The human colon is a dynamic organ that performs many functions including absorption of water and electrolytes, salvage of nutrients not absorbed in the small intestine, and transport of luminal contents. It is not an essential organ because life can be sustained after its removal. However, it has a major role in maintaining the health of the human body. Understanding the physiologic principles of its function is essential when treating diseases affecting it—both surgically and medically.

Embryology

To understand the colon, embryology is an important starting point. The midgut begins just distal to the entrance of the bile duct into the duodenum and ends at the junction of the proximal two-thirds of the transverse colon with its distal one-third. Over the entire length, the midgut is supplied by the superior mesenteric artery. The distal third of the transverse colon, the descending colon, the sigmoid, the rectum, and the upper part of the anal canal are derived from the hindgut. The inferior mesenteric artery supplies the hindgut.

The epithelial lining (mucosa) is derived from endodermal tissue. The muscular and peritoneal components are of mesodermal origin surrounding the endoderm. The primitive intestinal loops normally rotate 270 degrees counterclockwise around an axis formed by the superior mesenteric artery. Bowel loops are herniated outside the abdominal cavity. At the end of the third month, the loops return into the abdominal cavity and complete the rotation. The cecum normally rotates down to the right lower quadrant when the process is complete. The appendix begins as a bulge on the cecum at about the sixth week of intrauterine life. As the cecum grows, this bulge lags behind the elongation of the remaining portion of the cecum, forming the appendix.

Innervation

The innervation to the colon comes from two sources—one from outside (extrinsic) and the other inside (intrinsic). The extrinsic component comes from the autonomic nervous system and affects both motor and sensory functions. Parasympathetic fibers reach the proximal colon through the posterior vagal trunk running with the arterial blood supply (along the ileocolic and middle colic branches of the superior mesenteric artery). The distal colon receives parasympathetic fibers from the sacral parasympathetic nerves (S2-4) through the pelvic plexus. These pelvic splanchnic (splanchnic means visceral) nerves give off discrete branches, which run under the peritoneum and into the sigmoid mesocolon toward the left colonic flexure. The parasympathetic nerves are predominantly excitatory for the colon’s motor component via the neurotransmitter acetylcholine and tachykinins, such as substance P. The parasympathetic nerves also convey visceral sensory function.

Sympathetic input is inhibitory to colonic peristalsis (excitatory to sphincters and inhibitory to nonspincteric muscle). The effector cells originate from the thoracic and lumbar sections of the spinal cord. The thoracic splanchnic (visceral nerves) are divided into the greater (T4 to T10), lesser (T9 to T11), and least (T11 to L1). The lumbar input is L2-3. As they emerge via anterior spinal nerve roots they merge into paired paravertebral ganglia, which are located along the medial margin of the psoas muscle in the retroperitoneum. The nerve fiber enters these ganglia as a white ramus and does one of the following: 1) travels up or down the trunk to synapse at another level and supply a segment without its own sympathetic input (i.e., above T1 and below L2); 2) synapses in the ganglion and exits as a gray ramus to supply viscera; 3) passes through the ganglion to a “prevertebral” ganglion such as the celiac plexus where it synapses; or 4) synapses in the ganglion and rejoins its own segmental nerve as a gray ramus. Most preganglionic fibers serving the colon pass through to synapse in a prevertebral ganglia (number 3 above). They form a plexus around the superior and inferior mesenteric arteries where there are perivascular ganglia. Here they synapse and follow the arteries which supply the gut. The inhibition in tone to the colon from sympathetic input is believed to be mediated in part from alpha-2 adrenergic.
receptors.\textsuperscript{3} In one study in humans, alpha-2 agonists (clonidine) have been found to reduce colonic tone, whereas the alpha-2 antagonist (yohimbine) increased the tone.\textsuperscript{9} Alpha-1 agonist and beta-2 agonist did not affect tone.\textsuperscript{9} However, more research is needed. In other studies, beta-1, beta-2, and beta-3 adrenoreceptors detected on the human colon were tested in vitro. Agonists relaxed the colon.\textsuperscript{10}

