Chapter 2
Anatomy and Physiology of Anorectal Prolapse

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Abstract The pathophysiology of hemorrhoids and obstructed defecation requires a sound knowledge of the structure and function of the pelvic floor. The essential structure of this area is discussed with a specific emphasis on applied anatomy. Current theories regarding the physiology of continence and defecation are described. These concepts are then applied to explain the underlying pathophysiology of hemorrhoids and obstructed defecation.

Introduction

Anatomy and physiology sections of medical textbooks are generally dismissed as rather dull and largely unnecessary starters to the main course of sought-after clinical material. However, knowledge of the anatomy and physiology of the anorectum is crucial to understanding how normal continence is maintained, the mechanisms of defecation, and the concepts underpinning the evolution of stapling techniques described later in this book.

This chapter is deliberately selective in concentrating on those aspects of applied anatomy and pathophysiology which are relevant to the stapling procedures used in the treatment of hemorrhoids, anorectal prolapse, and obstructed defecation syndrome (ODS).

Essential Anatomy

Anal Canal

The anal canal extends posteroinferiorly from the lower extremity of the rectum to the anus. It is normally 2–4 cm in length, and its superior part lies in the pelvic cavity. It is surrounded by the internal anal sphincter, which is the thickened inferior extension of the circular muscle of the rectum, and longitudinal muscle fibers derived from the longitudinal rectal muscle coat. The levator muscles condense around the uppermost anal canal to form the puborectalis sling. Inferiorly, the puborectalisfuses with the deep external sphincter, which is contiguous with the superficial external anal sphincter below. These voluntary muscles surround and enclose the internal sphincter and anal epithelium in a cylindrical fashion, but are separated from them by the intersphincteric space, which is a largely avascular anatomic plane of areolar tissue, containing a series of anal glands, and also longitudinal muscle fibers. The inferior part of the anal canal lies in the perineum and is surrounded by the superficial part of the external sphincter which, together with fibers of the longitudinal muscle, is responsible for forming radial corrugations at the point of attachment to the perianal skin (Fig. 2.1).

It is important to clarify definitions. The anatomic anal canal is defined as the region from the dentate
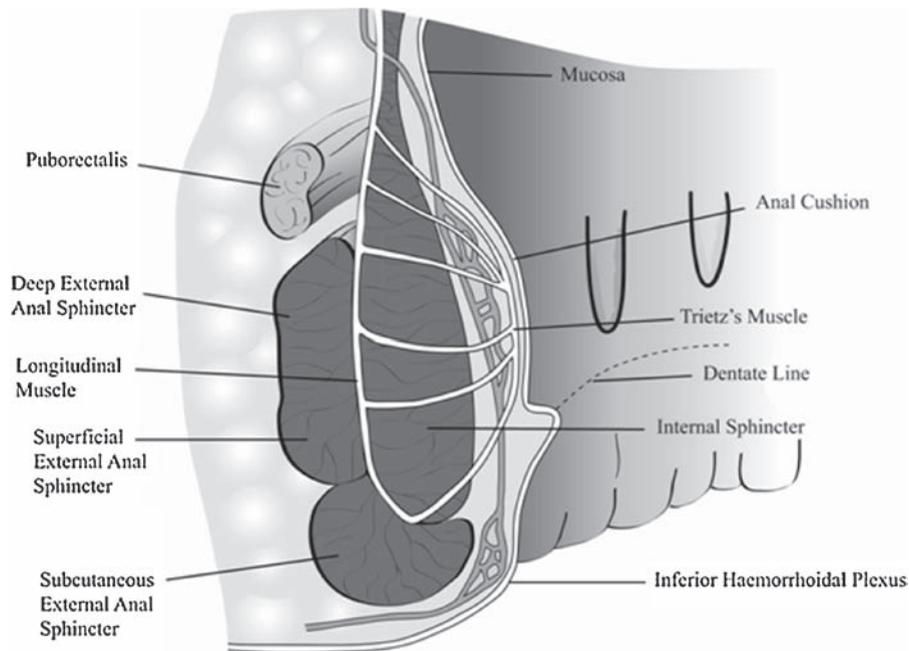
FIG. 2.1. Anatomy of the anal canal with particular reference to the structure of the anal cushions. Fibroelastic and muscular tissues form a scaffold that supports the vascular infrastructure of the cushions. Contraction leads to compression of the vascular anal cushions, increasing the luminal diameter of the anal canal and aiding evacuation. Rupture of the supporting scaffold results in prolapse of the cushions, failure of adequate compression during evacuation, and subsequent engorgement of the vascular component. Note also the fibers from the superficial external sphincter and the longitudinal muscle inserting into the perianal skin to form the anal skin corrugations

line to the anal verge, in contrast to the surgical anal canal which is the area extending from the anorectal junction to the anal verge.

