Fundamentals of Anorectal Surgery

David E. Beck
Scott R. Steele
Steven D. Wexner
Editors

Third Edition

Springer

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Originally published by WB Saunders, Philadelphia, 1998
ISBN 978-3-319-65965-7 ISBN 978-3-319-65966-4 (eBook)
https://doi.org/10.1007/978-3-319-65966-4

Library of Congress Control Number: 2018949946

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Preface to the Third Edition

It has been 25 years since the publication of the first, and almost 20 years since the publication of the second, edition of *Fundamentals of Anorectal Surgery.* The delay was not from a lack of interest by the editors or the authors, but the result of a period of inadequate interest by the publishers. Fortunately, this attitude has changed, resulting in a third edition. As with the previous editions, this volume was designed not to replace the numerous excellent textbooks of colon and rectal surgery, but to provide expanded coverage of the evaluation and management of the anus and rectum, as these disorders (rather than colonic pathology) often pose a greater difficulty to practitioners. The first two editions included many talented young surgeons from a variety of practices, all of whom shared the common goal of disseminating well-written information about anorectal topics.

Due to the plethora of new information and diagnostic and therapeutic maneuvers regarding disorders of the anorectum, a third edition was necessary. To accomplish this task, a bevy of eminently qualified, internationally acclaimed experts spent a considerable amount of time to completely rewrite the chapter that includes all of the new advances and updates pertinent to each respective subject area. The individuals who have made these contributions are exceptionally well qualified to have made these improvements. The text continues with thirty-two chapters, but there have been several changes. Significant progress has been made in the prevention and therapy of acquired immunodeficiency syndrome, so the latest information has been included into the sexually transmitted disease chapter. Advances in our knowledge of anal intraepithelial neoplasia and pelvic floor disorders each merit its own chapter. When the first edition was written, minimally invasive surgery was in its infancy. As the second edition was produced, laparoscopic colorectal surgery was routinely performed at numerous locations throughout the world. Today these techniques have matured and become integrated into routine colorectal practices. Accordingly, minimally invasive techniques are incorporated into individual management chapters.

Half of these 32 chapters provide a comprehensive in-depth survey of all anorectal topics, including both benign and malignant disorders. The first four chapters discuss anatomy including congenital disorders, as well as means of evaluating anorectal dysfunction and disease. The next two chapters (Chaps. 5 and 6) evaluate perioperative and operative techniques. Chapters 7 through 9 evaluate functional disorders including prolapse and incontinence; a variety of additional benign anorectal disorders are evaluated in Chaps. 10

through 18. Chapters 19 through 26 review anorectal neoplasia. Rectal cancer remains a major focus, with several world experts updating and emphasizing the critical need for a multidisciplinary approach.

Chapter 22 describes the newer transanal techniques of TEM and TAMIS. Chapter 27 delves into sexually transmitted diseases and AIDS. Chapter 28 evaluates anorectal trauma. Chapters 29 through 30 analyze inflammatory disorders of the anus and rectum. Chapter 31 examines pelvic floor disorders related to urology and gynecology and Chap. 32 outlines nursing considerations including ostomy management.

These chapters have been carefully crafted neither to stand independently nor to duplicate each other. One of the marked and immediate obvious advantages of a multiauthored textbook is the skillfully executed interplay among authors. These authors have deftly intertwined their chapters to provide the reader with all the current concepts, theories, and practices relative to anorectal surgery. Clearly, enunciation of this ideal and execution of the concept to fruition are not necessarily synonymous. The goal was realized only through the outstanding indefatigable efforts of the numerous practicing clinicians who authored the chapters contained herein. Specifically, the third edition of *Fundamentals of Anorectal Surgery* has been authored exclusively by energetic colorectal surgeons, gastroenterologists, and nurses. This composition has enabled the textbook to impart a uniquely current perspective on the evaluation and management of anorectal disorders. The chapters have been well written, amply illustrated, and comprehensively referenced. As such, this book provides an excellent resource for both the practicing surgeon as well as surgical residents and fellows in training and medical students in school.