The intrinsic innervation is called the enteric nervous system. The enteric nervous system has the unique ability to mediate reflex behavior independent of input from the brain or spinal cord.\textsuperscript{11} It does this through an abundance of different types of neurons within the walls of the intestinal tract. It has neuronal plexuses in the myenteric and submucosal/mucosal layers. The myenteric plexus regulates smooth muscle function. The submucosal plexus modulates mucosal ion transport and absorptive functions. There is substantial diversity within the enteric nervous system and all the modulators and transmitters of the central nervous system are found in the enteric nervous system.\textsuperscript{13} The amine and peptide neurotransmitters currently believed to be important are acetylcholine, opioids, norepinephrine, serotonin, somatostatin, cholecystokinin, substance P, vasoactive intestinal polypeptide, neuropeptide Y, and nitric oxide.\textsuperscript{12} Control of colonic motor function via the enteric nervous system remains poorly understood at this time.

**Colonic Function**

**Salvage, Metabolism, and Storage**

Even though the majority of our food undergoes digestion in the stomach and small intestine, the colon still has a major role in digestion. It processes certain starches and proteins, which are resistant to digestion and absorption in the foregut.\textsuperscript{13} The large quantity of heterogeneous bacteria in the colon is responsible for fermentation—the process by which these starches and proteins are broken down and energy is produced. There are more than 400 different species of bacteria, the majority of which are anaerobes.\textsuperscript{14} The bacteria feed upon mucous, residual proteins, and primarily complex carbohydrates that enter the colon.\textsuperscript{15} During fermentation of complex carbohydrates, short-chain fatty acids (SCFAs) are produced. More than 95\% of SCFAs are produced and absorbed within the colon.\textsuperscript{14,16} The principle ones are acetate, propionate, and butyrate. This process for the most part occurs in the right and proximal transverse colon. Protein residue, which reaches the colon is also fermented by anaerobic bacteria.\textsuperscript{17} Proteins are fermented in the left colon. Proteins are broken down into SCFAs, branched chain fatty acids, and ammonia, amines, phenols, and indoles. Part of these metabolites become a nitrogen source for bacterial growth.\textsuperscript{17,18} These products are either passed in feces or absorbed. Thus, the colon salvages and actively processes carbohydrates and proteins that reach the cecum. Dietary fat is probably not absorbed to any degree in the colon.\textsuperscript{14}

The colonic mucosa is unable to nourish itself from the bloodstream.\textsuperscript{19} Therefore, the nutrient requirements are met from the luminal contents. Butyrate (produced in the least amount) is important as the primary energy source for the colonocyte.\textsuperscript{13} Butyrate may also have a major role in cell proliferation and differentiation\textsuperscript{20,21} as well as being important in absorption of water and salt from the colon. Regarding the other SCFAs produced, propionate combines with the other 3 carbon compounds in the liver for gluconeogenesis. Acetate is the most abundantly produced SCFA. It is used by the liver to synthesize longer-chain fatty acids\textsuperscript{13} and as an energy source for muscle.\textsuperscript{15}

The proximal colon differs from the distal colon in many functions. Besides being derived from different embryologic origins, the proximal colon is more saccular and the distal more tubular.\textsuperscript{22} SCFAs are principally derived in the proximal colon and proteins degraded in the distal colon. When considering storage, the two parts also differ. The proximal colon acts as a reservoir and the distal colonic segments mainly act as a conduit.\textsuperscript{23} When confronted with large amounts of fluid, the fluid seems to move quickly into the transverse colon with the solid material catching up later.\textsuperscript{24,28} Even after right colon resection, the transverse colon can adapt to store colonic contents nearly as efficiently as the right colon.\textsuperscript{26} In addition, the haustral segmentation of the colon facilitates mixing, retention of luminal material, and formation of solid stool.\textsuperscript{4}

**Transport of Electrolytes**

The colon is extremely efficient at conserving sodium and water.\textsuperscript{4} Normally the colon is presented 1–2 L of water daily.\textsuperscript{15} It efficiently absorbs 90\% such that approximately 100–150 mL of fluid is eliminated in the stool. When challenged, it can increase the absorption to 5–6 L daily.\textsuperscript{27,28} Therefore, when the ileal flow of fluid and electrolytes exceeds the capacity of the colon, diarrhea will result.