Rectum

The junction of the sigmoid colon and rectum has always been a matter of debate between surgeons and anatomists. Although its distinction is of importance in rectal cancer surgery, the upper extent of the rectum is of limited relevance in the treatment of anorectal prolapse. A description of the lower rectum is highly relevant: its distal end is limited by the upper end of the anal canal, where the anorectal angle is directed posteriorly as it passes through the pelvic floor. This corresponds to the level of the puborectalis muscle. Active contraction of this muscle maintains the angle between the rectum and the anal canal, acting as a sling to pull the anorectal angle forward, and is the most important component of the continence mechanism (Fig. 2.2).

The rectum may be arbitrarily divided into three regions related to its coverings: the upper rectum is covered in peritoneum anteriorly and laterally, the middle third only anteriorly, and the lower third is devoid of a covering as the peritoneum is reflected forward onto the seminal vesicles and bladder in the male and vagina and uterus in the female. This forms a pocket of peritoneum, the pouch of Douglas, which is variable in its extent. The peritoneal reflection lies approximately 8 cm from the perineal skin in men and 5–8 cm in women. It is particularly deep in patients with a full-thickness rectal prolapse and in those patients with an enterocoele or sigmoidocoele. It is important to be aware of this when carrying out full-thickness rectal stapling techniques as incorporation of the peritoneum into the staple line increases the risk of small bowel or sigmoid colon being included in the staple line. Inclusion of the peritoneal lining in the staple line also carries a risk of peritonitis in the event of staple line dehiscence, when the rectal lumen will communicate directly with the pelvic cavity.
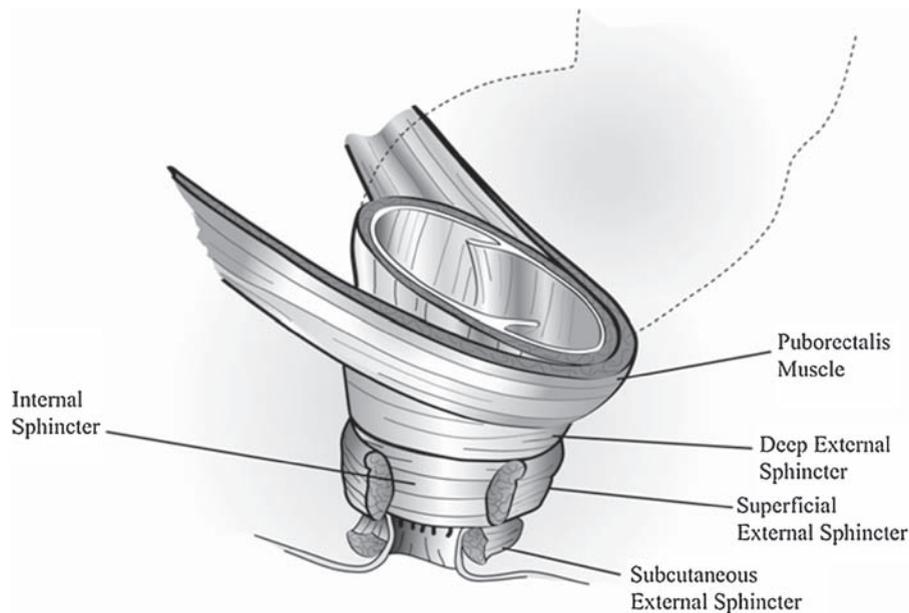
FIG. 2.2. Components of the anal sphincter. The puborectalis acts as a sling to pull the anorectal junction forward. This plays an important role in maintaining continence

A discrete layer of dense connective tissue, comprising collagen, smooth muscle, and elastin fibers, can be found between the rectum and the vagina, and has been well demonstrated by operative and cadaveric studies [1, 2]. The rectovaginal fascia extends from the cervix to the perineum, merging laterally into the fascial covering of the iliococcygeus and pubococcygeus muscles. It provides support for the rectal wall and, in the normal female, resists the effect of increased intra-abdominal pressure to form an anterior rectocele.

