Handbook of Colorectal Surgery

Second Edition,
Revised and Expanded

edited by
David E. Beck

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David E. Beck
Ochsner Clinic Foundation
New Orleans, Louisiana, U.S.A.

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To

My wife, Sharon
for her love, understanding, support,
ability, and ongoing efforts to improve me

My daughter, Allison
who desires to pursue a medical career
without having to deal with incompetence

My daughter, Lauren
for enjoying life and learning that
some people always get caught

My son, John
for demonstrating the primal forces
acting on young males in our culture:
music, girls, and sleep

My residents, fellows, and colleagues,
who challenge me on a daily basis

My patients who remind me of how much we still need to learn
while allowing me to assist in their
regaining and maintaining their health

Foreword

Despite the explosion of texts in colorectal surgery over the past few years, reflecting the importance of teaching and learning the essentials of care for patients with disease of the colon, rectum and anus, the *Handbook of Colorectal Surgery* is being released in its second edition just six years from its first publication. In the original preface, Dr. Beck indicated that the book was an ''ideal portable reference'' for nurses, students, and residents; time has proven this to be correct.

Organizational aspects of the book include a brief outline at the outset of each chapter and a list of classic references at the end. Tables are succinct and uniformly helpful, as are the illustrations. Although the order of chapters is exactly the same, nearly every one has been rewritten by Dr. Beck, with updating of information to incorporate new advances where appropriate. ''Rounds Questions,'' a list of often-asked questions that highlight the essential teaching points of each topic, have been continued and expanded in the new edition.

Other unique aspects of this book, in addition to its format, include its size and cost. It is indeed portable and affordable—in contrast to the usual comprehensive volumes most often encountered on colorectal topics. The book is intended to be not so much a reference book as a basic useful guide to the care of patients that will be used repeatedly in daily practice.

Experienced surgeons will find this a most useful adjunct to training medical students and residents of all levels, including colon and rectal residents. Physician extenders involved in colorectal and general surgery practices, as well as nurses, should find this book invaluable.

Dr. Beck should be congratulated for devoting considerable precious time to updating this singular contribution to the colorectal literature. Educators should not omit this book from their lists of required reading and should, in fact, consider providing copies of it to all their trainees.

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Preface

How do you evaluate and treat constipation?
What type of bowel preparation is used to clean a patient's colon before an operative procedure?
What is the follow-up for colorectal cancer?
What are the indications for colonoscopy?
What are the options for treating hemorrhoids?

Questions such as these are frequently asked by residents, students, and nurses working in our colorectal surgery program. It was the number and range of these questions that provided the initial impetus for writing the first edition of this book. Despite the availability of several outstanding texts covering our specialty, there was no current, affordable manual to recommend to residents, nurses, and other members of our health care team seeking this kind of information. The answers to the questions posed above, and many more, were covered in the first edition of *Handbook of Colorectal Surgery.* Each medical specialty—and colon and rectal surgery is no exception—has seen new developments and knowledge.

The second edition continues to serve as a basic guide to the management of patients with colorectal diseases. Each chapter was reviewed and in many

cases rewritten to present current concepts and recommendations. Key elements throughout the book are reinforced by the Rounds Questions following each chapter, and extensive updated references provide options for further study. The newest concepts in patient care and operative technique (such as laparoscopic surgery) are covered and profusely illustrated. The second edition of this handbook remains an ideal portable reference for residents, students, and nurses. Experienced surgeons will find this manual helpful in their training of residents and fellows, and it may serve as a stimulus for additional thought and research.

This book incorporates the collaborative efforts of many individuals. The contributors to the first edition did an excellent job, and their contributions served as a foundation for the updated second edition. Three of the original contributors have been retained and additional experts in specialized fields have lent their expertise. These talented and dedicated individuals are active in teaching medical students and residents and continue to shape the future of colorectal surgery. The illustrations were produced or review by Barbara Siede, Director of Ochsner Medical Illustrations, whose exceptional ability has clarified many difficult concepts. Marion R. Stafford, Director of Ochsner Medical Editing, and her staff, René M. Couvillion and Liana M. Tome, were instrumental in error correction and improving the readability of the text. My nurse, Andrea Thibodeaux, R.N., and clinic support staff assist me in patient management and helped me squeeze in time for these academic pursuits. Finally, my thanks to the staff of Marcel Dekker, Inc., who worked hard to make this second edition a reality.

David E. Beck

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1

Anatomy

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A knowledge of intra-abdominal anatomy is essential to understand and treat intestinal diseases. [1] This chapter briefly summarizes anatomical features and principles that are important to the colorectal surgeon. Discussions of greater depth are available in comprehensive anatomy and colon and rectal surgery texts. [2–7] Although study and experience will increase the surgeon's knowledge of expected anatomical findings, it must be remembered that variability is the rule in human anatomy. The abdominal cavity contains many structures (Figure 1). The abdominal portion of the intestinal tract starts with the esophagus, which connects to the stomach. Structures of major importance to the colorectal surgeon include the small bowel, colon, rectum, and anus.

MACROSCOPIC ANATOMY

Small Bowel

The small bowel starts at the stomach and connects to the large bowel or colon at the ileocecal valve. The small bowel has three parts, the duodenum, jejunum, and ileum. The proximal portion, the duodenum is approximately 25 cm in length.

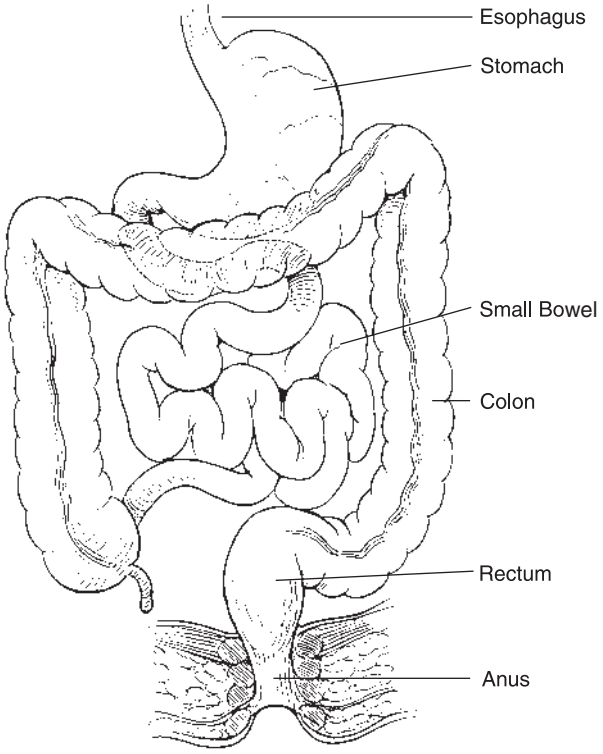


Figure 1 Normal gastrointestinal anatomy (midcoronal view).

The bile duct from the liver enters into the second part of the duodenum. The end of the duodenum ends at a fibrous band called the ligament of Treitz. Two fifths of the remaining small bowel is called the jejunum, and the distal three fifths is called the ileum. The total length of the small bowel varies from 250 to 800 cm, with a mean of 500 cm. It is folded in a variable fashion to accommodate the length of bowel in the abdomen. The major function of the small bowel is digestion and absorption of fluid and nutrients. The diameter of the small bowel is greatest at the duodenum and gradually narrows to the ileum.

Colon

The colon (large intestine) starts from the cecum (usually located in the right lower quadrant) and continues through all portions of the abdomen to the colorectal junction in the pelvis. The colon is about 1.5 m long [2] and classically has been divided into segments based on the vascular supply and location of each

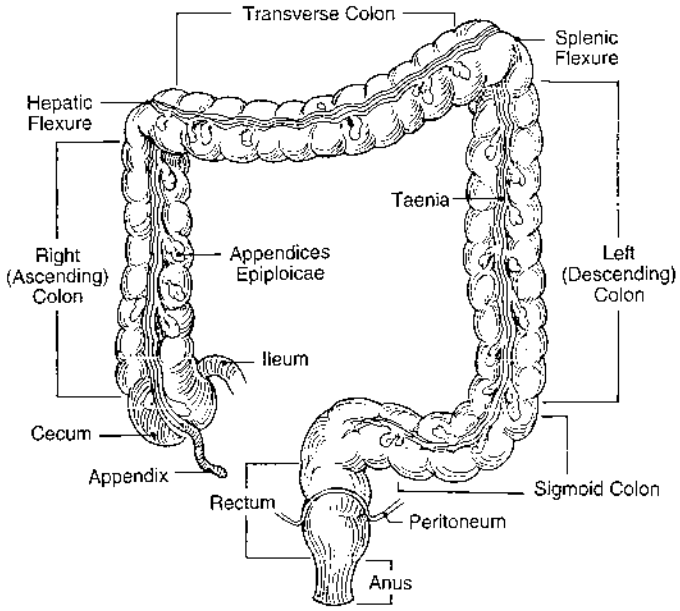


FIGURE 2 Topographic anatomy of the colon.

segment within the abdomen, as shown in Figure 2. The cecum, right colon (supplied by the right and ileocolic artery), and left colon (supplied by the left colic artery) are usually retroperitoneal and fixed. The transverse colon (supplied by the middle colic artery) and sigmoid colon (supplied by branches of the inferior mesenteric artery) are intraperitoneal and relatively mobile. The colon contains two flexures (or bends) in the right upper quadrant (hepatic) and left upper quadrant (splenic). When the colon is not unduly distended with feces, its diameter is largest at the cecum and gradually narrows to the distal sigmoid colon (the narrowest part of the colon).

The external wall of the colon is unique because of the presence of several appendages (taeniae, omentum, appendices epiploicae, and diverticula). The outer longitudinal muscle is thickened in three longitudinal bands called taeniae. These average 8 mm in width and are named in reference to their relationship to the bowel mesentery or omentum. Thus there is a taenia mesocolica (associated with the mesentery), a taenia omentalis (associated with the omentum), and a taenia libera (not related to either the mesentery or omentum). The three taeniae meet at the appendiceal orifice and continue to the colorectal junction, where they expand to form a solid layer. Intermittent contractions of the inner circular muscle

result in formation of semicircular folds called haustra, which are thought to aid in mixing the stool. The haustra are visible on the exterior surface of the colon.

The omentum is a sheet of fat and fibrous tissue that is well vascularized. It starts at the greater curvature of the stomach, attaches to the transverse colon at the taenia omentalis, and extends into the abdomen. It doubles back on itself and attaches again to the colon, dividing the abdomen into several spaces (Figure 3). This arrangement allows it to be detached from the colon with minimal dissection in an almost bloodless plane. It has been theorized that the omentum functions to localize inflammatory processes and to assist in healing. This is supported by clinical experience and the frequent finding of omental adhesions to other portions of the bowel. Because of its multiple important functions, I prefer to preserve the omentum in operations on patients in whom neoplastic lesions are not present. This is easily accomplished by elevating the omentum superiorly and dividing the thin avascular tissues that attach the omentum to the colon. With care, the omentum can be detached intact.

The appendices epiploicae are subserous pockets of fat that occur in two rows on the right and the sigmoid colon and in a single row on the transverse colon. Their only recognized role is to act as a storage site for fat cells. Many adults also have colonic diverticula (mucosal herniations) located adjacent to the taeniae (see Chap. 13).