Additionally, the colon is important in the recovery of salts. Under normal conditions, the colon absorbs sodium and chloride and secretes bicarbonate and potassium. Sodium is actively absorbed against a concentration and electrical gradient. This concept is extremely important for the colon’s ability to conserve sodium. The average concentration of sodium in the chyme which enters the colon is 130–140 mmol/L. Stool has approximately 40 mmol/L.\textsuperscript{17} As long as the luminal sodium content is more than 25 mmol/L, there is a linear relationship between luminal concentration and the amount of sodium absorbed.\textsuperscript{29} However, when the luminal concentration of sodium is less than 25 mmol/L, sodium is secreted.

Aldosterone is secreted by the adrenal gland in response to sodium depletion and dehydration. Aldosterone enhances fluid and sodium absorption in the colon. (This is in contrast to angiotensin, which also participates in fluid balance but via the small intestine.)\textsuperscript{30,31} SCFAs produced in the colon are the principle anions. They also stimulate sodium absorption.\textsuperscript{15}
Chloride is exchanged for bicarbonate, which is secreted into the lumen to neutralize organic acids that are produced.\textsuperscript{15} This occurs at the luminal border of the mucosal cells.\textsuperscript{17} Potassium movement, overall, is believed to be passive as a result of the active absorption of sodium. There is evidence that active potassium secretion occurs in the distal colon.\textsuperscript{31} This secretion combined with potassium in bacteria and colonic mucus in stool may explain the relatively high concentration of potassium, 50–90 mmol/L, in stool.\textsuperscript{32,33} Additionally, the colon secretes urea into the lumen. The urea is metabolized to ammonia. The majority is absorbed passively.\textsuperscript{17}

Similar to differences in salvage of food components that enter the colon, there exist qualitative differences in several ion transport processes between different segments of colon.\textsuperscript{34,35} Absorption of water and salt occurs primarily in the ascending and transverse colon.\textsuperscript{17,36} Active transport of sodium creates an osmotic gradient and the water passively follows. Additionally, there is a difference in the functional nature of the mucosal cells. The surface cells in the colon seem to be responsible for absorption whereas the crypt cells are involved with fluid secretion.\textsuperscript{30}

### Colonic Motility

#### Methodology for Determining Motility

Even though altered motility is thought to have a major role in some gastrointestinal disorders, it is surprising how little is known about colonic motility. This is because of the difficulty and inaccessibility of the proximal colon for direct study. Interestingly, stool frequency has been shown to correlate poorly with colorectal transit time.\textsuperscript{37} Early studies used barium but lacked the ability to give precise measurement of colonic motility.\textsuperscript{7}

**Marker**

Radiopaque markers orally ingested and followed sequentially through the intestinal tract via plain X-rays is one of the first methods used to actually measure transit time.\textsuperscript{28} This test is still used frequently to evaluate patients with severe constipation looking for slow transit through the colon. Variations in the protocol exist. Patients stop taking all laxatives 48 hours before the ingestion of the markers. One method calls for a capsule with 24 markers to be ingested and an X-ray obtained on day 5. This reflects the transit time of the entire gut. On day 5, 80% (17) of the markers should be expelled.

