3
Anal Physiology: The Physiology of Continence and Defecation

Vitaliy Poylin and Thomas E. Cataldo

Abbreviations

RAIR	Rectoanal inhibitory reflex
SNS	Sacral nerve stimulation
FI	Fecal incontinence
MR	Magnetic resonance

Key Concepts

- The innervation of the anal sphincter complex is a mixed sympathetic and parasympathetic crossed over system that provides redundant safeguards to continence.
- Normal continence and defecation require intact sensation and motor control and reflexes to sense, retain, and voluntarily expect the rectal contents at a socially appropriate time and place.
- The normal physiology of the anus can be disturbed in a variety of ways resulting in lack of control, inability to expel, or chronic pelvic pain.
- The process of childbirth can contribute significantly to alteration in anorectal anatomy and physiology resulting in a variety of disorders of defecation and/or incontinence.

Introduction

The physiology of the anus and its surrounding structures is in essence the physiology of continence and controlled defecation. This is a physiology of balance and continuous feedback and complex reflexes. Normal continence requires a balance between the pressure inside the rectum and the combined tone of the internal and external sphincters. Defecation and the controlled passage of gas or stool at socially appropriate circumstances required very fine sensation and ability to discern the rectal contents. Defecation requires the balance to tip in favor of the rectal pressure and contraction with simultaneous coordinated relaxation of the pelvic floor and internal and external sphincters. Disturbance in any part of this complex balance can result in incontinence either through reduced anal tone, excess rectal contraction, reduced sensation, or the inability to differentiate the consistency of the rectal contents. Alternatively, disorders tipping in the opposite direction may result in inability to properly or completely empty the rectum. Additionally, more proximal conditions resulting in chronic diarrhea or constipation may tip the balance. And forces even higher can contribute to the behavioral and psychosocial aspects of ordered and disordered function of the rectum and anal canal.

It is the patient and skilled practitioner who listens to what the patient can teach and tell about how and what they are doing combined with a good working knowledge of anorectal physiology that can effectively intervene in disorders of defecation.

Normal Anatomy and Physiology

For a detailed discussion on the anal anatomy, see Chap. 1. Briefly, the musculature of the anus is made up of three concentric cylindrical structures. The internal sphincter is derived as an extension of the involuntary circular smooth muscle of the rectum. The longitudinal muscle is derived from the outer longitudinal smooth muscle of the rectum, and ultimately does extend into the anus and turns medially through the internal sphincter to comprise the muscles of Treitz that support the internal hemorrhoids. Lastly, the external sphincter is derived from the voluntary striated muscle of the pelvic floor.

The internal sphincter begins as a condensation of the inner circular involuntary smooth muscle of the GI tract at the top of the surgical anal canal, as the top of the anorectal ring. It

Electronic supplementary material: The online version of this chapter (doi:10.1007/978-3-319-25970-3_3) contains supplementary material, which is available to authorized users.

S.R. Steele et al. (eds.), *The ASCRS Textbook of Colon and Rectal Surgery*, DOI 10.1007/978-3-319-25970-3_3

extends downward to just proximal to the end of the external sphincter in the non-retracted or effaced state. The length of the normal internal sphincter can vary from under 2 to over 4 cm. In the unstimulated state, the internal sphincter is chronically contracting and contributes approximately 50–75 % of the resting tone of the anus. It appears as a 2–3-mm hypoechoic band on transanal ultrasound imaging [1]. The internal sphincter may not represent a perfect cylinder in all patients. Proximal anterior defects have been demonstrated in nulliparous women [2]. Length and bulk of the sphincter can be reduced if deprived of innervation or hormones in postmenopausal women (progesterone).

The external sphincter is a cylinder of striated muscle that extends downward from the levator ani muscle to the distal anoderm. Like the internal sphincter, it exists in a chronically contracting state, but has the potential when stimulated under voluntary control, to more than double the tone of the anus above the resting state. It was initially considered to be divided into three separate segments, deep, superficial, and subcutaneous; this is no longer thought to be a meaningful distinction [3].

Between the internal and external sphincters is a layer of mixed smooth and striated muscle that is made up of an extension of the longitudinal outer muscle of the bowel and some striated extensions of the levator ani muscle. As it extends downward, some aspects of the muscle cross medially through the internal sphincter to contribute to the suspensory muscles that hold the hemorrhoid complex in place (Trietz's muscle). Distally, the conjoined muscle extends to the anoderm and through the external sphincter radially to form the corrugator cutis ani [1, 4, 5].