Posteriorly the rectum follows the curve of the sacrum, and as it passes downward and forward to meet the anal canal, it angulates sharply backward and inferiorly. This has significance for purse string insertion during stapling procedures, as there is a tendency to spiral the suture downward posteriorly, risking incorporation of part of the puborectalis muscle into the staple line. This may be a potential cause of postoperative pain.

Arterial and Venous Drainage

Anatomical descriptions of the arterial supply to the rectum and anal canal generally list five main arteries, which anastomose together to a variable extent.

Three of these arteries are usually encountered in various forms during mesorectal dissection. The inferior mesenteric artery continues as the superior rectal artery when it reaches the pelvic brim, and is the most consistent of these arteries, dividing into right and left branches at the level of S3. Two midrectal arteries (arising from the internal iliac arteries) are less consistent [3–5]. Two inferior rectal arteries are encountered in abdominoperineal resection, as they arise from the internal pudendal arteries high in the ischiorectal fossa.

A rich venous plexus surrounds the surface of the rectum in the submucosal plane and is relevant to stapling techniques. The plexus drains into the superior, middle, and inferior rectal veins, which follow the course of the arteries (Fig. 2.3). This is a well-known example of a portosystemic communication: the superior rectal vein drains into the portal system, whereas the middle and the inferior rectal veins enter the systemic system via the internal iliac veins. There is a free communication with other pelvic plexuses which explains an increased predisposition for engorgement and other hemorrhoidal symptoms in situations of increased pelvic blood flow, for example during pregnancy and, to a lesser extent, menstruation.
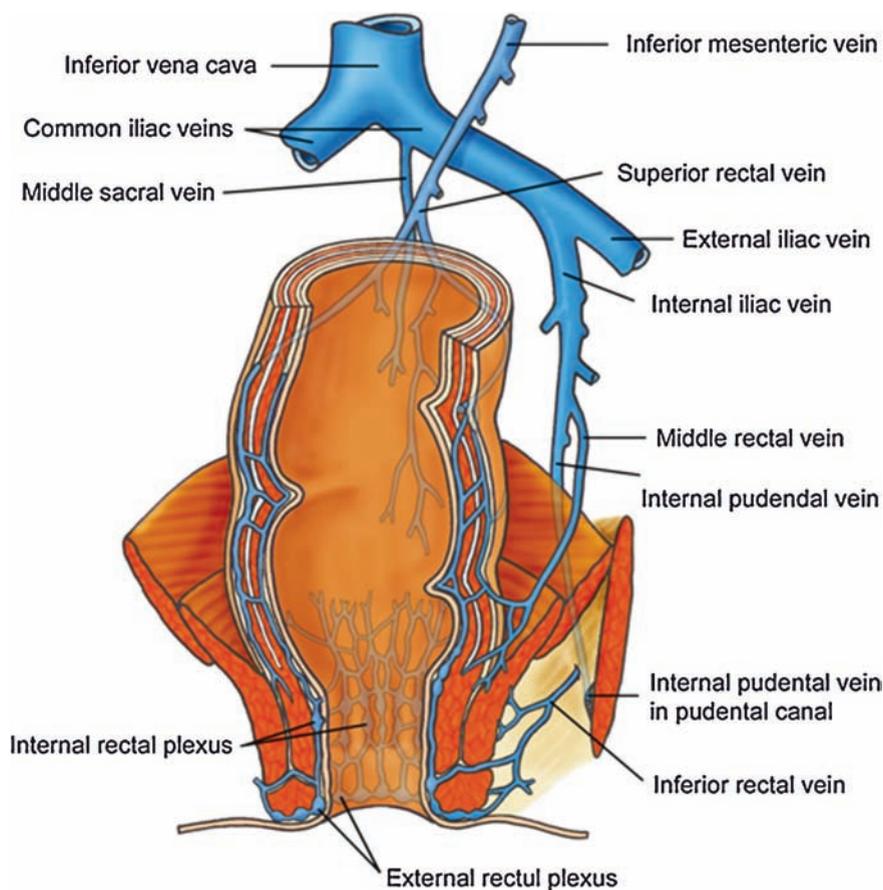
FIG. 2.3. Venous drainage of the rectum and anal canal. Note the rich venous submucosal plexus. Note also the drainage into portal and systemic systems and free communication with genital and visceral plexuses

Epithelial Lining

Skin extends into the distal anal canal as far as the lower border of the internal anal sphincter, and is lined by stratified squamous keratinizing epithelium, with sweat glands, hair follicles, and sebaceous glands. Stratified nonkeratinizing squamous epithelium extends from this level to the dentate line and is devoid of these structures. Immediately above the dentate line is the anal transition zone (ATZ), which is a layer of mixed stratified and columnar epithelium approximately 1 cm in length. The ATZ is important for anal sampling and the maintenance of continence (see later).