With sequential abdominal X-rays, markers can be localized to specific regions of the colon: right, left, and pelvis. One protocol asks patients to take one capsule with 20 markers and abdominal X-rays are taken every other day until all markers are passed. In an effort to decrease radiation exposure, patients ingest 84 markers on three successive days. (Some protocols call for markers to be different shapes on each of the 3 days.) Then one X-ray is obtained on day 4.\textsuperscript{29} Total colonic transit time is 30.7 (SD 3.0) hours for men and 38.3 (SD 2.9) hours for women.\textsuperscript{7}

**Scintigraphy**

Some centers favor colonic scintigraphy to study colonic transit. Patients refrain from taking laxatives or opiates 24 hours before the test. They remain on their normal diet throughout the study. Typically, a capsule coated with pH-sensitive polymer containing \textsuperscript{111}In-labeled radioisotope is ingested. The coating dissolves in the distal ileum and the radioactive material passes into the colon.\textsuperscript{25} Alternately, the patient will ingest the \textsuperscript{111}In-labeled material with water and serial images will be obtained with the gamma camera at specified hours (this varies but can be as frequent as twice daily or daily).\textsuperscript{40} Segmental transit is usually calculated for the right, left, and rectosigmoid regions of the colon. Results are expressed as the percentage of the total amount of isotope ingested in each segment or the geometric center of the isotope mass at any given time point.\textsuperscript{40} For clinical use, the total percentage retained compared with normal data seems to be the most convenient reporting system.

**Recording Techniques of Colonic Motility**

Most techniques that record colonic motility using some form of colonic manometry still remain in the researcher’s domain and have not been assimilated into the clinical armamentarium for the caregiver. Difficulty in accessing the colon is the obvious obstacle. However, significant information is being obtained from these types of studies in the research domain. Typically, a flexible catheter is placed into the colon. It is either a solid-state manometry catheter or a water-perfused system. It is argued that the water-perfused system increases the amount of fluid in the colon and may alter results. However, solid-state manometry catheters are fragile, expensive, and sensitive to corrosive damage from colonic irritants.\textsuperscript{41} The catheter is placed in the colon using one of several ways: it can be placed via the nasal-oral route and the position confirmed with fluoroscopy. The goal is to position the catheter so right colonic information can be obtained.

Alternately, it can be placed with a colonoscope using two methods. The catheter can be grasped with a biopsy forceps, which has been passed through the port of the colonoscope. It is then pulled along in a piggyback manner with the colonoscope. The scope is advanced via the anus and positioned in the colon usually as far as the transverse colon and then released. The colonoscope is carefully withdrawn in an effort to avoid displacing the catheter. The other method uses a guidewire threaded through a colonoscope. The guidewire remains as the scope is withdrawn. Then the catheter is threaded over the guidewire using fluoroscopic guidance. Initial studies asked patients to prep the entire colon. Because this is not physiologic, unprepared colons are more frequently used today (some still use enemas before colonoscopy). Retained stool can then hamper retrograde placement of the...
Peristalsis

Peristalsis is the waves of alternate contraction and relaxation of the muscles of the intestinal tube, which propels contents. Using transit studies, scintigraphy, and especially ambulatory colonic manometry, information has been learned about motor and pressure activity in humans that leads to peristalsis. Unfortunately, it is difficult to precisely define colonic contractions, pressure waves, and electrical events because no standard terminology or definitions exist. There is also no standardized way that the measurements are obtained. It is also difficult to study the colon because of the inaccessibility of the proximal portion. In contrast to the small intestine where contents are quickly propelled forward, the colon needs prolonged observation to be correctly studied.

In an effort to standardize observations, Bassotti and colleagues have proposed a classification system, which encompasses previous observations. Contractile events are divided as: 1) segmental contractions that are either single contractions or bursts of contractions, either rhythmic or arrhythmic contractions; 2) propagated contractions—low-amplitude propagated contraction (LAPC) (long spike bursts) and high-amplitude propagated contraction (HAPC) (migrating long spike bursts).

HAPCs have also been referred to in the past as large bowel peristalsis, giant migrating contractions, and migrating long spike bursts. HAPC is thought to be the equivalent of mass movement. The main function of HAPCs is to move large amounts of colonic contents toward the anus. They occur approximately five times daily. More than 95% of HAPCs propagate toward the anus (not retrograde). They usually occur upon awakening, during the day, and after meals. They are usually associated with abdominal sensation and defecatory stimulation (or defecation).

Less is known regarding LAPCs. They occur in all normal volunteers and are strongly related to meals and sleep–wake cycles. They may also be related to the passage of flatus. The mechanism regulating LAPCs and HAPCs remains unknown.