The colon connects to the small bowel at the ileocecal valve. Although lacking an anatomical sphincter, this functional valve is responsible for several

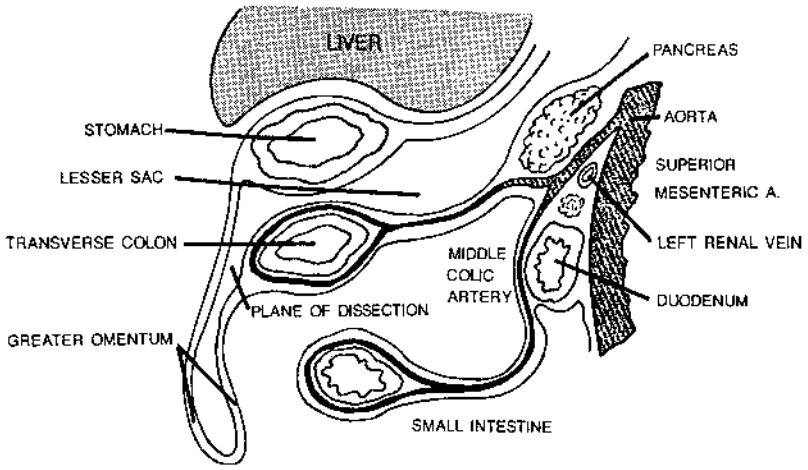


Figure 3 Sagittal section of the abdomen demonstrating attachments of the greater omentum.

physiological actions: it allows the digested contents of the small bowel to pass into the cecum at a controlled rate and acts as a relative barrier to prevent the large number of bacteria (concentration of 10^{10}) in the colon from moving to the distal small intestine (concentration of 10^3). Approximately 15% of patients have an incompetent ileocecal valve as demonstrated on barium enema studies.

Rectum

The rectum (Figure 4) is 12–15 cm long and can be divided into thirds based on its peritoneal relations. The upper third is intraperitoneal and covered anteriorly and laterally by peritoneum. At its middle portion the rectum passes through the peritoneal floor and is covered by peritoneum on the anterior surface. The lower third is extraperitoneal as it travels through the levators to the anus. The lower rectum is enveloped by visceral pelvic fascia. Anteriorly, Denonvilliers' fascia (Figure 5) separates the rectum from the seminal vesicles, prostate, and bladder trigone in males and the posterior vaginal wall in women. Posteriorly, Waldeyer's fascia separates the rectum from the presacral venous plexus.

The rectum can be differentiated from the colon by its lack of a posterior mesentery, sacculations, and appendices epiploicae. The outer longitudinal muscle layer of the rectum diffuses to form a solid, thick layer. Thus there are no

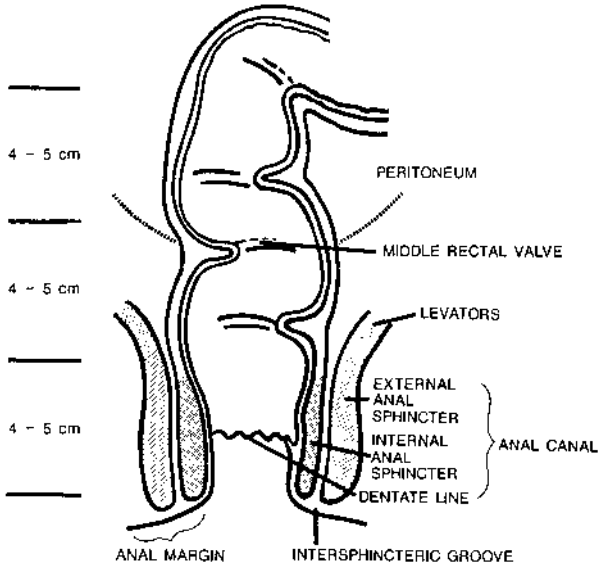
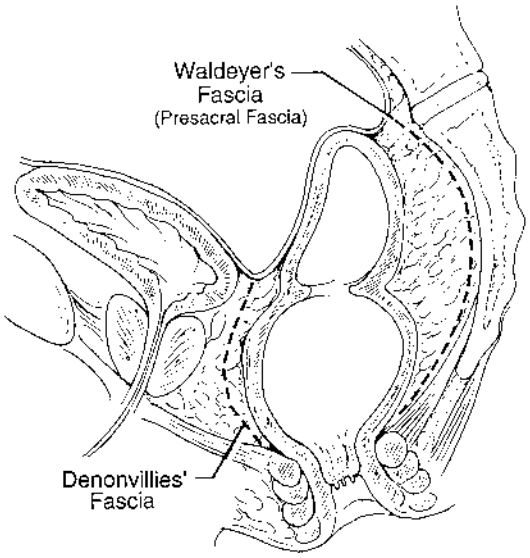
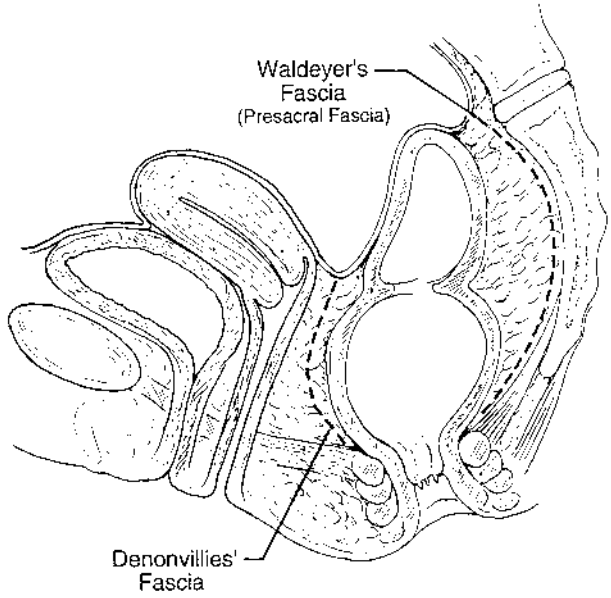


Figure 4 Rectum and anus (coronal section).



(a)



(b)

Figure 5 Sagittal section of the pelvis demonstrating anterior and posterior rectal fascia: (A) male, (B) female.

taeniae or diverticula. The rectum is also larger in diameter than the sigmoid colon.

The inner rectum contains three indentations or *valves of Houston*. These are composed of circular muscle only. The superior valve is located 4 cm below the rectosigmoid junction on the left side; the middle valve is located at the peritoneal reflection on the right side; the inferior valve is located 2–3 cm above the dentate line on the left side. These valves aid the surgeon in localizing lesions with respect to the peritoneal location.

Anus

The anal canal starts at the anorectal junction located at the palpable upper edge of the anal sphincter mechanism (junction of the puborectalis and the anal sphincter). The anal canal ends at the intersphincteric groove (approximately 2 cm distal to the dentate line). The anal margin is that portion of the perineum from the intersphincteric groove to approximately 5 cm out from the dentate line (see Figure 4).

The complex musculature of the anal canal can be thought of as composed of two tubes: the outer tube is funnel shaped, composed of skeletal muscle, and innervated by somatic nerves; the upper portion of this funnel is formed by the levator ani muscles. This sheet of muscle originates from the pelvic side wall (laterally), the sacrum (posteriorly), and the pubis (anteriorly) to the upper anus. Fibers of the levators can be grouped into three sections: the puborectalis (inner), pubococcygeus, and ileococcygeus muscles (posterolateral).

The lower portion of this outer cylinder of muscle is composed of the external anal sphincter. Although this voluntary muscle has been divided into three portions, clinically and physiologically it acts as a unit. Contraction of this muscle and the puborectalis produces the anal squeeze examined during the digital examination described in Chapters 2 and 3.

The inner tube of the anal canal is composed of visceral smooth muscle that is controlled by autonomic nerves. At the anus the inner circular muscle of the rectum thickens to become the internal anal sphincter. The longitudinal muscles of the rectum pass through the internal sphincter and attach to the perianal skin. The inner muscles of the anus are controlled by branches of the inferior rectal nerve and the perineal branch of the fourth sacral nerve. The internal anal sphincter is normally contracted and provides the resting anal tone felt during a digital anal examination. At rest, the lateral walls of the anal canal are opposed to form an anteroposterior slit [8].

The pelvic musculature and its attachments divide the pelvis into several spaces; these are described in Chapter 17.

VASCULAR ANATOMY

The colon receives its blood supply from branches of two major vessels, the superior and inferior mesenteric arteries (Figure 6). The *superior mesenteric ar-*

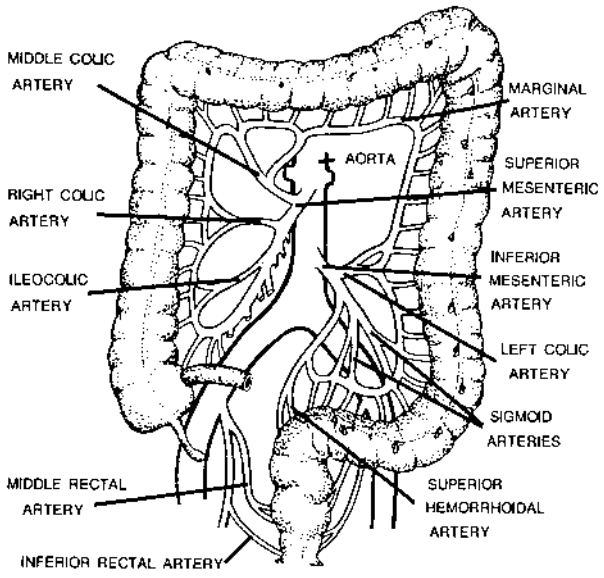


FIGURE 6 Arterial supply to the colon.

tery (SMA) originates on the anterior surface of the aorta, at the level of the first lumbar vertebrae, 1.25 cm caudal to the celiac artery, superior to the duodenum and pancreas [2]. Its first major branch is the middle colic artery. The middle colic artery divides close to its origin into an ascending and descending branch. After further branching it connects to the marginal artery and supplies the transverse colon. Distal to the marginal artery, end vessels travel in the mesentery to connect the marginal artery to the bowel (Figure 6).

The *inferior mesenteric artery* (IMA) originates 2–3 cm caudal to the SMA (inferior to the duodenum and pancreas). Its first major branch is the left colic artery. The left colic artery usually divides into two branches within 4–5 cm of its origin. This area is important in colonic operations. The next branches off the IMA are three to six sigmoid arteries. As branches of the artery approach the bowel, they communicate with the marginal artery. The distal branch of the IMA, the ileocolic artery supplies the cecum.

The IMA continues to the upper rectum, where it becomes the superior hemorrhoidal artery. As it courses distally it splits into multiple branches that enter the rectum laterally.

The venous drainage of the colon (Figure 7) goes to the portal system and tends to follow the arterial system. The ileocolic vein attaches to the superior mesenteric vein (SMV) approximately 3 cm before the SMV joins to the splenic

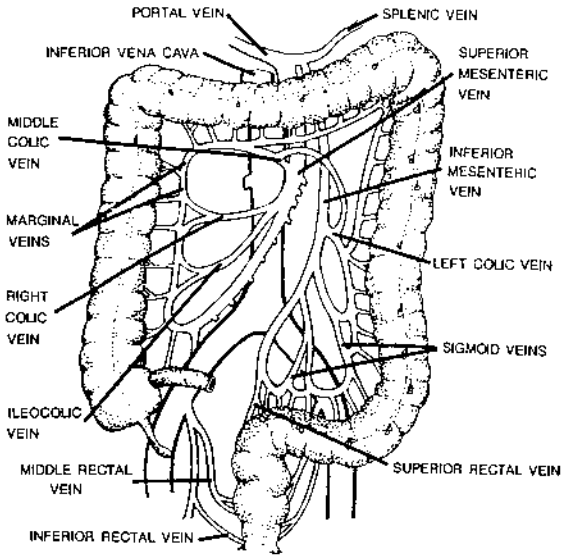


FIGURE 7 Venous drainage of the colon.

vein (inferior to the pancreas). The left colic vein enters the inferior mesenteric vein (IMV) at the level of the IMA origin. The IMV travels to the left of the IMA and continues to enter the splenic vein beneath the pancreas.

The lymphatic drainage of the colon follows the arterial supply. Major lymphatic chains are located along and named after the major named veins. The lymph nodes along these chains are important in colorectal cancer recurrence and prognosis.

The upper rectum receives blood from branches of the IMA. At the upper rectum this vessel is called the superior hemorrhoidal (rectal) artery. As it continues down the rectum the vessel splits, and branches move laterally and communicate with branches of the middle hemorrhoidal arteries. The distal rectum and anus are supplied by branches of middle and inferior hemorrhoidal arteries. As these vessels approach the the internal iliac arteries and the bowel, they split into multiple communicating vessels.

