Pelvic Floor Physiology: From Posterior Compartment to Perineal Body to Anterior Compartment

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Introduction

The primary physiologic function of the posterior pelvic floor is bowel continence and evacuation. The muscles of the pelvic floor act as both a supportive base for the abdominal viscera and provide mechanisms for continence. The bony pelvis provides the attachments for these muscles that surround the external orifices. These muscles are innervated by both the parasympathetic and sympathetic nervous systems.

Dysfunction of the pelvic floor contributes to morbidity and decreased quality of life in many patients, especially the geriatric population. Baseline pelvic floor muscle tone and neurologic integrity both play a role in the maintenance of fecal continence. In addition, there can be variation in the regulation of stool due to systemic disease, bowel motility, stool consistency, as well as cognitive and emotional factors. Understanding the anatomy, innervation, and reflexes of the pelvic floor and anal sphincters is the key to assessing disorders of continence and treating this patient population [1].

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Posterior Pelvic Floor Physiology

Pelvic Floor and Anal Sphincters

The muscular pelvic floor consists of both superficial and deep muscles, both of which play a role in continence. The *superficial pelvic floor muscles* are most relevant to anal canal function are the (1) external anal sphincter, (2) perineal body, and the (3) puboperineal transverse muscles, which are considered. The *deep pelvic floor muscles* consist of the (1) pubococcygeus, (2) iliococcygeus, (3) coccygeus, and (4) puborectalis muscles (Fig. 2.1). These muscles originate at the pectinate line of the pubic bone and obturator internus fascia, and insert at the *coccyx*. The puborectalis muscle may be more accurately described as *located between* the superficial and deep layers. The muscle originates at the inferior pubic ramus, tracks posteriorly, wrapping around the rectum as it descends, and attaches to the contralateral pubic ramus.

The *internal anal sphincter (IAS)* and *external anal sphincter (EAS)* are the major constrictors of the anal canal. The *IAS* develops from a thickening of the circular colonic muscle layer (Fig. 2.2). As it progresses distally, it thickens and develops an increased number of muscle fibers. This makes the IAS histologically different from the upper colonic circular muscle layer. Involuntary, autonomic innervation is divided between sympathetic and parasympathetic nerves. *Sympathetic* nerves arise from the lower thoracic ganglia, creating the superior hypogastric plexus. *Parasympathetic* innervation arises from sacral nerves 2 through 4, forming the nervi erigentes and the inferior hypogastric plexus (Fig. 2.3). The IAS maintains a constant level of tone, preventing fecal incontinence. It is believed that the interstitial cells of Cajal (ICC) maintain this tone. Unlike the remainder of the gastrointestinal tract, where the interstitial cells of Cajal create rhythmic muscle contractions, studies using imatinib mesylate have shown that the ICCs create a constant level of tone in the IAS [2].

D.A. Gordon, M.R. Katlic (eds.), *Pelvic Floor Dysfunction and Pelvic Surgery in the Elderly*,
DOI 10.1007/978-1-4939-6554-0_2

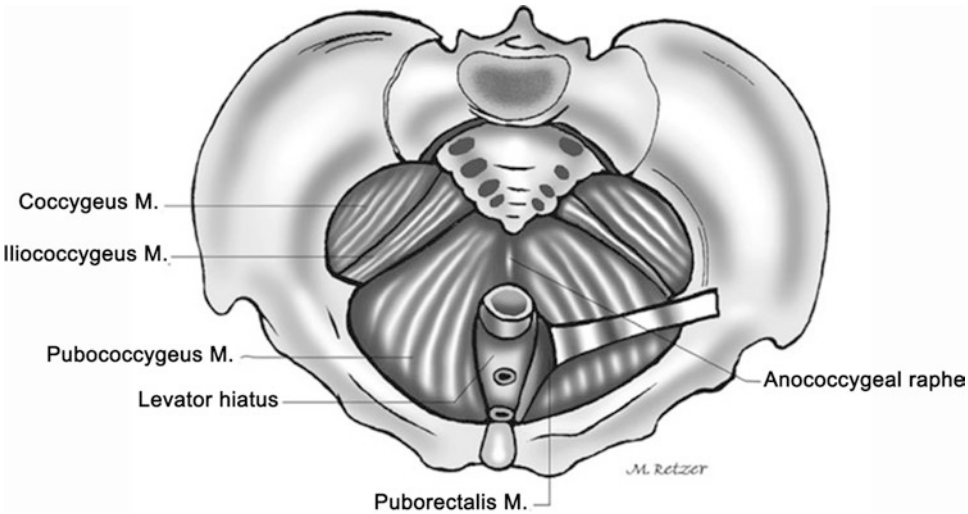

Fig. 2.1 Pelvic floor (*anterior* and *posterior*). With permission from Jorge JMN Habr-Gama A. Anatomy and Embryology of the Colon, Rectum, and Anus. In: Beck DE, Wexner SD, David E. Beck, Steven D. Wexner, Hull TL, Roberts PL, Senagore AJ, Stamos JM, Steele SR, eds. The ASCRS Manual of Colon and Rectal Surgery, 2nd Edn. Springer, New York, 2014; pp:1–25