Single segmental contractions also have been referred to as electrical response activity, contractile electrical complex, and short-duration contractions. Bursts of segmental contractions have been referred to as long-duration contractions, continuous electrical response activity, and short spike bursts. The majority of the colonic motility is represented by segmental contractions. This allows slow transit and the opportunity for the luminal contents to maximally come in contact with the mucosal surface.

The colon in humans differs from the small intestines and colons of other mammals in that there is no cyclic motility. Combining what is seen with contrast fluoroscopy with what is known myoelectrically, haustra appear as ring-like segmenting contractions. They are static and partially occluding. With peristalsis, the haustra disappear as concentric waves of contraction spread distally along the now unsegmented colon. This seems to correspond to the descriptions of mass movement when contents in the right colon could be propelled distally into the left colon in seconds.

Cellular Basis for Motility

Cells important for movement in the colon include the circular muscle, longitudinal muscle, and interstitial cells of Cajal (ICC). Electrical activity is associated with mechanical activity. Electrical activity, which generates motility patterns in the human colon, is poorly understood. All electrical activity in the human colon is dependent on stimulation by stretch or chemical mediation. Critical volumes of distention are needed for propulsion. Fiber may augment this degree of stretch.

ICC are the pacemaker cells of the gut that have a central role in regulation of intestinal motility. These are mesenchymal cells, which form a three-dimensional network, placed between and in smooth muscle layers. They are also in close association with elements of the enteric nervous system. They are electrically active and create ion currents for pacemaker function. ICC in the submucosal layer (of the circular smooth muscle) initiate slow waves in the colon. There is also an additional pacemaker in the colon in the septa separating individual circular muscle bundles. It is difficult to determine the exact role of ICC in spreading the waves, but slow waves appear to spread along the long axis and around the circumference of the colon with the ICC representing a basal pathway. Slow waves of circular and longitudinal muscle cells are in phase, which indicates that a link must exist between these layers.

Characteristics of Colonic Motility in Health

Using 24-hour manometry, it has been found that the colon is continually active. There is a well-established circadian
rhythm with marked diminution of pressure activity at night. Immediately after waking, there is a threefold increase in colonic pressure activity. This may account for bowel patterns in some individuals who move their bowels after awakening in the morning. Colonic pressure activity also increases after meals, which in one study lasted for up to 2 hours after a meal. Propagating pressure waves (probably HAPCs) were seen intermittently throughout the day and especially after meals or after waking. There was also regional differences in pressure activity. During the day, the transverse to descending colon had more pressure activity than the rectosigmoid colon. Even though activity decreased at night, the rectosigmoid region was the most active. Women had less activity in the transverse/descending colon compared with men. One other factor from this study was that even though scintigraphic studies have shown retrograde movement of radiotransducers, the type of wave occurred infrequently and usually after a meal or during the morning waking response.

Stress can influence gut function. One study found that psychological stress induced prolonged propagated contractions without appreciable autonomic response. These contractions propagated across several areas of the colon. The motor activity persisted after the stressor ceased. Physical stress induced simultaneous contractions defined as pressure waves occurring simultaneously in several areas of the colon. The motor activity ceased immediately after the activity stopped. In another study, it was found that acute physical exercise increased LAPCs and HAPCs.

The right colon and transverse colon are major sites of storage of solid stool. Solid residue remains in the right colon for extended periods allowing for mixing. There is also considerable variability among individuals as far as right colon transit. After eating, the proximal colon has an immediate increase in tonic contraction. There is also increased tone in the distal colon, but this is less pronounced than the one on the right. Therefore, well before the ingested food reaches the colon, there is an increase in colonic motility and tone. This is known as the gastrocolic reflex. The mediator of this response is unknown and neither a stomach nor intact nervous system is required for it to occur. (CCK) is a well-known colonic stimulator increasing colonic motility. It has been postulated to be the mediator of this postprandial colonic activity. However, CCK antagonists do not block the gastrically response and CCK infusion that maximally stimulates the pancreatic exocrine secretion and gallbladder contraction has no effect on motor function or transit in a prepared colon.

