Pathogenesis of Slow Transit and Pelvic Floor Dysfunction: From Bench to Bedside

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The colon and anorectum function together to provide intraluminal mixing, absorption of water, electrolytes, and short chain fatty acids, dehydration of fecal material, storage, and ultimately, elimination in a socially appropriate manner. Normal function and continence require accommodation of the colon and rectum to the entry of fecal materials, which includes receptive relaxation, perception, and discrimination of rectal contents, and voluntary and reflex motor function of the anorectum. Defecation, on the other hand, requires the reflex relaxation of the internal anal sphincter, voluntary and reflexive relaxation of the external anal sphincters and pelvic floor structures, and adequate rectosigmoid tone to allow funneling of contents through the anal canal. The sensation of urgency with rectal filling, and the motivation and prior learning of the appropriate responses are also required. Continence and defecation, therefore, involve complex sensory, structural, and motor mechanisms that involve both the colon and pelvic floor. These mechanisms and their relative importance to the pathogenesis of slow-transit constipation and pelvic floor dysfunction will be reviewed.


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Anatomically, the colon is constructed of four primary layers: the serosa, tunica muscularis, submucosa, and mucosa. The submucosa consists of a layer of dense connective tissues that contains the submucosal plexus of the enteric nervous system, blood supply, and lymphatic vessels that supply the mucosa. The mucosa, which lines the lumen of the bowel, contains epithelial cells that act as sensory transducers between the lumen and the enteric nerve plexuses within the bowel wall. Endocrine cells within the mucosa produce and release gut hormones that mediate motility, secretion, and absorption. Gastrointestinal
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motor function and transit are largely governed by the tunica muscularis, which consists of two relatively thick layers of smooth muscle fibers that are aligned circumferentially and longitudinally along the digestive tract. Coordinated contraction and relaxation of the circular and longitudinal layers create the motor patterns that control the movement of contents within the colon. These complex motor patterns are mediated by the myenteric neural plexus (enteric nervous system [ENS]) and the interstitial cells of Cajal (ICC), which are located between and around the inner circular muscle and the outer longitudinal muscle layers (Figure 1).

Enteric Nervous System
The ENS contains two neural networks embedded within the walls of the gastrointestinal tract. The myenteric plexus is located between the longitudinal and circular muscle layers and plays a principal role in the control of motor function, including peristalsis and the resulting colonic transit. The submucous plexus is located between the circular muscle layer and the mucosa and is involved in local reflex control of epithelial cell function, blood flow, secretion, and absorption. Sensory neurons in the ENS function to sense and transmit information from the lumen to the submucous and myenteric plexuses. Sensory information is transmitted from specialized receptors that respond to mechanical, thermal, osmotic, and chemical stimuli. In response to the activation of sensory neurons, motor neurons within the ENS initiate appropriate gastrointestinal motor and secretory functions. Within the ENS, interneurons integrate a variety of information from sensory neurons that subsequently act via hard-wired programs to activate enteric motor neurons and stimulate coordinated motility and secretion.

In the periphery, the ENS acts directly on effector systems, including smooth muscles, secretory endothelium, endocrine cells, and the vasculature. The central nervous system (CNS) modulates the ENS through both parasympathetic and sympathetic pathways. Afferent parasympathetic activity is processed through the nodose ganglia, whereas afferent sympathetic activity—particularly nociceptive activity—acts through the dorsal root ganglia. Local spinal reflexes can mediate direct effects on the ENS whereas long reflex pathways through vagovagal pathways can influence both systems. The ENS, functioning in a semiautonomous manner, can act locally on effector systems to influence both secretion and gastrointestinal (GI) motility. Intrinsic primary afferents from the effector systems (mucosa) also act on the ENS via local reflex pathways. The central autonomic neural network integrates communications between the CNS and the ENS in the periphery (Figure 2). These effector pathways are modulated via a wide array of neurotransmitters and neuromodulators, including serotonin (5-hydroxytryptamine, 5-HT), norepinephrine, dopamine, acetylcholine, and calcitonin gene-related peptide (CGRP) acting through intermediate cells or interneurons. Major mediators controlling GI motility are acetylcholine and substance P for excitation and nitric oxide (NO) and vasoactive intestinal peptide (VIP) for inhibition. The relevant receptors are muscarinic for acetylcholine, neurokinin (especially NK1 receptor) for substance P, VIP1 and VIP2 for vasoactive intestinal peptide (VIP), and cytosolic guanylate cyclase and other intracellular response systems for NO and CO. Investigators have begun to identify the distribution, chemical coding, and projections of enteric neurons in the human colon. Wattchow and colleagues reported that circular muscle motor

