

Fifth Edition

First Principles of Gastroenterology

The Basis of Disease and an Approach to Management

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The Colon

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1. INTRODUCTION

This chapter presents an overview of colonic physiology and the diseases that affect the colon. It discusses lower gastrointestinal bleeding, infectious diseases affecting the colon and diseases specifically involving the anus. The main inflammatory bowel diseases have been discussed in Chapter 9. Diarrhea and its causes are examined in Chapter 6, “The Small Intestine.” Other infections are presented in Chapter 6 and in Chapter 8, “Gastrointestinal Manifestations of Human Immunodeficiency Virus Infection.”

2. PHYSIOLOGY OF THE COLON / S.J. VANNER

2.1 Function

The colon contributes to three important functions in the body: (1) concentration of fecal effluent through water and electrolyte absorption, (2) storage and controlled evacuation of fecal material and (3) digestion and absorption of undigested food. Although the colon is not essential for survival, its functions contribute significantly to the overall well-being of humans. The colon can be functionally divided through the transverse colon into two parts, the right and left colon. The right colon (cecum and ascending colon) plays a major role in water and electrolyte absorption and fermentation of undigested sugars, and the left colon (descending colon, sigmoid colon and rectum) is predominantly involved in storage and evacuation of stool.

2.2 Functional Anatomy

The human colon is a muscular organ measuring approximately 125 cm in length

in vivo. Its wall consists of the four basic layers found in other GI hollow visceral organs – the mucosa, submucosa, circular muscle and longitudinal muscle – but several important differences exist. The mucosa lacks the villous projections found in the small intestine and presents a relatively smooth surface, but numerous crypts extend from its surface. Cell types lining the surface and the crypts resemble those in the small intestine but are composed of significantly greater numbers of goblet cells. These cells secrete mucus into the lumen, and mucus strands can often be identified in association with stool. This observation is misconstrued by some patients as a response to underlying colonic pathology. The haustral folds, which help define the colon on barium x-ray, are not a static anatomical feature of the colon but rather result from circular muscle contractions that remain constant for several hours at a time. The outer or longitudinal muscle is organized in three bands, called taeniae coli, which run from the cecum to the rectum where they fuse together to form a uniform outer muscular layer. These muscular bands and elongated serosal fat saccules, called appendices epiploicae, aid in the identification of the colon in the peritoneal cavity.

The colon is innervated by the complex interaction of intrinsic (enteric nervous system) and extrinsic (autonomic nervous system) nerves (Figure 1). The cell bodies of neurons in the enteric nervous system are organized into ganglia with interconnecting fiber tracts, which form the submucosal and myenteric plexi. These nerves are organized into local neural reflex circuits, which modulate motility (myenteric), secretion, blood flow and probably immune function (submucosal). Serotonin (5-HT), released from enterochromaffin into the surrounding lamina propria, is an important signaling pathway to these nerves in response to chemical and mechanical stimuli. Acetylcholine, substance P and serotonin (5-HT) serve to activate local circuits such as those innervating muscle contractions. Release of excitatory neurotransmitters such as acetylcholine, substance P and serotonin (5-HT) serves to activate local circuits such as those innervating muscle contractions. Their receptor subtypes provide pharmacological targets for the development of drugs designed to alter colonic functions such as motility. The major inhibitory neurotransmitter is nitric oxide. The importance of the enteric nervous system is exemplified by Hirschsprung's disease, where there is a congenital absence of nitric oxide – containing inhibitory neurons over variable lengths of the rectum and colon. This results in an inability of the colon to relax in the affected region. Infants typically present with bowel obstruction or severe constipation. Barium x-rays identify the affected region as a constricted segment because the excitatory effects of the neurotransmitter acetylcholine are unopposed as a result of the absence of inhibitory neurotransmitter.

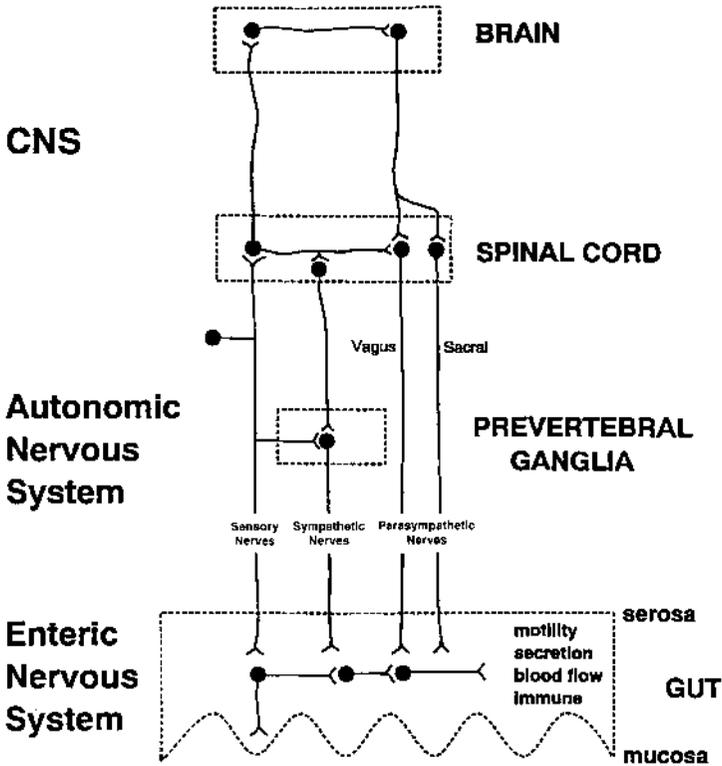


FIGURE 1. Innervation of the colon.

The autonomic nervous system comprises sensory nerves, whose cell bodies are found in the dorsal root ganglia, and motor nerves, the sympathetic and parasympathetic nerves. Parasympathetic nerves innervating the right colon travel in the vagus nerve, and those innervating the left colon originate from the pelvic sacral nerves. Parasympathetic nerves are predominantly excitatory, and sympathetic nerves inhibitory. Autonomic nerves modulate the enteric neural circuits within the colon and participate in neural reflexes at the level of the autonomic ganglia, spinal cord and brain. Brain–gut connections are important both for perception of visceral stimuli (sensory) and in modifying colonic function (motor) in response to central stimuli. An example of a central stimulus that can evoke significant changes in colonic activity through this

connection is acute stress. This stimulus provokes release of central hormones, such as corticotropin releasing factor. These hormones activate parasympathetic pathways that stimulate motility patterns in the colon and can result in diarrhea.

2.3 Absorption and Secretion

The colon is highly efficient at absorbing water. Under normal physiological conditions, approximately 1.5 L of fluid enters the colon each day, but only about 100–200 mL is excreted in the stool. The maximal absorptive capacity of the colon is up to about 4.5 L per day, so that diarrhea (increased water in stools) will not occur unless the ileocecal flow rate exceeds the absorptive capacity and/or the colonic mucosa itself is secreting. The fundamental feature of colonic electrolyte transport that enables this efficient water absorption is the ability of the colonic mucosa to generate a large osmotic gradient between the lumen and the intercellular space. This osmotic gradient is created by electrogenic sodium transport. This depends upon the energy-dependent Na^+/K^+ -ATPase pump on the basolateral membrane, which pumps sodium from inside the cell against a large concentration gradient into the intercellular space (see Figure 6 in Chapter 6, “The Small Intestine”). Luminal sodium in turn enters the apical membrane of the cell through sodium channels, flowing down the concentration gradient created by the pump. In contrast to the small intestine, where sodium in the intercellular space can diffuse back into the lumen and become iso-osmotic, hypertonic solutions are maintained in the intercellular space because the tight junctions are much less permeable to sodium diffusion. The net result is that the hypertonic fluid within the intercellular space draws water passively into the mucosa from the lumen. It also results in highly efficient absorption of sodium. Of the 150 mEq of sodium that enters the colon each day, less than 5 mEq is lost in the stool. The tight junctions are highly permeable to potassium, in contrast to sodium, allowing potassium to move from plasma to the lumen. Potassium pumped into the cell by the Na^+/K^+ -ATPase pump can also be secreted into the lumen. Potassium is normally secreted into the lumen unless intraluminal potassium rises above 15 mEq/L. This handling of potassium may account for hypokalemia seen with colonic diarrhea and may play a role in maintaining potassium balance in the late stages of renal failure. Other transport mechanisms, similar to those found in the small intestine (see Chapter 6, Section 5), are also found on colonic enterocytes, which maintain electrical neutrality, intracellular pH and secretion. Nutrient cotransporters, however, are not found in the colon.

The regulation of water and electrolyte transport in the colon also involves the complex interplay between humoral, paracrine and neural regulatory

pathways (see Chapter 6). One important difference is the effect of aldosterone, which is absent in the small intestine. This hormone is secreted in response to total body sodium depletion or potassium loading and stimulates sodium absorption and potassium secretion in the colon.

2.4 Motility of the Colon

Much less is known about the motility of the colon compared to other regions of the GI tract. The movement of fecal material from cecum to rectum is a slow process, occurring over days. Functionally, the contraction patterns in the right colon (cecum and ascending colon) cause significant mixing, which facilitates the absorption of water, whereas in the left colon (sigmoid and rectum) they slow the movement of formed stool, forming a reservoir until reflexes activate contractions to advance and evacuate stool.

Several fundamental contractile patterns exist within the colon. Ring contractions are due to circular muscle contraction and can be tonic or rhythmic. Tonic contractions are sustained over hours and form the haustral markings evident on barium x-rays; they appear to play a role in mixing. Rhythmic ring contractions can be intermittent or regular. Regular contractions are non-occlusive, occur over a few seconds, and migrate cephalad (right colon) and caudad (left colon). Presumably, they too play a role in mixing. Intermittent ring contractions occur every few hours, occlude the lumen, and migrate caudad. They result in the mass movement of stool, particularly in the sigmoid colon and rectum. Contractions of the longitudinal muscle appear to produce bulging of the colonic wall between the taeniae coli, but the importance of this action remains poorly understood. The origin of the contractions is not completely understood but depends upon the slow wave frequency of smooth muscle. Action potentials occur on the peaks of these membrane oscillations and hence they control the frequency of contractions. It is now recognized that these slow waves originate in the interstitial cells of Cajal (ICCs), which serve as the pacemakers. This network of cells is interposed between the enteric nerves and the smooth muscle cells. Contractions are also modulated by paracrine, humoral and other neural pathways.

The nature of the contractile patterns within the colon depends upon the fed state. This is best exemplified during eating when the “gastrocolic reflex” is activated. Food in the duodenum, particularly fatty foods, evokes reflex intermittent rhythmic contractions within the colon and corresponding mass movement of stool. This action, which is mediated by neural and humoral mechanisms, accounts for the observation by many individuals that eating stimulates the urge to defecate.

Figures 2 and 3 show normal images of the colon at colonoscopy.



FIGURE 2 (A and B). Normal colon appearance at endoscopy.

FIGURE 2A. Close-up view of the normal submucosal vessels visible through the healthy transparent mucosa overlying the vessels.

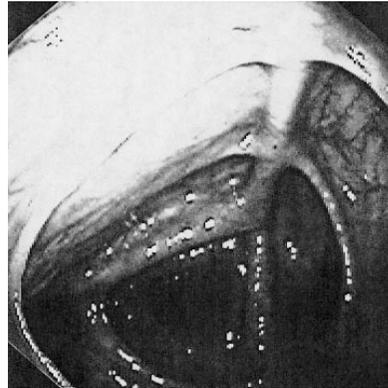


FIGURE 2B. Normal transverse colon with the normal triangular appearance to the fold pattern.

2.5 Digestion and Absorption of Undigested Food Products

Greater numbers of bacteria (more anaerobes than aerobes) are found within the colonic lumen than elsewhere in the GI tract. These bacteria digest a number of undigested food products normally found in the effluent delivered to the colon, such as complex sugars contained in dietary fiber.

Complex sugars are fermented by the bacteria, forming the short-chain fatty acids (SCFAs) butyrate, propionate and acetate. These SCFAs are essential nutrient sources for colonic epithelium, and in addition can provide up to 500 cal/day of overall nutritional needs. They are passively and actively transported into the cell where they become an important energy source for the cell through the β -oxidation pathway. The importance of this role is illustrated by the effects of a “defunctioning” colostomy, which diverts the fecal stream from the distal colon. Examination of this area typically reveals signs of inflammation, termed *diversion colitis*. This inflammation can be successfully treated with the installation of mixtures of short-chain fatty acids into the rectum.

Fermentation of sugars by colonic bacteria is also an important source of colonic gases such as hydrogen, methane and carbon dioxide. These gases, particularly methane, largely account for the tendency of some stools to float in the toilet. Nitrogen gas, which diffuses into the colon from the plasma, is

FIGURE 3 (A, B and C). The ileo-cecal valve. The three parts of this figure illustrate examples of the variation in appearance of the ileocecal valve.



FIGURE 3A. Normal ileocecal valve.

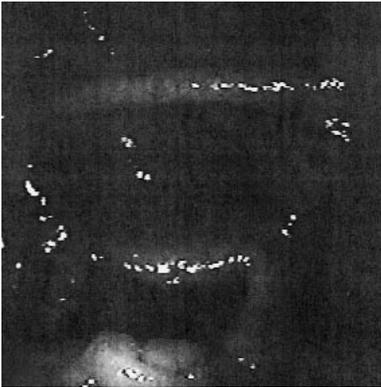


FIGURE 3B. Normal ileocecal valve with melanosis coli pigmentation of the colon (see Figure 4A).



FIGURE 3C. A normal variant of the ileocecal valve where fatty infiltration of the valve makes it appear more prominent; on x-ray it can be confused with a polyp. The appearance of the mucosa is normal. Biopsy confirms that the mucosa is normal – i.e., not a polyp.

the predominant gas. However, the ingestion of large quantities of undigested complex sugars such as found in beans or the maldigestion of simple sugars such as lactose can result in large increases in production of colonic gas. This can lead to patients' complaints of abdominal bloating and increased flatus.

When bile salts or long-chain fatty acids are malabsorbed in sufficient quantities, their digestion by colonic bacteria generates potent secretagogues. Bile salt malabsorption causing “choleraic diarrhea” typically occurs following terminal ileum resection, usually for management of Crohn’s disease. When the resection involves segments greater than 100 cm this problem is further complicated by depletion of the bile salt pool, because bile salt production cannot compensate for the increased fecal loss. In these circumstances diarrhea also results from fat malabsorption. The proposed mechanisms by which multiple metabolites of bile salts and hydroxylated metabolites of long-chain fatty acids act as secretagogues provide an example of how multiple regulatory systems can interact to control colonic function. These mechanisms include disruption of mucosal permeability, stimulation of Cl^- and water secretion by activating enteric secretomotor neurons, enhancement of the paracrine actions of prostaglandins by increasing production, and direct effects on the enterocyte that increase intracellular calcium.

Non-pathogenic bacteria also signal to mucosal cells and can evoke cytokine signaling from epithelial cells to effector cells within the colon wall (e.g., immune cells, nerves). Some species stimulate pro-inflammatory responses whereas others are anti-inflammatory. These signaling pathways are enhanced when the tight junctions between epithelial cells are altered. These junctions are formed by proteins (e.g., occludens) and can be disrupted by a growing list of processes, e.g., inflammation such as Crohn’s disease and also non-inflammatory states such as acute stress. This allows bacteria greater access to the epithelium and immune cells in the lamina propria. This bacterial-epithelial signaling underlies the rationale for the use of probiotics where “healthy” or anti-inflammatory bacteria are ingested (e.g., lactobacillus, bifobacteria) and alter the dynamic between the competing species of bacteria.