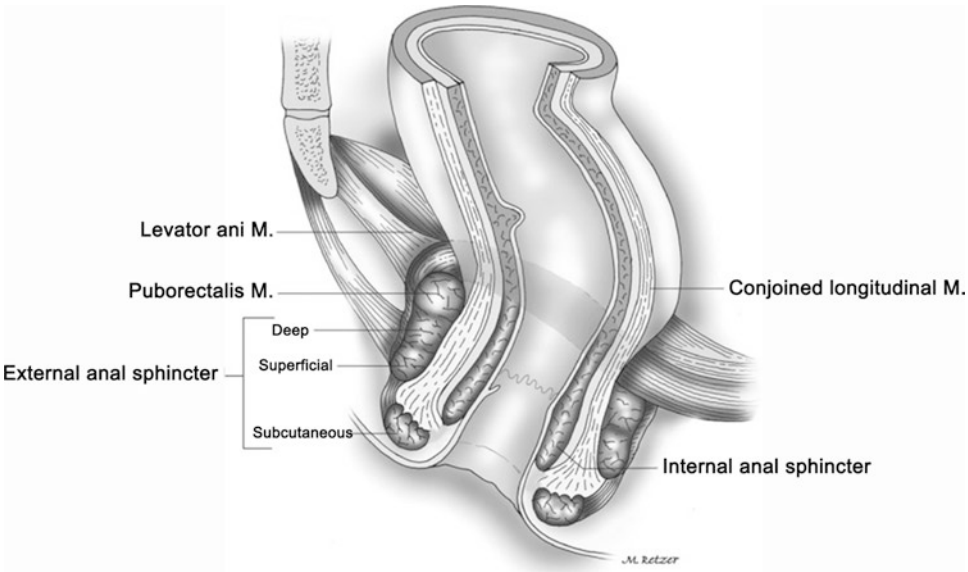

Fig. 2.2 Internal and external anal sphincter. With permission from Jorge JMN Habr-Gama A. Anatomy and Embryology of the Colon, Rectum, and Anus. In: Beck DE, Wexner SD,David E. Beck, Steven D. Wexner, Hull TL, Roberts PL, Senagore AJ, Stamos JM, Steele SR, eds. The ASCRS Manual of Colon and Rectal Surgery, 2nd Edn. Springer, New York, 2014; pp:1–25

The inferior hypogastric plexus further divides to form the superior, middle, and inferior rectal nerves. These nerves synapse with the myenteric plexus of the rectal muscle to regulate tone. Sympathetic activation, via β-adrenergic receptors, creates and maintains internal anal sphincter tonicity, and thus involuntary continence. Parasympathetic innervation utilizes nitric oxide to cause sphincter relaxation [3].

The external anal sphincter (EAS) is comprised of three (3) muscular layers: (1) subcutaneous layer, (2) superficial layer, and (3) deep layer (Fig. 2.2). The subcutaneous portion lies distal to the internal anal sphincter, the superficial surrounds it, and the deep portion merges with the puborectalis muscle. Some consider the deep portion to be a part of the puborectalis muscle rather than a muscular component of the EAS muscle complex [4]. The EAS attaches to the perineal body and transverse perinei muscle anteriorly, and moves posteriorly to attach to the anococcygeal raphae. Laterally the EAS connects with the transverse perinei muscle. New MRI/ultrasound work has suggested that the EAS muscle complex is actually a purse-string morphology, rather than a "donut" configuration. In this purse-string arrangement, the EAS musculature continues (with the transverse perinei muscle) to the contralateral attachment [4]. The concept of this configuration is further supported by the fact that both the anorectal angle changes and the coccyx is pulled anteriorly during contraction of the external anal sphincter.

Comprised of voluntary muscle fibers with resting tonicity, the EAS is innervated by sacral motor neurons that arise in Onuf's nucleus and travel through the pudendal nerve (S2–S4). This monosynaptic reflex creates resting sphincter tone. This tone is abolished with spinal anesthesia and impaired in disorders such as diabetes. The fact that EAS tone can be over-

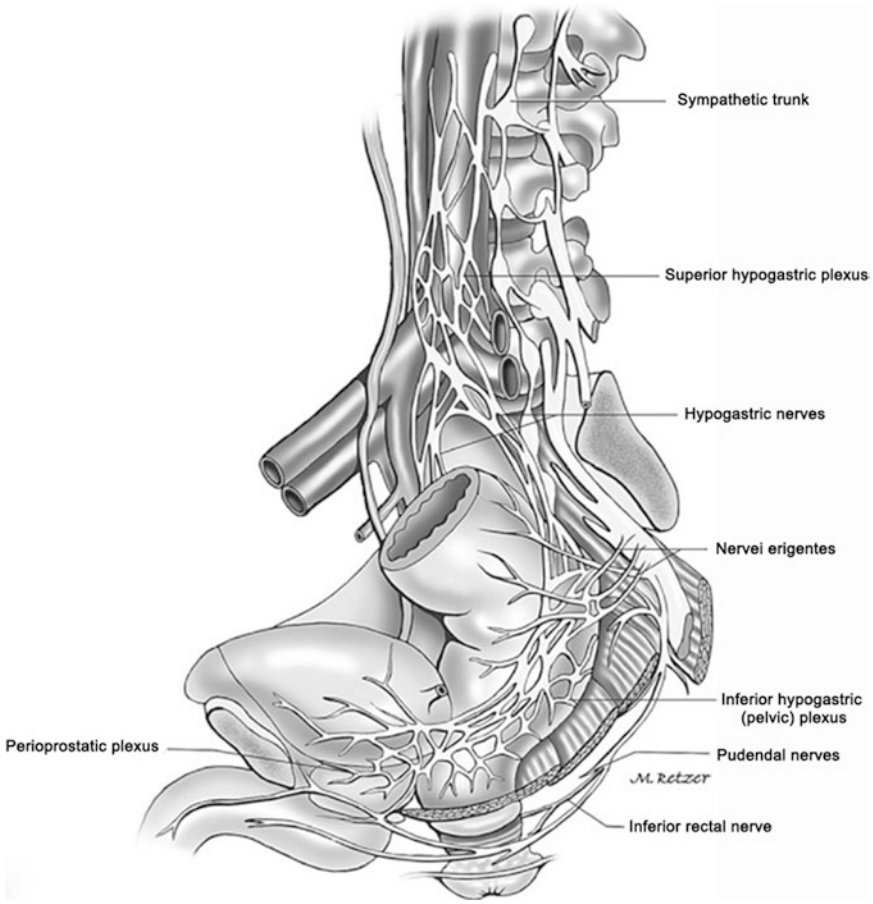

Fig. 2.3 Innervation of the posterior pelvic floor. With permission from Jorge JMN Habr-Gama A. Anatomy and Embryology of the Colon, Rectum, and Anus. In: Beck DE, Wexner SD, David E. Beck, Steven D. Wexner, Hull TL, Roberts PL, Senagore AJ, Stamos JM, Steele SR, eds. The ASCRS Manual of Colon and Rectal Surgery, 2nd Edn. Springer, New York, 2014; pp:1–25

come by spinal anesthesia suggests that there may be a spinal reflex that helps to maintain external anal sphincter tone, and thus continence. A study by Broens et al. suggests that, in addition to previously documented reflexes and autonomic and somatic innervation, an "external sphincter continence reflex" exists, due to a spinal reflex that utilizes receptors in the mucosa and submucosa of the distal anal canal. The concept of this reflex was supported by the knowledge that the incidence of incontinence increases following mucosectomy and that patients with high spinal cord injuries (above the level of T5) can maintain some degree of continence [5].