![Diagram of the gastrointestinal tract with layers labeled: Mucosa, EC Cells, Submucosa, Circular muscle, Myenteric plexus, Longitudinal muscle, Afferent nerves.](image)

Figure 1. The 4 primary layers of the colon include: serosa, tunica muscularis, submucosa, and mucosa. The mucosa contains endocrine cells, which produce and release gut hormones that mediate motility, secretion, and absorption. Gastrointestinal (GI) motor function and transit are largely governed by the tunica muscularis, which consists of two layers of smooth muscle fibers that are aligned circumferentially and longitudinally along the digestive tract. Coordinated contraction and relaxation of the circular and longitudinal layers create the motor patterns that control the movement of contents within the colon. The enteric nervous system and the interstitial cells of Cajal mediate these complex motor patterns. They are located between and around the inner circular muscle and the outer longitudinal muscle layers.
neurons and myenteric interneurons display distinct projections and polarity in the human colon. They also demonstrated two distinct classes of colonic motor neurons: one that is excitatory (ChAT-immunoreactive) and mainly projects orally and the other that is inhibitory (NOS active) and mainly projects anally. This configuration of excitatory and inhibitory pathways may facilitate the peristaltic reflex in the colon. Further exploration of neurochemical coding and projections are needed to evaluate changes in enteric neurons in patients with slow-transit constipation.

Neurotransmitters from enteric nerves might also interact with pacemaking ICC networks during peristaltic reflexes, inhibiting slow wave amplitude distal to and enhancing it proximal to the site activation of the reflex. This pattern would enhance proximal contractions and inhibit distal contractions, thus increasing the probability of action potentials on slow waves and contractions, promoting peristalsis. However, no empirical data are currently available to support this hypothesis.

**Interstitial Cells of Cajal**
Spontaneous electrical and mechanical activities are seen in smooth muscle cells of the gut in the absence of direct stimulation. These activities were originally thought to originate from the smooth muscle cells directly. However, recent work has demonstrated that activity in smooth muscles arises from ICC. ICC are anatomically associated with the myenteric plexus, submucosal plexus, and the circular muscle layer of the colon and are located in close proximity to the enteric nerves. The ICC form extensive networks of electrically coupled cells that are widely distributed within the submucosal (ICC-SM), intramuscular (ICC-IM, ICC-DMP) and intermuscular layers (ICC-MY) of the GI tract from the esophagus to the internal anal sphincter. ICC are also located in close proximity to enteric nerves.

ICC are essential for normal neuromuscular function and GI transit. Recent studies have shown that the ICC play an important role in neurotransmission within the gut. They are largely responsible for myogenic electrical slow waves activity (electrical control activity; ECA) from the distal stomach, intestine, and colon. Investigators have shown that ICC located near the myenteric plexus generate pacemaker potentials that are conducted passively into the adjacent muscle layers where they produce rhythmical membrane potential changes. Hirst and colleagues have recently shown that nerve terminals form synapses with ICC and that the ICC relay information directly to the smooth muscle cells. They concluded that ICC distributed among the smooth muscle cells of the gut are the targets of transmitters released by intrinsic enteric excitatory and inhibitory nerve terminals.