3. SPECIFIC COLONIC DISEASES / G.K. TURNBULL AND J. BURKE

3.1 Colon Polyps and Cancer

Colon cancer is the second most common cancer (after lung cancer) in men and women combined in Canada. Unlike lung cancer, it has a high survival rate in patients diagnosed before it has spread beyond the confines of the bowel wall. Since it is a very common cancer, has a high survival rate with early curative surgery and is poorly responsive to other forms of cancer therapy, a high index of suspicion must be maintained in approaching patients with symptoms of colonic dysfunction (Table 1), especially if they are over the age of 40, when the incidence of colon cancer begins to rise. Increased colon cancer risk is also seen in patients with ulcerative colitis or Crohn’s colitis, a history of uterine or ovarian cancer or a family history of colon cancer or colonic adenomas (including familial polyposis syndromes).

TABLE 1. Presenting features of colon cancer

Abdominal pain, including symptoms of bowel obstruction
Change in bowel habit
Abdominal complaints of recent onset
Rectal bleeding or melena stool
Abdominal mass
Iron deficiency anemia
Hypokalemia

TABLE 2. Colorectal adenocarcinoma staging

<i>TNM stage</i>	<i>Dukes' stage</i>	<i>Tumor invasion</i>	<i>5 year survival</i>
T0N0M0	N/A	Mucosa only	100%
T1N0M0	A	Submucosa No lymph nodes or distant metastases	90–95%
T2N0M0	B1	Muscularis involved No lymph nodes or distant metastases	85%
T3–4N0M0	B2	Tumor to serosa (T3) or through serosa (T4) No lymph nodes or distant metastases	70–75%
T2N1M0	C1	Muscularis involved but not to serosa Lymph nodes involved	35–65%
T3–4N1M0	C2	Tumor to serosa (see T3/4 above) Lymph nodes involved	35–65%
TxNxM1	D	Distant metastases (liver) regardless of extent of primary tumor	<5%

The tumor, node, metastasis (TNM) staging system has largely replaced the older Dukes' classification for staging colon cancer after surgical resection. Table 2 details the TNM staging system for colorectal cancer and compares it to the older Dukes' A to D classification. Other factors that increase the mortality from colorectal cancer besides the stage of the tumor, are poorly differentiated histology and vascular invasion by the tumor histologically at the time of resection. Other poor prognostic indicators for colon cancer are cancers that have perforated, adherence to adjacent organs and colon cancer presenting with complete bowel obstruction.

Early recognition is of the utmost importance to try to identify colon cancer at an early, curative stage. Therefore, patients with intermittent symptoms are

as important to investigate as patients with persistent symptoms, and the story of occasional blood in the stool in a patient over 40 years of age should not be attributed to local anorectal disease without excluding a more proximal lesion. Many patients may present with no gastrointestinal symptoms, but rather an iron deficiency anemia due to chronic bleeding from the tumor. Patients may not see blood in the stool or note a melena stool, particularly when there is a right-sided colonic lesion. A change in bowel habit, often with constipation alternating with diarrhea, may be the first sign of obstructive symptoms from a colon cancer, and should never be ignored in a patient over 40 years of age with a recent onset of these symptoms. Some patients may present with primarily diarrhea if they have a high output of mucus and fluid from the tumor; in this instance the tumor is often sessile in appearance (see below) and large, with the histology of a villous adenoma. Some patients may have hypokalemia due to the large amounts of mucus secretion from the tumor with loss of potassium.

Carcinoembryonic antigen (CEA) is a tumor marker that has limited use in diagnosing colon cancer but is often useful in following patients with colon cancer. A high CEA level before surgery often suggests a poor prognosis with probable metastases. A CEA level that does not fall to normal levels one month after surgery suggests that all the cancer has not been resected. After surgery, regular monitoring of CEA levels can identify patients with early recurrence. Sometimes a search for metastases will discover a solitary lesion in the liver that may be surgically resectable or with the early use of chemotherapy, may lead to a cure of the cancer.

3.1.1 POLYP-CARCINOMA SEQUENCE

It is now agreed that for the majority of colon cancer patients, the adenocarcinoma arises from an adenomatous polyp. Polyps of 2 cm or greater have about 50% incidence of cancer, compared to 1% in adenomas less than 1 cm. Adenomatous polyps are a premalignant condition, and their identification and removal before becoming malignant prevents the development of colon cancer. These polyps can arise anywhere in the colon, but (as is the case for colon cancer) they are more frequently seen in the left colon. The majority of polyps are completely asymptomatic, but the occurrence of occult bleeding does increase as they grow. Unfortunately, polyps can still be missed, even with occult blood testing of the stool, since the blood loss may be intermittent. Examples of different polyps are seen in Figure 4.

Three histologic types of adenomatous polyps occur: tubular, tubulovillous and villous. The malignant potential is greatest in villous polyps (40%) and lowest in tubular polyps (5%), with an intermediate risk in tubulovillous polyps (22%). The malignant potential may also be described pathologically

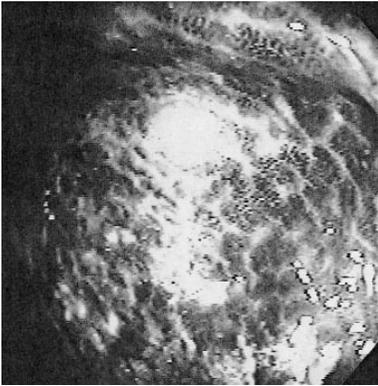


FIGURE 4 (A, B, C and D). Adenomatous polyps of the colon. B, C and D illustrate polyps of different size and morphology. Note in D that although the polyp is rather large, its flat sessile morphology makes it more difficult to see endoscopically.

FIGURE 4A. A very small adenoma made more obvious in a colon with melanosis coli, a pigment in the mucosa resulting from chronic laxative abuse. See how the adenoma is not pigmented, as its tissue is abnormal and the cells in the polyp divide more rapidly than cells in the “normal” pigmented mucosa.

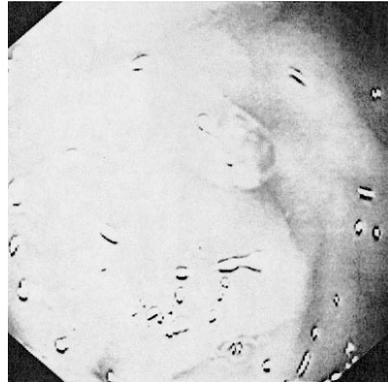


FIGURE 4B.



FIGURE 4C.

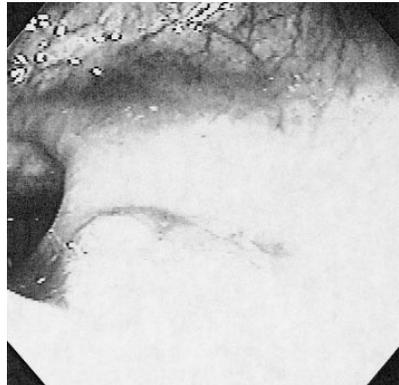


FIGURE 4D.

as the degree of “dysplasia”: the more severe the dysplasia, the greater the rate of malignancy. These tubular, tubulovillous and villous polyps can often be completely removed by snare polypectomy at colonoscopy if they are pedunculated on a stalk, but sessile polyps that carpet a wide area of colonic mucosa (often villous polyps) can usually be completely removed only by resection surgery. Since polyps precede cancer and removal of polyps “cures” the cancer, it has been hoped that screening colonoscopy may help reduce the incidence of cancer. Other polyps as well may be present at the initial or index colonoscopy, and polyps and cancer tend to recur. This sets the stage for the rationale for performing follow-up surveillance colonoscopies (colon cancer surveillance program). The best time interval for this surveillance is probably every five years; longer intervals between surveillance colonoscopies may be safe but have yet to be tested. The cost-effectiveness of screening all patients over the age of 50 has not been proven but screening has been shown to reduce the incidence of cancer especially in high risk groups for developing colon cancer. Screening for colon cancer has become very controversial as to the correct intervals, who to screen and the best tools to use for screening. The Canadian Association of Gastroenterology has recently published guidelines for colon cancer screening and they also refer to the guidelines published by the American Gastroenterological Association and the British Society of Gastroenterology.

3.1.2 COLON CANCER SCREENING

Particular conditions have been associated with an increased risk of colon cancer. It is clear that all patients with the following conditions require some form of regular colon surveillance to detect polyps/cancer at its earliest stage to improve survival. The “polyposis” syndromes of Familial Adenomatous Polyposis (FAP), Gardner’s syndrome and Turcot’s syndrome are now recognized to all be various expressions of disease caused by mutations of the APC gene (autosomal dominant inheritance). These conditions are manifested by early onset (usually before age 30) of innumerable colonic adenomatous polyps that eventually and invariably lead to colon cancer (usually before age 40). Since the colon has too many polyps to remove by endoscopy-guided polypectomy these patients are referred at an early age for total proctocolectomy to remove the risk of colorectal cancer. Most patients opt for an ileal pouch with anastomosis to the anal sphincters, rather than an ileostomy. After colectomy these patients still need regular upper GI endoscopic surveillance and patients also need the ileal pouch endoscopically examined to ensure there are no changes from the transitional mucosa left behind at the anastomosis of the ileal pouch to the anus. Biopsies are taken from the ampulla of Vater to look for adenomas that frequently occur in the proximal duodenum around the ampulla, and the stomach

is also examined endoscopically for evidence of adenomas of the stomach. An experimental approach at present is to do tests on blood monocytes looking for mutation of the APC gene, which is the cause of this autosomal dominant disease.

There are other families (site-specific colorectal cancer, family cancer syndrome) that have a high risk of colon cancer (autosomal dominant inheritance), with more than two first-degree relatives in at least two generations, having had colon cancer or adenomatous polyps and at least one of the relatives has to be under age 50. This disease is called hereditary nonpolyposis colorectal cancer (HNPCC). All patients should be entered into a colon cancer surveillance program of colonoscopy and/or air contrast barium enema starting at age 21. Patients with HNPCC are to have the colon screened every two years until age 40, when they should have yearly colon screening. Screening should be done with colonoscopy if possible as polyps are frequently encountered and need to be removed when found. Female patients with HNPCC also have an increased risk of endometrial and ovarian cancer and need yearly pelvic ultrasound and/or vaginal ultrasound after age 40 to identify suspicious lesions as early as possible. The other group of patients at increased risk of cancer who should all be screened are those patients who have had a colon cancer resected. Colonoscopy should be done either preoperatively or within a year of surgery. It should be repeated three years post surgery and then every five years if there are no polyps or evidence of recurrent tumor. If there is any concern about complete resection of the original tumor, earlier surveillance would be recommended (less than one year after surgery).

Also at a high risk for colon cancer are patients with chronic ulcerative colitis for more than 8 to 10 years; this risk also appears to be present in patients with Crohn's pancolitis. The patients at highest risk are those who have had total colon involvement and those with disease up to and including the hepatic flexure (subtotal colitis); patients with proctosigmoiditis are at least risk – probably not greater than the general population. Curiously, the risk of cancer does not correlate with the degree of disease activity. Therefore, patients with just one bout of proven subtotal ulcerative colitis would have an increased risk of cancer after 8 to 10 years of disease, and the younger the patient at the time of onset of his or her disease, the greater the cumulative risk of cancer will be for that patient. Unlike those who experience the “polyp–carcinoma sequence,” patients with colitis do not develop adenomatous polyps before they develop cancer; therefore they require colonoscopy about every one to two years, with endoscopic biopsies of the colon performed to identify dysplasia of the mucosa. Particular attention should be paid to “elevated” or “flat” lesions seen at colonoscopy where the incidence of early colon cancer is high. If there is dysplasia, either “high grade” or “low grade,” colectomy should be recommended to the patient.



FIGURE 5A. Single contrast barium enema demonstrating a large cecal mass which proved to be an adenocarcinoma.



FIGURE 5B. Double contrast barium enema showing multiple diverticula as well as a subcentimeter polyp (white arrow).



FIGURE 5C. Single contrast barium enema demonstrating multiple colonic diverticula (white arrow).

3.1.3 DIAGNOSTIC IMAGING OF THE COLON /J. BURKE

Imaging of the colon has traditionally been achieved by performing a barium enema. Early in the 20th century the single contrast technique was developed. After a suitable bowel cleaning preparation, a tube is placed in the rectum and the colon is distended with a large volume of low density barium. Multiple spot images are then obtained of the various segments in an attempt to visualize the entirety of the colon free from any overlapping loops. Later in the

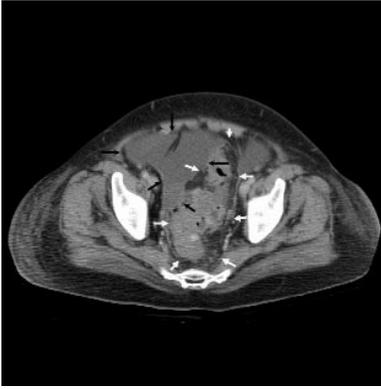


FIGURE 6A. CT scan demonstrating a case of diverticulitis with a thick walled loop of sigmoid colon (white arrows) with adjacent inflammatory stranding. There is also a considerable volume of free intraperitoneal fluid (black arrows).

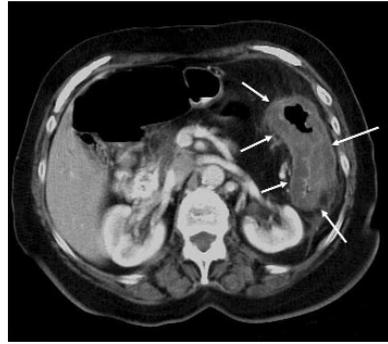


FIGURE 6B. CT scan demonstrating diffuse concentric wall thickening of the splenic flexure (white arrows) in a patient with ischemic colitis.

20th century, the double contrast technique was developed. It involves the introduction of a small volume of high density barium through a rectal tube. This is followed by the insufflation of a large volume of room air. This achieves good colonic distension and mucosal coating, which are prerequisites for this examination. Double contrast barium enemas are generally accepted as preferable for the assessment of mucosal abnormalities as well as the detection of small polypoid lesions. Single contrast examinations are adequate for the detection of larger lesions, obstructing lesions as well as the depiction of diverticular disease. Figure 5A is a single contrast study demonstrating a large cecal mass which proved to be an adenocarcinoma. Figure 5B is a double contrast barium enema showing multiple diverticula as well as a subcentimeter polyp (white arrow) which proved to be a tubular adenoma. Figure 5C is a single contrast barium enema demonstrating multiple colonic diverticula (white arrow).

In the 1980s, computed tomographic (CT) scanning became widely available and accepted in the evaluation of the abdomen and pelvis. While CT is relatively insensitive for the detection of intraluminal colonic abnormalities, it is well suited for the assessment of intramural and extracolonic pathology. Figure 6A demonstrates a thick walled loop of sigmoid colon (white arrows) with adjacent inflammatory stranding. There is also a considerable volume of free intraperitoneal fluid (black arrows). Figure 6B demonstrates diffuse



FIGURE 7A. Axial source image.

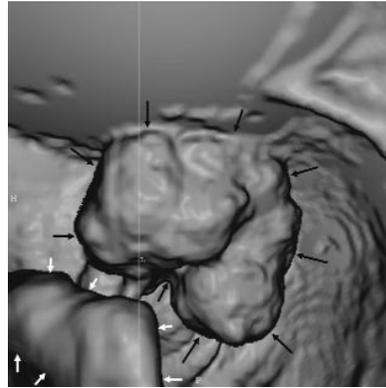


FIGURE 7B. Endoluminal reconstruction.

Figure 7A (axial source image) and figure 7B (endoluminal reconstruction) in a patient with an incomplete colonoscopy demonstrating a large polypoid mass (black arrows) subsequently proven to be an adenocarcinoma. The white arrows denote the ileocecal valve.

concentric wall thickening of the splenic flexure (white arrows) in this patient with ischemic colitis.