The anus receives blood from two sources: branches of the lower hemorrhoidal plexus (inferior hemorrhoidal arteries) communicate with the middle hemorrhoidal arteries (as described previously) and with branches from the pudendal arteries. The pudendal arteries branch from the internal iliac arteries. Venous and lymphatic drainage goes to both mesenteric and systemic veins.

NERVOUS SUPPLY

The colon and rectum are richly innervated by multiple nerves whose function is poorly understood. The majority of efferent fibers to the intestine originate in the hypothalamus. The parasympathetic efferent fibers exit the central nervous system in two areas (cranial and sacral). The foregut is supplied via the vagus nerves, and the hindgut fibers exit the sacral cord via the dorsal columns at sacral roots 2 through 4. Fibers from S3 and S4 are called the nervi erigentes [9]. After exiting the spinal cord, the fibers pass through a sacral plexus and then join with the hypogastric nerves (sympathetic nerves) to form the pelvic plexus. Parasympathetic nerves then pass upward in the inferior mesenteric plexus to be distributed to the superior hemorrhoidal artery and left colonic arteries. Other sacral fibers (S2–4) supply fibers to the levators, then enter the perineum via Alcock's canal as the pudendal nerve. At the anus the pudendal nerve becomes the hemorrhoidal nerve, perianal and dorsal penile nerve, or clitoral nerve.

The sympathetic efferent nerves exit the spinal cord at the thoracic and lumbar segments. The fibers pass through the splanchnic nerves to the mesenteric ganglia. Fibers then travel along the superior and inferior mesenteric arteries to reach the intestine. Additional fibers pass through the inferior hypogastric (pelvic) plexus, as previously described, to supply the rectum.

Afferent fibers from the intestine carry sensations of stretch, distention, and pain (anoxia or chemical damage) to the brain. The intestines are also affected by intrinsic innervation via the enteric plexus. These nerve cells and fibers are grouped into the myenteric (Auerbach) plexus and the submucosal (Meissner) plexus.

BOWEL WALL

The colon wall is composed of several layers (Figure 8). The innermost layer is the mucosa, a single layer of columnar cells with a cuticular border; it contains tubular pits and goblet cells. The submucosa is the strength layer of the bowel; this layer also contains blood vessels, lymphatics, Meissner's plexus, and solitary lymphatic nodules. There are two muscular layers: the inner layer, composed of muscle cells oriented in a circular fashion, and the outer layer of muscle, oriented in a linear fashion. In three areas the muscle fibers are thickened and fused to form the taeniae. The outermost layer is the serosa, which is composed of fibrous tissue.

The rectum contains layers similar to those of the colon, with two exceptions. The upper rectum contains a serosal covering on the anterior and lateral surface; however, this is lost as the rectum becomes extraperitoneal. The outer longitudinal muscle layer is thickened and diffused to form a solid sheet. The inner muscles are circular and, as described earlier, form three semicircular valves.

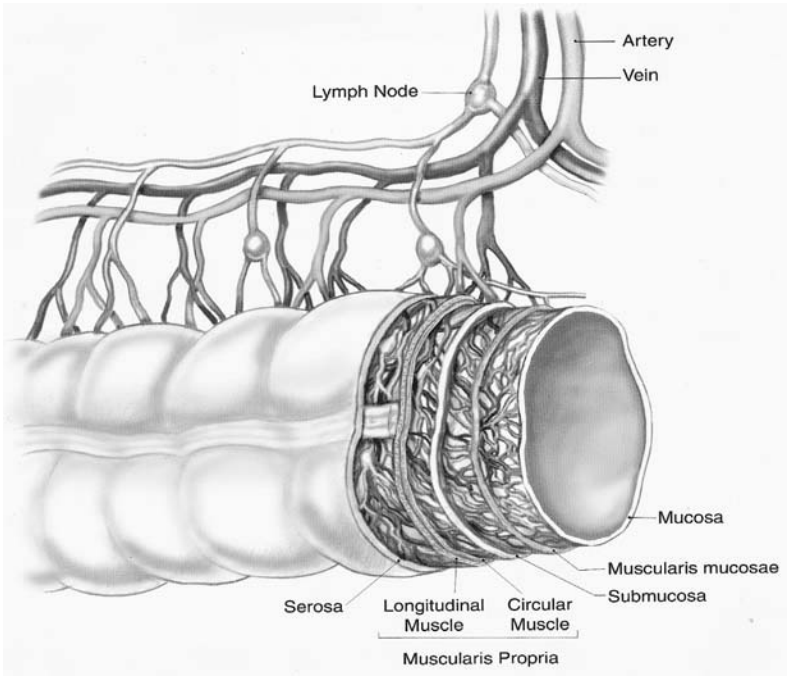


FIGURE 8 Bowel wall anatomy.

The inner and outer muscles contribute fibers to the formation of the internal anal sphincter, as described previously.

The lining of the anus is composed of a transitional zone, where the mucosa changes from a columnar cell layer to a squamous cell layer at the dentate line. The area distal to the anal canal is lined by modified squamous epithelium without hair or glands [8]. Farther caudally, the lining changes to squamous epithelium, with hair and glands at the anal verge.

The submucosa of the anal canal contains three bundles of vascular sinusoids, called hemorrhoidal tissue [10]. (For additional discussion of these structures, see Chapter 16.)

ROUNDS QUESTIONS

1. How many taeniae does the colon have and what are their names?
   There are three taeniae: the taenia mesocolica, the taenia omentalis, and the taenia libera (p. 3).

2. How do you identify where the colon ends and the rectum starts?
   The rectum is larger in diameter, has no taeniae, sacculations, or appendices epiploicae, and lacks a posterior mesentary (p. 5–7).
3. Define the cranial and caudal boundaries of the anal canal.
   The anal canal starts at the anorectal junction located at the palpable upper edge of the anal sphincter mechanism (junction of puborectalis and anal sphincter). The anal canal ends at the intersphincteric groove (approximately 2 cm distal to the dentate line) (p. 7).
4. What is the blood supply to the cecum?
   The cecum is supplied by the ileocolic artery (p. 8).
5. The inferior mesenteric vein empties into what vessel?
   The splenic vein (p. 9).
6. What type of mucosa does the colon contain?
   Columnar epithelium (p. 10).
7. Which layer of the bowel is the strongest?
   The submucosa (p. 10).

REFERENCES

1. Beck DE. Anatomy. In: Beck DE, Welling DR, eds. Patient Care in Colorectal Surgery. Boston: Little, Brown, 1991:3–9.
2. Gray H, Goss CM. The Digestive System. In: Anatomy of the Human Body. Philadelphia: Lea & Fehiger, 1974:1155–1264.
3. Hollinshead WH. The thorax, abdomen, and pelvis. In: Anatomy for Surgeons. New York: Harper & Row, 1971:676–718.
4. Goligher JC. Surgery of the Anus, Rectum, and Colon. 5th ed. London: Bailliere Tindall, 1984:1–47.
5. Netter F. Ciba Collection of Medical Illustrations. Vol 3. Part 11. Summit, NJ: Ciba-Geigy Corp, 1962.
6. Gordon PH, Nivatvongs S, eds. Principles and Practice of Surgery for the Colon, Rectum, and Anus. St. Louis: Quality Medical Publishing, 1992:3–38.
7. Beck DE. Anatomy. In: Beck DE, ed. Handbook of Colorectal Surgery. St. Louis: Quality Medical Publishing, 1997:3–12.
8. Phillips SF, Edwards DAW. Some aspects of anal continence and defecation. Gut 1965; 6:396–406.
9. Pemberton JH. Anatomy and physiology of the anus and rectum. In: Beck DE, Wexner SD, eds. Fundamentals of Anorectal Surgery. New York: McGraw-Hill, 1992:1–24.
10. Corman ML. Colon and Rectal Surgery. 3rd ed. Philadelphia: JB Lippincott, 1984: 1–48.

2

Pathophysiology

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As humans have become more "civilized," conditions of the colon, rectum, and anus have become more prevalent. Diseases such as irritable bowel syndrome, ulcerative colitis, Crohn's disease, diverticulitis, colorectal carcinoma, and constipation are now so common that they have supported the growth of gastroenterology and colon and rectal surgery as specialty practices.

Effective treatment of colorectal disorders depends on a sound understanding of basic physiology. Although little attention was previously given to the colon and anorectum compared with other portions of the digestive tract, our knowledge of the physiology of the colon and anorectum has increased substantially in recent years. The colon and anorectum are responsible for the storage, transport, processing, and timely expulsion of intestinal contents exiting the ileocecal valve. These functions depend on coordination of neural, hormonal, and muscular interactions both locally and centrally. This chapter reviews basic concepts of physiology of the colon, rectum, and anus in a normal individual and briefly discusses the altered physiology of severa1 disease states.

COLONIC MOTILITY

Motility is of central importance when discussing colonic function. Although to the layperson colonic motility may be categorized as "too fast," "too slow," or "just right," the actual study of colonic motility physiology is more complex. Colonic motility is more difficult to study than small bowel motility because of the great regional heterogeneity in the colon and the intermittent and unpredictable nature of colonic contractile waves. Even the normal pattern of motility remains the subject of debate. I have divided this review of colonic motility into sections on motor activity and myoelectric activity, although these functions are inextricably woven together in vivo. The coordinated process of defecation will be discussed later in the chapter.

Colonic Motor Activity

Our understanding of human colonic contractile activity was initially inferred from in vivo animal studies and then based on radiographic observations of ingested barium and radiopaque markers in the human colon and on manometric studies using balloon- or open-tipped catheters. To simplify a complex field of often conflicting evidence, it is helpful to refer to the work of Cannon [1] and Elliot and Barclay [2,3] at the turn of the century, much of which has been confirmed by later investigators [4].

Contractions in the proximal colon are characterized by antiperistaltic waves traveling from midtransverse colon toward the cecum. These waves were initially described as having a fundamental frequency of 5.5 cycles/min, lasting for 2–8 minutes, with 10–15 minutes of inactivity between episodes [1]. These waves are more prominent in herbivores than in omnivores and are thus thought to allow for return of complex polysaccharides toward the cecum for fermentation. They may also function to improve the efficiency of water and salt absorption in the proximal colon. The region extending from the midtransverse colon to the proximal rectosigmoid is characterized by intermittent contractile waves causing primarily segmental, nonpropulsive movement. There is, however, a slow net distal progression of feces toward the rectum. The proposed function of these segmental contractions is to mix the colonic contents to improve absorption.

The rectosigmoid and descending colon have been observed to have strong, organized contraction waves that propel a stool bolus distally through a long segment of colon. These so-called mass movements occur a few times daily and are associated with meals.

Colonic transit studies using radiopaque nonabsorbable markers reveal two areas of delayed colonic transit: the midtransverse colon and the rectosigmoid colon. The midtransverse colon is the area of transition where the proximal pattern of retrograde peristalsis changes to the distal pattern of antegrade peristalsis. For this reason, the midtransverse colon has been proposed to be the site of the colonic

pacemaker [5]. The second area of transit delay is the rectosigmoid, referred to as the rectosigmoid sphincter of O'Beirne [6]. In the nineteenth century, O'Beirne and others proposed that the thickened muscularis of the sigmoid functioned as an anatomical sphincter and was the major determinant of fecal continence. Although there is not a true sphincter in the rectosigmoid, the delay in stool transit at this level may serve a purpose, since it enables more complete water and sodium absorption by the left colon [7].

Many factors affect colonic motility. Emotions such as hostility, anger, and resentment are associated with hypermotility, whereas anxiety and fear are associated with hypomotility. Exercise has been shown to increase both segmental and peristaltic colonic activity; sleep is a depressant of colonic motility. Mechanical colonic distention stimulates motility and is the basis for the effect of bulking types of laxatives. Nondigestible polysaccharides and cellulose derivatives absorb water and increase fecal mass, thus stimulating colonic propulsion [8].