Innervation of the Anus and Pelvic Floor

The parasympathetic fibers to the rectum and anal canal emerge from the sacral foramina at the S2, 3, 4 levels. They join the sympathetic hypogastric nerves in the pelvic plexus. From there mixed postganglionic fibers extend to the lower rectum and anal canal. Thereby internal sphincter is innervated by L5–S4 mixed autonomic function in crossed fashion so that unilateral injury still results in preserved function. The external sphincter is similarly innervated from branches of S2–3 via the inferior rectal branch of the pudendal nerve and the perineal branch of S4. This nervous distribution also carries the nerves of sensation and contributes to the functional aspects of continence. The upper anal canal contains a high density of free and organized sensory nerve endings [1, 6, 7]. Organized nerve endings include Meisner's corpuscles (touch), Krause's bulbs (cold), Golgi-Mazzoni bodies (pressure), and genital corpuscles (friction).

Normal Continence

Rectal Capacity

Normal continence first requires a location to temporarily hold and assess the contents and expel them under control. The rectum therefore needs both a baseline capacity and the compliance to expand and the force to expel. The empty rectum is a low pressure vessel with the capacity to receive stool from the sigmoid. It must have the capacity to expand significantly to accommodate stool under pressure. Patients with diminished rectal capacity will suffer from fecal frequency, urgency and frequently may contribute to incontinence.

Pressure and Motility

Baseline pressure in the rectum is low, about 5 mmHg with frequent low amplitude contractions every 6–12 s. Occasional high pressure waves up to 100 mmHg have been demonstrated. The anal canal shows overlapping of resting tone with small oscillations of pressure and frequency of 15 cycles/min and cm H_2O. Pressure in the anal canal ranges 10–14 times that of the rectum. Motor activity is more frequent, and contractile waves are of higher amplitude in the rectum than in the sigmoid [6]. This reverse gradient provides a pressure barrier resisting forward motion of stool and may propel stool back into the sigmoid as part of delaying bowel movements when it is not convenient [7]. Slow waves are observed in the anal canal with increasing frequency distally. This gradient is thought to help maintain continence by propelling the contents back into the rectum and helps keep the canal empty.

Rectoanal Sensation and Sampling

The rectum does not itself have receptors for proprioception. The conscious sensation of the need to defecate lives in the levators and the anal canal, hence the preserved sensation in patients who have had complete proctectomies and anal anastomoses. Distention of the rectum triggers contraction of the external anal sphincter and significant internal anal sphincter contraction. As first described by Gowers in 1877 [8] the rectoanal inhibitory reflex (RAIR) is thought to allow the highly innervated sensitive epithelial lining of the upper anal canal to sample the contents of the distal rectum to determine its quality and consistency. This allows the patient to accurately discern flatus from stool, and liquid stool from firm. Alterations in this mechanism, either through reduced sensation, or impaired sampling can result in incontinence either through overflow or inability to

discern that defecation is occurring. Impaired anal sensation has been associated with childbirth, perineal descent, and mucosectomy [9–11].

Structural Considerations

In addition to the baseline resultant tone provided by the anal sphincter complex and the puborectalis sling, the entire structure is held closed by the angulation created by the puborectalis in its chronically contracted unstimulated state. This angle between the axis of the anus and the axis of the rectum is between 80° and 90° and is responsible for the majority of gross fecal continence. It may increase normally above 90 while sitting and will extend beyond 110° during normal defecation. In cases of dysfunctional defecation where the puborectalis does not sufficiently relax the angle can be enhanced by squatting and flexing the hips to an angle of less than 90°. The flap valve theory advocated by Parks suggests the anterior rectal mucosa constitutes a flap that lies over the upper end of the anal canal. Increased inter abdominal pressure not associated with defecation increased the angulation and closes flap more firmly over the upper anal canal. The flap is opened when the perineum descends and the anorectal angle is straightened. The anterior mucosal flap certainly seems to be a component of the issue when patients suffer from obstructed defecation and have evidence of internal rectal prolapse.

Role of Hemorrhoids in Normal Continence

It has also been postulated that the normal function of the hemorrhoids, in a non-pathologic state serve as an additional important component of normal continence. Stelzner referred to the hemorrhoids as the corpora cavernosum of the anus [12]. These vascular cushions have the ability to expand as needed to create a seal above the anus creating the fine tuning of continence. This concept is supported by the observation that after formal hemorrhoidectomy some patients experience minor alterations in continence.

Sensation and Innervation

Within the pelvis, the innervation of the proximal anal canal descends from the rectum. The rectum has a mixed sympathetic and parasympathetic innervation derived from the hypogastric nerves and the sacral parasympathetic nerves through the pelvic plexi. Extrapelvic innervation comes to the anus from the pudendal nerve derived from S2 to S4 via the inferior rectal nerve and ultimately spreads around the anus from both sides entering at lateral to slightly anterior positions. There is known to be significant crossover innervation around the anus as a complete disruption of either pudendal nerve does not result in asymmetric sphincter atrophy or fecal incontinence.