In the past 10 years, the main development in the radiographic evaluation of the colon has been CT colonography. CT colonography is a means of imaging both the intra and extraluminal portions of the colon. The technique involves the placement of a small catheter in the rectum. Room air or carbon dioxide is insufflated to achieve adequate distension. A CT scan of the abdomen and pelvis is then performed in both the supine and prone positions. The acquired axial images are then sent to a workstation for post processing. A hollow 3D model of the colon is built and an endoluminal fly through is performed. Work to date has shown that this technique is quite promising for the detection of colonic polyps. Figure 7A (axial source image) and figure 7B (endoluminal reconstruction) in a patient with an incomplete optical endoscopy demonstrates a large polypoid mass (black arrows) subsequently proven to be an adenocarcinoma. The white arrows denote the ileocecal valve.

3.2 Diverticulosis

In Western societies diverticulosis occurs in at least one person in two over the age of 50 years. The frequency increases with age. Diverticulosis or diverticular disease of the colon is due to pseudodiverticula in that the wall of the diverticulum is not full-thickness colonic wall, but rather outpouchings of

TABLE 3. Complications of diverticulitis

Abdominal abscess/Liver abscess
Colonic obstruction
Fistulas
Colovesical
Colovaginal
Colocutaneous

colonic mucosa through points of weakness in the colonic wall where the blood vessels penetrate the muscularis propria. These diverticula are prone to infection or “diverticulitis” presumably because they trap feces with bacteria. If the infection spreads beyond the confines of the diverticula in the colonic wall, an abscess is formed. Patients present with increasing left lower quadrant pain and fever, often with constipation and lower abdominal obstructive symptoms such as bloating and distention. Some patients with severe obstructive symptoms may actually describe nausea or vomiting. This can occur with or without abscess formation. Other causes of these symptoms include Crohn’s colitis with stricture formation, colonic cancer and ischemic colitis (see Section 3.3).

On physical examination the patient often has localized tenderness in the left lower quadrant, and with severe infection and an abscess may have rebound tenderness in the left lower quadrant. A palpable mass is often identifiable where the sigmoid colon (the most common site of diverticulitis) is infected. Treatment consists of intravenous fluids and bowel rest by placing the patient on no oral intake or just a clear liquid diet; broad-spectrum intravenous antibiotics are administered. Antibiotics selection should be to cover both gram-negative enteric bacteria and anaerobic bacteria that are normally found in the colon. CT scan may be helpful in outlining the colon and identifying an abscess, and is preferable to barium enema for diagnosis in patients with acute illness.

Many complications can occur in diverticulitis. These are listed in Table 3. Colonic stricture after resolution of diverticulitis is described further in Section 3.3.

Bleeding occurs in less than 5% of diverticulosis patients; it is abrupt in onset, painless, and often massive. A bleeding diverticulum can be from either the left or right colon. Even though bleeding is more likely to occur in right colonic diverticulosis, the bleeding frequency is approximately equal because of the much higher frequency of left colonic diverticulosis. It is rare for patients with diverticulosis to have significant bleeding. Over 80% of diverticulosis patients will stop bleeding, but the rest will continue and

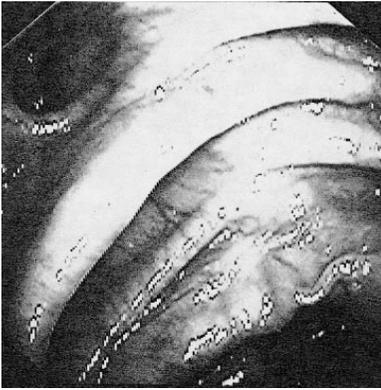


FIGURE 8 (A and B). Diverticulosis.

FIGURE 8A. The rounded opening of a single diverticulum is shown at the top left, with the colonic lumen at the bottom right.



FIGURE 8B. Two diverticulum openings are shown at the top, with the lumen in the lower half of the image. This shows how easy it is to confuse the lumen with a diverticulum opening. With insertion of the colonoscope into a diverticulum, perforation is the result.

require investigation and treatment (see Section 5). Segmental colonic resection is reserved for that small group of patients who continue bleeding or have recurrent bleeding. Patients under the age of 40 with symptomatic diverticulitis should have surgical resection because this small subgroup is at greater risk of complications.

Figure 8 shows examples of diverticulosis of the sigmoid colon at colonoscopy.

3.3 Colonic Obstruction

Acute colonic obstruction is a surgical emergency that must be recognized early and dealt with expeditiously in order to avoid the high fatality rate due to colonic perforation. The highest risk patients for colonic perforation are those with an intact ileocecal valve that does not allow air to reflux back into the small bowel from the obstructed colon. The cecum is the most frequent site of perforation, because wall tension is highest in the bowel with the largest diameter (Laplace's law).

Patients with colonic obstruction usually have pain as a prominent symptom, with constipation often preceding the complete obstruction. Patients may initially present with diarrhea as the bowel distal to the obstruction empties, but diarrhea may be persistent, especially with a partial obstruction, because

of the increased intestinal secretion proximal to the obstruction. The small intestine is the most common site of intestinal obstruction because of the narrower caliber of the bowel, and similarly the left colon is the most common site for colonic obstruction, especially since the stool is more formed in the left colon and unable to pass through a narrowed lumen.

On physical examination the general state of the patient depends upon the duration of the obstruction. With a recent sudden obstruction the patient will be in extreme pain, will often have distention of the abdomen if the ileocecal valve is intact and may describe initially diarrheal stool as the bowel distal to the obstruction is emptied. Abdominal palpation can often discern a mass lesion at the site of the obstruction. Prompt identification of the site of obstruction is mandatory, with the use of supine and erect abdominal x-rays. An urgent surgical consultation is required if the rectum is empty of air with dilation of more proximal colon, indicating a complete colonic obstruction.

Many patients present with a more gradual history. If they have had protracted diarrhea up to the point of obstruction, the amount of abdominal pain may be less; they may have abdominal distention, but be less tender on abdominal exam; and they will often show signs of dehydration. Fever and an abdominal mass is particularly common in patients with diverticulitis and a resulting colonic obstruction. A third type of colonic obstruction can be seen that is actually a form of ileus limited to the colon and is sometimes referred to as Ogilvie's syndrome. These patients are most often seen in intensive care units, but the condition can also occur postoperatively (even when no bowel surgery has been performed). As with a "mechanical" bowel obstruction described above, patients with Ogilvie's syndrome may have marked abdominal distention, but frequently they have little abdominal pain and the abdominal x-rays show a picture of dilated colon with impaired movement of air into the distal colon.

Once a diagnosis of colonic obstruction has been made, the site of obstruction should be determined by plain abdominal x-rays and/or with a water-soluble contrast enema (such as iohalamate meglumine) to identify whether urgent surgery is indicated.

If investigations do not confirm obstruction of the colon, colonic ileus can often be treated safely by neostigmine 2.0 to 2.5 mg IV. Bradycardia may occur with this medication, and all patients must receive cardiac monitoring. The majority of patients respond well to neostigmine and this avoids the need for urgent colonoscopy and the increased risk of perforation of the colon due to poor visualization in the unprepared colon. However, if the endoscopist is able to decompress the lumen by suctioning the excess air, a decompression tube can sometimes be placed high in the colon to facilitate removing colonic air following the procedure.

TABLE 4. Causes of colonic obstruction

<i>Common</i>
Left-sided cancer
Diverticulitis
Ogilvie's syndrome
<i>Others</i>
Hernia
Strictures
Crohn's
Postischemic
Postsurgical
Intussusception
Volvulus
Adhesions

There are many causes of colonic obstruction (Table 4). Colon cancer and diverticulitis are the most common causes. Most colon cancers that obstruct are in the left colon. They cause circumferential disease or “apple-core” lesions (so called because of the irregular mucosal appearance with luminal narrowing seen at x-ray). Diverticulitis commonly occurs in the sigmoid colon, where diverticular disease is most common; the acute abscess formation with swelling of the inflamed diverticulum compresses and obstructs the affected sigmoid colon. Ogilvie's syndrome may initially have been considered to be due to a cancer or diverticulitis, but contrast x-ray demonstrates a patent lumen.

Less common causes of colonic obstruction are hernias, in which a loop of colon (usually sigmoid) becomes strangulated and the bowel is acutely obstructed. This is a much more common cause of small bowel obstruction. Strictures in the colon can also be associated with obstruction, especially when they occur in the left colon. These can occur with Crohn's colitis, after a bout of ischemic colitis or at the site of anastomosis following colonic surgery. This latter cause of obstruction should always be visualized endoscopically if possible, since most colonic resections are for cancer and the possibility of a local cancer recurrence can complicate a postsurgical stricture.

Intussusception can occur in the colon, and in adults it almost always occurs at the site of a polyp, which “leads” the intussusception. Typically, this will cause intermittent acute bowel obstruction associated with severe pain and often rectal bleeding from the vascular compromise produced in the intussuscepting bowel. Because of the intermittent nature of the obstruction, a diagnosis may not be made until after repeated attacks. A barium enema

should always be considered in this setting, as it identifies the mucosal lesion “leading” the intussusception and can occasionally be used to reduce the intussusception without the need for urgent surgery.

Volvulus of the colon tends to happen in the cecum and/or the sigmoid colon, because the mesentery can be long and redundant in these areas and cause the bowel to rotate upon itself. This can be a surgical emergency, since the affected bowel will strangulate if the volvulus is not relieved quickly. Again, an urgent barium enema may be able to reduce the volvulus, thus allowing a more elective surgical procedure to correct the problem. A sigmoid volvulus will usually be reduced by this approach, and success with colonoscopic decompression of a sigmoid volvulus has been reported. A cecal volvulus may not be easily treatable with either a barium enema or colonoscopic therapy; thus, surgical advice should be sought urgently if cecal volvulus is diagnosed.

Adhesions are often described as a common cause of bowel obstruction, but this is probably true only for small bowel obstruction. Since much of the colon is retroperitoneal or on a limited mesentery, adhesive disease with obstruction of the colon is rare. However, it can occur, particularly in the sigmoid colon if the mesentery is quite long, particularly after pelvic operations.

3.4 Irritable Bowel Syndrome

Most commonly, patients exhibiting symptoms from the GI tract are suffering from the irritable bowel syndrome. This is a condition that may be a variant of normal function. Causes of irritable bowel are still being evaluated, but the syndrome does sometimes occur after an episode of infectious diarrhea. It appears that patients have no organic disease of the gastrointestinal tract, yet they experience frequent symptoms from the bowel. Large epidemiologic studies would suggest that the condition occurs in at least 15% of the population.

The commonest symptom that brings a patient to a doctor is abdominal pain. Criteria have been developed to identify with more certainty those patients who have the irritable bowel syndrome. A positive diagnosis can be made, particularly in women, if the abdominal pain is present for at least three months in the last year and the pain is relieved by defecation. The abdominal pain is also associated with a change in stool consistency and stool frequency. These criteria are termed the Rome II criteria, that have been shown to be reliable in making a positive diagnosis of irritable bowel syndrome. The following criteria are also used to help confirm a positive diagnosis. The more of these symptoms that are present, the more likely the diagnosis is irritable bowel. These symptoms are: abdominal bloating or distension, mucus in the stool, and difficult defecation.

Patients who have difficulty with defecation can have the following complaints. There can be “urgency,” with the sudden urge to pass stool and a fear

of incontinence if defecation is not performed immediately. Many patients with this symptom will relate that they always identify where the toilet is when they are away from home. The fear of incontinence can often greatly limit a patient's ability to function normally in society. Other patients with difficult defecation may have to strain – defined as having to hold their breath and push when attempting defecation. Straining is defined as “constipation” when a patient must strain 25% or more of the time when trying to defecate. Finally, some patients describe a feeling of incomplete emptying after passing stool. This symptom has to be asked for specifically, as most patients will not spontaneously report it. Nevertheless, the symptom is commonly reported by patients with an irritable bowel.

The presence of mucus in the stool can be alarming to some patients, since they may interpret this to mean they have “colitis.” In the past, some doctors used to refer to irritable bowel as “mucus colitis,” which is a misnomer since there is no “colitis” or inflammation of the colon in irritable bowel. Mucus is a normal product of the colon, and only if mucus and blood are seen together should other diagnoses such as “colitis” be considered.

The typical stool pattern described by patients with an irritable bowel is the change in stool character and frequency with the onset of abdominal pain. Typically, patients will pass a normally formed stool (sometimes even a constipated stool) first thing in the morning. Then with the attacks of abdominal pain the stools become more frequent and looser, sometimes becoming just liquid diarrheal stools. Once bowel movements cease the pain is relieved, but it can recur again later in the day, often precipitated by eating high-fat foods or other gut stimulants (e.g., coffee).

In men, the above criteria (called the Manning Criteria) may not be as helpful as they are in women. It is also important to note that the vast majority of people with an irritable bowel have their symptoms begin in young adult life. One should consider other colonic diseases in patients over the age of 40 who develop these symptoms for the first time without previous episodes suggesting irritable bowel. Sometimes later in life patients can develop irritable bowel after severe infectious diarrhea, but in this population as well, further investigations are warranted to ensure no other cause for the change in bowel function.

The irritable bowel syndrome is a disorder affecting the entire gut, and although many of the symptoms appear to arise from the colon, these patients frequently have symptoms from other parts of the GI tract as well as from other organs. Upper GI symptoms are very common in irritable bowel; these consist of increased heartburn and dyspepsia. Dyspepsia symptoms in general occur more commonly than lower bowel symptoms, but may be due to many other causes, including reflux esophagitis, gastritis, peptic ulcer disease and, less commonly, biliary tract and pancreatic disease. When upper GI symptoms

are associated with irritable bowel, other underlying diseases must be considered. Other associated symptoms include frequent headaches and urinary symptoms that are similar to bowel symptoms, in that patients can have urgency and frequency of urination. These symptoms are often worse at times when the bowel symptoms are troublesome. In women, irritable bowel symptoms can often be exacerbated or worsened around the time of menstruation. Studies suggest that bowel symptoms associated with menstruation occur in at least 50% of the normal female population.

When assessing a patient complaining of irritable bowel symptoms, remember that only a small proportion of patients with an irritable bowel present to doctors with these symptoms. Recent studies would suggest that patients who see doctors about their symptoms often have psychological problems, with increased levels of distress and depression as common findings. It is important to inquire about these problems, as successful treatment often consists of dealing with the distress and/or depression that exacerbates the irritable bowel symptoms. They may often be the reason that the patient has sought medical attention in the first place.

3.4.1 DIFFERENTIAL DIAGNOSIS

The Manning Criteria provide a positive diagnosis of irritable bowel: abdominal pain with the association of increased frequency and increased looseness of stool, relief of abdominal pain with defecation, abdominal bloating, mucus in the stool and defecation difficulties such as a sensation of incomplete rectal emptying after defecation. However, lactose intolerance is a common cause of change in bowel habit in young adults, particularly if their racial background is not northern European. Therefore, investigating for lactose intolerance in patients who present with increased frequency and looseness of stool is worthwhile, since the ingestion of lactose-containing foods may be the reason for their symptoms.

All patients should have a thorough physical examination, looking for evidence of disease in other organ systems such as the thyroid, which can present with a change in bowel habit. Patients with an irritable bowel will often have pain over the colon, particularly the sigmoid colon, on palpation. The identification of an enlarged liver or spleen or other abdominal masses necessitates further investigations. A barium enema is rarely required in a young healthy adult with new onset of irritable bowel symptoms. However, a patient over the age of 40 presenting with symptoms that may be irritable bowel yet of new onset and without previous complaints would warrant at least a barium enema and a sigmoidoscopic examination. The barium enema should also evaluate the terminal ileum if there is pain on palpation in the right lower quadrant. A complete blood count with platelet count should be done, as an elevated

platelet count is often a sensitive finding for underlying inflammation and in the presence of bowel symptoms could mean the presence of early inflammatory bowel disease. Crohn's disease is more likely to present this way than irritable bowel. The persistence of the abdominal pain, even though lessened after bowel movements, would suggest possible underlying inflammation of the gut rather than an irritable bowel. Ulcerative colitis usually presents with rectal bleeding. Rectal bleeding is not a symptom of irritable bowel and its cause must always be investigated. Fever, weight loss and symptoms that wake a patient from sleep, as opposed to early waking in the morning, are all symptoms that should be further investigated.