The pudendal nerve (via S3-S4) innervates the puborectalis and levator ani muscles. In cases of severe pudendal nerve injury, such as a traumatic injury, both the EAS and pelvic floor musculature can be affected and fecal incontinence may result.

The sensory function of the rectum is a very important component to consider. This allows for the discrimination of solid, versus liquid, versus gas components of stool. These specialized cells and their attendant histologic arrangement are constructed within the distal rectum. The distal rectum extends from approximately 2.5–15 mm above the anal valves and can sense prick, light touch, hot and cold. Again, it is this sensory ability that helps discriminate between gaseous flatus and solid stool. Above this level, the rectum is only able to sense distention. The inferior rectal branch of the pudendal nerve is responsible for this sensory ability in the lower rectum. Stretch receptors in the rectal wall and surrounding pelvic fascia, via S2-4 parasympathetic fibers, contribute to higher rectal sensation.

In addition to the musculature and neurologic components of continence, the physical orientation of the rectum and associated pelvic floor muscles creates an orientation, called the *Anorectal Angle* that ultimately develops a valve-like structure, believed to assist in continence. The puborectalis muscle, by nature of its attachments, pulls the rectum anteriorly. This causes apposition of the rectal mucosa. As intraabdominal pressure increases, pushing down on the rectum and contents, the anorectal angle becomes more acute, causing a tightening of the valve-structure. This development relies on the rectal reservoir to tolerate the rising volume of fecal material traveling into the rectum. These complex and mixed voluntary and involuntary movements facilitate the development of a stripping wave, which moves the stool from the rectum and relaxes the pelvic floor muscles and the anus resulting in stool evacuation [6, 7]. Figure 2.2 shows the anorectal angle at rest, constriction and defecation. There comes a point where rectal capacity will be reached, and overflow incontinence may ensue if defecation is not initiated voluntarily.

Anorectal Reflexes

There are multiple reflexes that assist in the maintenance of continence. The *cutaneous-anal reflex* is a contraction of the anal sphincter with scratching of the perianal skin. An S4 sensory and motor efferent and afferent from the pudendal nerve is responsible for this reflex. Due to the rapid fatigability of the anal sphincter, it is important to test this particular reflex early in sphincter testing proceedings. Patients suffering from cauda equina syndrome lack this reflex.

The *bulbocavernosus reflex* is the sensation of pelvic floor contraction with the squeezing of the glans of the penis or the clitoris. This reflex is perpetuated through the pudendal nerve (S2–S4).

The **R**ectal **A**nal **I**nhibitory **R**eflex (RAIR) is the act of IAS relaxation in response to distention of the rectum. RAIR plays an important role in fine adjustments of continence. The reflex starts with fecal material or flatus coming in contact with sensory receptors in the upper anal canal. These receptors sample the fecal contents and create a sense of awareness with regard to the contents (flatus versus stool). This reflex is responsible for one's ability to pass flatus and stool independently. This reflex is absent in patients with Hirschsprung's disease, and may be damaged by an overgenerous myotomy in a lateral internal sphincterotomy. While the relaxation of the IAS is a temporary phenomenon, thus preventing fecal incontinence, the IAS tone does not return fully to baseline, but rather a new "plateau pressure" which continues to maintain contraction of the sphincter complex [3].

The *Rectal Anal Excitatory Reflex*, in contrast, is the contraction of the EAS in response to rectal distention. This reflex prevents involuntary fecal incontinence, and is regulated by the splanchnic nerves (S2–S4 parasympathetic fibers). These splanchnic nerves may be considered to be associated with the pudendal nerve and as would follow, a pudendal nerve block will remove this reflex. The *cough reflex* is a polysynaptic reflex that develops in response to a rapid increase in intraabdominal pressure. This reflex causes contraction of the anal sphincters, thus preventing fecal incontinence during coughing, laughing, shouting, or any other activity causing a rapid increase in intraabdominal pressure.

Pelvic Floor Dysfunction

Physiology of the pelvic floor combines sensory input, anatomical variants, mechanical factors, and reflexes. Correlating physiologic properties with clinical pathology and patient symptoms has led to improvements in testing for specific defects. Pelvic floor dysfunctions (PFD) is common, affecting up to 10–15 % of the population with even higher incidence in women and the elderly, with significant impact on quality of life, emotional well-being, and ability to actively participate in society [8]. Etiologic risk factors for PFD include: (1) vaginal parity, (2) aging, (3) hormonal status, (4) pelvic surgery, (5) collagen diseases, (6) toilet training before complete myelination of the spinal tracts, and (7) depression. Clinical improvement requires a well-thought-out strategy. Definitive management and complete resolution of incontinence is rare and a combination of treatment options is essential for improving the quality of life in patients.

As with most clinical pathologies a complete history is essential in patients with pelvic floor dysfunction. Even more important is asking the right questions. Detailed questions about bowel habits may help to differentiate between pelvic floor dysfunction and other more concerning and immediate pathology such as obstruction due to cancer. Repeated visits to the bathroom with incomplete emptying, self-assistance in defecation with support of the perineum or posterior vaginal wall, and soiling in the absence of urge incontinence are all signs of pelvic floor pathology and are more chronic in nature. In addition, repeated questionnaires can be beneficial to systematically obtain history as well as provide quality of life information that may be followed through treatment modalities. There are multiple fecal incontinence scoring systems which are used to grade and categorize both the subjective and objective effects of incontinence.