The ICC regulate the ECA of the gut smooth muscle. In the colon, electrical activity consists of an oscillatory ECA interspersed with spike potentials that control smooth muscle contractions. The ICC facilitate the conduction of electrical currents within and along the colon, and they mediate neural signaling between enteric nerves and muscles. Several studies have demonstrated that the ICC are required for

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**Figure 2.** The central autonomic neural network integrates communication between the central nervous system (CNS) and the enteric nervous system (ENS) in the periphery. The ENS acts locally on effector systems, including smooth muscles, secretory endothelium, endocrine cells, and the vasculature. The CNS modulates the ENS through both parasympathetic and sympathetic pathways. Afferent parasympathetic activity is processed through the nodose ganglia, while afferent sympathetic activity acts through the dorsal root ganglia. Local spinal reflexes can mediate direct effects on the ENS while long reflex pathways through vagovagal pathways can influence both systems. The CNS, functioning in a semiautonomous manner, can act locally on effector systems to influence both secretion and gastrointestinal motility. Intrinsic primary afferents from the effector systems (mucosa) also act on the ENS via local reflex pathways. Adapted with permission from Goyal RK et al. N Engl J Med. 1996;334:1106-1115.
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normal neurotransmission and that they initiate slow waves in most areas of the GI tract. Other studies have shown the cyclic changes in intracellular calcium concentration recorded in ICC to be similar to the intrinsic electrical slow wave. The electrical slow wave activity determines the characteristic frequency of phasic contractions of the colon. Slow waves generated by the interaction of ICC and the ENS are also intricately involved in the determination of the direction and velocity of propagation of peristaltic activity.

ICC have been reported to be decreased in the left colon of patients with slow-transit constipation. Lyford and colleagues evaluated the distribution of ICC within the human colon to determine if ICC are decreased in slow-transit constipation compared to normal controls. They studied the cecum, ascending, transverse, and sigmoid colons from patients with slow-transit constipation and from controls. ICC were located within both the longitudinal and circular muscle layers. Two networks of ICC were identified, one in the myenteric plexus region and another, less defined network, in the submucosal border. Cecum, ascending colon, transverse colon, and sigmoid colon displayed similar ICC volumes. ICC volume was significantly lower in the slow-transit constipation patients across all colonic regions. They concluded that ICC distribution is relatively uniform throughout the human colon and that decreased ICC volume is pan-colonic in idiopathic slow-transit constipation.

Additional support for altered ICC in the colon of patients with constipation was reported by Yu and coworkers. They evaluated myenteric ganglion cells and ICC from patients with chronic idiopathic constipation and found that the number of ganglion cells was significantly reduced in the ascending and descending colon of the constipated group compared to controls. Ganglion cells were found to be similar in the sigmoid colon. Pan-colonic decreases were noted for ICC in the constipated group. A similar trend was noted for the submucosal border, circular muscle, and longitudinal muscle. However, no significant differences between the two groups were found in the myenteric plexus. These investigators concluded that a quantitative decrease in ganglion cells and ICC may be implicated in chronic idiopathic constipation.

**Colonic Peristalsis**

Peristalsis, the primary propulsive motor pattern in the colon, is orchestrated by neuronal excitation and inhibition of contractile activity that is mediated by the ENS and the ICC. The electrical slow waves generated by the ICC propagate into the mucoculture, thus initiating rhythmic contractile activity upon excitation by the enteric nerves. The ICC have recently been shown to be responsible for the rhythmic inward currents, which have not been identified in smooth muscle cells. Intrinsic and induced slow wave activity from the ICC exerts control of the progression of peristaltic motor patterns in the gut. In conjunction with the ICC, the ENS exerts control over motor function of the intestinal tract utilizing a number of neurotransmitters and neuromodulators.

Accumulating data support a role for 5-HT receptors in the mediation of reflexes controlling GI motility and secretion. The primary source of 5-HT in the gut is the EC cells located in the mucosal crypts. When the mucosa is stimulated mechanically or chemically, the EC cells secrete 5-HT and other peptides, which act as paracrine mediators that excite afferent nerves from the mucosa, thus initiating the peristaltic reflex. 5-HT in the gut is most often associated with increased gastrointestinal motility, but inhibitory actions may result in certain tissues, depending on the type and density of the local 5-HT receptors.