New Orleans, LA, USA — David E. Beck
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2019

Dedications and Acknowledgments

I thank our contributors for taking time from work and family to produce superb chapters, and my colleagues Steve and Scott for their efforts in editing this ongoing project. Steve and I remain lifelong friends and colleagues. Our relationship strengthened by the stresses and challenges of projects like Fundamentals. Scott has the enthusiasm of youth and his energy and ability will help mold the future of our specialty. Elektra did her usual outstanding job as a Developmental Editor. I remain indebted to my partners, colleagues, and trainees who continue to support and stimulate my clinical and academic efforts. Finally, I reaffirm my love and appreciation to my wife, Sharon, for her support and encouragement for all the nights and weekends spent in my office working on this project.

David E. Beck

I would first again like to thank our outstanding Developmental Editor, Elektra McDermott, for her extraordinary efforts in overseeing this edition and ensuring its timely completion and thoroughness. Having had the privilege of working with her on several texts, she never fails to amaze and deliver. I would also like to thank my fellow editors, Dave and Steve, for their tremendous vision and hard work throughout this entire process. They continue to be mentors, colleagues, and friends—and for the opportunities they have each given to me, I am forever grateful. Finally, and most importantly, thank you to Michele, Marianna (& Piper and Flynn) for supporting, encouraging, and giving me time to complete this wonderful project.

Scott R. Steele

I also echo my appreciation to Elektra McDermott for her superlative editorial skills without which this text would not have come to timely fruition. In addition I also thank Dave and Scott for their efforts, expertise, time, talent, and friendship; it has been a pleasure working with them on this project. I also express my eternal gratitude to the most important people in my life who have supported me with love as I have pursued my academic endeavors: Mariana Berho, Wesley, and Trevor for their love, understanding, and patience during the conception, creation, and completion of this book.

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1 Anorectal Anatomy and Physiology

Ravi Moonka and Joseph C. Carmichael

Introduction

The physiology of the pelvic floor is intrinsically related to its anatomy. Although, the basic anatomic concepts were established as early as 1543 by the anatomist Andreas Vesalius, many refinements were only appreciated after advances in surgery. Unlike the anatomist, the colorectal surgeon has the advantages of in vivo dissection as well as physiologic and endoscopic examinations.

Anatomy of the Anal Canal

The "anatomic" anal canal begins at the dentate line and extends distally to the anal verge. This definition is solely based on the embryology and histology of the anal canal and does not take into account the function of the anal canal as a whole. For surgeons, this strict anatomic definition of the anal canal bears little relevance in the practice of anorectal surgery. For this reason, in their 1934–1937 manuscripts, Milligan and Morgan [1, 2] advanced the argument that for clinical purposes, we must consider the anal canal in different terms. The "surgical" anal canal, as first defined by Milligan and Morgan, extends from the anorectal ring to the anal verge. The anorectal ring is a composite fibromuscular band composed of the upper portion of the internal anal sphincter, conjoined longitudinal muscle, puborectalis and external sphincter (Fig. 1.1) and is most easily identified posteriorly on rectal examination by palpating the sling-like fibers of the puborectalis portion of the levator ani [1]. This surgical definition of the anal canal takes in to account the surrounding musculature that is critical to consider during the conduct of operations from low anterior resection to anal fistulotomy. The surgical anal canal also more accurately reflects the physiology of anal continence. For these reasons, whenever the anal canal is referred to in this chapter, it is the "surgical" anal canal.

On average, the surgical anal canal is longer in males than in females. Intraoperative measurements of the posterior anal canal have estimated the surgical anal canal to be 4.4 cm in men compared with 4.0 cm in women [4]. In addition, the anal canal was shown to be a unique muscular unit in that its length did not vary with age.

The anatomy of the anal canal has also been characterized using magnetic resonance imaging. MR imaging did not show a difference in the length of the posterior anal canal in men and women, but did show that the anterior and posterior external anal sphincter length (not including puborectalis) was significantly shorter in women [5].

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D. E. Beck et al. (eds.), *Fundamentals of Anorectal Surgery*,
https://doi.org/10.1007/978-3-319-65966-4_1