Patients with pelvic floor dysfunction can usually be divided into one of two broad categories. The first and largest group is comprised of parous women suffering from long-term sequelae of pregnancy and childbirth. *The second* is men and nulliparous women who suffer from prior surgical intervention, connective tissue disorders, neuromuscular disorders, including adult neuromuscular sequelae of Hinman's syndrome or from more psychological and behavioral problems. The latter group tends to be misdiagnosed with irritable bowel syndrome (IBS) although significant life events such as physical or emotional abuse, eating disorders, and other psychological stress have been shown to have a strong association with pelvic floor dysfunction later in life. The most common conditions include constipation, obstructed defecation, fecal incontinence, and pelvic pain. Each of these can be very limiting to patient's ability to function normally in the world.

Immediate surgical intervention has lost appeal due to disappointing long-term improvement and morbidity. The 1980s utilized subtotal colectomy for slow transit constipation and postanal repair for incontinence. Therapeutic options have increased in the last decade with sacral nerve stimulation providing a tremendous option and preventing the need for a stoma or severely limiting conservative therapy. Surgical correction of rectal intussusception has also shown improvement in symptoms leading to further study on a perhaps unrecognized pathogenesis of incontinence. Providing patients with a multidisciplinary approach has shown improvements in quality of life. Early conservative management utilizes the assistance of a pelvic floor physiologist and specialist nurse to correctly diagnose underlying

pathology. Even after a patient has been diagnosed with a defecatory problem, conservative treatment (increased dietary fibers, removal of constipating medications, increased fluid intake) should be attempted first. If this does not improve the patient's condition, then laxatives (both osmotic and stimulant) should be trialed. If the patient continues to suffer from dysfunctional defecation, then more testing should be performed [9]. Surgical input is recommended early from a colon and rectal surgeon and a radiologist with expert training in defecography and pelvic anatomy.

Constipation

Chronic constipation can be described as reduced frequency or difficulty of defecation. There are two major types of constipation: slow transit and outlet obstruction. Slow transit is associated with decreased motility of fecal material within the colon. Outlet obstruction occurs when the patient has difficulty evacuating contents from the rectum. The normal act of defecation includes performance of a Valsalva maneuver, with an increase in intra-abdominal and rectal pressures, as well as relaxation of the rectal and anal sphincters.

Functional constipation is related to slow transit colonic constipation, which is rather uncommon, and evacuation problems or a combination in which the motility of the colon slows over time secondary to difficulty in evacuation. Patients with slow transit or true colonic motility disorders do not experience the same urge or call to stool. Bloating, heaviness, and abdominal discomfort become more apparent. High-grade internal rectal prolapse has been chronically misdiagnosed as IBS with delayed surgical consultation. Most patients with a functional disorder have fecal incontinence, need for digitization, incomplete evacuation, and toilet revisiting [1].

There is a potential third type of constipation that is essentially a combination of anatomic obstructive constipation and functional constipation. Essentially, this type of patient exhibits characteristics of both. It is the patient with levator based hypertonic pelvic floor dysfunction, with the hypertonicity focused at the level of the "puborectalis muscle." Here, the hypertonicity of the puborectalis changes the anorectal angle and makes it more acute thereby creating a type of anatomic obstructive constipatory effect. This type of obstruction then can lead to an exaggerated beta III effect which in turns slows down colonic transit time and further precipitates constipation (Figs. 2.4, 2.5, and 2.6).

Fecal Incontinence

Fecal incontinence can be described as either associated with urgency or occur as a passive event. Incontinence is the inability to defer the passage of gas, liquid or solid stool until the desired time. Fecal urge incontinence (FUI or *active incontinence*) is the loss of stool despite efforts to control it. It may be associated with inflammatory changes in the rectum such as prostatitis as well as carcinoma, or may be associated with a problem with the external anal sphincter. Disastrous events of

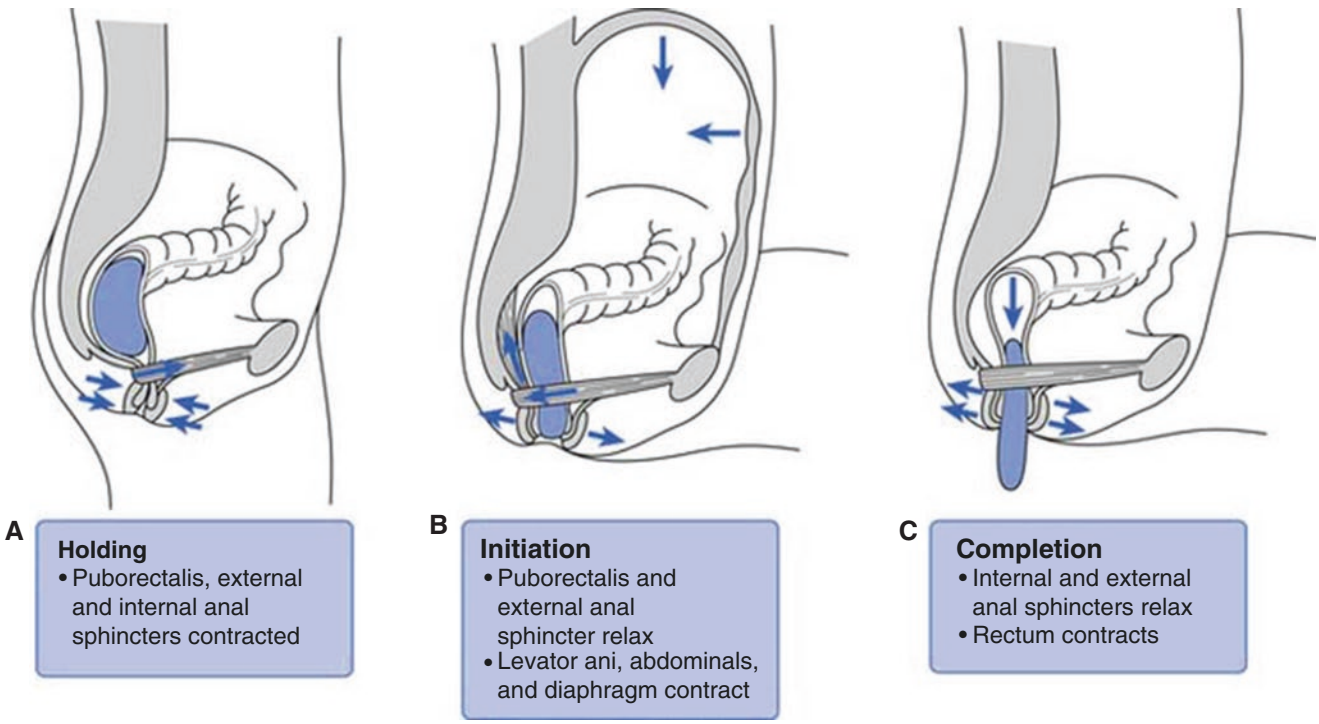

Fig. 2.4 Anorectal angle. Modified from Gianna Rodriguez, John C. King, Steven A. Stiens. Dysfunction and Rehabilitation. http://clinicalgate.com/neurogenic-bowel-dysfunction-and-rehabilitation/