Peristaltic reflexes are initiated by local mucosal stimulation or luminal distention and the subsequent release of 5-HT from EC cells within mucosal crypts. Recent investigations have suggested that 5-HT acts on intrinsic, primary afferent neurons that synapse in the myenteric plexus with ascending inhibitory and descending excitatory interneurons. Grider and colleagues have demonstrated the involvement of CGRP in this local reflex and have proposed that the primary intrinsic afferent involved is a CGRP neuron. They concluded that 5-HT released by mucosal stimulation initiates the

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peristaltic reflex by activating 5-HT$_4$/5-HT$_3$ receptors on sensory CGRP-containing neurons. These investigators also demonstrated that the specific 5-HT$_4$ agonist, tegaserod, stimulated ascending contraction and descending relaxation that was significantly inhibited by a CGRP antagonist. Tegaserod, a partial 5-HT$_4$ agonist, has been shown to be efficacious for the treatment of irritable bowel syndrome (IBS) symptoms in women whose primary bowel symptom is constipation. These effects may, in part, be explained by the acceleration of small bowel and colonic transit.  

Further support for the hypothesis that slowed colonic transit and symptoms of constipation may result from alterations in the peristaltic reflex has been reported. Prucalopride, a 5-HT$_4$ agonist, stimulates colonic peristalsis and high-amplitude propagating contractions in the colon. A preliminary clinical study reported that prucalopride improved symptoms in constipated patients with both slow and normal transit, and that it improved colon transit in patients with slow-transit constipation.  

Additional studies have demonstrated a role for the 5-HT$_3$ receptors in the gastrocolonic response and the peristaltic reflex. Bjornsson and colleagues compared descending colonic tone responses to antral distension, duodenal lipid perfusion, and colonic distension after double-blind placebo or granisetron in healthy volunteers and patients with slow-transit constipation. Antral distension and duodenal lipid perfusion increased colon tone in healthy volunteers, but failed to increase tone in constipated patients. The 5-HT$_3$ receptor antagonist, granisetron, reduced responses to antral distension and lipids in volunteers and to lipids in constipated patients. Additionally, the ascending contraction of the peristaltic reflex was blunted in constipated patients. They concluded that altered gastrocolonic responses and ascending contractions of the peristaltic reflex were impaired with slow-transit constipation and that loss of both 5-HT$_3$-dependent and -independent function was involved in the abnormalities of neural reflex modulation of colonic motor function.  

Altered motility associated with excitatory and inhibitory neurotransmission that involve inhibitory neurotransmitters such as NO, 5'-adenosine triphosphate (ATP), and VIP has also been proposed to be involved in slow-transit constipation. Experiments that antagonized or reduced the effect of putative inhibitory mediators have suggested that NO and ATP release was altered in patients with constipation. Increased NO-mediated relaxation was identified in patients with constipation compared to controls, supporting the hypothesis that excessive NO production may be involved in the pathophysiological mechanism of constipation. These findings suggest that the peristaltic reflex may be disrupted in patients with slow-transit constipation.  

### Colonic Function Motility  

Colonic motor activity promotes the mixing of intraluminal contents, absorption, and facilitates the control of distal propulsion of colonic contents. Colonic transit time is much slower than that found in the more proximal portions of the alimentary canal and may range from hours to days. Colonic transit, therefore, comprises the largest proportion of whole-gut transit time. The majority of contractile activity in the colon is nonpropagating segmenting contractions that move colonic materials over relatively short distances in a to-and-fro mixing manner, which results in enhanced absorption of fluids, electrolytes, and short chain fatty acids. Segmenting motor patterns slow colonic transit to facilitate absorptive functions and excessive segmenting contractions can contribute to slow-transit constipation. However, the net propulsion of intraluminal contents is largely determined by segmental pressure gradients within each colonic segment.  

Contractile activity within the colon may also be peristaltic, thus propelling contents distally. Two types of propagating pressure waves or peristaltic contractions have been described in vivo. These contractile patterns have been defined as low-amplitude propagating contractions (LAPCs; average amplitude < 50 mmHg) or high-amplitude propagating contractions (HAPCs; average amplitude > 75 mm Hg). LAPCs are thought to move contents over relatively short distances, orally in the ascending colon or aborally in the transverse and descending colon. They are also thought to be primarily associated with the movement of liquids and gas within the colon. HAPCs, or “mass movements,” propel colonic contents over relatively longer distances and are often associated with the gastrocolonic response and defecation. An excessive proportion of nonpropagating segmenting activity and a reduction in HAPCs has been described in patients with constipation; the reverse has been reported in patients with chronic diarrhea. HAPCs may be initiated by luminal distention or by contact stimulants and have been associated with defecation. In fact, contractile activity in response to contact stimulants has been suggested as a useful tool for the evaluation of neuromuscular function in the colon.  