At the dentate line is a series of approximately twelve papillae, which sometimes enlarge and fibrose in response to anal fissures or hemorrhoidal complications, such as thrombosis. Anal glands drain into a series of ducts lined by squamous epithelium, and which cross the internal sphincter to open at the dentate line behind the papillae. These are thought to predispose to anal sepsis and fistula formation [6].

Longitudinal mucosal columns, usually situated in the left lateral, right posterior, and right anterior aspects of the upper anal canal and ending at the dentate line, arise from the anal cushions above.

Microstructure of the Anal Cushions

The elegant anatomical injection studies of Thomson demonstrated the structure and function of the anal cushions [4]. All the arteries to the rectum supply the hemorrhoidal cushions. The cushions consist of a fibroelastic and muscular network within

vascular lakes, which drain directly into the superior, middle, and inferior rectal veins at various levels within the confines of the anal sphincters. Uniquely, there is no intervening capillary network. Radiological and serial section histological techniques have shown that arteriovenous channels communicate directly with the venous lakes via a system of arterioles passing through the muscle wall of the distal rectum. This explains why hemorrhoidal bleeding is bright red. These complex channels, interlaced by a fibroelastic and muscular network derived from the longitudinal part of the internal sphincter (Fig. 2.1), function as a scaffold, providing architectural support for the cushions. The scaffold concept was originally described by Trietz more than 150 years ago.

Anal Sphincters, Pelvic Floor Muscles, and Ligaments

The circular muscle coat of the rectum extends and thickens as it enters the anal canal to form the internal anal sphincter. It ends as a well-defined palpable rounded edge immediately superior to the anal margin.

The longitudinal muscle of the rectum blends with pubococcygeus at the anorectal junction, but some fibers continue downward in the intersphincteric space and, diverging beyond the lower border of the external anal sphincter, attach to the skin of the perianal region to function as an anchor.

The external sphincter, puborectalis, and levator animaintain voluntary continence. Levator ani is a muscular diaphragm and supports the pelvic floor. It consists of a cradle-like sheet of muscle (the levator plate), which arises from various bony prominences around the pelvis, and is deficient in the midline where the pelvic viscera pass through. The perineal bodyand anococcygeal ligament lie anterior and posterior to the anal canal, respectively, to form insertions for levator ani, transverse perineii, and the external anal sphincter. These condensations anchor the anal canal to the pelvic bones (Fig. 2.4).

Levator ani has four component parts, of which puborectalis is the most prominent, forming a sling around the anorectal junction and contiguous with the external sphincter as a functioning unit for maintenance of continence.

There are three levels of support for the vagina [7, 8]:

1. The cardinal-uterosacral complex supports the cervix and vagina above the levator plate.
2. Lateral connections to the arcus tendinous fascia of the pelvis support the midvagina.
3. Connections to the perineal membrane anteriorly and the perineal body posteriorly also support the mid vagina.
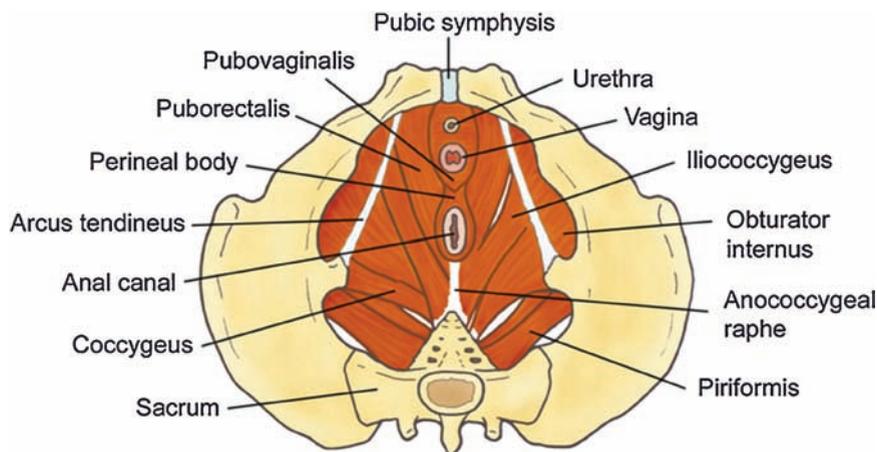