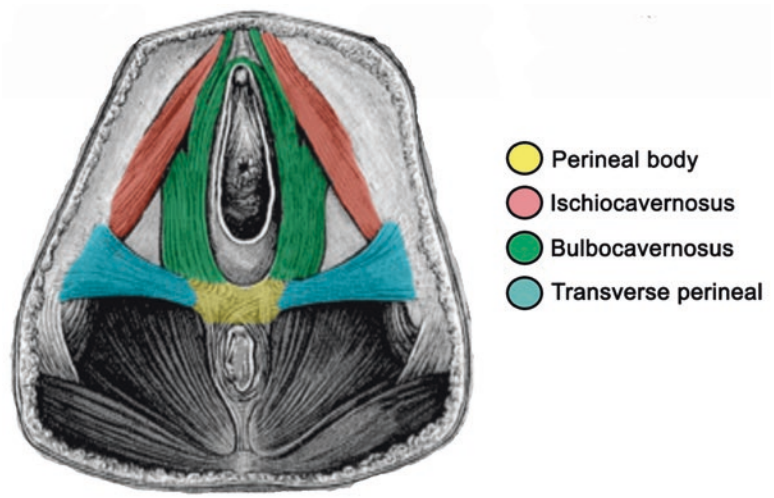

Fig. 2.5 Perineal body with pelvic floor (*anterior* and *posterior*). Modified from http://teachmeanatomy.info

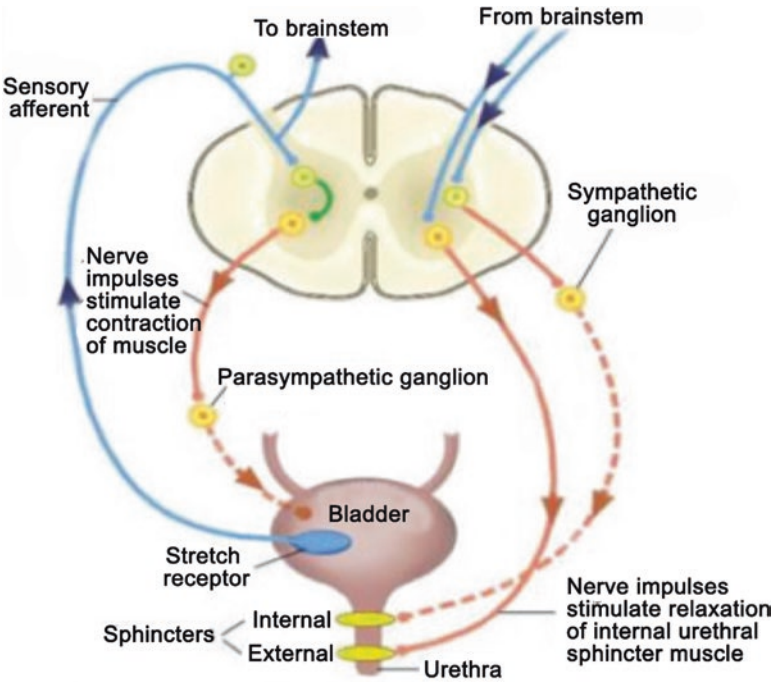

Fig. 2.6 Neuromuscular aspects of voiding

high volume incontinence can be socially crippling and associated with high levels of anxiety. Passive Incontinence is the loss of stool without awareness and is more often a physiologic outcome from a deficient internal anal sphincter or anatomic deformity. True anorectal prolapse or rectal intussusception results in a variable and unpredictable incontinence often resulting in underwear staining or release of small pellets rather than high volume. Abnormal rectal sensation can lead to incontinence due to hyperacute sensation in inflammatory pathology and blunted sensation leading to Overflow Incontinence.

Mixed symptoms may also occur as a consequence of passive loss of stool that is retained in the rectum or rectocele as a result of incomplete evacuation. This seems counterintuitive and results in misdiagnosis with only marginally reduced sphincter tone. These patients tend to benefit most from laparoscopic ventral rectopexy with improvement in continence.

Anal canal pressure is the major determinant of the strength of the anal continence mechanism. Pressures can be measured using anatomic and functional studies. Resting pressures are mostly related to the internal anal sphincter and voluntary squeeze pressure increase can be attributed to the external anal sphincter. At the same time, the anatomical location and association of the IAS, EAS, and puborectalis can be isolated based on pressure readings along the length of the anal canal. Pressure studies have found that fecal

incontinence is rarely associated with an isolated dysfunction but rather the degree of incontinence is associated with a composite effect of damage to the three continence muscles. Biofeedback therapy has resulted in improvement of fecal incontinence symptoms associated with improvement in levator ani function, rather than improved contraction of the IAS or EAS.

Pelvic Pain

Chronic pelvic pain is a challenging frustrating and usually multidisciplinary clinical issue. The component part that make up the bulk of chronic pelvic pain syndromes are addressed in other chapters in this text. This includes a special chapter on the biology of chronic pelvic pain. Nevertheless, suffice it to say that most patients have been assessed by multiple specialists including urologists, gynecologists, and colon and rectal surgeons. They often seek assistance from pain management specialists with variable improvement on a socially limiting chronic condition. The original pathophysiologic source of their pain (endometriosis, prolapse, postpartum pain) may become lost in a long series of surgical interventions that if not treated with care, logic and sophistication may actually make the whole process worse. Please refer to the other sections in this book for more relevant details on the pathogenesis and treatment on chronic pelvic pain.