Prolonged, ambulatory monitoring of colonic motor function has yielded important information regarding...
normal and abnormal colonic motility under more physiologic conditions. These techniques have been used to characterize the temporal frequency and circadian rhythms of contractile patterns in the colon in healthy patients as well as those with constipation and diarrhea. Low-amplitude propagating and nonpropagating segmenting contractions occur most frequently. HAPCs occur less frequently (approximately 6 times per 24-hour cycle) and are associated with the movement of colonic contents over longer distances.

Altered rectal compliance may result in enhanced (hypertonic rectum) or diminished (hypotonic rectum or megarectum) rectal tone, either of which may contribute to disturbed bowel function and the loss of continence.

These motor patterns have been shown to be associated with food ingestion and defecation. In the human colon, both LAPCs and HAPCs are related to sleep–wake cycles and meal ingestion. Contractile activity within the colon is strongly inhibited during sleep and increases significantly upon morning awakening.

Most HAPCs appear to terminate at the rectosigmoid junction with smaller pressure changes sometimes noted in the rectum. Although no anatomical sphincter has been identified in this region, smooth muscle tone, sigmoid angulations, and pressure gradients may create a functional sphincter or mechanical break to slow transit and coordinate rectosigmoid sphincter or mechanical break to slow tone, sigmoid angulations, and pressure changes sometimes noted in this region, smooth muscle anatomical sphincter has been identified in the rectum. Although no peripheral neuropathies or surgical interventions most often result in fecal incontinence. Loss of rectal sensation is also a poor prognostic indicator for biofeedback treatment of incontinence.

Rectal compliance is the ability of the rectum to adapt to increasing volumes of distention (increase in pressure per unit volume). The rectum is larger in diameter and much more compliant than the adjacent sigmoid colon due to anatomical changes in the circular and longitudinal muscle layers. Anatomic and physiologic differences between the rectum and sigmoid colon may provide an important pressure gradient to facilitate the net movement of contents in the caudal direction from the sigmoid to the rectum and prevent reflux of rectal contents into the pelvic colon during straining.

Altered rectal compliance may result in enhanced (hypertonic rectum) or diminished (hypotonic rectum or megarectum) rectal tone, either of which may contribute to disturbed bowel function and the loss of continence. Decreased compliance and lowered sensory thresholds result in frequent defecation, extreme urgency, rapid funnelling of rectal contents via increased rectal tone, and incontinence. Rectal compliance may be compromised secondary to inflammatory diseases, rectal ischemia, colorectal or
ileorectal anastomosis, abdominoperineal pull-through, and other surgical interventions. Elevated sensory thresholds associated with increased compliance (hypotonic rectum), as seen in conditions of megarectum, result in the loss of the sense of urgency, fecal impaction, and overflow incontinence.

Pelvic Floor Function and Dysfunction

Pelvic Floor Muscles and Innervation

The pelvic floor consists of striated muscles that form a support structure through which the viscera pass. The pelvic floor musculature consists of a paired set of levator ani muscles, which are further subdivided into four muscle groups by their respective attachments to the pubic bone. The attachments stretch from the pubis along the ischial spine and are called the pubococcygeus, ilioococcygeus, and ischiococcygeus. The pubococcygeus also includes the puborectalis muscle. The perineal body lies between the urogenital viscera and the anal canal.

The levator ani muscles are innervated by the fourth sacral nerve; the innervation of the puborectalis muscle is less clear. Some studies have suggested that the puborectalis is innervated only by the sacral nerve, whereas others have suggested additional innervation from the pudendal nerve. Whether the origin of the puborectalis is with the pelvic floor muscles or with the external anal sphincter is also unclear. Manometric and electrophysiologic studies of the external anal sphincter and puborectalis muscle suggest that the muscle groups are coordinated during cough and strain.