As anyone who has run to the toilet after breakfast can attest, eating is a potent colonic stimulant. This gastrocolic reflex, as described by Hertz and Newton [9], involves increased motor and electrical activity in the colon and causes the urge to defecate after a meal. The exact mechanism of this response is not known, but various neural and hormonal mediators have been implicated [10,11]. Fatty meals appear to have a greater effect on colonic motility than carbohydrate or protein meals.

Colonic Myoelectric Activity

Although the electrical activity of gastric and small intestinal smooth muscle has been well documented, that of colonic smooth muscle remains less well defined. As in the stomach and small intestine, two types of electrical signals are generated in the colon: slow waves or slow electrical transients and spikes or rapid transients. Because of the difficulties in measuring electrical activity in the human colon, much controversy exists regarding the origin, frequency, and incidence of slow waves [9,12]. It is thought that several slow wave pacemaker sites are present in the colon, one being in the midtransverse colon corresponding to the site of origin of retrograde peristalsis (as discussed earlier) [5]. Although slow wave activity often leads to uncoordinated smooth muscle cell depolarization (phase unlocked), it may propagate in such a way that depolarization proceeds with a constant time lag along a directional gradient causing coordinated colonic contractions (phase locked) [8].

Colonic spike activity occurs either as short or long bursts. Clusters of spike bursts may migrate in either direction in the colon. Long spike bursts that migrate rapidly in a distal direction are associated with passing flatus or defecating [12]. The relationship of slow waves to spike activity is unclear.

Marker Studies

Measurement of colonic motor and myoelectric activity does not always correlate with colonic transit, because electrical and contractile waves do not always propagate distally. Thus, measurement of colonic transit time does not involve measurement of colonic motor or myoelectric activity directly. The most common method involves ingestion of several radiopaque markers and sequential plain abdominal radiography. In individuals with normal gastrointestinal transit, the first markers are excreted at 36–48 hours, and 80% of the markers are excreted within 5 days. An alternate method involves ingestion of three different shaped radiopaque markers on 3 successive days, followed by plain abdominal radiography on the fourth day. If 24 markers are ingested on 3 successive days, the number of markers corresponds to the colonic transit time in hours. This method also permits the evaluation of transit through different areas of the colon [5].

COLONIC MOTILITY DISORDERS

Perturbations in colonic motility are associated with a number of clinical disorders, including irritable bowel syndrome, diverticular disease, idiopathic megacolon, constipation, diarrhea, postoperative ileus, and colonic pseudoobstruction. Some of these topics are covered in more detail elsewhere in this book.

Irritable Bowel Syndrome and Diverticular Disease

Irritable bowel syndrome is a disorder manifested by altered bowel habits and abdominal pain in the absence of other pathological findings. Patients with a diagnosis of irritable bowel syndrome have been shown to have increased slow wave activity (three cycles/min) in the rectosigmoid, corresponding to increased contractile activity at the same frequency [13,14]. A similar motility pattern has been noted in patients with diverticular disease, and some authors [8,15] have suggested that the underlying mechanism producing diverticula and irritable bowel syndrome is the same. Uncoordinated smooth muscle activity, followed by increased intraluminal pressure, may in part contribute to the pathogenesis of colonic diverticula [15].

It should be noted, however, that irritable bowel syndrome is a diagnosis of exclusion and has many different presentations. Thus, caution should be exercised when interpreting studies of patients who carry the diagnosis, because these patients may have different causes of their symptoms.

Postoperative Ileus

Postoperative ileus is a temporary impairment of intestinal motility after operation. Ileus is most commonly seen after laparotomy, but it may follow thoracot-

omy or other extraperitoneal procedures. In the past, the duration of postoperative ileus has been said to be proportional to the severity and duration of the surgical procedure. However, experimental evidence exists showing that the recovery of coordinated intestinal function is not influenced by either the magnitude or the length of an operative procedure [16,17]. The shorter period of ileus noted after laparoscopic gastrointestinal procedures has lent further credence to this concept.

A growing body of evidence implicates the colon, primarily the distal colon, as the most persistent site of postoperative ileus. Studies by Condon et al. [17,18] in monkeys and humans have shown that recovery from ileus is faster in the stomach and small bowel than in the colon and that the right colon recovers more rapidly than the left colon. The sequential return of motility in different segments of the gastrointestinal tract after operation may explain why a patient may have "active bowel sounds" postoperatively and yet have persistent colonic ileus, and why a trial of oral feeding fails see Chapter 10).

The pathogenesis of postoperative ileus is unclear. Several theories exist that attempt to explain the mechanism of ileus, including sympathetic hyperactivity inhibiting bowel motility, peritoneal irritation caused by foreign material, electrolyte imbalance, and the effects of anesthetics and narcotic analgesics. The traditional view of the effect of the autonomic nervous system on the intestine consisted of a prokinetic, secretagogue action of the parasympathetic system and an inhibitory, antisecretory action of the sympathetic system. Using this concept, investigators have tried to shorten the duration of ileus with adrenergic blockade and parasympathetic stimulation [19–21]. The results have been mixed. Although the treatment has simple physiological appeal, part of the lack of consistent success can be attributed to the complexity of the body's control of intestinal motility, which includes the effects of a plethora of intestinal hormones, such as vasoactive inhibitory peptide, motilin, peptide YY, cholecystokinin, and neuropeptide Y [5].

Electrolyte imbalances are thought to play a role in the prolongation of ileus. Hypokalemia, in particular, has been shown to reduce colonic contractile activity in monkeys [22]. Anesthesia was once thought to cause postoperative ileus. Although inhaled anesthetics, specifically enflurane and halothane, have been shown to reduce contractions in the colon, the effect is short lived and is rapidly reversed by cessation of the anesthetic [23]. Nitrous oxide has no effect on colonic contractile activity [23]. Narcotics have been shown to depress colonic motility, although not in a uniform fashion. Low doses of morphine increase the number of nonmigrating random colonic contractions. Higher doses of morphine, however, inhibit colonic electrical and contractile activity [17,24,25]. This narcotic effect may play a major role in the postoperative ileus seen after major laparotomies. The use of colonic morphine receptor agonists is being studied to reduce the incidence of postoperative ileus. Epidural morphine does not affect colonic motility, [24] suggesting that the opioid receptors in the spinal cord do not control intestinal motility.

Colonic Pseudo-obstruction (Ogilvie's Syndrome)

Intestinal pseudo-obstruction, a profound ileus without evidence of mechanical obstruction, was first described by Ingelfinger in 1943 [26]. The first description of the colonic variant of pseudo-obstruction is thought to be Sir Heneage Ogilvie's 1948 report [27] of two cases associated with malignant infiltration of the celiac plexus. Colonic pseudo-obstruction is associated with neuroleptic medications, opiates, malignancy, and severe metabolic illness. One mechanism thought to play a role in its pathogenesis is sympathetic overactivity overriding the parasympathetic system. This concept is supported by anecdotal reports of success with epidural anesthesia [28], which paralyzes the sympathetic afferent and efferent nerve fibers to the colon, and with neostigmine [29], which increases parasympathetic tone by its anticholinesterase effect. Prokinetic agents such as cisapride and erythromycin have also been used to treat pseudo-obstruction, although colonoscopic decompression has remained the primary treatment modality [5].

WATER AND ELECTROLYTE TRANSPORT

Absorption

The major absorptive function of the colon is the final regulation of water and electrolyte balance in the intestine, deemed colonic salvage. The colon reduces the volume of enteric contents by absorbing greater than 90% of the water and electrolytes presented to it. On average, this accounts for 1 or 2 L of fluid and 200 mEq of sodium and chloride per day. During a 24-hour period, 8 L of fluid enters the jejunum. In healthy individuals, the small bowel absorbs about 6.5 L and the colon 1.4 L, leaving 0.1 L of normal fecal water content. Under maximum conditions, the colon can absorb 5 to 6 L of fluid a day. Only if small bowel absorption is reduced to less than 2 L a day is colonic salvage overwhelmed and the resultant increase in fecal water content manifested as diarrhea [5].

The colon is able to absorb sodium against high concentration gradients, especially in the distal colon, which shares many basic cellular mechanisms of sodium and water transport with the distal convoluted tubule of the kidney [30]. The colonic response to aldosterone stimulation may be an important compensatory mechanism during dehydration.

Although active absorption of nutrients is minimal, the colon can passively absorb short-chain fatty acids formed by intraluminal bacterial fermentation of unabsorbed carbohydrates. This can account for up to 540 kcal per day of assimilated calories. The absorbed short-chain fatty acids, principally butyrate, are the major fuel sources of the colonic epithelium [31–33]. Evidence exists that short-chain fatty acid metabolism is impaired in patients with ulcerative colitis [34–39] and that intraluminal infusion of short-chain fatty acids can be of benefit in patients with colitis [40]. Short-chain fatty acids have also been shown to be

effective in treating diversion colitis, implicating colonocyte nutritional deficiency as the cause of this disorder [41–42].

Secretion

In healthy persons the colon absorbs water, sodium, and chloride while secreting potassium and bicarbonate. Potassium transport in the colon is mainly passive along an electrochemical gradient generated by the active transport of sodium. Bicarbonate is exchanged with chloride by an electroneutral mechanism [43].

A number of agents can stimulate fluid and electrolyte secretion in the colon, including bacteria, enterotoxins, hormones, neurotransmitters, and laxatives. The diarrhea associated with *Shigella* and *Salmonella* infection is caused by diminished absorption or increased secretion of water, sodium, and chloride. Intestinal hormones, particularly vasoactive intestinal polypeptide (VIP), have been shown to have significant effects on colonic absorption and secretion. Prostaglandins play a role in the pathogenesis of diarrhea associated with ulcerative colitis and several laxatives [8].

Any sort of irritation to the colon can cause increased secretion, which results in diarrhea. Common causes of this sort of diarrhea include bile salt malabsorption after resection of the terminal ileum and long-chain fatty acid malabsorption in steatorrhea. The induced colonic mucus and fluid are high in potassium and may result in potassium depletion in chronic cases.

BACTERIAL BARRIER

The human colon is sterile at birth, but within a matter of hours the intestine is colonized from the environment in an oral to anal direction. *Bacteroides*, destined to be the dominant bacteria in the colon, is first noted at about 10 days after birth. By 3–4 weeks after birth, the characteristic stool flora is established and persists into adult life. An individual's pattern of bacterial flora most closely resembles that of his or her mother.

The bacterial population of the colon is a complex collection of aerobic and anaerobic microorganisms. Nearly one third of the fecal dry weight consists of viable bacteria, with as many as 10^{11}–10^{12} bacteria present per gram of feces. Anaerobic bacteria dominate the flora by as much as 10,000:1 over aerobic organisms, but the mixture is diverse, with as many as 400 different species cultured from the stool of one individual. Knowledge of the types of normal colonic bacteria is of paramount importance to the surgeon who must use this information to guide the selection of antibiotic therapy, both for prophylaxis and treatment.

INTESTINAL GAS

Nitrogen, oxygen, carbon dioxide, hydrogen, and methane make up 99% of all the gas in the intestine [43]. Nitrogen and oxygen are found in the atmosphere

and appear in the colon by means of swallowing air. Hydrogen, methane, and carbon dioxide are produced by bacterial fermentation of carbohydrates and proteins in the colon. An eminent flatologist, Levitt [44], has shown that most patients who complain of excessive flatus have high concentrations of hydrogen and carbon dioxide in their intestinal gas. Since carbon dioxide is an end-product of bacterial fermentation, therapy consists of diet manipulation to decrease the amount of ingested carbohydrates, especially lactose, wheat, and potatoes.

One of the most important points for the surgeon to remember is the explosive nature of hydrogen and methane. Opening unprepared colon with an electrocautery device can have dramatic and disastrous consequences.