Sensory innervation within the rectum is sensitive only to stretch, resulting in vague sensation to visceral pelvic pain. Distal rectal stretch or distention can result in significant parasympathetic stimulation of the vagus nerve, thereby resulting in bradycardia and hypotension. The lack of pain-sensitive innervation proximal to a short distance from the dentate line is what allows some hemorrhoid treatments to be performed with relatively limited discomfort, e.g., elastic band ligation, injection sclerotherapy, and stapled hemorrhoidopexy. Somatic sensory innervation begins in the anal transitional zone proximal to the dentate line for a short variable distance 0.3–1.5 cm [13]. Within this zone, there is a dense collection of nerve endings for pain, touch, pressure, and temperature. As such they are theorized to be an integral part of the sampling aspect of the continence mechanism [14]. These fibers are derived from the pudendal branches, and complete anesthesia to this area can be provided by bilateral anal nerve blockade.

Normal Defecation

Normal defecation is a complicated mechanism that relies on a close interaction between the somatic and autonomic nervous system, which includes the conscious and unconscious control of both sensory input and muscle contraction. The process starts with stool arriving into the rectum and sampling as described above. If it is not an appropriate time for defecation, the anal sphincter will contract and rectum will start to distend [7]. This process continues with progressive distention of the rectum without a person's full awareness; patients are often unaware that they have stool in the vault during rectal exam. Conscious sampling, however, is also present during this process (one can differentiate between gas and stool and allow gas to pass, even with full rectum). As the rectum continues to expand, a person becomes aware (with continuous sampling) There is an urge defecate that usually lasts for a few seconds and can be controlled by further contraction of external anal sphincter (efferent nerve endings end in lumbosacral spine which is under higher control, that allows conscious suppression of the urge) [15, 16].

When it becomes socially appropriate to proceed, the defecation process again relies on both conscious and unconscious response. The process starts with contraction of abdominal musculature (Valsalva), which is also associated with contraction of the sigmoid colon to move stool forward. Pelvic floor musculature on the other hand relaxes, which is a combination of relaxation of puborectalis (releases sling around anorectal junction) and relaxation of remaining levator muscle. This allows the pelvic floor to descend slightly and straighten the anorectal angle. The rectum itself starts to contract and both internal and external sphincters relax. Even if the sphincters are not completely relaxed, at this point pressure in the rectum exceeds pressure in the anal canal and defecation will occur. This process can also be aided by assuming the squatting position, which increases

the intra-abdominal pressure and straightens the rectum further. If the conscious decision to defecate is made during sampling (rectum is contracting, internal sphincters already partially relaxed) allowing the external sphincters to relax, then defecation will occur [17–19]. Once begun a number of patterns can occur. There may be a single evacuation of the rectal contents accompanied by mass peristalsis of the left and sigmoid colon clearing the bowel in one continuous movement, or the passage of smaller volumes of stool individually over a short time requiring recurrent efforts and straining [20]. These two patterns and variations thereof are dictated by the habits of the patient and other factors including the overall consistency of the stool.

If a large volume of stool is delivered quickly to the rectum, normal rectal compliance and accommodation may be insufficient. In this case the patient with normal sensation and function will have a sense of acute urgency and can forestall defecation for 40–60 s with the use of voluntary contraction of the external sphincter to allow accommodation or move to a socially appropriate location to evacuate.

For obvious reasons, studying this process can be difficult, and thus our understanding of it relies on what is observed during testing (e.g., defecography—Video 3.1; and anal manometric studies) [2, 6], patients with neurologic deficits (specifically spinal injuries) [21] and animal studies. Animal studies revealed the presence of different, more sensitive mechanoreceptors in the rectum, when compared to the colon that are most responsive to tension and rapid distention [22–24]. These tension mechanoreceptors respond to both rectal distension and muscle contraction consistent with the observation that rectal filling sensation coincides with the period of raised rectal pressure during rectal distension [3–6].

Physiology of Tibial Nerve and Sacral Nerve Root Stimulation in Fecal Continence

For many years it has been recognized that chronic electrical stimulation of nerves entering the pelvis has had effects of visceral function and activity. Unilateral stimulation of the S3 or S4 nerve as it exits the foramen has been used for urinary incontinence for over 30 years; meanwhile benefits for fecal incontinence have been recognized as well. Most recently, sacral nerve stimulation has shown encouraging results for idiopathic constipation as well [25–27].

The exact mechanism of how sacral nerve stimulation creates its effect remains unclear. The physiological control of defecation relies on the coordinated sensory and motor efforts of the colon, rectum, and anus. Current opinion is that disordered defecation is secondary to several disturbances of anorectal and colonic physiology and not purely a sphincter disturbance in patients with FI or colonic transit failure in constipation. It is therefore likely that the therapeutic effects of SNS are due not only to peripheral motor stimulation of the anal sphincter complex in patients with FI as was initially proposed, but instead due to changes in the motor and/or sensory function of the combined functional anorectal unit. Such a hypothesis would explain the "paradox" of SNS effectiveness in both FI and chronic constipation, i.e., it is likely that SNS is effective in both conditions not due to paradoxical actions in each, but instead by improvement of common pathophysiologies. This hypothesis also explains why FI and disordered defecation so frequently coexist [28]. Similarly, intermittent stimulation of the posterior tibial nerve has a beneficial effect on fecal incontinence through a mechanism that is not fully understood [29].