FIG. 2.4. Structure of the pelvic diaphragm. The perineal body is formed by fusion of the levator ani, transverse perineii, and the external sphincter muscles. Together with the anococcygeal ligament, they act as anchors to give the funnel shape of the pelvic diaphragm

Nerve Supply

Sympathetic and Parasympathetic Innervation

The anorectum and the pelvic floor are supplied by sympathetic, parasympathetic, and somatic nerve fibers. Sympathetic innervation is derived from the sympathetic trunk via the superior hypogastric nerve plexus. Parasympathetic fibers originate from ventral rami of the second, third, and often the fourth sacral nerves (S2–4). Sacral parasympathetic pathways to the colon have excitatory and inhibitory components [9]. Excitatory pathways play an important role in colonic propulsive activity, especially during defecation. Inhibitory pathways allow colonic volume to adapt to its contents, and also mediate descending inhibition, which initiates colonic relaxation ahead of a fecal bolus.

Somatic Innervation

Somatic branches travel mainly in the pudendal nerve to supply motor fibers, mainly voluntary, to the external anal sphincter and levator ani, and also sensory fibers to the anus.

Anorectal Sensation

Modalities of anal sensation can be precisely defined [10]. The anal canal is extremely sensitive to touch, pain, temperature, and movement. For many of these stimuli, sensitivity is greater than that of the perianal skin, which has a similar level of sensitivity to the dorsum of the hand. Sensory perception varies throughout the anal canal and is maximal within the ATZ. Above the ATZ, sensation is similar to that within the rectum.. Potentially painful stimuli, such as band ligation or PPH, must therefore be carried out well above the upper limit of the ATZ. This upper limit can be very variable.

In contrast to anal sensation, certain modalities of rectal sensation are indistinct. The rectum is insensitive to stimuli such as pain or touch, but sensitive to distension. Distension of a rectal balloon gives a sensation of the need to defecate particularly in the ampulla. The course of the sensory fibers detecting distension is unclear, but they probably pass through the inferior hypogastric plexus to the spinal cord and are responsible for the rectoanal inhibitory reflex. Evidence from patients who have undergone rectal excision and coloanal anastomosis but who still have sensation to distension suggests that nerve endings are present in the surrounding pelvic floor structures [11]. Indistinct rectal sensation for pain allows surgery in this area to be carried out without the type of acute postoperative pain normally experienced after surgery below the dentate line. However, patients do sometimes experience aching pelvic pain after procedures such as banding and stapled hemorrhoidopexy (PPH), even when the intervention has been carried out well above the dentate line [12–14]. The etiology of this pain is unclear. Following band ligation, any pain appears to be related to the number of bands placed, is seldom severe, and often resolves within 24–48 h [12]. Persistent pain after PPH is uncommon but, in the absence of any other cause, may be related to sphincter spasm [14].

The rectoanal inhibitory reflex occurs when the internal anal sphincter relaxes almost immediately after distension of the rectum, and is dependent on mechanoreceptors in the rectum but independent of higher center control. Duthie and Bennett suggested that internal sphincter relaxation allows rectal contents to be "sampled" by the sensitive anal transition zone and the lower anal canal [15]. Sampling allows discrimination between feces and flatus and appears to be an important component of continence [16].

Physiology of Defecation

Maintenance of Continence

During the resting phase, the high-pressure zone in the anal canal can maintain continence, even when the rectum is full. Resting pressure results mainly from internal anal sphincter tone, but there is a 15–20% contribution from the external sphincter [17, 18] and up to 15% from the anal cushions [19–22]. The cushions act as a seal to create a watertight anal canal and function as a unit with the sphincters: without them the internal sphincter would lose much of its tone, as it needs the arteriolar pressure within the cushions to generate its resting pressure, until the two reach a state of tonic equilibrium at rest. A mucinous film covering the sealed cushions completes the seal by means of surface tension. Hemorrhoidal prolapse reduces the efficiency of the seal, due to reduced resting pressure, allowing seepage [23]. Similarly, excision of the anal cushions during hemorrhoidectomy will result in reduced efficiency of the seal which, together with the effects of scarring and deformity, may lead to postoperative soiling.

Although the external sphincter is in a state of tonic contraction and contributes to resting tone, its main function is voluntary contraction. Under central control, it maintains continence when it is socially inappropriate to defecate after initiation of the rectoanal inhibitory reflex. It also contracts as part of a reflex in unison with the other striated muscles of the pelvic floor to maintain continence during coughing, straining, or other instances of sudden rises in intra-abdominal pressure.