ANORECTAL PHYSIOLOGY

Fecal continence is the ability to defer the urge to defecate until a socially convenient time and place can be found. Many factors are involved in fecal continence, including anal canal pressures generated by the sphincter mechanism, anorectal angle formed by the pelvic floor musculature, anorectal sensation, rectal compliance, anorectal reflexes, colonic transit, and stool volume and consistency. This section focuses on the anorectal mechanisms that contribute to fecal continence.

Anal Canal Pressures and Anal Sphincters

The internal and external anal sphincters surround the anal canal and are responsible for maintaining resting pressure and generating squeeze pressures. The internal anal sphincter is composed of smooth muscle and is tonically contracted at rest. It contributes about 85% of the resting tone of the anal canal. Dividing the internal anal sphincter in the presence of an intact external anal sphincter weakens anal tone but does not abolish it [45]. The external anal sphincter is one of the only striated muscles in the body that maintains a constant tone. External anal sphincter tone is maintained during the day and, to a lesser extent, during sleep.

Anal canal squeeze pressures are generated by the puborectalis muscle and the external anal sphincter, which are under voluntary control. Squeeze pressures are more than twice the resting pressure during maximum effort. Maximum squeeze pressure can be maintained for less than 1 minute; the sphincter rapidly fatigues after that time.

The sphincter mechanism is not symmetric. Anal manometric measurements have shown that resting pressures posteriorly are highest proximally and lowest near the anal verge [46]. Anterior resting pressures vary between the sexes, being highest distally in women and highest proximally in men [47]. Squeeze pressures are also asymmetric. The high-pressure zone of the anal sphincter is

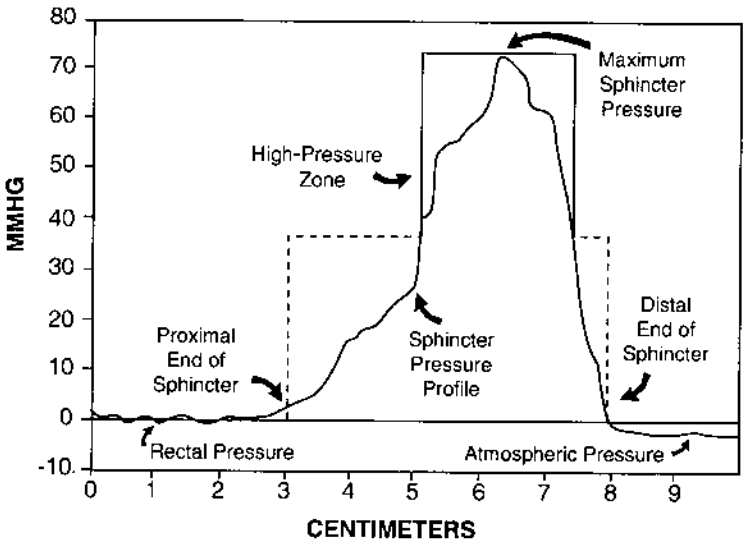

Figure 1 Characteristics of a typical longitudinal pressure profile of the resting anorectal sphincter. Pressures have been equated to a zero rectal pressure. The pressures from an eight-channel multilumen probe during continuous resting pull-out have been averaged at each point along the sphincter by microcomputer. (From Ref. 46.)

located distal to the midpoint of the sphincter (Figure 1). A transition from posterior predominance to anterior predominance occurs as one travels from proximal to distal in the anal canal [48].

The relative contributions of the internal and external anal sphincters to maintaining continence has been the subject of some debate. At one time, it was thought that the internal anal sphincter was not important in continence because of the reflex relaxation of the internal sphincter that occurs with rectal distention, the rectoanal inhibitory reflex [49] (see p. xx). However, after surgeons found that complete division of the internal sphincter for treatment of anal fissure resulted in a 40% risk of soiling or incontinence for flatus or liquid stool, [50] this view was modified. Loss of internal anal sphincter function can be compensated for by intact and well-functioning external anal sphincter and puborectalis muscles. However, if these muscles weaken with age or are subsequently injured, incontinence may result [51].

The external anal sphincter is important in maintaining continence. In one study, [52] a persistent defect of the external anal sphincter by ultrasonography

was associated with a 50% prevalence of incontinence to flatus or stool in patients who underwent primary suture repair of obstetric sphincter injuries. Further evidence of the role of the external sphincter in continence comes from the good results achieved by direct sphincteroplasty in incontinent patients with simple defects of the external anal sphincter [53].

Anorectal Angle

Another mechanism that helps to maintain fecal continence is the configuration of the pelvic floor, formed predominantly by the anterior pull of the puborectalis muscle at the level of the anorectal ring, producing the anorectal angle (Figure 2). The angle is between 60 and 105 degrees at rest and becomes more acute during squeeze and more obtuse during defecation. The flap valve theory, proposed by Parks et al. [54], suggests that the puborectalis pulls the anorectal junction anteri-

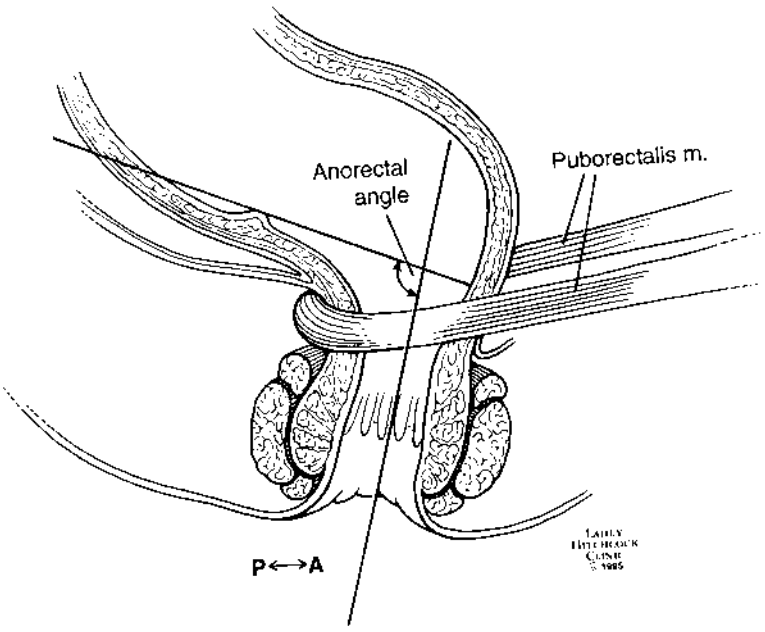

Figure 2 The anorectal angle is formed by the anterior pull of the puhorectalis muscle. The angle is measured at the intersection of lines drawn through the center of the anal canal and along the posterior wall of the rectum. The angle is between 60 and 105 degrees at rest and becomes more acute during squeeze and more obtuse during defecation. (Courtesy Lahey Hitchcock Clinic.)

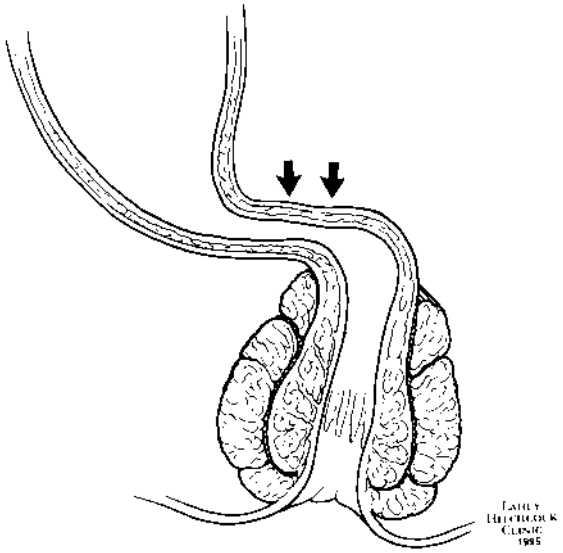

FIGURE 3 The "flap valve" effect of the puborectalis pulling the rectum anteriorly during squeeze, thereby permitting intra-abdominal pressures during the Valsalva maneuver to close off the rectum. (Courtesy Lahey Hitchcock Clinic.)

orly and that increases in abdominal pressure seal the anterior wall to the posterior wall of the anal canal (Figure 3). A Valsalva maneuver would thus occlude the lumen and protect the lower anal canal from the transmission of pressure and leakage of stool. The flap valve theory and the contribution of the anorectal angle to maintaining continence are controversial, however. Bartolo et al. [55] demonstrated that during maximum Valsalva maneuver, the anterior wall remains separate from the posterior wall and the lumen remains patent in normal subjects. Although the flap valve theory may not be entirely accurate, the puborectalis is an important part of the continence mechanism. This is illustrated by the fact that division of the puborectalis in the treatment of constipation is associated with a high degree of incontinence of flatus and liquid stool [56].

The flutter valve theory, proposed by Phillips and Edwards [57], suggests that the puborectalis flattens the walls of the rectum from side to side and creates a slitlike opening in the pelvic floor. They proposed that sudden increases in abdominal pressure force the opposing walls of the proximal anorectal canal together, thus helping to maintain fecal continence (Figure 4). Anteroposterior and lateral images of the barium paste–coated rectum tend to support this view.

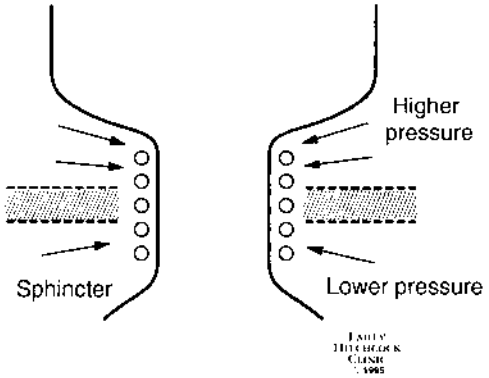

FIGURE 4 The "flutter valve" effect of increasing intra-abdominal pressure causes the walls of the anorectum to flatten together. (Courtesy Lahey Hitchcock Clinic.)

Anorectal Sensation

Sensory mechanisms exist that permit discrimination of the character of rectal contents (stool, liquid, or gas) and of the need to expel that content. These sensory receptors are located in the rectal muscularis, in the surrounding muscles of the pelvic floor, and/or in the anal canal mucosa [45]. A sampling response occurs in which transient relaxation of the upper part of the internal sphincter permits rectal contents to come into contact with the sensory epithelium of the proximal anal canal (the anal transition zone) for assessment of the nature of rectal content [58,59]. The ultimate importance of the sampling response is a matter of debate, however, since patients undergoing proctectomy and ileoanal anastomosis retain the ability to discriminate gas from stool, yet do not show evidence of a sampling response [60]. Furthermore, anesthetizing the anal canal mucosa has been shown not to affect continence to large-volume saline solution enemas [61].

Rectal Compliance

The rectum accommodates passively to distention. As intraluminal volume increases, intraluminal pressure remains low. In healthy individuals the rectum can accommodate a maximum tolerable volume of 400 mL while pressure remains low, less than 20 mmHg [45]. The rectum is also thought to have an accommodation response, which consists of receptive relaxation of the rectal ampulla to accommodate a fecal bolus [43]. Disease states that alter rectal compliance, such as inflammatory bowel disease and radiation proctitis, may result in frequency,

urgency, tenesmus, and incontinence as the rectum loses its ability to distend and becomes a stiff conduit.

Reflexes

A number of involuntary reflexes involve the anal sphincters. Testing of these reflexes may assist in evaluation of pelvic floor innervation. As mentioned earlier, the rectoanal inhibitory reflex is relaxation of the internal anal sphincter and brief contraction of the external anal sphincter with distention of the rectum. Rapid intermittent rectal distention causes prolonged relaxation of the internal anal sphincter, whereas continuous rectal distention initially causes internal anal sphincter relaxation, but the muscle gradually returns to its resting tone over time [62]. First described by Gowers [63] in 1877, this reflex is probably mediated by means of intramural nerve plexuses as it persists in patients with spinal cord and sacral nerve root lesions. The reflex is absent in patients with Hirschsprung's disease and may be used as an adjunct to rectal biopsy to make this diagnosis [62].