In 2014, Carrington et al. performed an exhaustive review of the scientific literature regarding sacral and peripheral nerve stimulation for fecal incontinence and constipation [15]. To summarize their findings, SNS had no demonstrable effect of rectal compliance or motility. It did seem to reduce hypersensitivity in those with reduced capacity and hypersensitivity, while increasing sensitivity in those patients with reduced sensitivity. Additionally sacral nerve stimulation increases mucosal blood flow when on and returns to baseline when off. There are higher levels of the neuropeptide substance P identified in rectal biopsies of those undergoing stimulation, which reverses after it is discontinued. The exact importance or impact of these two phenomena has not been identified as yet. Forty studies have examined changes in anal sphincter function through the use of anorectal manometry. Direct comparison between studies is difficult, as equipment specifications, study protocol, and method of results reporting is extremely variable between centers. Fourteen studies reported a significant increase in voluntary anal squeeze, with eight of these also reporting an increase in resting pressure.

Spinal Cord Injuries and Defecation

The most interesting and informative studies in normal and abnormal defecation are provided by patients with spinal cord injuries. However, it is important to remember that this is a very heterogeneous group of patients with degrees of injury that can vary significantly from patient to patient [7]. High spinal cord injuries (above T7) interrupt higher control and sensation of the abdominal and pelvic floor musculature as well as colon in rectum [12, 29, 30]. This combination allows for lower tone in the colon and rectum. The decrease in propulsive ability of the colon, the decrease in tone resulting in distention and slower transit through the colon explains the constipation that often accompanies high spinal cord injuries. These patients are often unable to generate adequate intra-abdominal pressure or take squatting position to aid defecation [11, 13, 31]. At the same time, there is an unopposed stimulation of the lower neurons that increase contraction and spasticity of the pelvic floor and external anal sphincters.

Sensation is often also impaired which can eliminate the normal urge to defecate. Interestingly, this often does not affect mechanoreceptors and some patients will report vague sensation of pressure that is then interpreted as a need to defecate [31–33]. As a result, these patients often have chronic constipation caused by both diminished sensation and inability to move stool forward [12, 13]. This is combined with pelvic floor dysfunction and the inability to identify the urge to defecate and an inability to relax the pelvic floor. They often rely on a strict bowel program, which is a combination of laxatives, rectal stimulation and manual disimpaction [11–13]. Rectal stimulation can allow some patents to have decreased anal sphincter pressure. They can also experience fecal incontinence as a result of overflow and overfill of the rectum and well as damage to sphincters from manual disimpaction [12–14, 34].

Patients with low spinal cord injuries such as Cauda Equina Syndrome often have impaired afferent fibers that results in loss of tone in the internal and external sphincter muscle as well as impaired sensation. This can result in significant incontinence since any generation of intra-abdominal pressure may result in bowel movement [11–13].

Obstructed Defecation

Obstructed defecation is a poorly understood group of disorders resulted from an alteration in sensation, muscle relaxation or both. In many patients with these problems, the exact cause is multifactorial and/or the inciting event is not easily identifiable [35]. It is possible that an abnormality in the sensory mechanism is the primary insult in a number of patients [36]. Normal sensation is an integral part of normal defecation. It allows for appropriate reflexes, mostly importantly the anal sampling RAIR. Some causes of abnormal sensation can be fairly evident in patients such as those with significant proctitis (infectious or inflammatory) or those after anorectal injury/surgery. In the absence of above, the etiology is less clear. Dysfunction may be associated with conscious/subconscious inhibition of the need to defecate during childhood [15, 16, 37, 38]. According to this theory, repeated delays in defecation result in altered sensation that eventually leads to dyscoordination between the anorectal and pelvic floor musculature. As this process continues, even though patient may continue to experience “normal” urge to defecate, changes in sensation cause an increase in stimulation of lower (lumbosacral) neuronal loop; the relaxing effects of the upper parts of the nervous system are insufficient to overpower the abnormal stimulation. Once this occurs, and pelvic floor musculature such as puborectalis and sphincter complex fail to relax appropriately, increasingly higher intra-abdominal pressure is needed to overpower the rectal/anal pressure to evacuate [39]. This failure can be associated with pain and a feeling of incomplete evacuation.