The presence of nocturnal symptoms, particularly with diarrhea waking the patient at night, is rarely due to an irritable bowel. Occasionally patients with depression who have early morning waking report this symptom, but in general further investigations are indicated. Celiac disease (gluten enteropathy) can also present with irritable bowel symptoms. If iron deficiency anemia is found without an explanation, (i.e., bleeding source), then a tTG serology test should be done to exclude celiac disease (see Chapter 6: The Small Intestine).

3.4.2 THERAPY

The therapeutic approach in irritable bowel is as much reassurance as any specific therapies, as most patients do not have any "disease." It is most important to do a thorough history and physical examination to ensure that the complaints are not due to any underlying disease. Once this has been confirmed, explain to the patient how the bowel can produce these symptoms and that there is no cause for concern. Since patients presenting with irritable bowel symptoms frequently have more distress and tend to be more prone to seek medical attention for other minor medical conditions than other patients (so-called illness behavior), these patients may require considerable reassurance to convince them that they do not have serious disease. Part of this reassurance will be provided by screening blood tests such as a complete blood count with platelet count. Sigmoidoscopic examination will rule out most underlying early inflammatory bowel disease and any rectal pathology, particularly in patients complaining of defecation difficulties or a sensation of being unable to empty the rectum adequately. The stool should be analyzed for pathogens if diarrhea is present. Following these initial screening tests emphasis should be placed on the stresses present in the patient's life. Evaluating the level of stress and taking steps to correct it will often be helpful. Many patients, particularly those who have symptoms of constipation, may be helped with a high-fiber diet (see Section 3.6).

Drug therapy for irritable bowel is usually empiric, directed at the most troublesome symptom. There is no single drug that treats all the varied symptoms in irritable bowel, but occasional patients will continue to have

intractable symptomatology. In this situation selected medications for specific symptoms may be helpful. Table 5 outlines some drugs that may be useful for specific symptoms. Drug therapy for irritable bowel should always be restricted to short periods during exacerbation of symptoms, and patients should be taken off medications when well. As irritable bowel is a chronic condition and is probably “normal” for these patients, the chronic use of medications often reinforces the notion that they have a “disease.” Reassuring the patient that there is no association between irritable bowel symptoms and the development of more serious bowel disease such as colon cancer or inflammatory bowel disease can often alleviate some of the unreasonable yet very real concerns of many patients who present to doctors with these symptoms.

3.5 Fecal Incontinence

Understanding fecal incontinence requires knowledge of the normal function of the anorectum. Anatomically, it consists of the internal anal sphincter surrounded by the external anal sphincter and puborectalis muscles. The internal anal sphincter consists of smooth muscle and is a continuation of the circular smooth muscle of the rectum. The external anal sphincter is made up of skeletal muscle and surrounds the internal anal sphincter, whereas the puborectalis (also consisting of skeletal muscle) is a large U-shaped muscle that wraps around the upper anal canal at the anorectal junction above the external anal sphincter and loops anteriorly to attach to the pubic bone. This creates an anatomical sling of muscle that pulls the anorectal junction forward when it tightens, thus closing the upper anal canal and creating the anorectal angle that is vital to the maintenance of fecal continence.

When stool (or gas or liquid) enters the rectum or sigmoid colon, a normal rectoanal inhibitory reflex (RAIR) or rectosphincteric reflex is initiated – that is, the internal anal sphincter relaxes and, if voluntary muscle action occurs, the rectum empties through the anal canal (Figure 9). Fecal continence is maintained by contraction under voluntary control of the striated-muscle sphincters – i.e., the external anal sphincter (EAS) and puborectalis (PR) – until the rectal pressure rise decreases and the resting tone of the internal anal sphincter is restored. Thus, the “voluntary” sphincters (i.e., the EAS and PR) have the ability to be maximally contracted for approximately one minute, beyond which fecal continence is lost as a result of fatigue in the muscle if the tone of the internal anal sphincter has not recovered.

Some patients with fecal incontinence will often describe the problem as “diarrhea” rather than loss of control of bowel function. All patients with a complaint of diarrhea should be asked if they have lost control of stool, as this may indicate where the problem actually lies. Once fecal incontinence has

TABLE 5. Drug therapy in irritable bowel syndrome

<i>Symptom</i>	<i>Drug</i>	<i>Dosage</i>
Abdominal pain	Anticholinergics	
	Hyoscyamine	0.125 mg sl q4h prn (only available in US)
	Dicyclomine	10–20 mg po tid – qid before meals
	Calcium antagonists	
	Pinaverium bromide	50–100 mg po tid before meals
	Antidepressants	
	e.g., Nortriptyline	10–25 mg po hs (increase by 10–25 mg increments every 5 to 7 days as tolerated)
	Enteric opioids	
	Trimebutine	100–200 mg po tid before meals
	Fedotozine	NOT available in Canada
Constipation	High fiber diet	≥ 30 g daily plus 2 L liquid daily
	Osmotic laxatives	
	Milk of magnesia	15–30 mL po bid – tid
	Prokinetic agent	
	Tegaserod	6 mg po bid
Other agents		
	Misoprostol	200 µg po bid – qid before meals
Diarrhea	Binding agent (resin)	
	Cholestyramine	4 g po once to four times daily
	Antimotility agents	
	Loperamide	2–4 mg po prn (maximum dose 16 mg/day)
	Diphenoxylate	2.5 mg po qid prn
Alosetron	1 mg po bid (only available in US)	
Abdominal bloating, “gas”	Simethicone	up to qid prn
	Motility agents	
	Domperidone	10–20 mg po qid
	Tegaserod	6 mg po bid
	Probiotics	
	VSL#3	1 cap po bid

been noted, it is then necessary to identify the frequency of the incontinence, whether both liquid and solid stool have been leaked and whether the individual has an urge to defecate before the leakage occurs. A history of previous anorectal trauma (surgical, obstetrical or otherwise) is important to note, as is the strength of voluntary anal canal tone on digital rectal exam.

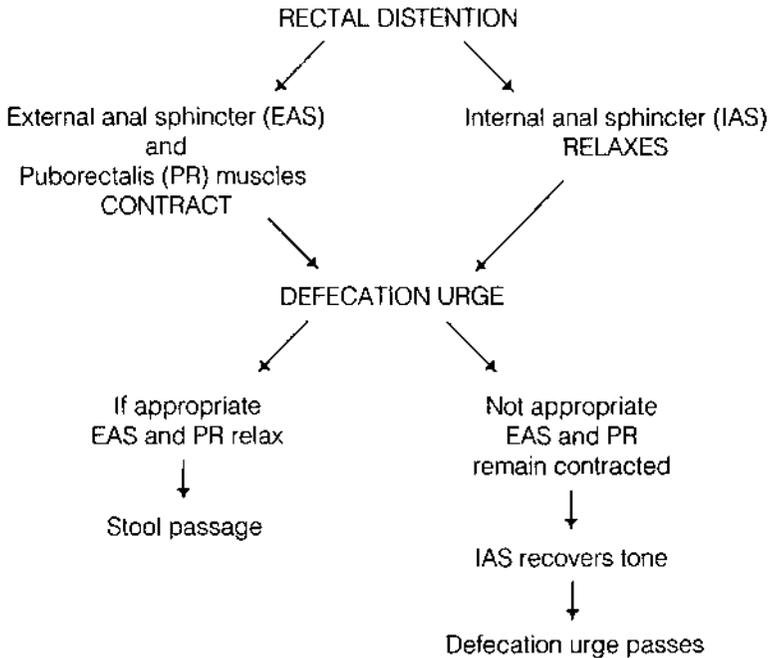


FIGURE 9. Physiology of defecation.

Most patients presenting with fecal incontinence have “idiopathic” fecal incontinence, but recent investigations in females with this complaint indicate they have suffered damage to the pudendal nerves during childbirth and, with time, this has led to gradual striated-muscle anal sphincter weakness. Many women suffer occult sphincter injury to the anal sphincters, both the internal anal sphincter and the external anal sphincters, with childbirth. The injury is often not recognized at the time of childbirth so the sphincter weakness and fecal incontinence only becomes symptomatic years later presumably with atrophy of the muscles with aging. Similar injury occurs with the urinary sphincters and many women with “idiopathic” from childbirth injuries present years later with both urinary and fecal incontinence. Sphincter injury at childbirth is more likely to occur with the first baby, if the baby is more than 4,600 g (10 lbs), if the second stage of labour is prolonged or if there are forceps or vacuum extraction used to help the delivery. Although an episiotomy is done to prevent tearing into the sphincters, recent reports have found this may even increase the risk of a sphincter tear.

Surgical trauma is the next most common cause of fecal incontinence; it should be remembered that surgery (e.g., a vaginal hysterectomy) can put excess stretch on the pelvic floor nerves and muscles, causing injury that may lead to weakness of the anal sphincters. Another common source of fecal incontinence is disruption of the internal anal sphincter, either during a lateral internal sphincterotomy to treat an anal fissure or, more commonly, with the older “Lord’s” procedure of forceful three- or four-finger dilation of the anal sphincter under anesthetic, where the extent of damage to the sphincters is not predictable. The finding of perineal descent can be noted on examination of the perineum when the patient is asked to strain and appears to be associated with weakness of the pelvic floor muscles as well as disruption of the normal anatomy. This gives rise to a mechanical disadvantage affecting the sphincter mechanism. Perineal descent may be associated with a rectocele or, in female patients, with a uterine prolapse. Rectal prolapse can also accompany weakness of the pelvic floor muscles and give rise to fecal incontinence.

Therapy of fecal incontinence has improved over the past decade, primarily because of the introduction of biofeedback training. This technique allows the patient to practice tightening of the striated-muscle portion of the anal sphincter, usually with a surface electromyography (EMG) plug electrode held in the anal canal with audio and visual feedback that the patient can see and hear to encourage maximal contraction of these muscles. Attention should also be given to increasing dietary fiber to help reduce the amount of liquid stool. Other drug therapy is limited, but loperamide has been shown to increase the resting tone of the anal sphincters and is a useful adjunct, especially if the stool frequency is increased (loperamide reduces this contributing factor). Cholestyramine may be useful when the patient has diarrhea or loose stool(s) since cholestyramine can make stool more solid (constipating effect). Surgery is sometimes required and is of greatest benefit in those patients who appear to have a mechanical problem such as rectal prolapse or disruption of the sphincter. Surgery to correct perineal descent is often less helpful, since the problem of muscle weakness that gives rise to the descent is not satisfactorily reversed by any of the surgical procedures currently used, and attempts to “suspend” the pelvic floor muscles cannot strengthen these muscles. Patients should refrain from excess straining if they have significant perineal descent, because this will serve only to worsen the pelvic floor muscle weakness.

3.6 Constipation

In the approach to a patient with constipation, it is first necessary to define what the patient means by the term. Many definitions exist, but the best clinical definition is that over 95% of the North American population has a stool frequency from three times a day to three times a week: therefore, patients

who have a bowel frequency less than three times a week would be defined as being constipated. Many patients will describe their stool as “constipated,” usually meaning that the stool is hard or in pellets (scybalous), while other patients may have a stool frequency that falls within the “normal” range yet feel that their bowels have not completely emptied. This latter symptom is a frequent complaint of the irritable bowel syndrome, and many patients with this disorder will describe a constipated bowel habit. Those constipated patients who have infrequent stool alternating with occasional diarrheal stool have the most common presentation of irritable bowel syndrome. Yet there are a great many patients, almost all female, who have infrequent stool passage, and this group must be considered as separate from the usual irritable bowel syndrome patient for they may be among those rare patients with a secondary cause of constipation.

In Western culture the most frequent cause of constipation is a lack of dietary fiber. The concept of “fiber” has become quite confusing to many patients with the increased emphasis on “oat fiber” for elevated cholesterol treatment. Many foods that patients consider to be high in fiber (e.g., salads, lettuce, tomatoes and celery) are in fact mainly water, and some vegetables may aggravate their symptoms. Fiber consists of complex carbohydrates that are incompletely digested by the small bowel and are then “digested” by colonic bacteria, liberating short-chain fatty acids and gases from fermentation, that may provoke and aggravate many of the associated abdominal symptoms (e.g., abdominal pain, “gas” and bloating). Cereal grain fibers that have more insoluble fiber (as opposed to soluble oat bran fiber) are best to increase stool frequency, but should be added gradually over 8 to 12 weeks to a daily dose of about 30 g. Other fibers in the form of “bulk laxative” preparations containing psyllium, methylcellulose, sterculia or isphagula may be added to wheat bran fiber to accomplish this level of fiber without completely altering a patient’s diet. Many patients who are constipated continue to pass dry, hard stool despite an increase in dietary fiber because they do not increase the water content of their diet. Fiber works in the gut by holding onto water and keeping the stool soft; to achieve this effect, the intake of liquids must be increased. For a 30 g/day fiber diet, it is recommended that patients drink eight 8 oz. glasses (i.e., 2 L) of non-caffeine-containing beverages per day.

Secondary causes of constipation must be excluded. The patient with bowel obstruction can present with constipation, and this possibility should always be considered in a patient with the onset of constipation after the age of 40 years (when the incidence of colon cancer rises). A rarer cause of constipation is hypothyroidism; not infrequently, patients with an underactive thyroid will present with a primary symptom of constipation. Hypercalcemia rarely reaches levels that produce constipation but should always be considered, since it can

be a life-threatening disorder; constipation in this setting is always resistant to therapy until the hypercalcemia is treated. Proctitis can present with a complaint of infrequent stool passage due to the functional obstruction caused by the inflammation of the rectum; the colon more proximally continues to produce formed stool, which cannot pass easily through the inflamed rectum. Proctitis will usually be associated with excess mucus production, with or without blood in the stool, and proctosigmoidoscopy will always diagnose this entity.

Another cause of constipation is diabetes mellitus, often as a result of impaired motility; dietary factors may also play a role, along with autonomic neuropathy of the enteric nervous system. A small proportion of these diabetic patients with constipation can go on to develop diarrhea, which again has been linked to the autonomic neuropathy seen with long-standing diabetes mellitus.

Inactivity from whatever cause seems to increase the likelihood of a patient's complaining of a constipated bowel habit. This is presumed to be secondary to reduced colonic activity but could be aggravated by a low fiber intake in many of these patients. Severe cardiopulmonary diseases of whatever cause that limit activity can result in constipation. Neurologic disorders that cause the patient to have a reduced ability to ambulate can have constipation as a feature. Some patients with diseases of the nervous system may have impaired awareness of rectal distention to signal a need to defecate, and nerve dysfunction (both peripheral and central) may impair normal colonic propulsion. Finally, the elderly may develop problems with defecation, and although constipation with fecal impaction occurs they may complain of "diarrhea" or "soiling" due to overflow incontinence of stool from the fecal impaction of the rectum inhibiting the normal resting tone of the anal sphincter. Not surprisingly, many of these patients may respond to laxative therapy after the fecal impaction is removed, since this prevents the recurrence of the fecal impaction with overflow incontinence. Some patients can aggravate long-standing constipation with regular laxative abuse, and some theoretical concerns remain that this practice may indeed damage the normal innervation of the colon, rendering it atonic and nonfunctional.

The physical findings are often minimal in the majority of patients with constipation, but specific secondary causes must be looked for. Signs of hypothyroidism may be present; signs of dehydration should be sought, as this may be an early indicator of hypercalcemia. Thorough cardiopulmonary and neurologic examinations are necessary to pick out associated diseases that may be treated, thereby improving the patient's overall health and thus improving bowel function. On abdominal examination, inspection for evidence of distention or hyperperistalsis or masses may point out the source of the impaired stool passage. Localized tenderness of the abdomen must be

noted, along with any evidence of liver, spleen or renal enlargement. A complete rectal examination and proctosigmoidoscopy is required in any patient with constipation so that the presence or absence of a fecal impaction, dilation or enlargement of the rectum or the presence of proctitis can be determined.