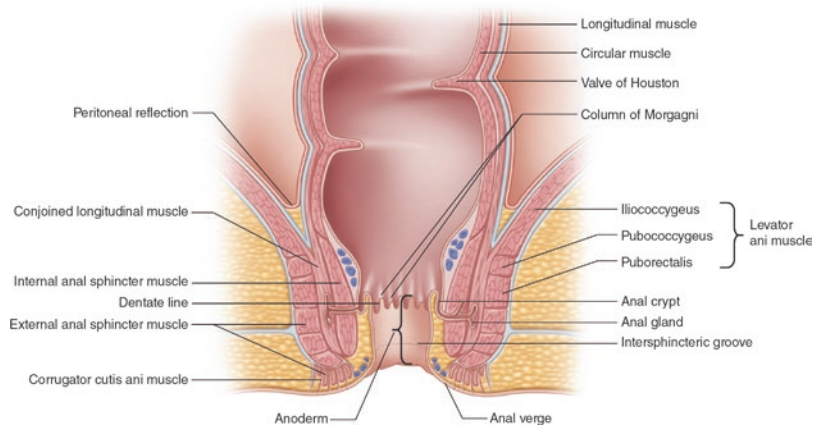

Fig. 1.1 Anal canal. From [3]. With permission © 2016 Springer

The anal canal forms proximally where the rectum passes through the pelvic hiatus and joins with the puborectalis muscle. Starting at this location, the muscular anal canal can be thought of as a "tube within a tube". The inner tube is the visceral smooth muscle of the internal anal sphincter and longitudinal layer that is innervated by the autonomic nervous system. The outer muscular tube consists of somatically-innervated, skeletal muscles including the components of the puborectalis and external anal sphincter [6]. It is the outer muscular tube that provides conscious control over continence and is strengthened during Kegel exercises. The external anal sphincter extends distal to the internal anal sphincter and the anal canal terminates at the anal verge where the superficial and subcutaneous portions of the external anal sphincter join the dermis.

Anal Canal Epithelium

The proximal anal canal has a pink appearance and is lined by the columnar epithelium of the rectal mucosa. Approximately 6–12 mm proximal to the dentate line, the anal transition zone (ATZ) begins, which appears purple in color and represents an area of gradual transition of columnar epithelium to squamous epithelium. The columns of Morgagni are noted in this area where redundant columns of tissue are noted with anal crypts at their base. This forms the rippled dentate line (or pectinate line), which can be most easily identified by locating the anal crypts at the base of the Columns of Morgagni.

From a histologic standpoint, the anal canal has three zones. The proximal zona columnaris is lined with simple columnar epithelium and extends from the apex of the anorectal ring to the dentate line. Below the dentate line, is the zona hemorrhagica that is lined by stratified squamous non-keratinized epithelium that ends at the intersphincteric groove, also referred to as Hilton's white line [7]. Below the intersphincteric groove is the zona cutanea that is lined by stratified squamous keratinized epithelium.

Anal crypts connect through anal ducts to underlying anal glands (Fig. 1.1), which are the presumed source of sepsis in the majority of anorectal abscesses and fistula. On average, there are six anal glands surrounding the anal canal (range 3–12) [6–9] and they tend to be more concentrated in the posterior quadrants. More than one gland may open into the same crypt and some crypts may not be connected to anal glands. The anal gland ducts proceed inferior and lateral from the anal canal and enter the submucosa where two-thirds enter the internal anal sphincter and half terminate in the intersphincteric plane [8]. It is theorized that obstruction of these ducts leads to anal fistula and abscess [6]. Knowledge of the anatomy also explains why the internal opening of a "cryptoglandular" anal fistula should typically be at the dentate line.

Distal to the dentate line, the anoderm begins and extends for approximately 1.5 cm. Anoderm has squamous histology and is devoid of hair,

sebaceous glands and sweat glands. At the anal verge, the anal canal lining becomes, thickened, pigmented and contains hair follicles—this represents normal skin.

The dentate line represents a true division between embryonic endoderm and ectoderm. Proximal to the dentate line, the innervation is via the sympathetic and parasympathetic systems, with venous, arterial and lymphatic drainage associated with the hypogastric vessels. Distal to the dentate line, the innervation is via somatic nerves with blood supply and drainage from the inferior hemorrhoidal system. In clinical practice, this anatomy is why malignant tumors below the dentate line can metastasize to superficial inguinal lymph nodes and external hemorrhoids (that always originate below the dentate line) are painful.

Internal Anal Sphincter

The internal anal sphincter (IAS) is the downward continuation of the circular smooth muscle of the rectum and terminates with a rounded edge approximately 1 cm proximal to the distal aspect of the external anal sphincter. The terminus of the internal anal sphincter is easily palpated on digital rectal exam and marks the intersphincteric groove. 3D imaging studies of this muscle demonstrate the overall volume does not vary according to gender, but the distribution is different with women tending to have a thicker medial/distal internal anal sphincter [10]. Overall, the IAS was found to be approximately 2 mm in thickness and 35 mm in length. The authors note that on any study, it is difficult to identify the proximal portion of the IAS as it is a continuation of the wall of the lower rectum.