Defecation and Colonic Sensation

The process of defecation seems to involve the entire colon. It has been shown to begin up to an hour before stool elimination—a preexpulsive phase. It is characterized by increased propagating and nonpropagating activity in the entire colon and is largely unperceived. This early component may result in stool contents being propelled into the distal colon and stimulating distal colonic afferent nerves. However, scintigraphic studies have also shown that the right colon can also be emptied during one episode of defecation. This could be associated with a total colonic propulsive activity that in some manner is associated with defecation. A second component begins approximately 15 minutes before stool expulsion. Propagating sequences during this time are associated with an increasing sensation of an urge to defecate. Even though several studies have shown that caudally propagating HAPCs occur in close temporal association with defecation, not all HAPCs end in defecation and defecation is not always preceded by HAPCs. However, it does appear that usually at least one very high amplitude HAPC occurs with the sensation of the urge to defecate.

Colonic sensation is complicated and poorly understood. The colon has no specialized sensory end organs. There are naked nerve endings within the wall and Pacinian corpuscles in the mesentery. Afferent fibers reach the central nervous system via sympathetic and parasympathetic pathways. Parasympathetic fibers convey nonconscious sensory information to the brainstem. Pain from abdominal viscera is almost exclusively conducted through the sympathetic afferents to the spinal cord via the dorsal root ganglia. The afferent neuron can mediate conscious perception of visceral events by synapsing in the dorsal portion of the spinal cord and then exiting back to the viscera, ascending within the spinohypothalamic or spinoetocellular tract toward the thalamus or reticular formation of the brain, or ascending directly to higher sensory centers of the brain.

Modulation of visceral sensation occurs through several methods. The first allows for enteroenteric reflexes that are mediated in the spinal cord to alter smooth muscle tone thereby increasing or decreasing the activation of the nerve endings in the gut or mesentery. Another method involves direct central modulation of pain. This can occur through the descending noradrenergic and serotonergic pathways from the brainstem. These projects in the dorsal horn and can modify the actual afferent input. This is the suspected mechanism by which wounded soldiers in the midst of battle will feel no pain. A further method explains “referred pain.” To initially understand this phenomena, it is recognized that somatic afferent nerves enter the same dorsal portion of the spinal cord as the visceral nerves. There is a wide overlap over multiple spinal lamina and some changes may occur in the ascending projection of the visceral stimuli. The dorsal horn may function as a “gate” controlling central transmission or changing excitability of the neuron. When the overlap of input appears more recognized by higher central brain forces from somatic input, referred pain may occur. The input is actually occurring in the visceral structure, but is perceived to be from the somatic structure. It is of note that when pain is referred it is usually to a structure that developed from the same embryonic dermatome. And lastly, visceral sensation...
can relay information via collaterals to the reticular formation and thalamus. This can induce changes in affect, appetite, pulse, and blood pressure through autonomic, hypothalamic, and limbic system connections.\textsuperscript{4,76}

Disturbances in Colonic Physiology

Physiology of Constipation

Constipation refers to stools that are infrequent or hard to pass (or both). Arbitrary definitions have been used. Individuals with constipation are an incredibly heterogeneous group. Distinct subtypes of constipation occur and require different treatment modalities, but even within these subtypes there can be wide variability in the clinical presentation and pathophysiological etiology. There may be dietary, pharmacologic, systemic, or local causes. Many people have constipation caused by dietary and lifestyle neglect. Two primary functions of the colon, solidifying chyme into stool and laxation, are interdependent on adequate dietary fiber. Dietary fiber “normalizes” large bowel function.\textsuperscript{77,78} Recommendations for adequate fiber intake ranges from 20 to 35 g per day for adults.\textsuperscript{79} Fiber is generally soluble or insoluble and seems to improve stool weight by different mechanisms. Oat bran, which is soluble, seems to increase stool weight by providing rapidly fermenting soluble fiber to the proximal colon. This allows for bacterial growth which is sustained until excretion. It seems that the increase in stool mass is from higher bacterial content and increased excretion of lipid and fat.\textsuperscript{80} Insoluble fiber such as wheat bran increases stool weight by increasing dietary fiber (undigested plant material) in the stool. Wheat bran also increases fat excretion, but not to the extent of oat bran.\textsuperscript{80} Interestingly, fiber intake in the United States is low. One explanation is that to achieve 15 g of fiber intake daily, 11 servings of refined grains and 5 servings of fruit and vegetables are needed for individuals consuming 1500–2000 kcal daily.\textsuperscript{77}

