostomy or pyloroplasty may relieve the excessive physiologic reflux or rumination that seems likely to be caused by increased stomach squeeze without abnormal outflow obstruction. Propantheline or dicyclomine to tolerance may reduce stomach squeeze enough for the medication to be worthwhile, even though the patient may suffer some side effects. Exaggerated physiologic reflux, especially rumination, can be very distressing, may occasionally be associated with esophagitis, may make the breath smell of vomit, and may even destroy a marriage. At this degree it is an intractable medical problem and there is not sufficient experience with depressurizing surgery to know its value. Neither hiatal herniation nor reflux of barium can be induced at a radiologic examination in these conditions by definition, and there is no indication for hiatal or hernial repair. Nor do they help. Physiologic reflux may be stopped by a full Nissen fundoplication, but such success is likely to stop vomiting and belching from the stomach as well.

Because normal sphincter squeeze is low to start with, doubling it with cholinergic agents, such as bethanechol or metoclopramide, is not enough to enable it to withstand common increases in abdominal pressure. To do so, medication would need to increase sphincter squeeze by 7 to 10 times and act in a way that did not interfere with reflux relaxation on swallowing. It must not increase the amplitude of gastric contractions because this might increase stomach squeeze. Metoclopramide may increase sphincter squeeze, but it also increases the amplitude of gastric contractions, which may explain why it is not helpful to all the patients the author sees with reflux, nor to those who ruminate or are "upright refluxers." Drugs designed to increase sphincter squeeze have not reduced the problem of reflux. Failure of the normally placed sphincter mechanism in the author's view, with some exceptions, is not an important nor intractable problem.

The hiatal mechanism breaks down commonly, for no defined reason, and probably with increasing incidence with age in what is called hiatal hernia. In the earliest or mildest stage the attachments of the esophagus to the hiatus become stretched or attenuated and allow herniation of the sphincter and a loculus of stomach of 0.5 to 1.0 cm diameter. Many people can herniate to this degree when they are tipped head down or bend over to touch their toes. Often the amount of herniation does not change for many years, if at all. The hiatus in these patients is not measurably enlarged, and barium cannot be made to flow from abdomen to thorax by tipping the patient or increasing abdominal pressure. The hiatus is competent during a barium examination, but symptoms may be provoked sometimes when the patient is lying or bending, suggesting the capacity for intermittent abnormal reflux.

In the next level of damage to the hiatal attachments herniation is increased to 1.0 to 2.5 cm diameter. This amount of herniation also tends not to change in size over 10 to 20 years, but is associated with some enlargement of the hiatus. When the patient is placed in some positions a flutter valve is formed, or the hiatus squeezes the tube of stomach enough to stop hiatal flow from abdominal to thoracic stomach when intra-abdominal pressure is increased. For example, with a stomach full of barium, hiatal flow may not occur with the patient tipped head down 10°, lying on the left side, nor on the back with or without straight leg raising, which increases the gradient of pressure from abdomen to thorax to 80 mm Hg or more. Yet in the same patient there may be enough flow to flood the gullet as the patient turns from the left side onto the back and particularly as he or she turns from the back onto the right side. Flow may continue or cease when the patient has stopped turning and rests on the right side. The gradient during turning is no greater than at rest. It seems that the shape of the hiatus changes, which is sufficient either to stop the flutter valve ef-