Conjoined Longitudinal Muscle

The conjoined, "combined" or "conjoint" longitudinal muscle (CLM) measures approximately 0.5–2.0 mm in thickness and lies in between the internal and external anal sphincters. It begins at the anorectal ring as an extension of the longitudinal rectal muscle fibers and descends caudally joined by fibers of the puborectalis muscle [11]. In this respect, the CLM is composed of longitudinal rectal muscle fibers and levator ani muscles. The extent to which the CLM is composed of smooth longitudinal rectal muscle fibers versus skeletal levator ani muscle fibers is a point of debate. A recent study using a novel immunohistochemistry technique to analyze cadaveric specimens found that the muscle tissue between the internal anal sphincter and external anal sphincter was not a conjoined muscle at all, but consisted mainly of smooth muscle from the longitudinal rectal muscle fibers [12]. These authors concluded that the levator ani muscle attaches directly to the longitudinal rectal muscle and a mixed layer of smooth and skeletal muscle fibers does not exist between the internal and external anal sphincter. This significant departure in interpretation of the anatomy would seem to require further validation in future studies.

At its most caudal aspect, some of the conjoined longitudinal muscle fibers (referred to as *corrugator cutis ani muscle*) traverse the distal external anal sphincter and insert into the perianal skin and some of the fibers enter the fat of the ischiorectal fossa. Fibers of the conjoined longitudinal muscle also pass obliquely and caudally through the internal anal sphincter to interlace in a network within the subepithelial space. These subepithelial smooth muscle fibers were originally described by Treitz in 1853 [13] and have been referred to as Treitz's muscle. They have also been referred to *corrugator cutis ani*, *musculus submucosae ani*, *mucosal suspensory ligament* and *musculus canalis ani* [14] It has been hypothesized by Thomson that disruption of Treitz's muscles results in anal cushion prolapse, vascular outflow obstruction and hemorrhoidal bleeding and thrombosis [15]. Haas and Fox have hypothesized that the conjoined longitudinal muscle, and the network of connective tissue that it supports, plays a role in minimizing anal incontinence after sphincterotomy.

External Anal Sphincter

The external anal sphincter (EAS) is composed of striated (skeletal) muscle that forms an ellipti-

cal tube around the internal anal sphincter and conjoined longitudinal muscle. As it extends beyond the distal most aspect of the internal anal sphincter the intersphincteric groove is formed. At its distal most aspect, *corrugator cutis ani muscle* fibers from the conjoined longitudinal muscle traverse the external anal sphincter and insert into the perianal skin. Milligan and Morgan described the external anal sphincter as having three distinct divisions from proximal to distal that were termed: sphincter ani externus profundus, superficialis, and subcutaneus [1]. However, with time, this theory of three distinct divisions of the external anal sphincter was proven invalid by Goligher who demonstrated that the external anal sphincter was truly a continuous sheet of skeletal muscle extending up to the puborectalis and levator ani muscles [16]. While the external anal sphincter does not have three distinct anatomic layers, it is not uncommon to still see the proximal portion of the EAS referred to as deep EAS, the mid-portion referred to as the superficial EAS and the most distal aspect as the subcutaneous EAS. The mid EAS has posterior attachment to the coccyx via the anococcygeal ligament (discussed below) and the proximal EAS becomes continuous with the puborectalis muscle. Anteriorly, the proximal EAS forms a portion of the perineal body with the transverse perineal muscle (Fig. 1.2). There are clear differences in the morphology of the anterior external anal sphincter that have been demonstrated on both MRI and three dimensional endoanal ultrasound studies in normal male and female volunteers [17, 18]. The normal female external anal sphincter has a variable natural defect occurring along its proximal anterior length below the level of the puborectalis sling that was demonstrated in 75% of nulliparous volunteers. This defect correlated with findings on anal manometry and the authors noted that it can make interpretation of an isolated endoanal ultrasound difficult resulting in over-reporting of obstetric sphincter defects [17]. This natural defect of the anterior anal sphincter provides justification why anterior anal sphincterotomy is not routinely recommended in women.