Physiologic Testing (Fecal Incontinence v. Constipation)

A variety of testing options now exist for definitive diagnosis of physiologic deficits leading to fecal incontinence and constipation. A combination of defects may contribute to the clinical symptoms, including fecal retention, prolapse, and incontinence. Less invasive testing methods are being developed with increased use of imaging modalities rather than direct physiologic testing with proven success. Diagnostic testing now includes:

1. Anorectal manometry
2. Balloon expulsion testing
3. Saline continence testing
4. Neurophysiologic testing
5. Pelvic floor imaging
6. Dynamic functional testing

Anorectal physiologic testing concentrates on two major areas: neurologic function and muscular function. If one or both of these is altered, clinical pathology occurs as retention, constipation, incontinence, or an array of symptoms. Muscles have a relatively simple function: they shorten from the insertion point towards the origin. In the case of the pelvic floor, the pubococcygeus, iliococcygeus, and ischiococcygeus contracting result in the coccyx moving anteriorly (ventrally) toward the pubic bone, thus transforming a basin into a dome and lifting the contents of the abdomen, providing support. Laxity of this muscular support results in perineal descent and may lead to pelvic floor dysfunction [10].

Anorectal Manometry

The techniques for anorectal manometry have evolved drastically over the years. Such testing has progressed from balloons or water sacs to water perfusion and most recently microtransducers. Manometry measures the pressures within the anal canal and distal rectum that provides information about the function of the internal and external anal sphincters. Anal manometry testing is most useful in evaluation of fecal incontinence with suspected sphincter dysfunction. The testing involves an anorectal probe, pressure-recording device, balloon for inflation within the rectum, and a monitoring system. Stool evacuation or enema before the test is optimal to prevent interference of the recordings. Anal canal pressures are then measured using the pull-through technique that creates a reflex sphincter contraction. The probe begins measurements at 6 cm and is subsequently removed at one-centimeter increments from the rectum to the anal verge [11–14].

Pressures that may be recorded during anorectal manometry include squeeze pressure, which is attributed to an increase in pressure as the external anal sphincter is voluntarily contracted, as well as resting pressures. *Resting pressures* are usually around 40–70 mmHg and the majority comes from the IAS. If IAS is compromised, the mean pressure may be lower. *Squeeze pressures* are normally 2–3 times the baseline resting pressure and the EAS is the main contributor. Obstetric and traumatic defects can result in decreased squeeze pressure.

New 3D high definition anorectal manometry (3DHRAM) has the advantage of providing a pressure recording over the entire length and circumference of the anal canal, allowing a more useful physiological assessment of anorectal function. One study with 3DHRAM established normal values in asymptomatic volunteers. Increasing age was associated with lower maximum resting pressure, mean resting pressure, and maximum squeeze pressure [15]. This study also showed that there are significant gender differences concerning squeeze pattern—maximum squeeze pressure, residual anal pressure, and intrarectal pressure were significantly higher in males compared to females [15].

The *cough reflex* can be assessed with a rapid increase in intraabdominal pressure with associated increase in contraction of the EAS to maintain continence. A sphincter defect or innervation injury may result in a weaker cough reflex response. *Rectal sensation* can be measured by distending the rectal balloon and assessing the patient's response including

first sensation of urge and maximal tolerable volume. This gives the clinician insight into the sensory perception of the patient. Rectal hypersensitivity is associated with fecal incontinence with increased frequency of defecation. Incontinent patients may have hyposensitivity and suffer from passive incontinence from overflow [16–20].

Balloon Expulsion Testing

Balloon expulsion testing can be used to evaluate rectal expulsion ability by inflation of a water-filled rectal balloon. This test, when performed correctly, should be able to uncover those patients who suffer with obstructed defecation. Normal patients should be able to expel a balloon containing 50–150 mL of water. Patients with enlarged rectums may have normal range of intrarectal pressure, but are unable to expel the balloon. This study may assist in evaluation of a patient with a hypertonic nonrelaxing pelvic floor muscle tone or even spasm in conjunction with other modalities [21, 22].

Saline Continence Testing

Saline continence testing evaluates the ability of the sphincters to remain continent during continuous infusion of saline into the rectum. Normal patients can accommodate approximately 1.5 L of saline without significant leakage. Patients with weak sphincter function or reduced rectal compliance can begin to leak with as little as 250–600 mL [7, 23–26].

Neurophysiologic Testing (PNTML & PFM EMG)

Neurophysiologic testing includes *Pudendal Nerve Terminal Motor Latency* (PNTML) and Pelvic Floor Muscle *Electromyography (PFM EMG)*. PNTML sends electrophysiologic impulses out to follow the course of the pudendal nerve and then return along its "reflex pathway," the Bulbocavernosus Reflex (BCR). The time course to complete this reflex is measured. The pudendal nerve innervates the EAS, urethral sphincter, perineal musculature, mucosa of the anal canal, and the perineal skin. Nerve conduction velocity can be measured with a disposable, finger-mounted electrode (the St. Mark's Electrode) placed in the rectum with stimulating and recording components placed near the ischial spine. The time for response at the level of the EAS is measured and is normally 2.0 ms. The other way to measure PNTML is to use a device that can electrophysiologically stimulate the more distal aspects of the pudendal nerve (the dorsal genital branches) and then follow the course through the sacrum and back to the recording electrodes anteriorly. Naturally, since the impulse is traveling a much longer distance, the time course is also increased. Normal values for this more anterior testing scenario vary from 35 to 50 ms. It is important to remember that PNTML cannot be interpreted in a vacuum. This is a complementary tool in the physiologic evaluation of anorectal function especially in patients with known neuropathy or injury to the pudendal nerve [27, 28].

Pelvic Floor Muscle Electromyography (PFM EMG) samples activity of the striated pelvic floor muscles and is primarily used to identify EAS activity. Needle EMGs can provide information on nerve injury (denervation–reinnervation potentials) and aid in locating the muscle in the EAS although this has been largely replaced by endoanal ultrasound. Patients with fecal incontinence have high fiber density and longer motor unit potentials than in controls. EMG may be useful in locating the striated EAS muscles prior to surgical intervention in imperforate anus [29–32]. Many colon and rectal surgeons utilize EMG for functional information and endoanal ultrasound for anatomic information.