The anocutaneous reflex consists of a visible contraction, the so-called anal wink, with stimulation of the perianal skin. The pudendal nerve supplies both the afferent and efferent pathways through sacral segments S1–S4. Testing of this reflex is useful when evaluating a patient for fecal incontinence, because it can give information about pudendal nerve function. The bulbocavernosus reflex consists of contraction of the bulbocavernosus muscle, external anal sphincter, and urethral sphincter with stimulation of the glans penis or clitoris. The vesicoanal reflex is inhibition of external anal sphincter activity and increased internal anal sphincter activity during micturition.

DEFECATION

Defecation commonly begins with distention of the left colon by stool. Although the individual may not be aware of any discomfort, the colon responds to this distention by generating mass movement waves that carry the stool from the descending and sigmoid colon into the rectum. The rectal distention may or may not be sensed by the individual if the amount of stool entering the rectum is small. The reflex response to rectal distention is inhibition of the internal anal sphincter and contraction of the external anal sphincter.

If it is a socially acceptable time for defecation, the individual assumes a squatting or seated position. This action straightens the anorectal angle and facilitates passage of stool. Intrarectal and intra-abdominal pressures then rise, resulting in reflex relaxation of the external and internal anal sphincters and puborectalis muscles. A conscious relaxation of the external anal sphincter also occurs. Some individuals may pass stool without straining. Others, however, must strain to

initiate rectal emptymg. Straining causes the external and internal anal sphincters and puborectalis muscles to relax further. If mass movement peristalsis occurs simultaneously with rectal emptying, the entire left colon may be emptied. If not, the bowel is evacuated in piecemeal fashion. At the end of defecation, the pelvic floor musculature, sphincters, and anorectal angle return to their normal configuration.

If rectal distention occurs at an inopportune time, the rectoanal inhibitory reflex provides brief automatic protection by means of contraction of the external anal sphincter. Continued voluntary squeeze of the external sphincter permits further deferment of defecation. This mechanism alone would soon fail because of the rapid fatigue of skeletal muscle were it not for the rapid, receptive relaxation of the rectum. The accommodation response of the rectum permits the external sphincter to relax after the pressure in the rectum has decreased. At this point, both the awareness of stool in the rectum and the urge to defecate decrease. The accommodation response can be overwhelmed, however, if the volume of stool coming into the rectum is great or if the rectum is already near maximum distention, as in the case of fecal impaction.

ROUNDS QUESTIONS

1. What is the function of segmental colonic contractions?
   The function is to mix the colonic contractions to improve absorption (p. 14).
2. Where is the proposed site of the colonic pacemaker?
   The midtransverse colon (pp. 14–15).
3. What is postoperative ileus?
   Temporary impairment of intestinal motility after operation (p. 16).
4. What is intestinal pseudo-obstruction (Ogilvie's syndrome)?
   A profound ileus without evidence of mechanical obstruction (p. 18).
5. What does the colon absorb and secrete?
   The colon absorbs water, sodium, and chloride and secretes potassium and bicarbonate (pp. 18–19).
6. Which muscle is mainly responsible for the resting tone of the anal sphincter?
   The internal sphincter (p. 20).
7. What is the sampling response?
   Transient relaxation of the upper part of the internal sphincter, which allows the rectal contents to come into contact with the sensory epithelium of the proximal anal canal (p. 24).
8. What disease is associated with an absence of the rectoanal inhibitory reflex?
   Hirschsprung's disease (p. 25).

REFERENCES

1. Cannon W. The movements of the intestine studied by means of roentgen rays. Am J Physiol 1902; 6:251–277.
2. Barclay A. Radiological studies of the large intestine. Br J Surg 1915; 2:638–652.
3. Elliot T, Barclay S. Antiperistalsis and other activities of the colon. J Physiol 1904; 31:272–303.
4. Ritchie J. Colonic motor activity and bowel function. Gut 1968; 9:442–456.
5. Armstrong D, Ballantyne G. Physiology of the small and large intestines. In: Mazier W, Luchtefeld M, Levien D, Senagore A, eds. Surgery of the Colon, Rectum, and Anus. Philadelphia: WB Saunders, 1995:40–65.
6. Ballantyne G. Rectosigmoid sphincter of O'Beirne. Dis Colon Rectum 1986; 29: 525–531.
7. Debongnie J, Phillips S. Capacity of the human colon to absorb fluid. Gastroenterology 1978; 74:698–703.
8. Frantzides CT. Physiology of the colon. In: Zuidema G, ed. Shackelford's Surgery of the Alimentary Tract. 4th ed. Philadelphia: WB Saunders, 1995:17–22.
9. Hertz A, Newton A. The normal movements of the colon in man. J Physiol 1913; 45:57.
10. Snape W, Matarazzo S, Cohen S. Effect of eating and gastrointestinal hormones on human colonic myoelectric and motor activity. Gastroenterology 1978; 75:373–378.
11. Strom J, Condon R, Schulte W, Cowles V, Go V. Glucagon, gastric inhibitory polypeptide and the gastrocolic response. Am J Surg 1982; 143:155–159.
12. Frantzides C, Condon R, Cowles V. Early postoperative colon electrical response activity. Surg Forum 1985; 36:163–165.
13. Snape W, Carlson G, Matarazzo S, Cohen S. Evidence that abnormal myoelectrical activity produces colonic motor dysfunction in the irritable bowel syndrome. Gastroenterology 1977; 72:383–387.
14. Taylor I, Darby C, Hammond P. Comparison of rectosigmoid myoelectriccal activity in the irritable colon syndrome during relapses and remissions. Gut 1978; 19: 923–929.
15. Painter N, Truelove S, Ardran G, Tuckey M. Segmentation and the localization of intraluminal pressures in the human colon, with special reference to the pathogenesis of colonic diverticula. Gastroenterology 1965; 49:169–177.
16. Graber J, Schulte W, Condon R, Cowles V. Relationship of duration of postoperative ileus to extent and site of operative dissection. Surgery 1982; 92:87–92.
17. Condon R, Cowles V, Schulte W, Frantzides C, Mahoney J, Sarna S. Resolution of postoperative ileus in humans. Ann Surg 1986; 203:574–581.
18. Woods J, Erickson L, Condon R, Schulte W, Sillin L. Postoperative ileus: A colonic problem? Surgery 1978; 54:527–533.
19. Heimbeck D, Crout J. Treatment of paralytic ileus with adrenergic neuronal blocking drugs. Surgery 1971; 69:582–587.
20. Neely J, Catchpole B. The restoration of alimentary tract motility by pharmacological means. Br J Surg 1971; 58:21–28.
21. Catchpole B. Ileus: Use of sympathetic blocking agents in its treatment. Surgery 1969; 66:811–820.

22. Schulte W, Cowles V, Condon R. Hypokalemia and the gastrocolic response [abst]. Dig Dis Sci 1984; 29:551.
23. Condon R, Cowles V, Ekbom G, Schulte W, Hess G. Effects of halothane, enflurane and nitrous oxide on colon motility. Surgery 1987; 101:61–85.
24. Frantzides C, Cowles V, Salaymeh B, Tekin E, Condon R. Morphine effects on human colonic myoelectric activity in the postoperative period. Am J Surg 1992; 163:144–149.
25. Frantzides C, Condon R, Schulte W, Cowles V. Effects of morphine on colonic myoelectric activity in subhuman primates. Am J Physiol 1990; 21:247–252.
26. Ingelfinger E. The diagnosis of sprue in nontropical cases. N Engl J Med 1943; 228: 180–184.
27. Ogilvie H. Large intestinal colic due to sympathetic deprivation: a new clinical syndrome. Br Med J 1948; 2:671–673.
28. Lee J, Taylor B, Singleton B. Epidural anesthesia for acute pseudo-obstruction of the colon (Ogilvie's syndrome). Dis Colon Rectum 1988; 31:666–691.
29. Stephenson B, Morgan A, Salaman J, Wheeler M. Ogilvie's syndrome: a new approach to an old problem. Dis Colon Rectum 1995; 38:424–427.
30. Frizzel R, Schults S. Effect of aldosterone on ion transport by rabbit colon in vitro. J Membr Biol 1978; 39:1–26.
31. Cummings J. Short chain fatty acids in the human colon. Gut 1981; 22:763–769.
32. Clausen M, Mortensen P. Kinetic studies on the metabolism of short chain fatty acids and glucose by isolated rat colonocytes. Gastroenterology 1994; 108:423–432.
33. Roediger W. Role of anaerobic bacteria in the metabolic welfare of the colonic mucosa in man. Gut 1980; 21:793–798.
34. Chapman M, Grahn M, Boyle M, Hutton M, Rogers J, Williams N. Butyrate oxidation is impaired in the colonic mucosa of sufferers of quiescent ulcerative colitis. Gut 1994; 35:73–76.
35. Chapman M, Grahn M, Hutton M, Williams N. Butyrate metabolism in the terminal ileal mucosa of patients with ulcerative colitis. Br J Surg 1995; 82:36–38.
36. Roediger W. The colonic epithelium in ulcerative colitis: an energy-deficient disease? Lancet 1980; 2:712–715.
37. Roediger W. The starved colon-diminished mucosal nutrition, diminished absorption, and colitis. Dis Colon Rectum 1990; 33:858–862.
38. Vernia P, Gnaedinger A, Hauck W, Breuer R. Organic anions and the diarrhea of inflammatory bowel disease. Dig Dis Sci 1988; 33:1353–1358.
39. Vernia P, Caprilli R, Latella G, Barhetti F, Magliocca F, Cittadini M. Fecal lactate and ulcerative colitis. Gastroenterology 1988; 95:1564–1568.
40. Senagore A, MacKeigan J, Scheider M, Ehrom S. Short-chain fatty acid enemas: a cost-effective alternative in the treatment of nonspecific proctosigmoiditis. Dis Colon Rectum 1992; 35:923–927.
41. Harig J, Soergel K, Komorowski R, Wood C. Treatment of diversion colitis with short chain fatty acid irrigation. N Engl J Med 1989; 320:23–28.
42. Kissmeyer-Nielsen P, Mortensen F, Laurherg S, Hessov I. Transmural trophic effect of short chain fatty acid infusions on atrophic, defunctioned rat colon. Dis Colon Rectum 1995; 38:946–951.

43. Schouten W, Gordon PH. Physiology. In: Gordon PH, Navatvongs S, eds. Principles and Practice of Surgery for the Colon, Rectum, and Anus. St. Louis: Quality Medical Publishing, 1992:39–79.
44. Levitt M. Intestinal gas production—recent advances in flatology. N Engl J Med 1980; 302:1474–1475.
45. Pemherton J, Meagher A. Anatomy and physiology of the anus and rectum. In: Condon R, ed. Colon. Vol 4. In: Zuidema G, ed. Shackelford's Surgery of the Alimentary Tract. 4th ed. Philadelphia: WB Saunders, 1995: 275–309.
46. Coller J. Clinical application of anorectal manometry. Gastroenterol Clin North Am 1987; 16:17–33.
47. McHugh S, Diamant N. Anal canal pressure profile: a reappraisal as determined by rapid pullthrough technique. Gut 1987; 28:1234–1241.
48. Taylor B, Beart RJ, Phillips S. Longitudinal and radial variations of pressure in the human anal sphincter. Gastroenterology 1984; 86:693–697.
49. Goligher J, Hughes E. Sensibility of the rectum and colon: its role in the mechanism of anal continence. Lancet 1951; 1:543–548.
50. Bennett R, Goligher J. Results of internal sphincterotomy for anal fissure. Br Med J 1962; 2:1500–1503.
51. Rasmussen O. Anorectal function. Dis Colon Rectum 1994; 37:386–403.
52. Nielsen M, Hauge C, Rasmussen O, Pedersen J, Christiansen J. Anal endosonographic findings in the follow-up of primarily sutured sphincteric ruptures. Br J Surg 1992; 79:104–106.
53. Jorge J, Wexner S. Etiology and management of fecal incontinence. Dis Colon Rectum 1993; 36:77–97.
54. Parks A, Porter N, Hardcastle J. The syndrome of the descending perineum. Proc R Soc Med 1966; 59:477–482.
55. Bartolo D, Roe A, Locke-Edwards J, Virjee J, Mortensen N. Flap-valve theory of anorectal continence. Br J Surg 1986; 73:1012–1014.
56. Barnes P, Hawley P, Preston D, Lennard-Jones J. Experience of posterior division of the puborectalis muscle in the management of chronic constipation. Br J Surg 1985; 72:475–477.
57. Phillips S, Edwards D. Some aspects of anal incontinence and defecation. Gut 1965; 6:396–406.
58. Duthie H, Bennett R. The relation of sensation in the anal canal to the functional anal sphincter. A possible factor in anal continence. Gut 1963; 4:179–182.
59. Miller R, Bartolo D, Cervero F, Mortensen N. Anorectal temperature sensation: a comparison of normal and incontinent patients. Br J Surg 1987; 74:511–515.
60. Beart RJ, Dozois R, Wolff B, Pemberton J. Mechanisms of rectal continence: lessons from the ileoanal procedure. Am J Surg 1985; 149:31–34.
61. Read M, Read N. The role of anorectal sensation in preserving continence. Gut 1982; 23:345–347.
62. Burleigh D, D'Mello A. Physiology and pharmacology of the internal anal sphincter. In: Henry M, Swash M, eds. Coloproctology and the Pelvic Floor. Cambridge, England: Cambridge University Press, 1985:22–41.
63. Gowers W. The autonomic action of the sphincter ani. Proc R Soc Med 1877; 26: 77–84.