The external anal sphincter is innervated on each side by the inferior rectal branch of the pudendal nerve (S2 and S3) and by the perineal branch of S4 (Fig. 1.3). There is substantial overlap in the pudendal innervation of the external anal sphincter muscle on the two sides which enables re-innervation to be partially accomplished from the contralateral side following nerve injury [19].

Anatomy of the Pelvic Floor

Perineal Body

The perineal body (Fig. 1.2) represents the intersection of the external anal sphincter, superficial transverse perinei, deep transverse perinei and bulbospongiosus (also referred to as bulbocavernosus) muscles. Recent research, based on advanced magnetic resonance and ultrasound imaging, has suggested that the transverse perinei (TP) and bulbospongiosus (BS) muscles contribute significantly to anal incontinence [20]. It has been proposed that the EAS, TP and BS muscles be collectively referred to as the "EAS complex muscles". In this theory, the EAS complex morphology is "purse string" shaped rather than the typical "donut" shape previously considered. When these muscles are considered as a functional unit, it lends further support to the idea that it is critical to attempt to repair the perineal body during overlapping sphincter reconstructions.

Anococcygeal Ligament

Cadaveric studies reveal the anococcygeal ligament is composed of two layers: a thick ventral layer extending from the presacral fascia to the conjoint longitudinal layer of the anal canal and a thin dorsal layer extending between the coccyx and external anal sphincter [21]. The clinical implication of this is that the thick ventral layer requires division during intersphincteric proctectomy or very low anterior resection. Both the ventral and dorsal layers would be divided during abdominoperineal resection [21]. Due to

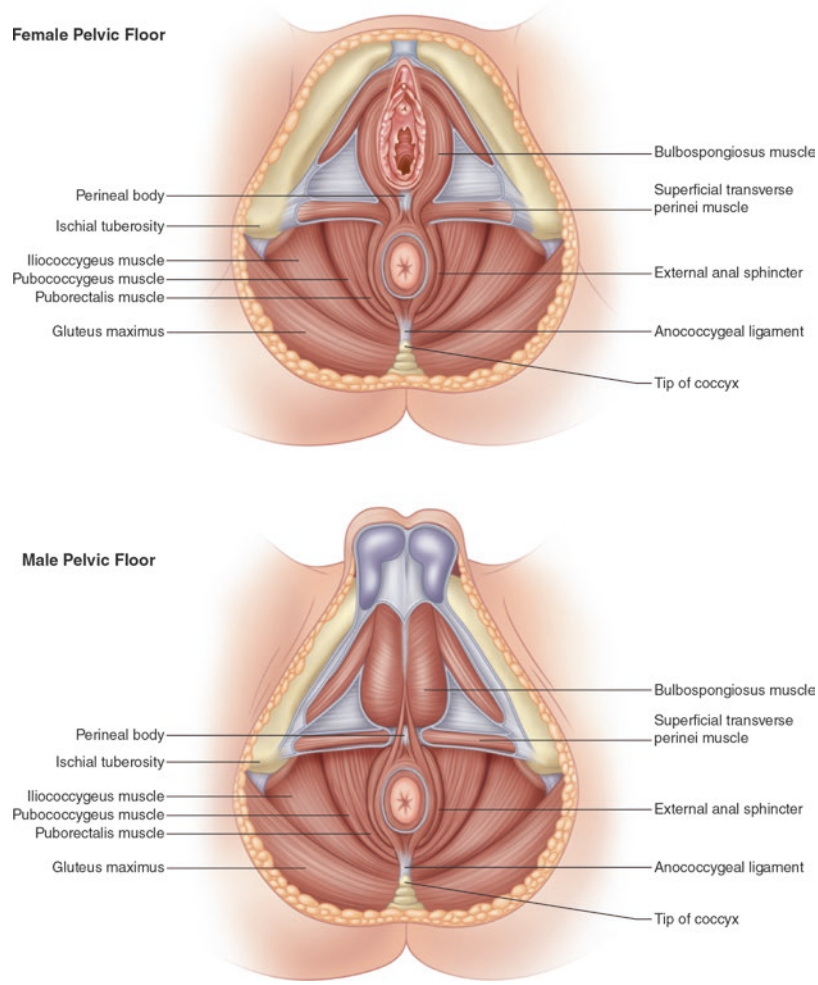

Fig. 1.2 Pelvic floor muscles. From [3]. With permission © 2016 Springer

the weak insertion into the coccyx and wavy course, it is felt that the superficial (dorsal) anococcygeal ligament is unlikely to provide a stable mechanical support to maintain configuration of the external anal sphincter [22].