Independent of what part of normal defecation was affected first, over time there is probably significant damage to the sensory pathways including receptors, efferent nerves and muscles. With time, this process will also start affecting the structural integrity of the pelvic floor. Obstructed defecation disorders include intussusception, rectocele, nonrelaxing puborectalis/levator muscle spasm, dyssynergic puborectalis, as well as enterocele and rectal prolapse. Although causes of enterocele and rectal prolapse may be complex, these disorders in their pure form are mechanical obstructions to defecation and thus beyond the scope of this chapter. Here we describe a few pathological conditions that are more directly affected abnormalities in sensory-muscular neurological loop.

Intussusception is mucosal descent causing blockage of the lower rectum/anal canal. It is possible that it is a primary process in some patients arising from redundancy of mucosa, possibly poor tone, and pelvic floor descent (either primary structural problems or as a result of childbirth and muscle/nerve damage in women). In most patients it is likely a secondary process resulting from increased pushing and decreased relaxation. Once developed, intussusception itself generates mechanical blockage to defecation and further attempts to generate more pressure to evacuate stool [17–19, 40].

Rectocele likely develops by a similar process. It is defined as greater than 2 cm of rectal wall out pouching or bowing anteriorly while straining. It can be accompanied by intussusception. Rectoceles are caused by abnormal relaxation of the pelvic floor/sphincter complex or structural defects in the rectal wall created during childbirth. As a result, when a patient attempts to evacuate, generated pressure delivers stool anteriorly towards the weakened portion of the wall that is not contracting appropriately. This generates a sensation of bulge and incomplete evacuation and can be at least in part relieved in women by pushing on the vagina in the initial stages of the disease (Figure 3-1**;** Video 3.2). However, a rectocele itself is a very common finding on the exam and only a small proportion of patients who have it will ever have symptoms. Most symptomatic patients likely have a combination of a weaker rectal wall as well as dyssynergy of the sphincters or puburectalis [15, 41].

Pelvic floor dyssynergy (pelvic outlet obstruction) results from a failure of the puborectalis and/or sphincter complex to relax. It can also be caused by an abnormal contraction during evacuation. As a result, when a patient tries to evacuate the anorectal angle may not increase or may even become sharper. A patient’s natural response is to generate higher pressures in which only further worsens the symptoms. Over time, these changes likely cause more damage to the musculature and nerves. Similar to the rest of the disorders in this group, rectal sensation is also impaired, but whether it is a result of long-term damage or from an inciting event is unclear [15, 16, 18].

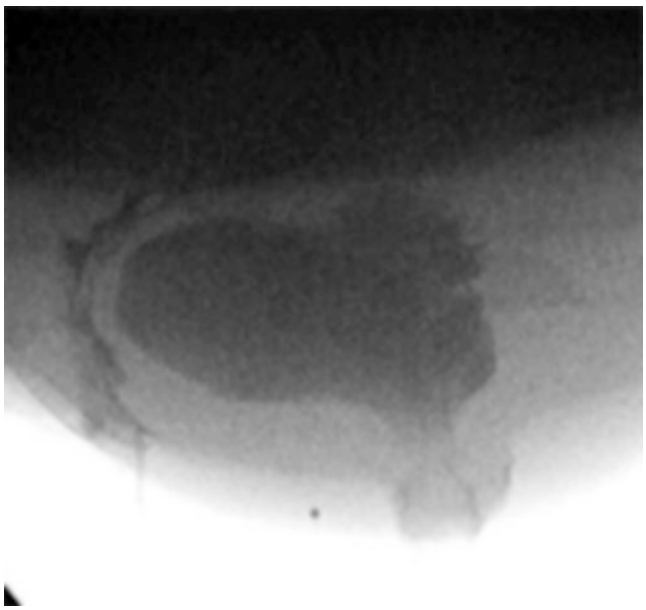

FIGURE 3-1. Defecography still image of a rectocele.

Functional Anorectal Pain

Most causes of anorectal pain can be routinely ascribed to such common conditions as anal fissures, hemorrhoidal disease, or inflammatory bowel diseases (see Chap. 11). There is a small group of disorders, however, that seem to be related to more functional, rather than structural problems [42].

Levator ani syndrome (levator spasm, puborectalis syndrome) is often described as dull pain, high in the rectum that is often made worse with sitting. By definition, it should last more than 20 min at the time and other causes are excluded [43]. Etiology of this condition is unclear. Interestingly, even though episodes may be triggered by difficult defecation (along with emotional stress among other things) it is not always associated with difficulty evacuating. Similar to other functional disorders, it is possible that alternations in sensation, and perhaps behaviors (deferring defecation, damage with hard stool) could contribute to the development and propagation of this problem. In addition, it is thought that prolonged muscle contraction may result in compression of vasculature, which then leads to relative ischemia and an increase in anaerobic consumptions. That in turn can cause activation of nociceptors in the muscle (bradykinin, Substance P), and further decrease in relaxation with spasm and pain [15, 16, 22].

Proctalgia fugax is a sudden severe anal pain, lasting seconds to minutes, that disappears completely. The etiology is unknown, but it seems to be related to stress. It is associated in some patients with a thickened internal sphincter muscle. Some studies suggest smooth muscle contraction is responsible for this pain [15, 16, 44].