3

History and Physical Examination

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A compassionately taken thorough history, complemented by a directed but gentle physical examination, is usually more revealing than a battery of sophisticated diagnostic tests in evaluating the patient with colorectal and anal complaints. Disorders of this "unmentionable" part of the body are often embarrassing for the patient to discuss and require great tact on the part of the examiner. This chapter focuses on features of the patient encounter unique to the colorectal patient. [1].

HISTORY

The value of a carefully taken history cannot be overemphasized. It often uncovers pieces of the puzzle that allow for proper diagnosis. One of the rewards in medicine is finding on physical examination the problem that was suspected on taking the history. [2].

Present Illness

The patient should first be asked to describe the problem in his or her own words. Duration of symptoms is important; incontinence may date to the birth of a child, possibly signifying an obstetric sphincter injury. When did the patient first notice his or her symptom? Exacerbating or alleviating circumstances should be sought, as should prior treatment attempts. Is this the first episode, or is it a recurring problem? Questions need to direct the patient to the present circumstance and should be tailored to the age and educational level of the patient.

Pain is the presenting symptom of many disorders. Abdominal complaints are often nonspecific; colonic distention causes hypogastric pain, whereas rectal conditions may be felt in the sacral or perineal areas. Crampy, colicky pain usually accompanies obstruction, possibly from a tumor, or excessive contraction of the colon, seen with diarrheal illnesses. Inflammatory conditions such as diverticulitis may cause peritoneal irritation, which is more readily localized, since this type of pain is carried by the somatic inervation. Discomfort that is worsened by hitting bumps during the car ride to the examination often signifies peritoneal irritation. It is important to uncover associated initiating or relieving factors such as relief with passage of stool or flatus and changes in symptoms with posture or medication. In women, pain that is cyclical with menses may be caused by endometriosis. Finally, the character of the pain (sharp, dull), any movement (radiation), and intensity are explored.

To many patients, any anorectal condition is thought to be caused by hemorrhoids. The nature of the discomfort should be elicited. Sharp pain that follows a bowel movement is indicative of a fissure, whereas a throbbing pain often accompanies an abscess. Tenesmus, the urge to defecate, is found in inflammatory or neoplastic conditions. Swelling may represent hemorrhoids or rectal prolapse [3]. Bleeding is often quite worrisome to patients but usually represents benign disease. Is the blood bright red, dark blue, accompanied by clots, mixed with stool, or is it on the toilet tissue only (denoting an anal source)? Melena usually denotes a proxima1 source but may come from the right colon. Bloody diarrhea is seen in inflammatory or ischemic colitis; a combination of blood and mucus suggests neoplasia.

No evaluation of colorectal complaints is complete without an inquiry into bowel habits. Consistency, frequency, and size of stool as well as recent changes should be noted. What is the patient's normal bowel pattern? What has changed? Constipation, the infrequent bowel movement, should be distinguished from regular bowel movements that are hard to pass. Any maneuvers that the patient performs, such as abdominal or vaginal pressure or digitalization, need to be sought out. The degree of incontinence is assessed, and one should determine whether the patient is incontinent of flatus or liquid or solid stool as well as the number of incontinent episodes per day. The ability to sense stool in the rectum but inability to reach the toilet in time is differentiated from incontinence without

warning. The relation of any changes to events such as childbirth, pelvic surgery, or irradiation or conditions such as diabetes is also important [4].

Review of Systems

A brief systems review is useful in evaluating the colorectal patient [1]. Unexpected weight loss may herald an underlying malignancy. Inflammatory bowel disease can manifest itself by a number of extraintestinal complaints—arthritis, uveitis, skin lesions, or jaundice. Recent travel to areas of poor sanitation could explain new-onset diarrhea. A tactful exploration of sexual contacts and practices may be warranted. Dietary practices and any recent changes are documented. Symptoms of systemic diseases that may have an intestinal component are also reviewed. Weight gain, letheragy, and constipation might suggest hypothyroidism, whereas weight loss, rapid heart rate, skin changes, and diarrhea could result from hyperthyroidism.

Past Medical History

A survey of the patient's medical history should he included, with particular attention to prior colorectal problems, previous abdominal and anorectal operations, difficult labor or childbirth, and prior infections. Current prescription and over-the-counter medications must be reviewed. Laxative use or abuse is important to ascertain. Previous radiation therapy to the pelvis for gynecologic, prostatic, or rectal malignancy may explain certain symptoms, such as tenesmus. A recent course of antibiotic therapy could be the cause of diarrhea. Immunosuppression from steroidal medications or antirejection medications, chemotherapy, or acquired immunodeficiency syndrome (AIDS) can make conditions that are usually easily managed life-threatening [5]. Medical conditions such as diabetes or thyroid abnormalities can produce intestinal problems.

Family History

A pertinent family history must be included in the patient interview. Familial adenamatous polyposis (FAP) is an inherited condition of colonic polyps that leads to early colorectal carcinoma. It is inherited in an autosomal dominant pattern; screening of family members should begin at age 10. Sporadic colorectal carcinomas (those without an inherited or identified cause) also show a familial tendency, especially for first-degree relatives (parents, siblings, or children) [6]. Periodic colonoscopic screening is currently recommended for patients beginning at age 50 or at an age 10 years before the age at which their relative was diagnosed with colorectal cancer, whichever comes first. When a positive family history involves multiple family members over two or more generations, the possibility of an inherited cancer syndrome must be considered [7].

The two inherited colorectal cancer syndromes are FAP (previously described) and the much more common hereditary nonpolyposis colorectal cancer (HNPCC). As many as 9% of patients with colorectal cancer may have HNPCC [7]. While FAP has the diagnostic findings of colonic polyposis, HNPCC is usually diagnosed on the basis of the family history using the Amsterdam or Bethesda criteria [8,9]. Patients with HNPCC also have an associated increased risk of gynecologic and urinary tract cancers. Patients with HNPCC have a 70% lifetime risk of developing colorectal cancer and a 90% risk of developing one of the HNPCC-related cancer [7]. First-degree relatives (parents, siblings, and children) of patients with HNPCC have a 50% risk of also having the syndrome. Patients with HNPCC and their at-risk relatives need frequent screening for colorectal and associated malignancies. Some patients may also choose prophylactic surgery.

A history of more distant relatives with colorectal cancer or a history of sporadic polyps in a close relative does add risk, but it is not as high as that with a first degree relative. Inflammatory bowel disease may also show a tendency to run in families. Do other family members or close associates have similar symptoms?

PHYSICAL EXAMINATION

Physical assessment begins as soon as the patient is seen [1]. Is the patient uncomfortable walking or sitting? Are the clothes too loose, from recent weight loss, or too tight, from abdominal distention? Is the skin discolored from jaundice or renal failure? Does the face show the effects of long-term steroid use? Much about the patient's overall state of health can be gleaned from careful observation during the interview.

Abdominal Examination

A complete abdominal examination is indicated in all patients with new complaints and as part of routine cancer follow-up. It should consist of inspection, auscultation, percussion, and palpation. This section will not describe examination techniques in detail but will focus on key points for the evaluation of colorectal patients. The patient is positioned flat on the examination table, with the entire abdomen and the inguinal region accessible. The contour of the abdomen as well as any surgical scars and stomas should be noted. Auscultation assesses the timbre and vigor of bowel activity. The patient is asked to identify the location of the pain. Gentle percussion should always precede vigorous palpation. The skilled examiner can elicit signs of peritoneal irritation through gentle percussion with ease; aggressive, deep palpation to find "rebound tenderness" only hurts the patient and makes subsequent examinations difficult. If no significant tenderness or tympany is found on percussion, the abdomen may be palpated for masses

and organomegaly. Each incision should be carefully palpated with the patient straining to check for incisional hernias. Appliances should be removed from stomas and a gentle digital examination performed to assess for stenosis or parastomal hernia. Finally, the inguinal region should be examined for hernia or adenopathy [10].

Anorectal Examination

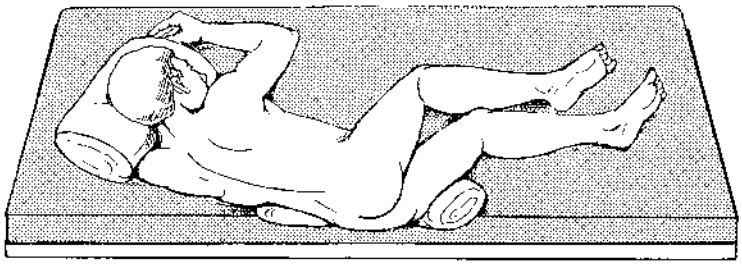
Examination of the perineal region consists of inspection and digital palpation, complemented by anoscopy, proctoscopy, or biopsy as indicated by findings. Before touching the perineal region, the examiner should warn the patient that the lubricant feels wet and cool and that the digital examination may produce mild pressure or a sensation similar to having a bowel movement.

Patient Positioning

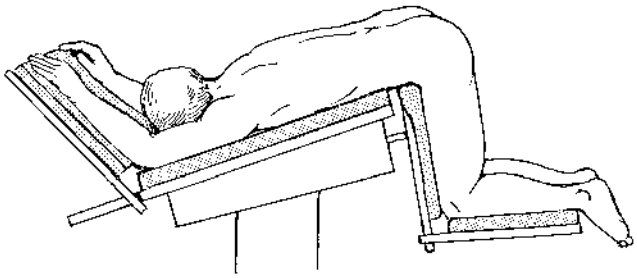
Several positions can be used for the examination and should take into account patient and examiner comfort, the equipment available, and exposure (Figure 1). The prone jackknife position is usually used with a movable procedure table. Patients wearing slacks are asked to kneel on the table platform (shelf) before dropping their slacks and underwear to prevent their trousers from dragging the floor, loss of pocket contents, and to avoid unnecessary undressing. After kneeling on the shelf, the patient bends forward and places his or her chest on the table with the elbows forward, palms on the table, and the back in a slight swayback position. The shelf is positioned to allow the abdomen to remain slightly off the table (Figure 1b). A sheet drapes the back and upper legs, preserving modesty and keeping the patient warm and comfortable.

After the patient is warned, the table is raised and tipped forward. Patients are asked not to straighten their legs, because this might cause them to slip off the table. The prone position allows better access to the perineum. For patients who have difficulty with the prone position (e.g., those who have undergone recent joint replacement surgery or who have arthritis or cardiovascular disease), other positions (such as Sims' or modified left lateral decubitus) are used.