Pelvic Floor Imaging

Endo-anal ultrasound is useful for imaging the sphincters and detecting defects in the IAS and/or EAS. It is one of the diagnostic cornerstones for evaluation of patients with pelvic floor dysfunction, especially incontinence. This may aid in decisions regarding surgical repair and preoperative planning [33–36]. Ultrasonography has emerged as a simple technique that is subject friendly, inexpensive and may be performed in an office setting.

Advancements in pelvic floor imaging have allowed new insight into the function of the pelvic floor muscles. Through the use of MRI, CT, and 3D ultrasound, there is now a better understanding of the dynamic processes that occur during defecation. Changes seen in the size of the pelvic floor hiatus are related to the puborectalis muscle and reflect the constrictor function. Ascent and descent of the pelvic floor as well as craniocaudal movements of the anorectal angle are predominately related to the contraction and relaxation of the pubococcygeus, iliococcygeus, and ischiococcygeus muscles [1].

Dynamic Functional Testing (Defecography)

Dynamic functional testing can be achieved with fluoroscopic defecography or MR defecography. The latter has been shown to demonstrate more detailed anatomic information and be more accurate at diagnosis of intussusception although both may be useful in detection of anorectal angles, rectal emptying, rectal intussusception, rectal prolapse, and rectocele. During defecography, contrast is used to fill the rectum and is most useful for evaluation of outlet obstruction and prolapse. Patients may limit the study with false positive

results due to embarrassment and inability to relax the pelvic floor. The ability to relax the puborectalis muscle and increase the anorectal angle from 75 to 90° at rest to 110–180° is necessary for evacuation [37–40].

Posterior Physiology: In Conclusion

The normal functioning of bowel continence relies on the complex interactions of various neural pathways with the rectum and anus and the pelvic floor. A thorough comprehension of these relationships is important in the diagnostic and therapeutic approach to the patients who have pelvic floor dysfunction.

The Perineum and Perineal Body

The perineum is a diamond shaped area between the coccyx posteriorly and the pubis anteriorly. This area is flanked by the ischial spines laterally. The diamond is further divided into two triangles, anterior and posterior. The anterior triangle is known as the urogenital triangle while the posterior triangle is known as the anal triangle. The perineal body, also known as the central tendon of perineum is a pyramidal fibromuscular mass of tissue located in the middle line of the perineum at the junction between the anterior and posterior triangles. The location of perineal body is different in males and females. In males, it is found between the bulb of the penis and the anus, while in females it is found between the vagina and anus, and about 1.25 cm in front of the latter. Most importantly, it must be noted that the perineal body is essential for the integrity of the pelvic floor musculature.

The pelvic floor musculature is integral in maintaining stability and functionality of the entire anterior abdominal wall with the posterior wall. Without an intact, strong pelvic floor muscular system our entire structure would fall apart. This strong, intact pelvic floor muscular system is made up of two (2) layers of expansive musculature with robust puborectalis muscle in between, almost making up a third layer. This large volume of musculature attaches to bone and ligaments anteriorly, posteriorly and laterally. However, for these large muscles to function well in both anterior and posterior positions there must be a central anchoring point. Obviously, an island of bone cannot really exist in that position. Consequently, a solid, strong, fibrous piece of tissue in the middle of the pelvis MUST exist. This tendinous center, or perineal body, is a median, fibromuscular mass between the urogenital and anal triangles. Several muscles and fasciae are anchored to it, including the levator ani and the external anal sphincter. Perineal body is essential for the integrity of the pelvic floor, especially in females. It provides attachment to the following muscles:

- EAS muscle
- Bulbospongiosus muscle
- Superficial transverse perineal muscle
- Levator ani muscle
- EUS
- Deep transverse perineal muscle

Understanding the relationships of its components is crucial for successful pelvic reconstructive surgical procedure. Now, the best way to understand this area from an anatomical perspective, as well as to help direct any surgical repair is to utilize MRI. Today in 2015, the use of advanced thin-slice MR images to identify structures within this region, define their 3 dimensional location, and provide a framework for visualizing this region's complex anatomy.

Anterior Pelvic Floor Physiology

Pelvic Floor and Urinary Sphincters

The muscular pelvic floor consists of both superficial and deep muscles, both of which play a role in continence. The superficial pelvic floor muscles most relevant to anal canal function are the (1) external anal sphincter, (2) perineal body, and the (3) puboperineal transverse muscles. The deep pelvic floor muscles consist of the (1) pubococcygeus, (2) iliococcygeus, (3) coccygeus, and (4) puborectalis muscles (Fig. 2.1). These muscles originate at the pectinate line of the pubic bone and obturator internus fascia, and insert at the coccyx. The puborectalis muscle may be more accurately described as located between the superficial and deep layers. The muscle originates at the inferior pubic ramus, tracks posteriorly, wrapping around the rectum as it descends, and attaches to the contralateral pubic ramus.

Lower Urinary Tract Physiology

The lower urinary tract is predominantly comprised of the bladder and the urethra. The nature of the bladder is essentially storage. In fact, it is the major storage organ for all liquid waste (urine) that is generated by our body's tissue. The base of the bladder begins to funnel into a **"bladder neck"** area which then segues into the urethra. It is this urethra that is a tubular conduit designed to facilitate the expulsion of urine. Now, at the bladder neck, there is a circular collar like smooth muscle known as the "internal urinary sphincter" (IUS). It is important to note here that the IUS is completely involuntary. There is no volitional control of continence at the bladder neck area. As one moves more distally down the urethra, there is a second collar like condensation of muscle tissue. The condensation of muscular tissue at this level is

known as the external urinary sphincter (EUS). The EUS is more accurately described as a rhabdosphincter. This rhabdosphincter is composed in part of striated muscle. Moreover, a significant portion of this musculature is under volitional control. The bladder neck and proximal urethra down to the most distal portion of the EUS, which includes these two sphincter rich areas is known as the continence zone (CZ). If one looks at this area as a whole, that is, the bladder muscle and the CZ. If taken together, their function is twofold, (1) urine storage and (2) urine expulsion (voiding). The storage phase (of urine) requires low bladder pressure that does not exceed bladder outlet resistance. Voiding requires (1) intact neurological pathways which govern volitional triggers for voiding, combined with (2) autonomic bladder muscle contraction, (3) adequate bladder contractility, and possibly, the most important aspect of all which is (4) the coordinated relaxation of the bladder outlet and pelvic floor.