Other factors influencing continence include rectal filling and compliance. As the rectum fills, it relaxes to maintain a fairly constant intrarectal pressure, until a compliance threshold is reached after which any further rise in pressure results in a desire to defecate. Progressive reduction in compliance results in urgency and eventually frank incontinence. Loss of sensation to rectal filling, as seen in cases of pudendal neuropathy or cauda equina lesions, leads to incontinence when rectal pressure exceeds anal pressure, and before the subject is even aware of the need to defecate.

Stool consistency and volume are also important for continence. Small pellet stools are more difficult to expel than large deformable stools. In addition pellet stools are more likely to result in soiling due to difficulty in evacuating completely. Small hard pellets accumulate in the rectum causing abdominal distension and sometimes incontinence, without even stimulating the urge to defecate [24]. Bulking agents avoid pellet stool formation and enable stool to be evacuated from the rectum more easily. Liquid stool is more likely to lead to incontinence, even in the normal subject. Rectal hypersensitivity is also associated with fecal incontinence and aggravated by loose stool, for instance in irritable bowel syndrome. In inflammatory bowel disease incontinence results from a combination of hypersensitivity, diarrhea, and loss of rectal compliance.

Defecation

Rectal distension produces a sensation of rectal fullness [25] and is probably mediated through receptors in the pelvic floor muscles, as it remains after coloanal anastomosis [10, 26]. Distension triggers the rectoanal inhibitory reflex, which permits sampling of rectal contents by the nerve-rich ATZ of the anal canal and allows discrimination between feces and flatus. Reflex contraction of the external sphincter maintains continence. Two examples of

the importance of the rectoanal inhibitory reflex in maintaining continence are given. Firstly reduced continence is seen in some patients after low anterior resection when the reflex may be lost [27]. Secondly some surgical procedures which restore the ATZ from a prolapsed state into a more correct anatomical position (e.g., sphincter repair and rectopexy) have been shown to restore continence by improving anal sensation [28, 29]. This second example may also apply to stapled hemorrhoidopexy.

When the internal sphincter relaxes there is a conscious desire to defecate and evacuation occurs, unless the call to stool is inconvenient. As the external sphincter relaxes, stool evacuates and is helped by voluntarily increasing intra-abdominal pressure. Squatting increases the anorectal angle and produces more efficient stool transmission. Completion of defecation is achieved by a closing reflex, when the external anal sphincter contracts and propels residual fecal material back into the rectum leaving an empty anal canal [20].

An understanding of the microstructure of the anal cushion sex plains their role in maintaining continence and aiding evacuation. Abundant venous drainage channels allow passive compression and widening of the anal canal as stool passes through the relaxed internal sphincter. Contraction of the fibroelastic muscle also actively compresses the venous dilatations. Finally, alteration in shape and eversion of the cushions aid evacuation as stool passes through (Fig. 2.5).
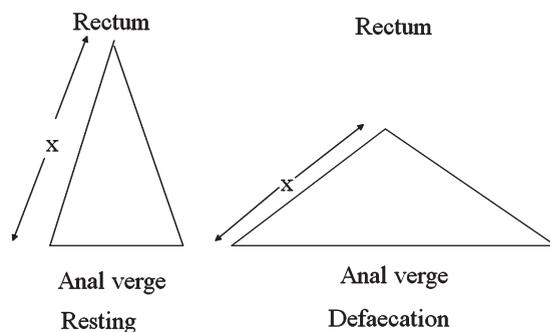

FIG. 2.5. Diagrammatic illustration of how the altered position and the form of the anal cushions aid evacuation. The length of the anal cushions (x) remains constant during defecation. However, the lumen of the anal canal dilates by relaxation of the internal and external sphincters and increased tension in anal cushion fibroelastic microstructure, resulting in venous lake compression. Consequently the cushions shorten and stool expulsion is augmented

Pathophysiology of Hemorrhoids

The anal cushions play a significant role in maintaining continence and facilitating evacuation. The term "hemorrhoids" should only be used if there is change in cushion morphology or function, resulting in hemorrhoidal symptoms such as prolapse, fresh bleeding, seepage, perianal irritation, or thrombosis.