3.6.1 MEGARECTUM

When the rectum is enlarged, further investigations are required to exclude other causes, particularly Hirschsprung's disease (Section 3.6.2). The majority of patients with constipation and a dilated rectum and/or colon at proctosigmoidoscopy or barium enema have idiopathic or acquired megarectum. A useful guideline for the diagnosis of a "megarectum" is a rectal diameter of greater than 6 cm on a lateral film at the level of the S2 vertebral body. These patients can often present in childhood (many of them presenting with encopresis) and in the elderly with a fecal impaction. The cause of the megarectum is unknown, but if the onset is in childhood it may be the result of chronic stool holding by the child, leading to progressive distention of the rectum and eventual loss of awareness of rectal distention. Once this has occurred the patient can no longer recognize when stool is present in the rectum; the distention of the rectum causes chronic inhibition of the resting tone of the internal anal sphincter. This leads to the loss of control of liquid or semisolid stool that passes by the fecal impaction without the patient being aware of it.

3.6.2 HIRSCHSPRUNG'S DISEASE

The majority of patients with this disorder present soon after birth or in early childhood. This is a lesion, present at birth, where variable lengths of distal colon have no myenteric plexus. The distal colon remains contracted and the normal proximal colon dilates as it fills with stool. Most of these patients present early in life with obstipation and colonic obstruction, and require surgery. However, a few patients have a very short segment of denervated distal colon, so that they can overcome the obstruction and force stool out of the rectum. They usually have lifelong constipation; the normal rectum proximal to the denervated segment dilates over time so that the patient presents with constipation and a "megarectum." These patients rarely have fecal incontinence or fecal "soiling" as seen with idiopathic megarectum, since the internal anal sphincter and denervated distal rectum maintain a high resting tone. This condition can be diagnosed by anorectal manometry in that a normal rectoanal inhibitory reflex cannot be identified (Figure 9). However, a definite diagnosis requires deep rectal biopsy from the denervated segment, which will show absence of the myenteric plexus ganglion cells and hypertrophy of nerve fiber bundles. It should be added that an identical condition can be acquired with Chagas' disease from South America, which attacks the myenteric plexus and

other autonomic ganglion cells; patients with this condition can also present with achalasia or intestinal pseudo-obstruction as well as cardiac arrhythmias. These patients will also have an absent rectoanal inhibitory reflex if the disease involves the rectal myenteric plexus.

3.6.3 PELVIC FLOOR DYSSYNERGIA

The majority of patients with constipation have a form of irritable bowel syndrome, but there is a small subgroup of patients who may have a specific disorder in colonic and/or anorectal function that produces constipation. These patients are almost all female, may have delayed colonic transit or present with anorectal dysfunction with impaired awareness to rectal distention (without a megarectum), or may demonstrate a phenomenon of rectal outlet obstruction due to inappropriate contraction of the voluntary anal sphincters during defecation. This has been termed pelvic floor dyssynergia or anismus. These patients can present major therapeutic dilemmas and warrant further investigation in specialized coloproctology units involved in the care of such patients.

3.7 Infections of the Colon

3.7.1 SHIGELLA

This infectious diarrhea is the classic cause of “bacillary dysentery.” Typical presentation is with fever, abdominal cramping and watery diarrhea that usually becomes bloody within 24 to 48 hours of onset. The incubation period is from 36 to 48 hours. As the disease progresses the symptoms become typical of colonic dysentery, with small, frequent stools and cramping and tenesmus that may lead to rectal prolapse in some individuals with prolonged straining.

The causative organism is a gram-negative bacterium with only humans as its host. The organism is well adapted to causing disease in humans. As few as 200 organisms are needed to cause infection as compared with other enteric infections requiring 10^6 organisms or more. It can persist in food for weeks and on contaminated body surfaces for several hours. Pathogenesis is through production of a cytotoxin called Shiga toxin or similar toxins that are both cytotoxic and neurotoxic, very similar to the toxin produced by *E. coli* 0157:H7 species. *Shigella* is a microinvasive bacterium that enters the host via the M cells in the intestine and then spreads laterally through the colonic mucosa to involve the basolateral membrane of the surrounding cells. It is seen mostly in travelers returning from endemic areas (tropical and subtropical). There is also a higher incidence in male homosexuals who practice oral–anal sex.

Treatment depends on the antibiotic resistance of the infecting strain of bacteria. *Shigella* species quickly develop antibiotic resistance; patients

should be encouraged to complete their course of antibiotics to prevent this. Antibiotics shorten the duration of both symptoms and carriage of the organism. Fluoroquinolones are the antibiotics of choice because of the low incidence of resistance at present, but this may change. Ampicillin and trimethoprim-sulfamethoxazole are also effective against sensitive strains. If infection has been acquired overseas, a confirmed *Shigella* infection is best treated with a fluoroquinolone twice daily for 5 days, but there are reports of a single large dose eradicating infection. Antimotility agents such as loperamide, diphenoxylate or narcotic analgesics are contraindicated with this infection because of the risk of a toxic colon. In general, antimotility agents should never be used in acute infectious diarrhea when bloody stool is present.

3.7.2 *SALMONELLA*

Infection with nontyphoidal strains of *Salmonella* results from ingesting foods contaminated with organisms. These bacteria are endemic to poultry and cattle populations. Large epidemics have resulted from undercooked eggs, and these bacteria are also frequently found in reptiles and amphibians. *Salmonella* contamination of marijuana can be an important infection source in young adults. The usual incubation period is from 8 to 48 hours after ingestion.

S. typhi, which causes typhoid fever, is found only in humans. It will not be discussed further other than to emphasize that all *Salmonella* species are related and therefore can cause systemic illness of similar severity, especially in patients who are immunocompromised and those at the extremes of age (i.e., those under 2 and the frail elderly).

Salmonella is an invasive bacterium that can cause septicemia after first multiplying in the mesenteric lymph nodes. Our resistance to infections is first a result of the presence of gastric acid and then of the integrity of the intestinal flora and of motility. Increased infection is associated with the use of purgatives, antimotility agents and broad-spectrum antibiotics along with acid-suppression therapy. Bowel surgery will also increase the chance of symptomatic infection. Diseases that predispose to infection as a result of impaired host defenses include sickle cell disease, systemic lupus erythematosus (SLE) and AIDS.

Treatment is usually symptomatic. Antibiotics should be used only if the patient is showing signs of bacteremia; antibiotics often increase the development of a chronic carrier state. The antibiotics of choice are ampicillin and trimethoprim-sulfamethoxazole, and the fluoroquinolones and the third-generation cephalosporins (especially ceftriaxone with its high biliary excretion) have been shown to be very effective in patients who need antibiotic therapy. Treatment with antibiotics should normally be considered only for patients under the age of 2 years and elderly patients with vascular disease, as

well as patients with metal implants in bones, lymphoproliferative disease, sickle cell disease or AIDS. The site of chronic infection is usually the biliary tract. Disease of the biliary tree, especially cholelithiasis, requires surgery to correct the disease followed by a two-week course of therapy, which often leads to resolution of the chronic carrier state.

3.7.3 *CLOSTRIDIUM DIFFICILE*

This spore-forming anaerobic gram-positive bacterium is the commonest cause of infectious diarrhea in hospitalized patients. The organism is not invasive, but with reduction of the normal colonic bacterial flora it multiplies and produces two toxins, known as toxins A and B. Toxin A causes colitis. Toxin B is a cytotoxin that is often used as a diagnostic test for this infection.

Most commonly, infection is preceded by antibiotic therapy. Outbreaks in hospital frequently occur among the sickest patients, some not receiving antibiotics beforehand. Penicillins, cephalosporins and clindamycin are more likely to be associated with *C. difficile* infection, but all antibiotics, including metronidazole and vancomycin, have been associated with it. Other risk factors include agents that affect gut motility such as enemas and anti-diarrheal medications, and intensive chemotherapy. Patients with severe illnesses and advanced age are also more prone to manifest disease symptoms.

Diarrhea is the commonest symptom of presentation and is usually non-bloody, but with prolonged diarrhea some blood can result from local anorectal irritation. The typical appearance at endoscopy of the colon and rectum is of "pseudomembranes" or whitish plaques on the surface of the colonic mucosa with intervening areas of mucosa that appear almost normal. For this reason, infection is often called "pseudomembranous colitis" (PMC). Unfortunately, these characteristic changes may not be present in the rectum, so diagnosis is usually confirmed by the presence of cytotoxin in the stool placed on tissue culture. The clinician must be alert to the possibility of this infection in susceptible patients since in some patients, neither culture of *C. difficile* nor the presence of the cytotoxin in the stool is positive. A careful inventory of any antibiotic therapy in the last three months is crucial in considering this cause for diarrhea, as many patients will have taken the offending antibiotic several days to weeks before symptoms begin.

Metronidazole (Flagyl®) treatment is preferred to vancomycin because both antibiotics show similar efficacy in treating this infection and metronidazole is about one-tenth the cost of vancomycin. Treatment is for 10 to 14 days, usually in a dose of 500 mg p.o. t.i.d. The vancomycin dosage is 125 mg p.o. q.i.d. but is effective only via the oral route, whereas metronidazole is also effective intravenously in the occasional patient with postoperative ileus. With both regimens there is a high relapse rate of

infection, of up to 20%. The best method to prevent relapse is unknown, but relapsing symptoms may respond to retreatment of the infection with either metronidazole or vancomycin.

3.7.4 *ENTAMOEBIA HISTOLYTICA (AMEBIASIS)*

Entamoeba histolytica, the parasite that causes amebiasis infection, appears to be the only ameba that causes disease in humans. Other amebas are often found in the colon as normal commensals. *E. histolytica* is a cyst-forming parasite, but the cysts do not cause disease. The cysts are ingested and are resistant to destruction by gastric acid; then the trophozoite develops in the colon from the ingested cyst. The cysts spread disease to others, and frequently unaffected carriers spread disease by excreting cysts. The trophozoites, which invade the colonic mucosa, cause disease, but trophozoites passed in the stool of symptomatic individuals cannot survive outside the body and rarely transmit infection. The disease is most prevalent in areas of the tropics where sanitation is poor.

The colon is the usual site of initial disease. Invasion of the mucosa by trophozoites is due to the production of an “amebapore” molecule that causes the lining colonocytes to lyse; the lysed colonocytes are then ingested by the amebas, leading to ulceration of the colon and dissemination throughout the body. The amebas infect the colon and rarely the ileum, but the cecum is usually involved. *E. histolytica* is an invasive pathogen and can spread hematogenously to other organs, especially the liver.

Diagnosis is usually made by identification of *E. histolytica* on microscopic analysis of stool, but can also be made on identification of the ameba on histological diagnosis of colonic biopsies. There is a decreased yield on stool analysis after barium studies and if antibiotics or mineral oil is used prior to collection. At endoscopy the ulcers of the rectum and colon may appear characteristic with undermined edges, and sometimes the intervening mucosa looks normal in contrast to acute bacillary dysentery (see Section 3.7.1) and ulcerative colitis. Diagnosis can also be made by indirect hemagglutination and ELISA tests on serum to detect infection, but if the patient is a carrier with only cyst excretion these tests are often negative. Chronic infection of the cecum leads to a “coned” appearance on x-ray. Other colonic complications include perforation, ameboma (a granulomatous tissue reaction in the colon; the mass effect can lead to obstruction or be mistaken for colonic malignancy), pericolic abscess and fistulas. The liver is the commonest extra-intestinal organ infected, but *E. histolytica* can also spread to the brain, lungs, pericardium and eyes. There is an increased risk of disseminated disease and abscess formation if the patient is on steroids, is pregnant or is immunocompromised.

Treatment is usually metronidazole 400–750 mg t.i.d. for 5–10 days for acute colitis. If the patient has chronic colonic disease with chronic shedding of cysts, diloxanide 500 mg t.i.d. for 10 days is the drug of choice. If this cannot be obtained, then iodoquinol 650 mg t.i.d. for 20 days can be used, but this is the maximal dose because it can cause optic neuritis. Patients with amebic liver abscess should first be treated with metronidazole for 10 days and then be given 10 days of diloxanide. All patients should be reassessed two to three months after treatment to ensure clearance of the parasite and that there is no chronic carrier state with cyst excretion.

3.7.5 *BALANTIDIUM COLI*

A very large, ciliated protozoan that uncommonly causes an illness similar to amebic dysentery, *B. coli* is usually easy to identify in stool samples owing to its large size. It is acquired in tropical or subtropical countries from exposure to pigs, which frequently carry this organism without signs of illness. Treatment is with tetracycline 500 mg q.i.d. for 10 days. *B. coli* is also sensitive to ampicillin and metronidazole.

3.7.6 *BLASTOCYSTIS HOMINIS*

A yeast frequently found in asymptomatic individuals, recently *B. hominis* has been suggested as a cause of unexplained diarrhea in some patients found to have large numbers of this protozoan in the stool. Treatment appears to be with either metronidazole 750 mg t.i.d. for 10 days or iodoquinol 650 mg t.i.d. for 20 days. Iodoquinol may be more successful, but the best treatment has not been identified to date.

3.8 Intestinal Nematode Infections

3.8.1 *ROUNDWORM (ASCARIS LUMBRICOIDES)*

Roundworm or ascaris, one of the more common nematodes found in humans, is most often found in the tropics. Usually eggs are ingested from contaminated foods or dirty hands. The eggs hatch in the intestine and spread by the blood to the liver and then to the lungs. An eosinophilic pneumonitis can develop and then the larvae migrate through the alveoli, up to the trachea and through the larynx where they are swallowed. They develop into adult worms in the small intestine. The adults can cause intestinal obstruction symptoms if large numbers are present, and can cause biliary symptoms if they migrate into the common bile duct.

3.8.2 HOOKWORM (*ANCYLOSTOMA DUODENALE*; *NECATOR AMERICANUS*)

Hookworm can infiltrate skin from contaminated earth and is prone to be found in areas with fecal contamination of the soil. A pruritic rash develops at the site of entry into the body. The filarial larvae then travel to the lungs, migrate through the alveoli and then up through the larynx where they are swallowed. After being swallowed, they cause nausea, diarrhea, vomiting, abdominal pain and flatulence. Many patients present with iron deficiency from a daily blood loss of 0.1–0.4 mL with each worm.

3.8.3 WHIPWORM (*TRICHURIS TRICHIURA*)

Whipworm can also cause iron deficiency if large numbers infect the GI tract. It primarily invades the colon. Bloody diarrhea develops with larger infestations. It is easily diagnosed by stool analysis looking for the typical eggs, but is increasingly diagnosed at colonoscopy during investigation of the bloody diarrhea, where the worms are easily seen if present.

3.8.4 PINWORM (*ENTEROBIUS VERMICULARIS*)

Pinworm is probably the commonest nematode worldwide. It usually causes pruritus ani, often worse at night when the worms migrate onto the perianal skin and lay their eggs. Pinworm is probably the most common nematode encountered in Canada, especially in children. Diagnosis is by identification of the eggs from the perianal skin, usually collected in the early morning before defecation.

3.8.5 *STRONGYLOIDES STERCORALIS* (*STRONGYLOIDIASIS*)

Strongyloides stercoralis is widely found in the tropics. It is the only nematode that can multiply and reproduce its entire life cycle within the human host, thus causing persistent reinfection over many years after the original infection. Larvae can penetrate intact skin or the eggs can be ingested. Filariform larvae that penetrate the skin travel hematogenously to the lungs and then, as with the other worms, travel into the airways and are swallowed. In the intestine the larvae become adult worms. When the eggs are ingested, they become filariform larvae in the intestine; then the larvae invade the blood vessels, thus reinfecting the host.