Anal Canal Muscles and Innervation

The anal canal is a specialized, sphincteric segment that is approximately 2 to 7 cm in length and composed of both smooth and striated muscle. The anal canal is generally closed by tonic contraction of the smooth muscle internal anal sphincter (IAS) and the partially contracted striated external anal sphincters (EAS). In healthy controls, tonic pressure at rest in the high-pressure zone of the anal canal ranges from approximately 30 mm Hg to 80 mm Hg and provides a passive barrier to the movement of rectal contents. The evaluation of neuromuscular function in the rectum and anal canal provides valuable diagnostic information for the patient with spastic constipation, delayed outlet constipation, or anal incontinence.

Internal Anal Sphincter

The smooth muscle IAS receives sympathetic innervation via the hypogastric and pelvic plexus. Parasympathetic innervation is from S1, S2, and S3 via the pelvic plexus. Most data suggest that sympathetic innervation is excitatory but there has been some conflicting data regarding the parasympathetic effects. Yamanouchi and colleagues recently evaluated the lumbar sympathetic neuromodulation of the rectoanal inhibitory reflex. They found that the extrinsic lumbar inhibitory outflow caused marked inhibition of the rectoanal reflex via the lumbar colonic nerves. Recent data support the innervation of the IAS from nonadrenergic, noncholinergic (NANC) pelvic nerves. Spinal anesthesia decreases rectal tone by 50% and the decreased resting tone seen in diabetic patients may be due to an autonomic neuropathy. The internal anal sphincter contributes 55% to the anal resting pressure (myogenic activity contributes 10%; 45% is due to the sympathetic innervation). The remainder of the resting tone is from the hemorrhoidal plexus (15%) and the external anal sphincter (30%). The internal anal sphincter has slow waves occurring 6–20 times each minute. Ultraslow waves have been reported and generally occur less than 3 times a minute, but often approximate 1 cycle per minute. Ultraslow waves are associated with higher resting pressures, hemorrhoids, and anal fissures. This pattern...
may also be associated with symptoms of straining and difficult defecation.

Both extrinsic and intrinsic innervations control the complex sensory/motor functions of the IAS during continence and defecation. Sympathetic afferent nerves arise from S2 and S3 of the spinal cord. Additionally, the myenteric plexus provides intrinsic control via local reflex mechanisms. Small volumes of rectal distention result in a brief contraction of the EAS, followed by the reflex inhibition of IAS resting tone (Figure 3). The reduction in resting anal tone is thought to facilitate normal defecation by reducing the pressure gradient when fecal material enters the rectum. This reflex has been labeled the Rectoanal Inhibitory Reflex (RAIR). Increasing volumes of distention result in a progressively larger RAIR of the IAS (Figure 4). Reflex inhibition is relatively brief in healthy controls (< 30 seconds), but may be prolonged in patients with fecal incontinence. The RAIR is dependent on an intact intrinsic nervous system and is absent in patients with Hirschsprung’s disease (congenital aganglionosis), Chagas disease, and following rectal mucosal excision. Low rectal anastomosis often leads to a loss of the RAIR, but the reflex has been reported to return post-anastomosis. These observations suggest that the afferent receptors are located both in the anorectal epithelium and rectal wall, but probably also in the pelvic floor. However, the RAIR is largely controlled intramurally and involves myenteric plexus neurons that are noncholinergic and nonadrenergic.

Local pathways also influence rectoanal reflexes. Serotonin, which exerts significant influences throughout the GI tract, appears to influence rectal and anal reflexes. In a guinea pig model, rectoanal reflexes and the rectoanal inhibitory reflex were both enhanced by the 5-HT4 receptor agonist, mosapride. A specific 5-HT4 receptor antagonist blocked the enhancement of these reflexes. The authors suggested that their findings support a role of 5-HT4 receptors in the mediation of rectorectal reflexes and the RAIR.

External anal sphincter

The external anal sphincters are composed of striated, skeletal muscle innervated primarily by the perineal and inferior hemorrhoidal branches of the pudendal nerve (S2, S3, S4). The EAS has been considered to consist of three relatively discrete muscle bundles: the deep bundle, the superficial bundle, and the subcutaneous bundle. The deep and superficial bundles surround the internal anal sphincter, whereas the subcutaneous bundle lies caudal to the IAS and creates the distal anal canal. However, more recent data suggest that the EAS may be one continuous muscle group that is functionally associated with the puborectalis muscle. The puborectalis muscle interdigitates with the deep bundles of the EAS and maintains the anorectal angle at approximately 90’ to 105’ at rest. The angle is voluntarily relaxed (becomes more obtuse) to approximately 120’ to 130’ during normal defecation. Tonic contraction of the EAS is also inhibited with higher levels of rectal distention.