There are many theories about the evolution of hemorrhoids [4]. Portal hypertension and varicose veins have been discounted by detailed anatomical and histological studies, demonstrating that venous dilations are integral to normal anal cushion structure. Patients with portal hypertension have a higher incidence of anorectal varices, but these are an entirely distinct anatomical and clinical entity. In fact, hemorrhoids per se are seen less frequently in patients with portal hypertension than in the general population. Recurrent infection of the anal lining has also been proposed as a cause of hemorrhoids but is untenable as the anal canal is very resistant to infection due to its excellent blood supply.

Epidemiology plays an important role in the etiology of hemorrhoids, which occur less commonly in geographical areas where diet is rich in fiber [30]. Harder, drier pellet stools, or large fecal masses result in straining with obstructed venous return and engorgement. However, the prevalence of constipation does not correlate particularly well with the occurrence of hemorrhoids, as many with the condition have a normal bowel habit.

The sliding mucosa theory [4] proposes that hemorrhoids arise from degeneration of Trietz's muscle, the fibromuscular scaffold forming the supporting tissue of the anal cushions. The cushions prolapse and engorge due to interruption of venous return and pressure from the anal canal, with the potential to precipitate thrombosis.

The following observations support the sliding mucosa theory:

1. Anal cushions are present in both the asymptomatic individual and fetus. Their structure is similar to that found in patients with symptomatic hemorrhoids [4].
2. Smooth muscle is often noted in excisional specimens.
3. Fiber-poor diet and straining are likely to result in degeneration and rupture of Trietz's muscle, making prolapse more likely.
4. A genetic predisposition to hemorrhoids can be explained by the presence of an inherited connective tissue weakness, perhaps also explaining the association of hemorrhoids with genital prolapse and hernias.
5. Aging is associated with fragmentation of the fibromuscular scaffold and correlates with an increase in hemorrhoidal symptoms.
6. Outpatient hemorrhoidal treatments, such as injection sclerotherapy, infrared coagulation, or elastic band ligation, fix the mucosa to the underlying rectal muscle wall, with restoration of the function of the suspensory muscle of Trietz.
7. Hemorrhoids resolve after abdominal rectopexy for rectal prolapse.

The rationale for stapled hemorrhoidopexy fits well with the sliding mucosa theory, as it aims to restore the prolapsed "hemorrhoids" to their original anatomical position. It restores anal cushion function by correcting anal sphincter pressure above the prolapse and reducing the submucosal arterial supply from the rectum. The level of the stapled anastomosis is placed well above the upper limit of somatic pain sensation within the anal canal, and is the reason for reduced postoperative pain and more rapid recovery after stapled hemorrhoidopexy as compared with excisional hemorrhoidectomy.

Pathophysiology of ODS

Normal defecation requires normal colonic transit, intact anorectal sensation, appropriate expulsion forces and coordinated pelvic floor function. Disturbance of any of these factors can lead to constipation. Obstructed defecation syndrome(ODS) arises when pelvic floor dysfunction results in an impaired ability to satisfactorily evacuate the rectum. The diagnosis should only be made after other organic causes, such as tumor or stenosis, having been excluded.

Different causes of ODS are listed in Table 2.1 [31] and discussed in more detail.

TABLE 2.1. Mechanisms of ODS and associated disorders.

Causes of ODS	Examples
Diminished rectal sensation	Megarectum
	Large rectocele
	Cauda equina lesion
	Pudendal neuropathy
	Diabetes mellitus
	Demyelinating neurological disease
Functional outlet obstruction	Hirshsprung's disease
	Chagas' disease
Failure to relax IAS	Anismus
Failure to relax pelvic floor	Spinal cord lesions
	Multiple Sclerosis
Mechanical outlet obstruction	Intussusception
	Full-thickness rectal prolapse
	Enterocele or sigmoidocele
	Uterine descent
Inefficient force vector	Rectocele
	Descending perineum
	Full-thickness rectal prolapse

Diminished Rectal Sensitivity

Sensory awareness of the presence of feces in the rectum is necessary to initiate the rectoanal inhibitory reflex and the sampling reflex. Whether or not defecation follows depends on central mechanisms and the conscious desire to defecate. Hyposensitivity occurs when the rectum is too large and more compliant, with raised sensory thresholds, or when there is a defect in the receptor or neural pathways. Both result in failure to initiate defecation.

Failure to Relax the Internal Sphincter

Relaxation of the internal sphincter is essential for evacuation to occur. Relaxation is coordinated via the sympathetic trunk and interruption of any part of the reflex pathway will result in constipation.