Physiology of Voiding

The basic process of micturition can be broken down into two broad categories or phases which are subsequently broken down into a total of six steps. The two phases are basically (1) urine storage and (2) bladder emptying. The six steps are as follows: (1) Urine is made at the kidneys and stored in the bladder. (2) The bladder fills with urine allowing for increasing volumes at low bladder pressures. (3) At a certain volume, the viscoelastic properties of the bladder wall muscle are met and a baroreceptor mediated sensory signal of fullness is generated. (4) The rhabdosphincter muscle voluntarily relaxes followed by the remainder of the CZ, (5) The bladder muscle (detrusor) then contracts in a coordinated fashion, (6) The bladder is emptied through the urethra and urine is removed from the body.

Now it is important to realize that even though we can break this process down into these six discreet steps as articulately, accurately, and logically as we have, that it is still over simplified. The physiology of voiding is an extremely complex process, beyond these six steps there are elaborate layers of control. The text below attempts to better explain the micturition process, and there is more depth in the graphic above.

The bladder is composed of bands of interlaced smooth muscle (detrusor). The innervation of the body of the bladder is different from that of the bladder neck. The body is rich in beta adrenergic receptors. These receptors are stimulated by the sympathetic component of the autonomic nervous system (ANS). Beta stimulation, via fibers of the hypogastric nerve, suppresses contraction of the detrusor. Conversely, parasympathetic stimulation, by fibers in the pelvic nerve, causes the detrusor to contract. Sympathetic stimulation accounts for baseline bladder muscle tone which is predominant during bladder filling, and the parasympathetic stimulation causes emptying.

The continence zone (CZ) which is composed of two sphincters control the bladder outlet. The internal sphincter is composed of smooth muscle like the detrusor and extends into the bladder neck. Like the detrusor, the internal sphincter is controlled by the ANS and is normally closed. The primary receptors in the bladder neck are alpha-adrenergic. Sympathetic stimulation of these alpha receptors, via fibers in the hypogastric nerve, contributes to urinary continence. The external sphincter is histologically different from the detrusor and internal sphincter. It is striated muscle. Like skeletal muscle, it's under voluntary control. It receives its innervation from the pudendal nerve, arising from the ventral horns of the sacral spinal cord. During micturition, supraspinal centers block stimulation by the hypogastric and pudendal nerves. This relaxes the internal and external sphincters and removes the *sympathetic* inhibition at the level of the detrusor to allow for unopposed parasympathetic tone via muscarinic receptor activation. The result is unobstructed passage of urine when the detrusor contracts.

Now bladder filling begins with the kidneys. Blood is filtered and urine is made at which point the urine is passed on to the ureters. The ureters then course down from the kidneys down along the ventral surface of the psoas and beneath the uterine pedicle to finally enter the bladder into the trigone via the intramural ureter between the layers of the detrusor. The ureters use its peristaltic activity to propel urine into the bladder. The bladder then passively expands to accommodate increasing volumes of urine at low pressures. As the bladder expands and intravesical pressure rises, the ureters are compressed between the layers of muscle, creating a valve mechanism. This valve mechanism limits the backflow of urine. The normal adult bladder can hold about 500 cc of urine. After emptying, the bladder may still retain about 50 cc residual volume. At about 150 cc of volume, stretch receptors in the detrusor begin signaling the CNS via afferent nerves; at 400 cc we are "seeking" an appropriate toilet.

Physiology of Urinary Continence

Now it is extremely important to understand the mechanism or process of urinary continence. Moreover, this process of urinary continence is *NOT* synonymous with the CZ as described above. The CZ is just one of the three major components required to maintain urinary continence. These are, respectively, (1) proximal urethral support, (2) the CZ, i.e. internal sphincter activity, and external sphincter function, (3) bladder muscle stability with intravesical pressure all contribute to continence. Any one alone may not be able to keep a patient dry. The pressures generated during a cough may easily overcome the internal and external sphincters closing powers, and the normal supportive mechanism works in such a way as to increase closure during increases in abdominal

pressure. Normal support, conversely, is not sufficient in and of itself to maintain continence, and must have sufficient resting sphincteric activity along with bladder muscle stability to be effective. When one element is abnormal, the other mechanisms may be able to compensate and maintain continence. It is because there are these several interdependent parts of the continence mechanism that no single urodynamic parameter is predictive of continence. Each different etiologic type of incontinence reflects the malfunction of one of the anatomic or physiologic components of continence. Therefore, a knowledge of this mechanism's structure and function is fundamental to an understanding of this common clinical problem. Technologic advances in the neuro-diagnostic assessment of the lower urinary tract have allowed for a much more sophisticated understanding of the process and more importantly, the ability to create and effective interventional strategy to control urine leakage.

Anterior Physiology: In Conclusion

The normal functioning of bladder continence relies on the complex interactions of various neural pathways with the bladder, CZ, and the pelvic floor. In the normal condition, we are able to control where and when we void. This is largely because the CNS is able to suppress the sacral micturition reflex. If the sacral reflex is unrestrained, parasympathetic stimulation via the pelvic nerve causes detrusor contraction. Detrusor contraction is suppressed via the sympathetic side of the autonomic nervous system. More precisely, detrusor muscle stabilization is achieved with baseline *sympathetic* stimulation mediated through beta receptors via the hypogastric nerve. In response to afferent stimulation, the centers above the brainstem become aware of the need to void. If it is appropriate, the somatic and parasympathetic nervous system relaxes the external sphincter which in turn inhibits the sympathetic inhibitory effect on the bladder muscle. Finally, the end result is that the bladder contracts and urine is released.

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Pelvic Floor Dysfunction and Pelvic Surgery in the Elderly
An Integrated Approach
Gordon, D.A.; Katlic, M.R. (Eds.)
2017, XIII, 425 p. 180 illus., 117 illus. in color.,
Hardcover
ISBN: 978-1-4939-6552-6