Pelvic Floor Dyssynergia

Paradoxical contraction of the puborectalis muscle and anal sphincters during attempted defecation (Figure 5) may result in outlet obstruction, dyschezia (straining to start or finish a bowel movement), and constipation. Pelvic floor dyssynergia (PFD) is a common clinical problem and may affect up to 50% of patients.
presenting with chronic constipation. Dyssynergia may overlap with slow-transit constipation in many of these patients. The primary pathophysiologic feature of PFD is the loss of rectoanal coordination during defecation. The onset of PFD may result from various causes including organic neuromuscular dysfunction and painful defecation. Pelvic floor spasticity has been shown to correlate with the presence of detrusor-external sphincter dyssynergia and with more severe spinal cord disease in patients with multiple sclerosis. PFD has also been reported in up to 60% of patients with early or late stage Parkinson’s disease. Although the mechanism is not known, PFD is a common finding in elderly patients with chronic constipation and excessive straining with defecation and is a poor prognostic indicator for standard medical treatments.

Habitual alterations in defecatory function also can be learned by intentional or unintentional aberrant behaviors. It has been suggested that paradoxical contractions of the pelvic floor can be conditioned by attempted defecation during periods associated with active hemorrhoids or anal trauma with pain. Unfortunately, few empirical data are available to support these contentions. However, the voluntary suppression of normal defecation can result in altered colorectal function. Klauser and colleagues paid healthy individuals to voluntarily suppress defecation and measured the effects on colonic transit. They concluded that behavioral control over defecation can induce changes in colonic function such as those seen in constipation, and that functional anorectal outlet obstruction may (probably by reflex mediation) affect right colon transit. Therefore, PFD and aberrant behaviors

Main Points

- Complex motor patterns control movement of material within the colon. These patterns are mediated by the enteric nervous system (ENS) and the interstitial cells of Cajal (ICC).
- Communication between the central nervous system and the ENS in the periphery are integrated by the central autonomic neural network. The ENS acts locally on effector systems; the CNS modulates the ENS through sympathetic and parasympathetic pathways.
- There are two distinct types of motor neurons in the colon: excitatory and inhibitory. The configuration of these two motor pathways may facilitate the peristaltic reflex in the colon.
- Normal neuromuscular function and gastrointestinal (GI) transit depend greatly on the ICC. The ICC near the myenteric plexus produce pacemaker potential, that, once conducted into the adjacent muscle, produces rhythmical membrane potential changes. The ICC in the smooth muscle cells of the gut are the targets of transmitters released by intrinsic enteric excitatory and inhibitory nerve terminals.
- There are 2 types of contractile pattern activity in the colon: low-amplitude propagating contractions, which are thought to move contents over short distances and may be linked to liquid and gas movement within the colon, and high-amplitude propagating contractions, which are thought to move contents over long distances and are frequently associated with the gastrocolonic response and defecation.
- Internal anal sphincter (IAS) innervation derives from nonadrenergic, noncholinergic pelvic nerves. Extrinsic and intrinsic innervations control the complex sensory/motor functions of the IAS during continence and defecation.
- Pelvic floor dyssynergia may affect almost 50% of patients with chronic constipation; onset may result from a number of causes. It is frequently found in elderly patients with chronic constipation and excessive straining with defecation and is a poor prognostic indicator for standard medical therapy.
can slow proximal colonic transit and present as slow-transit constipation.

Although PFD has generally been reported in association with symptoms of straining and the feeling of incomplete evacuation, not all data support the current diagnostic methods. Schouten and colleagues found that contraction of the puborectalis muscle during straining is not an abnormality found exclusively in patients with constipation and/or obstructed defecation. They also reported that the three tests most commonly used for the diagnosis of anismus showed extremely poor agreement. Based on these findings, concern has been raised over the clinical significance of anismus.

References