Additionally, constipation may be seen more frequently in sedentary people. In fact, abdominal cramps and diarrhea are reported more frequently in runners.\textsuperscript{81,82} Acute graded exercise has been shown to actually decrease phasic colonic motor activity. However, after the exercise, there was an increase in the number and amplitude of propagated pressure waves. It is believed that this post-exercise pattern may increase the propelling activity and propel stool.\textsuperscript{83}

Idiopathic slow transit constipation involves a measurable delayed movement of material through the colon. These patients are not helped (in fact may be made worse) with increased dietary fiber. They seem to have altered colonic motor response to eating and impaired or decreased HAPCs of the colon.\textsuperscript{50,64} This leads to reduced or absent colonic propulsive activity.\textsuperscript{83,84} Abnormalities in the neuronal network are suspected and recently a pan-colonic decrease in the ICC has been shown.\textsuperscript{56} As with other areas of colonic study, this one also needs much more investigation.

Irritable bowel syndrome (IBS) can manifest with multiple forms. It usually is characterized as altered bowel habits and pain directly related to the altered bowel habits. In one form, constipation can be the predominant feature. This may encompass about 30% of the IBS population and traditionally overwhelmingly affects women. This group of patients can show an overlap with those having slow transit constipation, but may have a normal transit study.\textsuperscript{85} Pharmaceutical companies have targeted drugs that affect metabolism of serotonin, which seems to be involved in the regulation of motility, sensitivity, and intestinal secretions. The specific 5-hydroxytryptamine (5-HT)\textsuperscript{4} receptor is involved in intrinsic sensory reflexes within the gut. Tegaserod is a 5-HT\textsuperscript{4} agonist that has been approved by the Food and Drug Administration (FDA) (July 2002) for treatment of this group of patients.\textsuperscript{87} Additionally, cholecystokinin-1 antagonists are in trials for treatment of patients with constipation-predominant IBS.\textsuperscript{87}

Obstructed Defecation

Obstructed defecation usually results from abnormalities in pelvic function versus colonic function. Typically this problem is associated with failure of the puborectalis to relax with defecation, rectocele, perineal descent, or other pelvic- and rectal-associated issues. Failure of the rectum to evacuate may lead to marker studies which also show marker collection in the left colon.\textsuperscript{88} This may also be associated with colonic total inertia.\textsuperscript{89}

A colonic source, which is a variant in obstructed defecation, is a sigmoidocele. Although rare, the sigmoid is seen to migrate into the pelvis with defecation and obstruct evacuation of stool. This form can be relieved and treated with a sigmoid resection, but the clinician should be aware of other pelvic floor abnormalities.

Ogilvie’s Syndrome

Ogilvie’s syndrome was described initially in 1948. It is also known as acute colonic pseudoobstruction. The pathophysiology is not clearly understood. Based on evidence from pharmacologic studies, it seems that Ogilvie’s original hypothesis is as correct as the current facts; namely, there seems to be an imbalance of autonomic innervation to the gut. The parasympathetic nerves, which are responsible for stimulating gut motility, have decreased function or input and the sympathetic nerves, which are inhibitory, increase their input.\textsuperscript{90} Because of the law of Laplace, the cecum can be the site of extreme dilatation (it requires the smallest amount of pressure to increase in size and therefore increase the wall tension). Treatment has focused on ruling out a distal obstruction with a Gastrografin enema and if needed colonoscopic decompression. However, pharmacologic treatment with neostigmine has been successful.\textsuperscript{95} This drug is a cholinesterase inhibitor that allows more available acetylcholine for neurotransmission in the parasympathetic system (excitatory) to promote contractility.\textsuperscript{92}
Irritable Bowel Syndrome