Pelvic Floor Muscles

In addition to the anal sphincter and perineal body, the levator ani (LA) muscles contribute to pelvic organ support. For example, injury to the LA is seen in 55% of women with pelvic organ prolapse, but in only 16% without prolapse [23]. The LA has three subdivisions including the pubococcygeus (aka pubovisceral), puborectalis, and iliococcygeus (Figs. 1.2 and 1.4). Some authors had previously suggested that the puborectalis was part of the deep portion of the EAS [24]; however, a significant amount of evidence has been presented to the contrary. In vivo MRI measurements in women have shown distinct, visible muscle fascicle directions for each of the three LA component muscles [25]. Embryology studies have also demonstrated that the puborectalis muscle is a portion of the LA muscle and shares a common primordium with the iliococcygeus and pubococcygeus muscles [26]. Histologically, the three component muscles of the levator ani muscle (puborectalis, iliococcygeus and pubococcygeus) cannot be distinguished from one another [12].

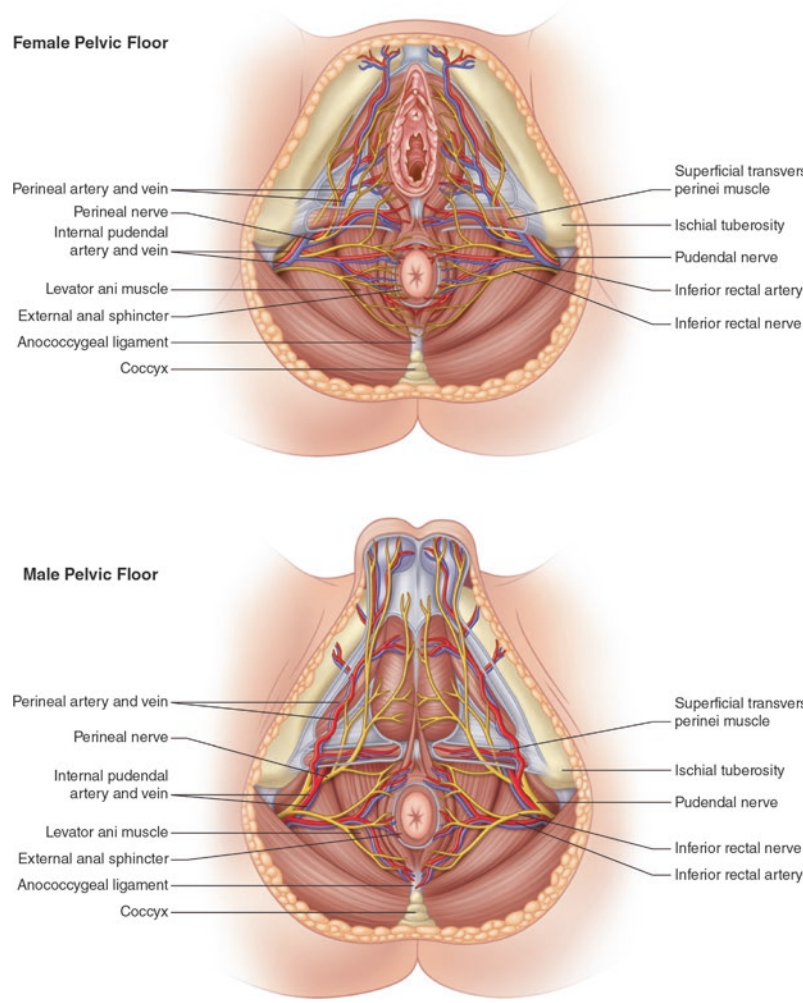

Fig. 1.3 Pelvic floor nerves and blood supply. From [3]. With permission © 2016 Springer

Innervation of the levator ani muscles has been described in detailed cadaveric studies [27]. The contemporary cadaveric studies suggest that the LA muscles are innervated by the pudendal nerve branches: perineal nerve and inferior rectal nerve as well as direct sacral nerves S3 and/or S4 (aka levator ani nerve) [28]. The pubococcygeus muscle and puborectalis muscle are primarily innervated by the pudendal nerve branches while the iliococcygeus muscle is primarily innervated by the direct sacral nerves S3 and/or S4.