Failure to Relax the Pelvic Floor

A spastic pelvic floor will also result in difficulty in evacuation. In this group anismus(anal dysynergia, spastic pelvic floor syndrome, puborectalis syndrome or paradox) deserves specific mention. It is defined as an increase in anal pressure during attempted defecation in conjunction with an impairment of rectal emptying and is a functional disorder characteristically seen in younger women, who often have normal anatomy.

It is thought to be due to abnormal rectoanal coordination with inappropriate contraction of the puborectalis and external anal sphincter muscles during straining [32, 33] Precise definition of the condition is difficult, as anorectal manometry, electrophysiology, and dynamic proctography may show the same abnormalities in patients with other conditions or normal individuals without obstructed defecation [34–36]. In some cases a finding of puborectalis paradox may be artifactual, due to defecation being attempted under nonphysiological laboratory conditions [37]. Defecationis a learned behavioral response, and habitual events that invoke altered behavior, such as anxiety, may be sufficient to change colonic and pelvic floor function. Paradoxical contractions are more likely to be representative of inappropriate defecation behavior rather than a true underlying pathology, with anismus being part of a more complex derangement of higher center control. Behavioral therapy (biofeedback) is an appropriate therapeutic strategy in these patients.

Mechanical Outlet Obstruction

Another prerequisite for effective evacuation is a clear channel for stool to progress from the rectum through the anal canal. Anal strictures or stenosing rectal cancers are obvious organic causes of obstruction. Other anorectal abnormalities such as intussusceptionmay be associated with obstructed defecation, although even large intussusceptions may sometimes cause no symptoms at all. Extrinsic obstruction can result from straining in the presence of an enteroceleor sigmoidocelewithin in a deep pouch of Douglas, when defecation can become difficult to initiate and complete [38]. An enlarged uterus can produce a similar effect.

Inefficient Force Vector

Increased intra-abdominal pressure forcefully expels rectal contents and is often necessary for successful evacuation. However, it requires channeling of the force that increases intrarectal pressure, which in turn is dependent on a degree of resistance from the supporting structures of the rectum and pelvis. A "loss of push" occurs if the force is dissipated by bulging of the rectum forward (rectocele), or downward (intussusception and descending perineum).

Rectocele

A rectocele is a type of pelvic organ prolapse in which there is herniation of the posterior vaginal wall forward, with the anterior attenuated rectal wall in direct contact with the vagina. Straining results in bulging of the rectocele at the expense of efficient evacuation, and stool is more likely to be directed into the rectocele rather than the anal canal. This is not always the case, as sometimes a normal defecation pattern is found in the presence of even a large rectocele

There are various levels of support for the vagina and rectum. Defects in any or all will result in a rectocele. A high rectocele is often associated with loss of uterine support and genital prolapse. A rectocele at an intermediate level may be associated with a defect in the rectovaginal septum and causes bulging of the posterior vaginal wall. A low rectocele is often associated with disruption of the perineal body and direct tear from obstetric injury [39]. Childbirth results in stretching and distortion of the pelvic floor, and in some cases disruption of the endopelvic fascia, including the rectovaginal septum and the perineal body. Apart from damage to the supporting structures, another factor predisposing to rectocele development is pudendal neuropathy, caused by stretching, traction, and direct pressure on the pudendal nerve as it traverses the pelvic side wall. This results in partial denervation of the levator ani and sphincter muscles and can occur during childbirth or from long-term straining during defecation.

Both of these changes can progress to perineal descent, widening of the genital hiatus through the levator ani, and generalized perineal laxity, due to degeneration and attenuation of the pelvic floor muscles. Because the vaginal opening can no longer completely close, the posterior vaginal wall is then subjected to an even higher pressure gradient. Similar damage also leads to obstructed defecation, but it is often difficult to distinguish cause and effect, as the anatomical changes themselves perpetuate the need to strain.

Descending Perineum Syndrome

The most prominent symptom of descending perineum syndrome is a permanent and intractable difficulty with evacuation, and is due to injury to the pudendal nerves from trauma, childbirth, or chronic straining to defecate [40, 41]. The perineum bulges on straining, reflecting a lack of pelvic floor support for the rectum and perirectal structures. Not only is there a "loss of push" but the anterior rectal wall also frequently intussuscepts into the anal canal with straining and acts as a plug preventing evacuation [42, 43].

Summary

An understanding of the anatomy and physiology of the pelvic floor, maintenance of fecal continence, and the process of defecation gives some insight not only into the possible mechanisms of system failure but also the underlying principles behind surgical correction. Restoration of normal anatomy and physiology is the ultimate goal if optimal function is to be achieved.

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