Pathophysiology of Obstetric-Related Problems

One of the worrisome potential sequelae of pregnancy and delivery is fecal incontinence. It can develop as a result of direct disruption of the anal sphincter, muscle, connective tissue or pudendal nerve injury [45]. During pregnancy, there is direct pressure on the pelvic floor as well as hormonal changes. Progesterone, released during pregnancy, acts by suppressing contraction of smooth muscle and prevents premature uterine contraction. This leads to decreased gut motility (that can contribute to constipation) and diminished tonic contraction of anal sphincters [25, 46]. Androgen, progesterone, and estrogen receptors are found in squamous epithelium of the anal canal, indirectly supporting possible effects of this hormone on the sphincters [47]. In addition, progesterone causes ligamentous laxity [48]. When combined with increased intra-abdominal pressure, these changes contribute to stretching of the pelvic floor musculature, widening of the levator hiatus, and potentially pudendal nerve injury. The pudendal nerve can be affected during pregnancy by stretching as well as traction injury during delivery as described below [49]. Pudendal nerve injury can affect both external sphincters by de-innervating them and causing muscle atrophy as well as by affecting sensory components and altering RAIR. Evidence of neuropathy in pelvic floor musculature has been found after delivery as well as in idiopathic FI and constipation.

Labor further complicates issues of continence. Pushing during labor can significantly exacerbate the above problem [50]. It can be associated with further muscle stretching or even evulsion and pudendal nerve injury [25]. This explains why a longer second stage of labor (pushing) is associated with higher rates later in life. In addition, there is likely effects of traction injury (increased baby weight is associated with higher chances of immediate and long-term problems). Use of additional devices to aid labor such as forceps and vacuum is associated with increased incidence of FI [25, 51]. This is likely related to direct damage to the sphincters as well as traction injury. Tearing and episiotomy are additional risk factors for FI and related to direct damage to the sphincter complex. Cesarean section is associated with lower incidences of flatus and stool incontinence, but this difference is smaller when comparing emergent Cesarean sections and vaginal deliveries. Emergent cesarean are often initiated after failure of labor to progress following significant pushing [52]. Although many women experience immediate mild problems with incontinence to flatus or stool, most have enough reserves to compensate. Presence of symptoms after delivery is an additional risk factor for developing significant incontinence in the future when age further weakens already damages muscles and nerves.

Urogynecological Considerations and Pelvic Pain

With all its complexity, the pelvic floor is anatomically very small area. It includes pelvic musculature and their corresponding nerves responsible not only for maintenance of continence and normal defecation, but also normal urinary gynecologic function. Not surprisingly, although dysfunction in any single system is common, more than one system is frequently affected. For example, physiologic and muscular changes associated with pregnancy and labor which effects the posterior compartment often has similar effects on middle and anterior compartment structures as well. Uterine prolapse is more common in multiparous women, especially in complicated deliveries. Urinary problems including incontinence are also common [16, 25]. The mechanism for urinary issues is likely the same as in posterior compartment problems, which is a combination of hormonal effects as well as direct damage to the pelvic floor muscle, nerves, and sphincters. Widening of the levator hiatus has been shown to affect middle and anterior compartments as well as posterior one. This can result in uterine and bladder prolapse in addition to rectal prolapse, intussusception, and rectocele [21]. Pregnancy and delivery effects on anal sphincters can affect urinary sphincters as well. It is common for women presenting with urinary incontinence to report fecal incontinence as well [16, 25]. As a result, urogynecologists see and treat a number of patients with anorectal problems, especially since the treatments available are similar between specialties (e.g., pelvic floor physical therapy, sacral nerve stimulation). Pelvic floor prolapse problems, especially of the middle compartment, may contribute to obstructed defecation. For this reason care should be taken to obtain full history of pelvic floor problems. Otherwise one risks missing significant contributors to patients' symptoms and may compromise success of treatment.

Another common problem is pelvic pain, and women with these symptoms are often referred directly to gynecologists, although underlying cause could be levator spasm or pelvic floor dyssynergy [23]. These problems are also commonly treated by our urogynecology colleagues utilizing similar techniques including physical therapy and other pelvic floor relaxation techniques. Diagnostic techniques employed by urogynecologists to diagnose anterior pelvic problems are often the same (MR defecography and conventional cine defecography, anal manometry). As a result, when patients present with anorectal problems related to pelvic floor issues, one has to maintain vigilance in identifying related problems with anterior and middle compartment since they can affect overall symptom control as well as how these problems are ultimately addressed.