Symptoms of strongyloidiasis vary and may include abdominal pain, diarrhea, nausea and vomiting. With mostly intestinal involvement diarrhea can develop; especially in children, a syndrome similar to celiac disease with protein-losing enteropathy can develop. The majority of adult infections are asymptomatic or are only intermittently symptomatic. Recurrent urticaria can develop where the worms infiltrate the skin, particularly the perianal skin and gluteal areas.

Diagnosis can be confirmed by stool analysis but can be negative in up to 25% of cases, even after repeated stool analysis. The larvae look similar to hookworm. An ELISA test is useful for diagnosis, but there may be overlap with the presence of *Filaria* species. Eosinophilia is often present, even in asymptomatic individuals.

Thiabendazole is usually used to treat, 25 mg/kg twice daily to a maximum of 3 g daily for two days, or five days for disseminated disease. Albendazole or ivermectin may be used if the patient is unable to tolerate thiabendazole, but these drugs appear to be less effective against *S. stercoralis*. With the hyperinfection syndrome, when large numbers of the worms are present (often in association with immune suppression, as with steroid therapy), antibiotics are often needed to treat the septicemia that results if the intestinal damage allows secondary bacterial invasion.

3.9 Microscopic Colitis

This condition has been recognized increasingly in which the patient presents with usually painless diarrhea. Investigations often find signs of inflammation, but the colon appears normal on both radioscopic and colonoscopic examination. This condition is sometimes called “lymphocytic colitis” and may also be part of a spectrum of colitis conditions that include “collagenous colitis.” The natural history of these diseases is unclear and no infective agent has been found. These disorders can be diagnosed only by colonoscopic biopsy. The colonic mucosa appears normal, yet on histological examination there is an increase in the inflammatory infiltrate of the lamina propria. In collagenous colitis the basement membrane of the colonic mucosa is thickened by a band of collagen. In most patients the disease appears to follow a benign course, but about half of patients continue to have significant diarrhea for more than two years. The disease is controlled by antimotility agents such as loperamide or by use of 5-aminosalicylic acid–based therapies directed at the colon (see Chapter 9, “Inflammatory Bowel Disease”), which often help to lessen the diarrhea. Glucocorticoids also control the diarrhea, but in view of the benign course of this illness in most patients, steroid therapy should be used only in severely symptomatic patients who cannot be controlled by other therapy.

3.10 Eosinophilic Colitis

Eosinophilic gastroenteritis is an uncommon inflammatory condition that affects primarily the upper GI tract and small intestine (see Chapters 4, 5 and 6). However, there have been recent reports of an apparently separate condition called “eosinophilic colitis” in which patients with connective tissue disease present with diarrhea of uncertain cause with negative stool investigations. These patients have all been diagnosed at colonoscopy by biopsy of

essentially normal-looking mucosa, yet with increased eosinophils in the lamina propria. All patients respond to steroids, but it would appear that not all patients resolve over time and some may need prolonged steroid therapy.

4. THE ANAL CANAL / M. BURNSTEIN

4.1 Functional Anatomy of the Anal Canal and Anorectal Spaces

4.1.1 THE ANAL CANAL

The anal canal begins where the terminal portion of the large bowel passes through the pelvic floor muscles, and it ends at the anal verge. It measures roughly 4 cm in length. The wall of the anal canal is formed by a continuation of the circular muscle of the rectal wall; the smooth muscle is thickened in this area to form the internal anal sphincter. This smooth-muscle sphincter is wrapped by skeletal muscle, the external anal sphincter. The top of the external anal sphincter is formed by the U-shaped puborectalis muscle, which loops around the anus, arising and inserting on the pubis. This is felt posteriorly and laterally as the anorectal ring on digital examination. The longitudinal muscle coat of the rectum descends in the plane between the sphincters as the conjoined longitudinal muscle, and it sends fibers across the lower part of the external anal sphincter to insert on the skin (corrugator cutis ani, responsible for the anocutaneous reflex or “anal wink”). These fibers also traverse the internal anal sphincter to insert on the submucosa (“mucosal suspensory ligament”).

In approximately the mid-anus there is a rolling line of demarcation called the dentate line. Above the line is columnar epithelium; below it is squamous epithelium without appendages (the anoderm). The demarcation does not really occur at a line, but at a transitional zone of 0.5–1 cm in length.

As the rectum narrows into the anal canal, the mucosa develops 6 to 14 longitudinal folds, Morgagni’s columns. Between the distal ends of the columns are small crypts. Anal glands open into the crypts. There are 4 to 10 glands, and they are lined by stratified columnar epithelium. About half of these tubular glands end in the intersphincteric plane.

Blood is supplied to the anus via the inferior rectal artery, a branch of the internal pudendal artery. The inferior rectal artery crosses the ischiorectal fossa. The superior rectal vein drains the upper part of the anal canal via the inferior mesenteric vein to the portal vein. The middle and inferior rectal veins drain the upper and lower anal canal into the systemic circulation via the internal iliac and internal pudendal veins, respectively.

Lymphatic drainage above the dentate line is via the superior rectal lymphatics (accompanying the superior rectal vessels) to the inferior mesenteric

nodes, and laterally along the middle and inferior rectal vessels to the internal iliac nodes. Lymphatic drainage from the anal canal below the dentate line may be in a cephalad or lateral direction, but is primarily to the inguinal nodes.

Motor innervation of the external anal sphincter is via the inferior rectal branch of the pudendal nerve and the perineal branch of the fourth sacral nerve. The internal anal sphincter has sympathetic (motor) and parasympathetic (inhibitory) innervation. Parasympathetic supply is from the nervi erigentes (S2, S3, S4). Sympathetic innervation is from the first three lumbar segments via the preaortic plexus. Fibers from the preaortic plexus ultimately join the nervi erigentes to form the pelvic plexuses. Sensation below the dentate line (and for up to 1.5 cm above the dentate line) is carried by the inferior rectal nerve. Above the level of the inferior rectal nerve sensory distribution, there are only dull perceptions, mediated by parasympathetic fibers.

4.1.2 ANORECTAL SPACES

Around the anorectum are a number of potential spaces filled with fat or connective tissue. These may become the sites of abscess formation. The perianal space is at the anal verge, and is continuous with the intersphincteric space. The pyramid-shaped ischiorectal (ischioanal) fossa is medially bounded by the external anal sphincter and the levator ani muscles. The lateral wall is the obturator internus muscle and fascia. The inferior boundary is the skin of the perineum, and the apex is the origin of the levator ani from the obturator fascia. Posteriorly is the gluteus maximus muscle, and anteriorly the transverse perinei muscles. On the obturator fascia is Alcock's canal, containing the internal pudendal vessels and pudendal nerve. The fossa is filled with fat and also contains the inferior rectal nerve and vessels, and the fourth sacral nerve. The two ischiorectal spaces communicate with one another behind the anal canal.

4.2 Evaluation of Anorectal Complaints

This section will review the symptoms associated with anorectal pathology and the techniques of anorectal examination.

4.2.1 HISTORY

As in most of medicine, taking a careful history is the most productive step in leading to a diagnosis. In the evaluation of the patient with anorectal complaints, there are a limited number of questions to be asked:

4.2.1.1 Pain

There are three common lesions that cause anorectal pain: fissure in ano, anal abscess and thrombosed external hemorrhoid. If the pain is sharp and occurs

during, and for a short time following, bowel movements, a fissure is likely. Continuous pain associated with a perianal swelling probably stems from thrombosis of perianal vessels, especially when there is an antecedent history of straining, either at stool or with physical exertion. An anal abscess will also produce a continuous, often throbbing pain, which may be aggravated by the patient's coughing or sneezing. Anorectal abscesses are generally associated with local signs of inflammation. The absence of an inflammatory mass in the setting of severe local pain and tenderness is typical of an intersphincteric abscess; the degree of tenderness usually prevents adequate examination, and evaluation under anesthesia is necessary to confirm the diagnosis and to drain the pus.

Anal pain of any etiology may be aggravated by bowel movements. Tenesmus, an uncomfortable desire to defecate, is frequently associated with inflammatory conditions of the anorectum. Although anal neoplasms rarely produce pain, invasion of the sphincter mechanism may also result in tenesmus. Tenesmus with urgency of evacuation suggests proctitis.

Transient, deep-seated pain that is unrelated to defecation may be due to levator spasm ("proctalgia fugax").

Anorectal pain is so frequently, and erroneously, attributed to hemorrhoids, that this point bears special mention: pain is *not* a symptom of uncomplicated hemorrhoids. If a perianal vein of the inferior rectal plexus undergoes thrombosis, or ruptures, an acutely painful and tender subcutaneous lump will appear. This is the "thrombosed external hemorrhoid." Internal hemorrhoids may prolapse and become strangulated to produce an acute problem of anorectal pain, tenderness, and mucous, bloody discharge. Gangrene and secondary infection may ensue.

4.2.1.2 *Bleeding*

The nature of the rectal bleeding will help determine the underlying cause. However, the clinician must remember that the historical features of the bleeding cannot be relied upon to define the problem with certainty. Bright red blood on the toilet paper or on the outside of the stool, or dripping into the bowl, suggests a local anal source, such as a fissure or internal hemorrhoids. Blood that is mixed in with the stool, or that is dark and clotted, suggests sources proximal to the anus. Melena is always due to more proximal pathology.

The associated symptoms are very helpful. A history of local anal bleeding, as described above, associated with painful defecation, suggests a fissure. The same bleeding pattern without pain suggests internal hemorrhoids; this may be associated with some degree of hemorrhoidal prolapse. Bleeding and diarrhea may occur with inflammatory bowel disease. When bleeding is associated with a painful lump and is not exclusively related to defecation, a

thrombosed external hemorrhoid is likely. Bleeding associated with a mucopurulent discharge and tenesmus may be seen with proctitis, or possibly with a rectal neoplasm.

Bleeding per rectum is an important symptom of colorectal cancer, and although this is not the most common cause of hematochezia, it is the most serious and must always be considered. This does not mean that every patient who passes blood must have contrast radiography of the colon or total colonoscopy. If the bleeding has an obvious anal source, it may be prudent not to proceed with a total colon examination, especially in a patient at low risk for colorectal neoplasms (i.e., age under 50 years; no history of Crohn's or ulcerative colitis; no family history of colon cancer; and no personal history of colorectal neoplasms). However, if bleeding persists after treatment of the anal pathology, more ominous lesions have to be excluded.

4.2.1.3 Prolapse

In evaluating protrusion from the anal opening, there are several relevant questions: Is the prolapse spontaneous or exclusively with defecation? Spontaneous prolapse is less characteristic of internal hemorrhoids than of hypertrophied anal papillae or complete rectal prolapse. Does the prolapsing tissue reduce spontaneously (as may be the case with second-degree internal hemorrhoids) or does it require manual reduction (as with third-degree internal hemorrhoids or complete rectal prolapse)? The patient may be able to describe the size of the prolapsing tissue, and this may suggest the diagnosis.

Complete rectal prolapse (proctidentia) must be distinguished from mucosal prolapse or prolapsing internal hemorrhoids. Proctidentia occurs mainly in women (female:male = 6:1), with a peak incidence in the seventh decade. Proctidentia is often associated with fecal incontinence. In later stages, protrusion occurs even with slight exertion such as coughing or sneezing. The extruded rectum becomes excoriated, leading to tenesmus, mucus discharge and bleeding. (Examination of the patient with proctidentia usually reveals poor anal tone, and with the tissue in a prolapsed state, the mucosal folds are seen to be concentric, whereas with prolapsed hemorrhoids there are radial folds.) Rarely, a large polypoid tumor of the rectum may prolapse through the anal canal.

4.2.1.4 Perianal mass

A painful perianal lump may be an abscess or a thrombosed external hemorrhoid. Knowing whether there has been a discharge of blood or pus may be helpful. An intermittent mass suggests a prolapsing lesion.

External or "skin" tags are very common deformities of the anal margin. They may be the result of previous or active fissure disease, or the sequelae of a thrombosed external hemorrhoid. Condylomata acuminata – or

venereal warts – are caused by a sexually transmitted virus. The perianal skin is frequently affected, and the condition occurs with greatest frequency in gay men.

The differential diagnosis also includes benign and malignant neoplasms.

4.2.1.5 *Pruritus*

Itching is a common associated feature of many anorectal conditions, especially during the healing phase or if there is a discharge. But pruritus ani may also be an isolated symptom or the patient's primary complaint. As a chief complaint, pruritus may be caused by infections (e.g., pinworms, condylomata, *Candida*) or skin conditions (e.g., contact dermatitis, psoriasis). More commonly, no specific underlying pathology is identified, and the problem is idiopathic.

Idiopathic pruritus ani is more common in men, and is typically worse at night. When chronic, the characteristic changes of hypertrophy and lichenification, nodularity, scarring and fissuring of the skin become apparent.

4.2.1.6 *Discharge*

Although mucus is a normal product of the colorectal mucosa, it is not normally seen in the stool. Increased mucus may be the result of proctocolitis or a colorectal neoplasm, especially a villous adenoma of the rectum. Both inflammatory and neoplastic conditions may present with mucus and blood. Phosphate enemas are irritating and often elicit copious mucus production. Patients with the irritable bowel syndrome may complain of mucous stools.

Mucus staining of the underclothes may be associated with prolapsing tissue. When the staining has a fecal component, or when there is associated inability to control gas or to discriminate gas from solids within the rectum, a disturbance of the continence mechanism exists. A history of "accidents," or the need to wear pads during the day or night, will help indicate the magnitude of the problem. The discharge may arise from an obvious external lesion – e.g., blood from a thrombosed external hemorrhoid, or pus from an abscess, from the external opening of a fistula, from a pilonidal process or from perianal hidradenitis suppurativa.

Other issues that will prove helpful in coming to a diagnosis of anorectal pathology include bowel habits, associated medical conditions and medications, sexual practices, travel history and family history.

4.2.2 *EXAMINATION*

The patient about to undergo examination of the anorectum may not only be embarrassed, but also afraid of impending pain and discomfort. Explanation of the examinations to be performed, and reassurance, will lessen the patient's anxiety and contribute greatly to patient cooperation.

The four steps in anorectal evaluation are inspection, palpation, anoscopy and proctosigmoidoscopy.

4.2.2.1 Positioning

The patient is placed either in the left lateral position or (preferably) in the prone-jackknife position. The prone-jackknife position requires a special table that tilts the head down and raises the anorectal region, with the buttocks tending to fall apart. This provides the best and easiest access for the examiner, although patient comfort may be less.

The left lateral (Sims') position has the advantages of patient comfort and of being suitable for any examining table, bed or stretcher. The patient's buttocks are allowed to protrude over the edge of the table, with hips flexed and knees slightly extended. The examiner may sit or stand.

The patient is unable to see "what's going on back there," and it is important to continually explain what you are doing and what can be expected.

4.2.2.2 Inspection

Looking at the anal area may reveal obvious external pathology. The resting anal aperture should be observed: a patulous opening may be seen with procidentia, sphincter injury or neurologic abnormality. Straining and squeezing by the patient may provide information about anorectal function.

Gentle spreading of the buttocks may elicit pain in a patient who has an anal fissure. Asking the patient to strain down may provide information: internal hemorrhoids may protrude or procidentia may be seen. However, if procidentia is suspected, it should be sought with the patient squatting or sitting at the toilet.

4.2.2.3 Palpation

A disposable plastic glove and water-soluble lubricant are required. The patient is told that a finger will be gently placed into the rectum. While one hand separates the buttocks, the index finger is placed on the anal verge, and with the patient bearing down, thereby relaxing the anus, the digit is advanced into the anal canal.

A methodical approach is best. Palpation anteriorly checks the prostate in males, and the cervix in females. The finger then sweeps backward and forward to palpate the rest of the circumference of the anorectum. This may be the only part of the examination that identifies submucosal lesions, which may easily go undetected by endoscopy. Resting tone and ability to squeeze should also be assessed. The location of tenderness or a palpable abnormality should be precisely recorded.