As stated above, IBS is characterized by altered bowel habits associated with pain. Besides the constipation-predominant type described above, there can be a diarrhea-predominant type and a mixed type. The pathophysiology of IBS has received extensive study, but it remains unclear. Abnormal motility, visceral hypersensitivity, inflammation, abnormalities in extrinsic autonomic innervation, abnormal brain–gut interaction, and the role of psychosocial factors have been investigated. If IBS is found in men it tends to be more diarrhea-predominant type. Treatment is based on the nature and severity of symptoms. Education, reassurance, and dietary modification (elimination of foods that aggravate the problem) are the first steps. For those who do not respond, medication is considered. Antispasmodics (anticholinergic) medication is considered for those with pain and bloating that is especially aggravated by meals. Usually, antispasmodics and anticholinergic agents are considered on an as-needed basis. Low-dose tricyclic antidepressants may be considered when the pain is more constant and perhaps disabling.

Considering specific types, no good pharmacologic research is available for the mixed-type IBS patients. However, for the diarrhea prone, 5-HT3 antagonists have been found to be effective. Alosetron was initially FDA approved (March 2000) only to be withdrawn after some patients suffered ischemic colitis and even death. In June 2002, it was reapproved with restrictions that require the prescriber to demonstrate educational understanding regarding the drug. Additional drugs are also undergoing trials.

Implications of Colonic Physiology for the Surgeon

Why is colonic physiology important for the surgeon? Recognizing the innervation and differences in embryologic development may be important in colon resections when considering nerve preservation, blood flow, and resection margins. Colonic motility is poorly understood. However, as knowledge is gained through research, the surgeon will be asked to evaluate and use pharmaceutical products to reduce ileus and treat other conditions.

Resection of all or a portion of the colon can have profound ramifications for the patient. It is the surgeon’s responsibility to understand the physiologic possibilities, recognize, and manage the outcome. For instance, this may be important for patients with a new ileostomy who may need counseling regarding fluids and increased salt intake to compensate for the colon, which has been resected.

Disorders or colonic motility are numerous in the human species. Surgeons will be consulted regarding surgical intervention. Knowledge of basic physiology will prepare the surgeon to make decisions regarding which patients are appropriate for medical treatment and the treatments available. Surgical intervention will then be reserved for appropriate patients.

In the colon, many metabolic processes can be influenced by food components. Prebiotics are “non-digestible food ingredients that beneficially affect the host by selectively stimulating the growth and/or activity of one or a limited number of bacteria in the colon, that can improve the host health.” The most common area has been stimulation of the growth of lactic acid–producing bacteria. This growth changes the colonic environment and may reduce the ability of carcinogens to form or lead to cancer. Probiotics are “a live microbial feed supplement which beneficially affects the host by improving its intestinal microfloral balance.” With increasing resistant bacteria in our hospitals, the World Heath Organization has recommended trying to combat this problem by using microbial interference therapy or nonpathogens to eliminate pathogens. Work is underway with probiotics in this manner in an effort to reduce potentially pathogenic microorganisms. Currently, probiotics may be used in cases of disturbed microbial balance, such as antibiotic-associated diarrhea, to lessen the risk and duration. In the future, pre- and probiotics may become important supplements administered to patients to promote health and prevent complications from illness.

Conclusion

In conclusion, the colon is a mysterious organ. It salvages water and electrolytes, which have passed through the small intestine. It produces SCFAs, which nourish its mucosa and provide substrate for energy. It propels its contents slowly toward the anus, continuously mixing them and exposing them to the luminal surface. Its ultimate task is to store stool until it is socially acceptable to eliminate.

References


The ASCRS Textbook of Colon and Rectal Surgery
2007, XXIV, 810 p. 384 illus., 65 illus. in color., Hardcover