References

1. Jorge JMN, Habr-Gama A. Anatomy and embryology of the colon rectum and anus. In: Wolff BG, Fleshman JW, Beck DE, Pemberton JH, Wexner SD, editors. The ASCRS textbook of colon and rectal surgery. New York, NY: Springer; 2007. p. 1–11.
2. Bollard RC, Gardiner A, Lindow S, Phillips K, Duthie GS. Normal female anal sphincter: difficulties in interpretation explained. Dis Colon Rectum. 2002;45:171–5.
3. Gordon PH. Anatomy and physiology of the anorectum. In: Fazio VW, Church JM, Delaney CP, editors. Current therapy in colon and rectal surgery. 2nd ed. Philadelphia, PA: Elsevier Mosby; 2005. p. 1–4.
4. Milligan ETC, Morgan CN, Jones LE, Officer R. Surgical anatomy of the anal canal and the operative treatment of haemorrhoids. Dis Colon Rectum. 1985;28:620–8.
5. Morgan CN. The surgical anatomy of the anal canal and rectum. Postgrad Med J. 1936;12:287–314.
6. Taylor I, Duthie HL, Amallwwod R, et al. Large bowel myoelectrical activity in man. Gut. 1975;16:808–14.
7. Gordon PH. Anorectal anatomy and physiology. Gastroenterol Clin North Am. 2001;30:1–13.
8. Gowers WR. The automatic action of the sphincter ani. Proc R Soc Lond. 1877;26:77–84.
9. Cornes H, Bartolo DCC, Stirra T. Changes in anal canal sensation after childbirth. Br J Surg. 1991;78:74–7.
10. Miller R, Bartolo DCC, Cervero F, Mortenson NJ. Differences in anal sensation in continent and incontinent patients with perineal descent. Int J Colorectal Dis. 1989;4:45–9.
11. Keighley MRB. Abdominal mucosectomy reduces the incidence of soiling and sphincter damage after restorative proctocolectomy and J-pouch. Dis Colon Rectum. 1987;39:386–90.
12. Stelzner F. The morphological principles of anorectal continence. In: Rickham PP, Hecker WSH, Prevot J, editors. Anorectal malformations and associated diseases, Progress in pediatric surgery series, vol. 9. Munich: Urban & Schwarzberg; 1976. p. 1–6.
13. Kaiser AM, Ortega AE. Anorectal anatomy. Surg Clin North Am. 2002;82:1125–38.
14. Duthie HL, Gairns FW. Sensory nerve-endings and sensation in the anal region of man. Br J Surg. 1960;206:585–95.
15. Sangwan YP, Solla JA. Internal anal sphincter: advances and insights. Dis Colon Rectum. 1998;41:1297–311.
16. Palit S, Lunniss PJ, Scott SM. The physiology of human defecation. Dig Dis Sci. 2012;57:1445–64.
17. Bajwa A, Emmanuel A. The physiology of continence and evacuation. Best Pract Res Clin Gastroenterol. 2009;23:477–85.
18. Brookes SJ, Dinning PG, Gladman MA. Neuroanatomy and physiology of colorectal function and defaecation: from basic science to human clinical studies. Neurogastroenterol Motil. 2009;21 Suppl 2:9–19.
19. Gurjar SV, Jones OM. Physiology: evacuation, pelvic floor and continence mechanisms. Surgery. 2011;29(8):358–61.
20. Lubowski DZ, Meagher AP, Smart AC, et al. Scintographic assessment of colonic function during defecation. Int J Colorectal Dis. 1995;10:91–3.