4.2.2.4 Anoscopy

The anoscope is the optimal instrument for examining lesions of the anal canal. It is not a substitute for proctosigmoidoscopy, and the proctosigmoidoscope does not provide as satisfactory a view of the canal as does the anoscope. Many anoscopes are available; the best instrument is end-viewing, with an attached fiberoptic light source.

4.2.2.5 Proctosigmoidoscopy

The rigid 25 cm sigmoidoscope (or proctoscope) is the best instrument for examining the rectum. A barium enema, because of the balloon-tipped catheter used in administering the contrast material, does not adequately evaluate the rectal ampulla and is never a sufficient workup of a lower GI complaint.

A variety of rigid sigmoidoscopes are available: disposable or reusable, in a range of diameters (1.1 cm, 1.9 cm, 2.7 cm) and with proximal or distal lighting. The 1.9 cm instrument provides good visibility with minimal patient discomfort. The instrument includes a 25 cm tube, a magnifying lens, a light source, and a bulb attachment for air insufflation. Long swabs may be helpful in maintaining visibility, but suction is best.

A single Fleet[®] enema provides excellent preparation of the distal bowel and should be used just before the examination. The Fleet[®] enema may produce transient mucosal changes, and if inflammatory bowel disease is suspected, it should be avoided.

The digital examination has set the stage for instrumentation by permitting the sphincter to relax. With the tip well lubricated, the sigmoidoscope is inserted and passed quickly up the rectum. As always, the patient is informed of what is being done, and is reassured that the sensation of impending evacuation is caused by the instrument, and that the bowels are not about to move.

Air insufflation should be kept to a minimum, as it may cause discomfort, but it is of value both on entry and on withdrawal in demonstrating the mucosa and lumen and in assessing rectal compliance and the presence of normal sensation of rectal distention. Advancement should occur only with the lumen clearly in sight. When the lumen is "lost," withdraw and redirect to regain it.

As the rectosigmoid is reached (approximately 15 cm along), the patient should be warned of possible cramping discomfort that will disappear as the scope is removed. Frequently, even with experience, the rectosigmoid angle cannot be negotiated, and the examination should be terminated. Most importantly, the patient should not be hurt or caused significant discomfort. The scope should be withdrawn making large circular motions, carefully inspecting the circumference of the bowel wall, flattening the mucosal folds and valves of Houston. The posterior rectal wall in the sacral hollow must be specifically sought out, or it will be missed.

In most large studies, the average depth of insertion is 18–20 cm; the full length of the instrument is inserted in less than half the patients.

Perforation of the normal rectum by the sigmoidoscope is extremely rare (1 in 50,000 or less). However, advancing the instrument or insufflating air may be hazardous in settings such as inflammatory bowel disease, radiation proctitis, diverticulitis and cancer. Of course, biopsy and electrocoagulation have to be performed with care and with knowledge of the technique and equipment.

The incidence and significance of bacteremia following anorectal manipulations is controversial, and has been reported in 0–25% of proctoscopies. Prophylactic antibiotics should be considered in patients with prosthetic heart valves.

4.3 Specific Anorectal Problems

This section will briefly review some of the more common anorectal problems.

4.3.1 HEMORRHOIDS

4.3.1.1 Background

The upper anal canal has three sites of thickened submucosa containing arterioles, venules and arteriovenous communications. These three vascular “cushions” are in the left lateral, right anterior and right posterior positions. Minor cushions may lie between the three main ones. The cushions are held in the upper anal canal by muscular fibers from the conjoined longitudinal muscle of the intersphincteric plane.

Hemorrhoids exist when the anal cushions prolapse after disruption of their sensory mechanism, or when there is dilation of the veins and arteriovenous anastomoses within the cushions. Various theories can be put forward for the development of internal hemorrhoidal disease: raised intra-abdominal pressure, pressure on the hemorrhoidal veins by an enlarging uterus, poor venous drainage secondary to an overactive internal anal sphincter, straining at stool with a resultant downward displacement of the cushions, etc.

Skin tags are projections of skin at the anal verge. They may be the result of previous thrombosed external hemorrhoids, fissure in ano, or inflammatory bowel disease.

External hemorrhoids are dilated veins of the inferior hemorrhoidal (rectal) plexus. This plexus lies just below the dentate line and is covered by squamous epithelium.

Internal hemorrhoids are the symptomatic, enlarged submucosal vascular cushions of the anal canal. The cushions are located above the dentate line and are covered by columnar and transitional epithelium. The patient’s history allows internal hemorrhoids to be subdivided. First-degree hemorrhoids produce painless bleeding but do not protrude from the anal canal; at anoscopy,

they are seen to bulge into the lumen. Second-degree hemorrhoids protrude with bowel movements, but reduce themselves spontaneously. Third-degree hemorrhoids prolapse outside the anal canal, either spontaneously or with bowel movements, but require digital reduction. Fourth-degree hemorrhoids are always prolapsed, and cannot be reduced.

4.3.1.2 *Diagnosis and treatment*

4.3.1.2.1 *Thrombosed external hemorrhoids* As a rule, external hemorrhoids are asymptomatic until the complication of thrombosis (intravascular clot) or rupture (perianal hematoma) supervenes. In either case, the presentation is severe pain with a perianal lump, often after straining. The natural history is one of continued pain for 4 to 5 days, then slow resolution over 10 to 14 days. The treatment depends on the severity of the pain and the timing of presentation. A patient who presents within 24 to 48 hours and with severe pain is best dealt with operatively. Under local anesthesia, the involved perianal vessel and clot are excised. The wound may be left open or may be closed. Simple evacuation of the thrombus is less effective. A patient presenting later, after 3 to 4 days, is advised to take frequent warm baths, a bulk laxative, a surface-active wetting agent, and oral analgesics. This regimen is also prescribed post-excision.

4.3.1.2.2 *Internal hemorrhoids* Painless, bright red rectal bleeding (usually with or following bowel movements) is the most common symptom of this condition. Blood appears on the toilet paper or on the outside of the stool, or drips into the bowl. It is very rare for the volume of blood lost from internal hemorrhoids to be sufficient to explain iron deficiency anemia; further workup is always indicated.

Prolapse with defecation or other straining activities is also a common symptom of internal hemorrhoids. Chronic prolapse is associated with mucus discharge, fecal staining of the underclothes and pruritus.

Anal sphincter spasm may result in thrombosis and strangulation of prolapsed hemorrhoids. This presents as an acute problem of a painful, discharging, edematous mass of hemorrhoids.

Inspection will identify the later stages of the disease, especially when the patient is asked to bear down. Digital examination can rule out other pathology, as well as assess the sphincters. A palpable abnormality suggests some other process. Anoscopy provides a diagnosis in first- and second-degree disease. With the anoscope in place, the patient is once again asked to strain, and the degree of prolapse observed. Proctosigmoidoscopy should always be performed to exclude other diseases, particularly rectal neoplasms and inflammatory bowel disease.

If the symptoms are at all atypical, or the physical findings leave any doubt about the source of blood, a colon-clearing examination (either colonoscopy or barium enema) should be performed.

In patients over the age of 50, it is reasonable to take the opportunity to screen (or to practice “case-finding”) for colorectal cancer by performing sigmoidoscopy with the 60 cm flexible instrument. If risk factors for colorectal neoplasia are present, then colonoscopy or barium enema should certainly be performed.

Occasional bleeding, especially if it is related to hard stools or straining, should be managed by improving bowel habits using high-fiber diet and bulk agents (e.g., psyllium). If bleeding persists or is frequent, intervention is indicated, and in most cases should take the form of rubber-band ligation. Prolapsing hemorrhoids that reduce spontaneously, or can be easily reduced, are also nicely treated by rubber-band ligation. If prolapsing tissue is not easily reduced, or if there is a significant external component, surgical hemorrhoidectomy offers the best cure. Similarly, prolapsed, thrombosed internal hemorrhoids should be surgically excised.

4.3.1.2.3 Rubber-band ligation In this technique, strangulating rubber bands are placed at the cephalad aspect of the internal hemorrhoids. The absence of somatic pain fibers above the dentate line renders this a relatively painless procedure, as long as the rings are properly positioned. The banded tissue infarcts and sloughs over the next week, resulting in reduction of hemorrhoidal tissue, as well as fixation of the residual hemorrhoid in the upper anal canal. It is a simple office procedure requiring an anoscope and ligator. In general, only one or two areas are banded at a time, so that several treatments are often required. Long-term success is expected in approximately 75% of patients with second-degree hemorrhoids. Pain, bleeding and infection are rare complications.

4.3.1.2.4 Hemorrhoidectomy Since the popularization of rubber-band ligation, excisional hemorrhoidectomy has been much less frequently performed. The important principles of all excisional procedures are removal of all external and internal hemorrhoids, protection of the internal anal sphincter from injury, and maintenance of the anoderm, so as to avoid anal stenosis.

4.3.2 FISSURE IN ANO

This is a linear crack in the lining of the anal canal, extending from the dentate line to the anal verge. It is seen equally in men and women, and at all ages, but is a common entity in young adults. It is encountered mainly in the posterior midline, but also occasionally in the anterior midline. If a fissure persists, secondary changes occur. These include the “sentinel pile” at the distal end of

the fissure and the “hypertrophied anal papilla” at the proximal end. They are due to edema and low-grade infection.

4.3.2.1 Pathogenesis

Fissure in ano is probably the result of trauma during the passage of hard stool, but not all patients with fissure in ano give a history of “constipation.” While most fissures will readily heal with an appropriate change in bowel habits, some will persist. This may be due to continued trauma or to spasm of the internal anal sphincter.

There is an association between fissures and inflammatory bowel disease, particularly Crohn’s disease, and this should be kept in mind.

4.3.2.2 Diagnosis

Pain with defecation is the chief complaint. The pain may persist for minutes to hours. Bright red blood is often seen on the toilet paper and on the stool. The patient with an edematous, tender skin tag (sentinel pile) may complain of a painful hemorrhoid. The patient may be constipated in response to painful defecation.

With gentle separation of the buttocks, most fissures will be visible. The sentinel pile of a chronic fissure may be the initial finding. With acute fissures, digital and anoscopic examination are usually not possible because of local tenderness. However, these examinations should be performed later to rule out other pathology. With chronic fissures, anoscopy reveals the defect in the anoderm, with exposed muscle fibers of the internal anal sphincter at the fissure base. The hypertrophied anal papilla may be seen.

Fissures off the midline should raise the possibility of other diseases. Crohn’s disease may be associated with atypical-looking fissures that are off the midline and have atypical symptoms. Anal and rectal carcinoma should be palpably different from fissures, but if any doubt exists, a biopsy should be done. A syphilitic chancre may occasionally look like an idiopathic fissure.

4.3.2.3 Treatment

The mainstay of therapy for acute fissures is to achieve daily soft bowel movements. This will prevent further tearing and relieve the anal spasm, allowing most acute fissures to heal within one to two weeks. Warm tub baths are soothing and cleansing, and may also reduce spasm. A high-fiber diet supplemented with bulk agents and surface-active wetting agents will accomplish the desired effect.

If the history is longer than a few weeks and the physical findings suggest chronicity (i.e., exposed sphincter fibers, hypertrophied papilla, sentinel pile and palpable induration), this conservative therapy may not help.

If symptoms warrant, such a fissure should be treated operatively, generally by lateral internal sphincterotomy. This relieves the internal anal sphincter spasm and allows the fissure to heal in over 90% of cases. Minor disturbances of continence, especially for flatus, may complicate a sphincterotomy in 5–10% of patients.

4.3.3 FISTULA-ABSCESS DISEASE

Anorectal abscess and fistula are the acute and chronic phases, respectively, of the same disease. The disease begins as an infection in the anal glands and initially presents as an abscess. When the abscess is surgically drained, or drains spontaneously, a communication (i.e., a fistula) exists between the gland of origin and the perianal skin.

The infection begins in the intersphincteric plane, where many of the anal glands terminate. The infectious process may remain in this plane as an intersphincteric abscess, or, more commonly, it may track downward in the intersphincteric plane to present as a perianal abscess. Similarly, infection may penetrate the external sphincter to enter the ischiorectal fossa. Many complex variations are seen, determined by the direction of spread and sometimes by inappropriate intervention. The infection may track circumferentially from one side of the anal canal to the other to cause a “horseshoe” abscess. Perianal and ischiorectal abscesses account for at least three-quarters of anorectal abscesses.

The classical signs of inflammation are generally present, although with an intersphincteric abscess there may be nothing to see. In the case of intersphincteric abscess, the patient will be too tender for adequate examination, and examination under anesthesia will be necessary.

Management of the abscess consists of incision and drainage, and this can usually be accomplished under local anesthesia. To ensure adequate drainage, a cruciate or elliptical incision is made. For the one-half to two-thirds of patients who go on to develop a fistula in ano, a fistulotomy, or laying-open, with curettage of the track is required. The wound heals secondarily. Non-healing or recurrence of the fistula usually indicates a failure to destroy the gland of origin. In performing fistulotomy, the utmost attention must be paid to the anatomic relationship between the fistula track and the sphincter mechanism. Excessive division of muscle contained within the fistula can lead to partial or complete fecal incontinence.

4.3.4 PILONIDAL DISEASE

This is an acquired condition in which body hair is drilled into the skin of the natal cleft by the back-and-forth motion of the buttocks. This produces a primary midline opening or track, from which abscesses and secondary tracks and openings may form.

The disease is mainly seen in young, hirsute males. It commonly presents as an acute abscess, but may also present as a chronic “sinus,” usually with multiple openings.

The abscess stage is treated by incision and drainage, usually under local anesthesia. After the abscess has healed, some of these patients will require definitive surgery to deal with the primary and secondary tracks. The preferred treatment consists of opening the anterior wall of the tracks and suturing the edge of the track to the skin edge. This technique is called “marsupialization.”

4.4 Sexually Transmitted Diseases of the Anorectum

There is an increasing incidence of venereal infections of the anorectal region, mainly accounted for by sexual practices among gay men. Many of these diseases may mimic nonvenereal conditions of the anorectum, and multiple venereal infections may coexist.

While immunocompetent gay men are subject to infection with the usual venereal pathogens, AIDS patients may additionally suffer from opportunistic infections of the gut.

The common anorectal venereal infections seen in North America are discussed here.

Condylomata acuminata, or venereal warts, are seen in the perianal region and anal canal, as well as the vulva, vagina and penis. They are most often seen in male homosexuals. The causative agent is believed to be a papilloma virus with an incubation period of one to six months. Symptoms are generally minor – itching, and occasionally bleeding. Perianal warts are frequently accompanied by warts within the anal canal, and these must be looked for at anoscopy.

Many treatments exist. None has a better than 70% chance of eradicating the disease by a single application. For perianal and anal canal warts, electrocoagulation or laser destruction is preferred. For extensive persistent disease, immunotherapy with an autologous vaccine has been very successful.

Squamous cancer has been seen to arise in *condylomata acuminata*.

Neisseria gonorrhoeae may produce proctitis. The incubation period of *gonococcal proctitis* is five to seven days. Gonococcal proctitis is most often asymptomatic; symptoms may include mucopurulent discharge and tenesmus. Proctoscopy reveals a thick, purulent discharge on a background of mild, non-ulcerative inflammation of the distal rectum. Gram’s stain is unreliable, but culture of the pus confirms the diagnosis. Serologic testing for syphilis should be carried out. Treatment for homosexual men is ceftriaxone, 250 mg IM once.