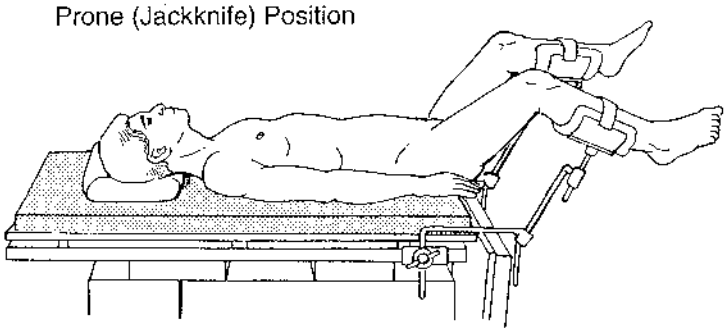
The Sims' or modified left lateral decubitus position (Figure 1a) is used if a movable procedure table is not available or if the patient cannot tolerate the prone position. The patient's head is placed on the opposite corner with the back angling across the table and the buttocks extending off the table. The hips are flexed and the knees are bent. This position is comfortable and prevents the patient from falling off the table. Having the buttocks extend off the table allows the buttocks to be easily spread and the end of an instrument such as a proctoscope, if it is to be used, to be manipulated in any direction. The end of the scope or the examiner's head (when looking through the end piece) is not hindered by the bed. Finally, this positioning allows any anal discharge to drop to the floor and not pool on the table, where it could contaminate the examiner's head or face.



A Left Lateral (Sims) Position



B Prone (Jackknife) Position



C Lloyd-Davies (Modified Lithotomy) Position

FIGURE 1 Patient positions for the anorectal examination.

If the patient's back is placed parallel to the side of the table, the patient has a tendency to slip and the exposure to the perineum is limited. The modified lithotomy position (Figure 1c) is rarely used in the office setting because the exposure of the perineum is limited. This position is helpful, however, if a pelvic examination must also be performed and in the operating room if abdominal exposure is required.

Physical Inspection

The buttocks should be inspected first for scars from prior abscess drainage, skin lesions, or sinus openings. The sacrococcygeal region is searched for signs of pilonidal disease : pits, cysts, or scars. Next the buttocks are gently retracted and the perianal skin inspected. Is there evidence of dermatitis or excoriation and linear scratches from pruritis (see Figure 1, Chapter 18)? Is there fecal or mucous soiling, indicating incontinence or prolapse? Swellings and protrusions are noted and characterized: condyloma (see Figure 1, Chapter 20), hypertrophied anal papillae (see Figure 5, Chapter 18), sentinel piles, external hemorrhoids (see Figure 4, Chapter 16), prolapsed internal hemorrhoids, or skin tags (Figure 2). Fissures can be seen by gentle distraction on the anus while the patient strains. They are usually found on the posterior midline; fissures found off the midline

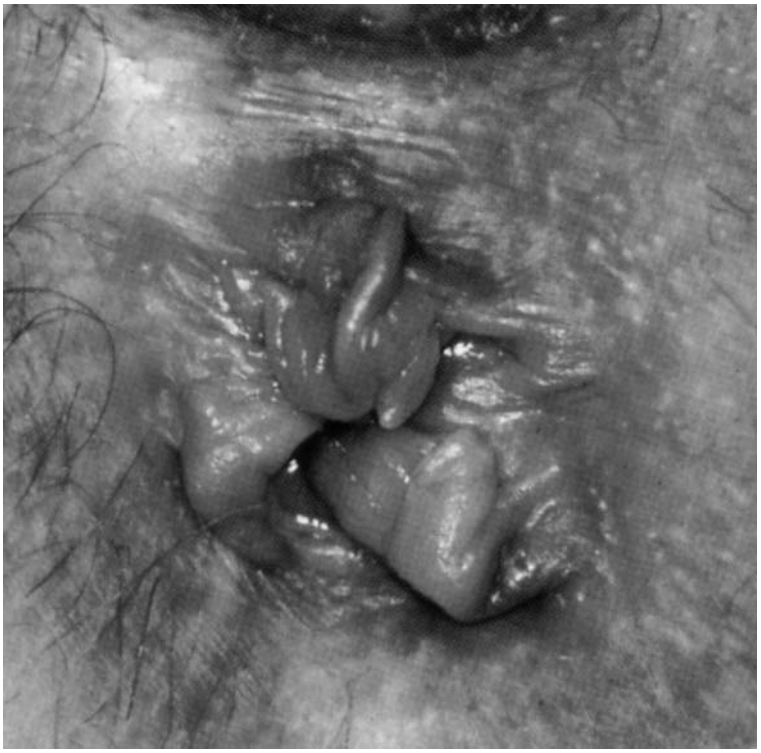


Figure 2 Anal skin tags.

and accompanied by abscesses raise the suspicion of Crohn's disease. External openings of fistulas should be noted, as should scars and any muscular asymmetry from prior anorectal surgery. These findings can be elicited by having the patient squeeze and strain. Sensation is assessed by light stroking or pinprick.

To avoid confusion for subsequent examiners, the position of significant findings is recorded by using left, right, anterior, and posterior, not by the face of a clock. A mass felt at "2 o'clock" in the prone jackknife position becomes "8 o'clock" when the patient is in the lithotomy position. A sketch in the patient's chart is also helpful.

Prolapsing conditions suggested by the patient's history may not be evident on initial inspection, especially when the patient is in the prone jackknife position. A patient with a prolapsing condition is asked to sit on the toilet and strain and is reexamined before he or she rises. Other options include the use of an extendable mirror or a flexible endoscope that can be passed into the toilet to visualize the prolapse (see Chap. 15).

Digital Palpation

Digital palpation of the anus and rectum is performed carefully with a gloved hand; finger cots are no longer recommended. The well-lubricated index finger is placed on the anal opening and gradually advanced. Having the patient bear down, which relaxes the external sphincters somewhat, may make this easier. The tone and symmetry of the sphincter complex is noted as the anal canal and dentate line are examined for masses, stenosis, scarring, or areas of tenderness. A fissure can often be palpated in the posterior midline as a rough region in the otherwise smooth anal canal. The anorectal junction is identified by the puborectalis sling posteriorly. Once the rectum is entered, palpation begins anteriorly. In men the prostate is examined; in women the cervix may be felt, or the defect of a rectocele may be appreciated. The examination continues circumferentially within the rectum to assess for any pathological condition both within and outside the rectum. The position, consistency, and fixity of masses should be noted. Laterally, extrarectal adenopathy or pelvic abscesses can sometimes be felt. Posteriorly, sacral masses may be detected. The cul-de-sac is searched for a tumor shelf. Before the examiner withdraws the finger, the patient is asked to repeat a squeeze and strain to assess the function and symmetry of the sphincter once more.

Anoscopy

Anoscopy completes the examination. It allows assessment of the anal canal and distal rectum and requires no special preparation. Severa1 styles of anoscope are available (Figure 3). The anoscope is lubricated generously and advanced slowly with the patient bearing down, which facilitates insertion by relaxing the anal canal. The anus is examined circumferentially. Some anoscopes allow this without

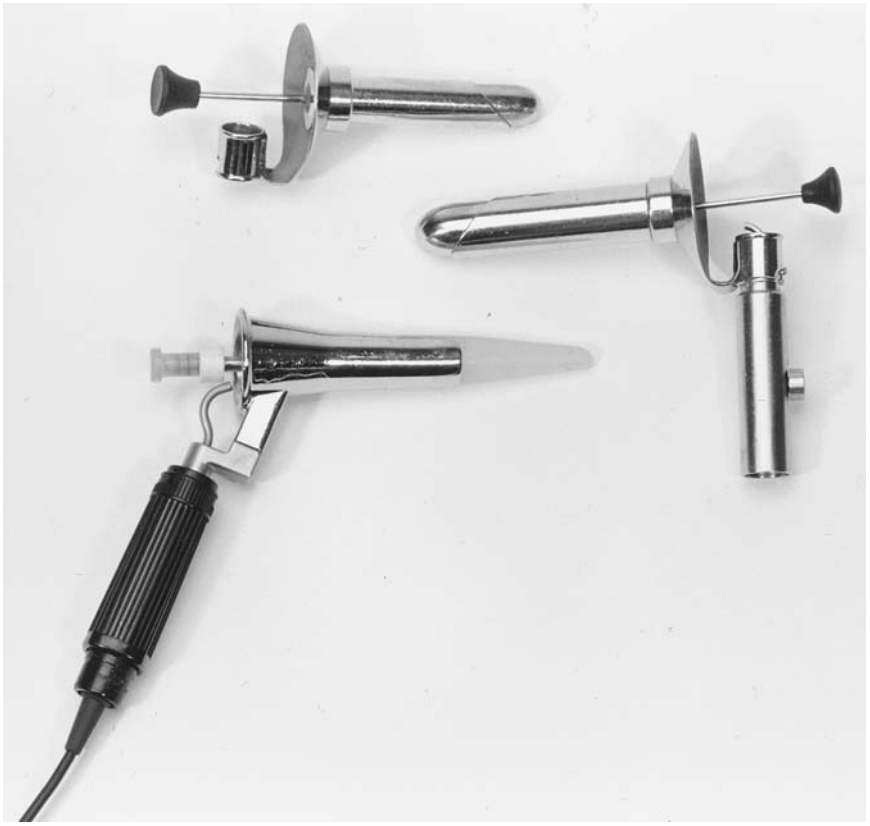


Figure 3 Anoscopes: (top) modified Buie-Hirchmann, small and medium; (bottom) Welch-Allen, slotted medium with light source attached.

having to be withdrawn; others need to be reinserted with an obturator to avoid pinching the sensitive anoderm. The anoderm is inspected for fissures, which usually lie on the posterior or anterior midline. Odd-appearing lateral fissures, especially those associated with edematous skin tags or abscess, are hallmarks of Crohn's disease. Hemorrhoids are typically found in three bundles: right posterior, right anterior, and left lateral. They should be graded with respect to the dentate line. If a fistula or abscess is encountered, the offending anal gland may sometimes be apparent by drainage or with gentle probing. Aggressive probing of fistulas is contraindicated, because false passages can be created. Other lesions that may be seen include condylomata involving the anal canal, epidermoid carcinoma, or melanoma [11].

To complete the anorectal examination, additional endoscopy is usually required. These important diagnostic and therapeutic procedures are covered in Chapter 5.

ROUNDS QUESTIONS

Explain why the following statements are true or false.

1. Abdominal pain is usually quite specific, and the location of the pain is seldom referred to other areas.
   False; abdominal pain is usually ill defined and is often referred to areas on the surface removed from the site of pathology (pp. 32).
2. Tenesmus, the feeling of the urge to defecate, may accompany rectal cancer.
   True; this symptom may also be seen with inflammatory conditions or following pelvic irradiation (p. 32).
3. Melena always comes from an upper or proximal source, whereas bright red blood is always from benign anal conditions.
   False; melena may be from the right colon, and although bright red bleeding is usually anal, a more proximal source may need to be excluded (p. 32).
4. A 44-year-old patient whose mother had a cancer diagnosed on a recent colonscopy and whose uncle was diagnosed with colon cancer at age 54 needs full colonoscopic screening.
   True; a family history in a first-degree relative increases the patient's risk of colorectal cancer (p. 33).
5. Findings of peritoneal irritation must be confirmed by eliciting rebound tenderness.
   False; if peritonitis is detected by gentle percussion, further vigorous palpation only hurts the patient and adds nothing to the clinical picture (p. 34).
6. Typical anal fissures cause painful bleeding that follows bowel movements and are found in the posterior midline by gently spreading the buttocks.
   True; atypical lateral fissures may indicate inflammatory bowel disease, malignancy, or infectious diseases (p. 37).
7. The only contraindications to properly performing a digital anorectal examination are (1) no finger, (2) no anus, and (3) no glove.
   True; when correctly done, the anorectal examination is no more stressful than any other part of the examination and may yield vital information about the patient‘s condition (p. 38).