21. Brading AF, Ramalingam T. Mechanisms controlling normal defecation and the potential effects of spinal cord injury. In: Weaver LC, Polosa C, editors. Progress in brain research 2006; vol 152:p. 345-358 (Chapter 23).
22. Broens PMA, Penninckx FM, Ochoa JB. Fecal continence revisited: the anal external sphincter continence reflex. Dis Colon Rectum. 2013;56:1273–81.
23. Lynn PA, Olsson C, Zagorodnyuk V, et al. Rectal intraganglionic laminar endings are transduction sites of extrinsic mechanoreceptors in the guinea pig rectum. Gastroenterology. 2003; 125:589–601.
24. Lynn PA, Blackshaw LA. In vitro recordings of afferent fibres with receptive fields in the serosa, muscle and mucosa of rat colon. J Physiol. 1999;518(Pt 1):271–82.
25. Tanagho EA, Schmidt RA. Electrical stimulation in the clinical management of the neurogenic bladder. J Urol. 1988;140: 1331–9.
26. Ganio E, Luc AR, Clerico G, Trompetto M. Sacral nerve stimulation for treatment of fecal incontinence: a novel approach for intractable fecal incontinence. Dis Colon Rectum. 2001;44:619–29.
27. Malouf AJ, Wiesel PH, Nicholls T, Nicholls RJ, Kamm MA. Sacral nerve stimulation for idiopathic slow transit constipation. Gastroenterol Clin North Am. 2001;118:4448–9.
28. Carrington EV et al. A systematic review of sacral nerve stimulation mechanisms in the treatment of fecal incontinence and constipation. Neurogastroenterol Motil. 2014;26(9):1222–37.
29. Thumas TO, Dudding TC, et al. A systemic review of posterior tibial nerve stimulation for faecal incontinence. Colorectal Dis. 2012;15:519–26.
30. Ebert E. Gastrointestinal involvement in spinal cord injury: a clinical perspective. J Gastrointestin Liver Dis. 2012;21(1): 75–82.
31. Lynch AC, Frizelle FA. Colorectal motility and defecation after spinal cord injury in humans. In: Weaver LC, Polosa C, editors. Progress in brain research 2006;vol 152:193–203 (Chapter 23).
32. Nout YS, Leedy GM, Beattie MS, Bresnahan JS. Alterations in eliminative and sexual reflexes after spinal cord injury: defecatory function and development of spasticity in pelvic floor musculature. In: Weaver LC, Polosa C, editors. Progress in brain research 2006;vol 152:359–273 (Chapter 23).
33. Preziosi G, Raptis DA, Raeburn A, Panicker J, Emmanuel A. Autonomic rectal dysfunction in patients with multiple sclerosis and bowel symptoms is secondary to spinal cord disease. Dis Colon Rectum. 2014;57:514–21.
34. Valle's M, Mearin F. Pathophysiology of bowel dysfunction in patients with motor incomplete spinal cord injury: comparison with patients with motor complete spinal cord injury. Dis Colon Rectum. 2009;52:1589–97.
35. Bharucha AE, Rao SSC. An update on anorectal disorders for gastroenterologists. Gastroenterology. 2014;146:37–45.
36. Bharucha AE, Wald A, Enck P, Rao S. Functional anorectal disorders. Gastroenterology. 2006;130:1510–8.
37. van Ginkel R, Reitsma JB, Buller HA, et al. Childhood constipation: longitudinal follow-up beyond puberty. Gastroenterology. 2003;125:67–72.
38. Rao SSC, Tuteja AK, Vellema T, et al. Dyssynergic defecation: demographics, symptoms, stool patterns and quality of life. J Clin Gastroenterol. 2004;38:680–5.
39. Rao SS, Welcher KD, Leistikow JS. Obstructive defecation: a failure of rectoanal coordination. Am J Gastroenterol. 1998;93: 1042–50.
40. Andromanakos N, Skandalakis P, Troupis T, Filippou D. Constipation of anorectal outlet obstruction: pathophysiology, evaluation and management. J Gastroenterol Hepatol. 2006;21: 638–46.
41. Felt-Bersma RJ, Tiersma ES, Cuesta MA. Rectal prolapse, rectal intussusception, rectocele, solitary rectal ulcer syndrome, and enterocele. Gastroenterol Clin North Am. 2008;37: 645–68.
42. Atkin GK, Suliman A, Vaizey CJ. Patient characteristics and treatment outcome in functional anorectal pain. Dis Colon Rectum. 2011;54:870–5.
43. Hull M, Cort MM. Evaluation of the levator ani and pelvic wall muscles in levator ani syndrome. Urol Nus. 2009; 29(4):225.
44. Eckardt VF, Dodt O, Kanzler G, Bernhard G. Anorectal function and morphology in patients with sporadic proctalgia fugax. Dis Colon Rectum. 2004;39:755–62.
45. Shin GH, Toto EL, Schey R. Pregnancy and postpartum bowel changes: constipation and fecal incontinence. Am J Gastroenterol. 2015;110:521–9.
46. Chiloiro M, Darconza G, Piccioli E, et al. Gastric emptying and orocecal transit time in pregnancy. J Gastroenterol. 2001;36: 538–43.
47. Oettling G, Franz HB. Mapping of androgen, estrogen and progesterone receptors in the anal continence organ. Eur J Obstet Gynecol Reprod Biol. 1998;77:785–95.
48. Shultz SJ, Wideman L, Montgomery MM, et al. Changes in serum collagen markers, IGF-I, and knee joint laxity across the menstrual cycle. J Orthop Res. 2012;30:1405–12.
49. Parks AG, Swash M. Denervation of the anal sphincter causing idiopathic anorectal incontinence. J R Coll Surg Edinb. 1979; 24:94–6.
50. Bharucha AE, Fletcher JG, Melton III LJ, et al. Obstetric trauma, pelvic floor injury and fecal incontinence: a population-based case–control study. Am J Gastroenterol. 2012;107: 902–11.
51. Dudding TC, Vaizey CJ, Kamm MJ. Obstetric anal sphincter injury incidence, risk factors, and management. Ann Surg. 2008;247(2):224–37.
52. Pretlove SJ, Thompson PJ, Toozs-Hobson PM, et al. Does the mode of delivery predispose women to anal incontinence in the first year postpartum? A comparative systematic review. BJOG. 2008;115:421–34.