Syphilis can affect the anal region. The incubation period ranges from 9 to 90 days. The primary lesion is a chancre, and because it is painful, it may be mistaken for a fissure. However, chancres are off the midline, are often

multiple, and have an atypical appearance. Bilateral inguinal lymphadenopathy may be present. The chancre regresses over 6 weeks. *Treponema pallidum* is demonstrated from the primary lesion by darkfield microscopy. Serologic testing will be positive within a few weeks of the appearance of the chancre. If untreated, the secondary stage of syphilis may involve the anal area 6 to 8 weeks after healing of the chancre. This takes the form of a rash or of condylomata lata – flat, wart-like lesions teeming with *Treponema pallidum*. Treatment of primary and secondary syphilis is with benzathine penicillin G, 2.4 million units IM once. Sexual contacts should be treated prophylactically.

Herpes simplex 2 may infect the anorectum. The incubation period is 4 to 21 days. Constitutional symptoms are followed by severe anorectal pain. Small vesicles and aphthous ulcers are seen perianally and in the anal canal and lower rectum. Examination may reveal tender inguinal lymphadenopathy. Viral cultures of the vesicular fluid will be positive and rectal biopsy has a characteristic appearance. Spontaneous resolution occurs over several weeks. Recurrences are frequent but less severe. Immunosuppressed patients may develop a severe, destructive process. Treatment is with tub baths and analgesics. Topical acyclovir q8h for 5 days shortens the symptomatic period and the duration of viral shedding. Intravenous acyclovir is used when there is proctitis in addition to anal and perianal disease. In the AIDS patient, acyclovir is used intravenously in the acute phase, followed by oral acyclovir for 6 months.

Chlamydia proctitis with non-LGV (lymphogranuloma venereum) serotypes is almost identical to gonococcal proctitis. However, the LGV serotypes are invasive and produce a severe proctocolitis with pain, tenesmus, discharge and diarrhea. Chlamydia is isolated from the rectum. Treatment is with tetracycline.

5. LOWER GASTROINTESTINAL BLEEDING / S.J. VANNER

Lower GI bleeding often presents as a medical emergency. Like other medical emergencies, optimum patient care requires careful assessment and resuscitation. The history and physical findings provide important clues to the etiology and are critical for determining the severity and location of the bleeding site.

Lower GI bleeding can be classified arbitrarily as major or minor. Patients presenting with the passage of significant amounts of bright red blood per rectum and hemodynamic compromise have major GI blood loss and are at risk of life-threatening hypovolemia. Be wary of the patient who may have stabilized temporarily or received intravenous fluids prior to a full clinical assessment. Historical clues to a major bleed include the occurrence of syncope or presyncope prior to seeking medical care. The vital signs, with particular

attention paid to postural changes, are crucial to assessing severity. *The passage of bright red blood per rectum almost always originates from the colon. However, it is important to remember that brisk bleeding from a site in the upper GI tract may masquerade as a major lower GI bleed.* In contrast to the patient with major lower GI bleeding is the patient who describes the passage of bright red blood per rectum as blood on the tissue paper or on the outside of formed stool in the absence of other symptoms. Such patients, whose general physical examination is normal, usually have a minor lower GI bleed. Most often this is due to local perianal pathology.

5.1 Determining the Site of Bleeding (Upper or Lower GI Tract)

In the clinical setting of a major lower GI bleed with the passage of bright red blood per rectum and hemodynamic compromise, there are a number of important clues that may raise the suspicion of an upper GI source. These include a past history or symptoms of peptic ulcer disease, NSAID use, prior abdominal aneurysm repair, alcohol abuse and coexisting liver disease. Unfortunately, the lack of upper GI symptoms does not exclude peptic ulcer disease, as a number of duodenal ulcers present as major GI bleeds without a previous typical ulcer history. On physical examination, the finding of hypovolemic shock, particularly in a young person, should trigger immediate consideration of a proximal source of bleeding. Features of chronic liver disease and portal hypertension suggest varices as a possible cause. Most major upper GI bleeding, even in a young person, is accompanied by a transient rise in the BUN (blood urea nitrogen), whereas this is not typical in a lower GI bleed unless there is renal comorbidity.

When an upper GI source is considered, several actions are necessary. A nasogastric tube returning bloody gastric aspirate positively identifies a proximal source of bleeding, but a negative aspirate may not. A negative aspirate will exclude significant bleeding from the esophagus or stomach but may fail to identify bleeding from the duodenum. Even aspirates with bile staining and no blood may fail to identify 5–10% of bleeding duodenal ulcers. When an upper GI source cannot be excluded with confidence, urgent upper endoscopy is required.

Another potentially confusing scenario involves the patient presenting with melena. Melena results from the digestion of blood as it travels through the GI tract, and almost always originates from the upper GI tract. However, occasionally transit of blood from a bleeding right colon is sufficiently slow that stool can appear as melena or melena mixed with dark red blood.

Positive fecal occult blood tests are another clue to lower gastrointestinal bleeding. Many results prove to be false positives; testing should be done with

TABLE 6. Causes of major lower GI bleed

Very common

Diverticular disease

Angiodysplasia

Less common

Ischemia

Neoplasia

Inflammatory bowel disease

Hemobilia

Perianal disease

Aortoenteric fistula

Solitary rectal ulcer

patients on a controlled diet (no red meat, vitamin C or aspirin) to minimize this possibility. Occult positive stools can result from bleeding sites in either the upper or lower GI tract.

5.2 Major Lower GI Bleeding

Angiodysplasia and diverticular bleeding are the two most common causes of major lower GI bleeding, accounting for up to 60–70% of cases.

Angiodysplastic lesions result from dilation and tortuosity of submucosal veins associated with small arteriovenous communication with submucosal arterioles. These lesions are typically multiple, less than 5 mm in diameter, and are most commonly found in the right colon and cecum. The pathogenesis of these lesions is unknown but they occur most commonly in elderly patients and differ from congenital vascular lesions. Diverticula are located predominantly in the left colon, but angiographic studies have shown that those in the right colon bleed more frequently. The pathophysiology underlying diverticular bleeding is also uncertain but is thought to result from rupture of arteries that penetrate the dome of the diverticulum.

A number of other possible but less common causes exist (Table 6), but many of these more typically present with minor lower GI bleeding and a clinical picture dominated by other features such as diarrhea. Angiodysplasia, unlike diverticular bleeding, can also present with minor chronic GI bleeding, and may even present as chronic anemia secondary to microscopic blood loss. In contrast to angiodysplasia and diverticular bleeding, which are relatively painless, bleeding secondary to colonic ischemia is typically preceded by minutes to hours of significant abdominal pain. Abdominal x-rays may demonstrate “thumb-printing,” but this finding is neither specific nor sensitive.

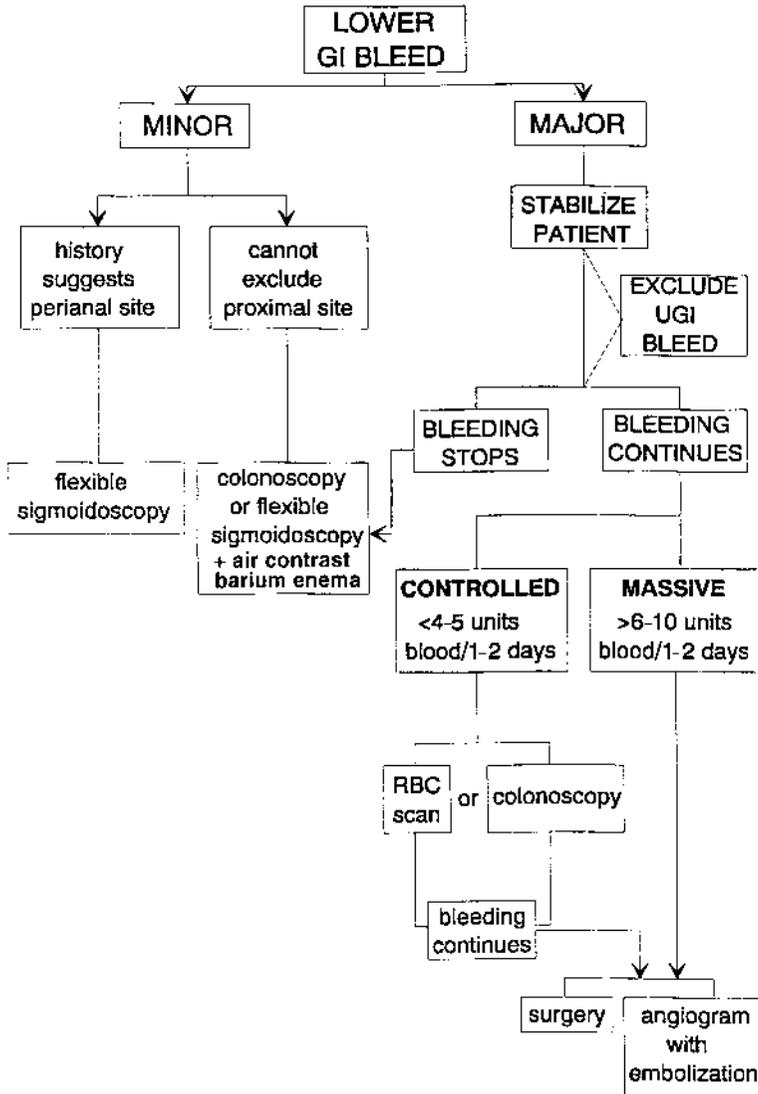


FIGURE 10. Flowchart for investigation of lower GI bleed.

Most major lower GI bleeding will stop without intervention and can be investigated electively, but up to 25% will continue to bleed and require immediate investigation and treatment (Figure 10). After resuscitation, the next priority is to identify the site of bleeding. Radionuclide scanning using technetium-labeled red blood cells is least invasive and readily available in most centers, but interpretation is fraught with false negative and positive results. Although angiography is less available and more invasive, it is more accurate and has the advantage of therapeutic intervention with embolization of the arteriole feeding the bleeding lesion. Colonoscopy can also be attempted to identify the bleeding lesion, and if angiodysplasia is evident it can be treated with electrocautery. However, unless the rate of bleeding is relatively slow, ongoing bleeding usually obscures the lumen, making it difficult to identify the responsible lesion and technically difficult to advance the colonoscope to the site of bleeding. In some cases, continuing bleeding (requiring transfusions of 6–10 units of blood) requires either urgent angiography with embolization or surgical resection with a subtotal colectomy.

5.3 Minor Lower GI Bleeding

Minor bleeding from the lower GI tract is a common complaint and requires a careful approach (Figure 10) to differentiate minor pathology such as hemorrhoids and fissures from serious problems such as colonic tumors. Patients may notice blood only on the outside of formed stool or on the tissue paper, suggesting that the blood originates from the anal canal or the rectosigmoid region. Alternatively, some patients notice that the blood is mixed in the stool, suggesting that bleeding is more proximal within the colon.

Hemorrhoids are the commonest cause of minor bleeding (Table 7), but even when the history is very suggestive, proctoscopic or sigmoidoscopic assessment should be carried out to ensure that a rectal lesion such as proctitis (Figure 11) or a tumor is not mimicking this presentation. Patients with ulcerative proctitis often have frequent bowel movements but pass only bright red blood and mucus on many occasions. Radiation proctitis can present shortly after radiotherapy treatment but is often delayed by many months or years. This condition results from chronic inflammation within the blood vessels, called endarteritis obliterans, and this indolent process underlies the delayed presentation.

TABLE 7. Causes of minor lower GI bleed

Very common

Hemorrhoids
Fissures
Other perianal disease
Proctitis

Less common

Neoplasia
Inflammatory bowel disease
Infectious colitis
Radiation colitis
Angiodysplasia
Ischemia
Rectal ulcer

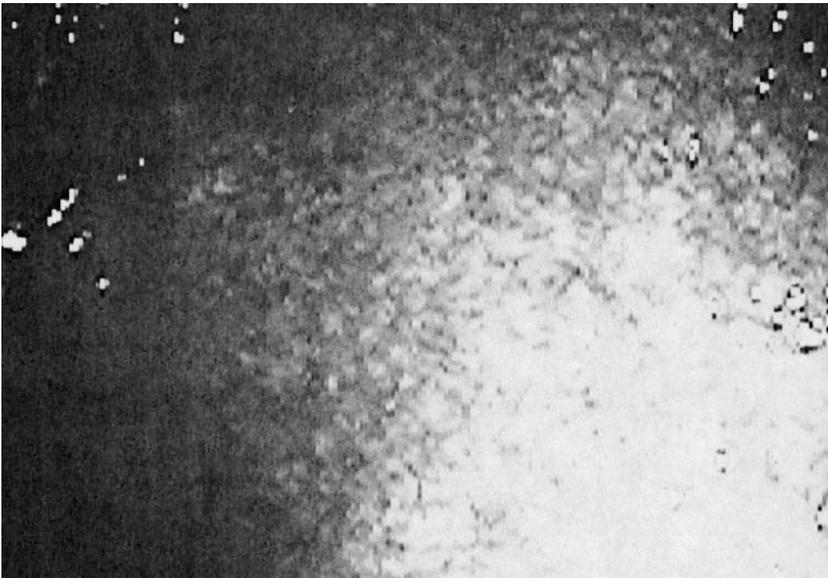


FIGURE 11. Endoscopic appearance of colitis. Note the diffuse reddening of the mucosa, which will bleed if touched. This patient had moderately active ulcerative colitis, but infection and other causes of colitis (e.g., ischemia) can look identical.

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OBJECTIVES

The student should be able to discuss the following with regard to colonic and anorectal function and disease.

Physiology

1. The role of the colon in the intestinal transport of fluid and electrolytes.
2. The mechanism of defecation.
3. The different motility patterns in the right and left colon and how they determine colonic function.
4. The coordination of colonic motility with eating and the innervation of the colon and its relationship with the central nervous system.
5. The role of the colon in digestion.

Carcinoma of the Colon

1. The epidemiology of colonic carcinoma and the predisposing causes to colon cancer.
2. The use of CEA in diagnosis and follow-up of patients with colon cancer.
3. The Dukes' classification of carcinoma of the colon and the percentage survival after five years for each group in the classification.
4. The role of diet in the etiology of colon cancer.
5. The role of chemotherapy and radiation therapy in carcinoma of the colon.
6. Classification of colonic polyps and how to determine their malignant potential.
7. How polyposis syndrome differs from other conditions associated with polyps.

Diverticular Disease

1. The pathophysiology of diverticular disease.
2. The complications and management of diverticular disease.

Colonic Obstruction

1. The symptoms and signs of colonic obstruction.
2. The causes of colonic obstruction in adults and children.
3. The diagnostic approach to a patient with presumed large bowel obstruction.
4. The x-ray findings of partial large bowel obstruction.

Irritable Bowel Syndrome

1. The Manning criteria and how to make a positive diagnosis of an irritable bowel.
2. The symptoms that are not associated with an irritable bowel.
3. When a patient with an irritable bowel should have further investigations to confirm the diagnosis and the appropriate screening tests to rule out other diseases.
4. The factors that influence patients with an irritable bowel seeking medical attention.
5. The treatment of irritable bowel and the approach to differential diagnosis.

Fecal Incontinence

1. The pathophysiology of fecal incontinence.
2. The mechanism by which the anal sphincter maintains continence.
3. The investigation and management of fecal incontinence.

Constipation

1. The etiological classification of constipation.
2. The investigation and differential diagnosis of constipation.
3. The management of constipation.
4. The identification of “laxative abuse.”
5. The causes of solitary rectal ulceration.

Colonic Ischemia

1. The colonic blood supply and the areas of the colon at greatest risk for ischemia.
2. The symptoms and signs of colonic ischemia.
3. The diagnosis and management of ischemia.

Infectious Diarrhea

1. The common causes of dysentery in the tropics.
2. The treatment of common bacterial infections of the colon and choice of the most appropriate antibiotic.
3. The infectious causes of persistent diarrhea.
4. The nematode infections that cause diarrhea and the presentation of nematode intestinal infections.

Perianal Disease

1. The proper techniques of examining the perineum and doing a complete rectal examination (students should be able to demonstrate these techniques).
2. The symptoms of hemorrhoids and their management.
3. The difference between internal and external hemorrhoids.
4. The symptoms and management of fissure in ano.