Puborectalis Muscle

The puborectalis muscle (PRM) fibers arise from the lower part of the symphysis pubis and from the superior fascia of the urogenital diaphragm and run alongside the anorectal junction. Posterior to the rectum, the fibers join forming a sling. The "anorectal ring" is composed of the upper borders of the internal anal sphincter and puborectalis muscle [1]. Contraction of the PRM sling causes a horizontal force [25] that closes the pelvic diaphragm and decreases the anorectal angle during squeeze. This is widely considered the most important contributing factor to gross fecal continence.

Iliococcygeus Muscle

Iliococcygeus muscle (ICM) fibers arise from the ischial spines and posterior obturator fascia, pass inferior/posterior and medially and insert into the distal sacrum, coccyx and anococcygeal raphe.

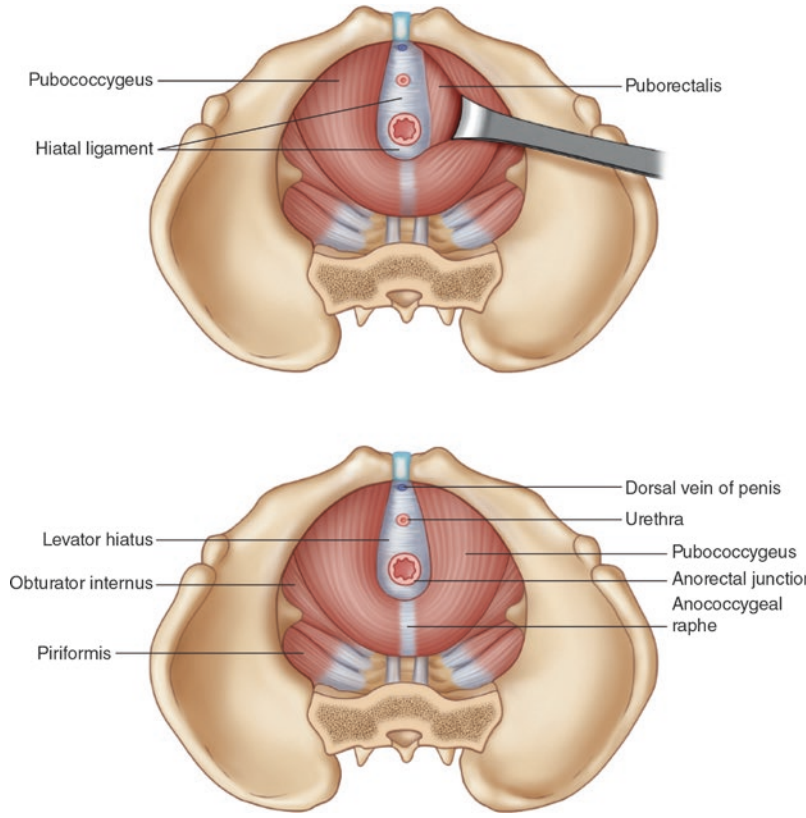

Fig. 1.4 Pelvic floor anatomy, abdominal view. From [3]. With permission © 2016 Springer

The ICM, along with the pubococcygeus muscle, contributes to "lifting" of the pelvic floor [25].

Pubococcygeus Muscle

The pubococcygeus (PCM) muscle lies medial to the PRM. PCM fibers arise from the anterior half of the obturator fascia and the high posterior pubis. The PCM fibers are directed posterior/inferior and medially, where they intersect with fibers from the opposite side and form the anococcygeal raphe (or anococcygeal ligament). PCM muscle fibers insert in the distal sacrum and tip of the coccyx. Portions of the PCM contribute to the conjoined longitudinal muscle. The PCM forms the "levator hiatus" (Fig. 1.4) as it ellipses the lower rectum, urethra, and either the vagina in women or the dorsal vein of the penis in men. The levator hiatus is connected to the intrahiatal organs by a fascial condensation called the "hiatal ligament". The hiatal ligament arises circumferentially around the hiatal margin as a continuation of the fascia on the pelvic surface of the levator muscle [29]. Enlargement of the levator hiatus has been implicated as a cause of female pelvic organ prolapse [30]. The PCM is the portion of the levator ani that is typically injured during traumatic vaginal delivery [31].

Anatomy of the Rectum

The rectum is arbitrarily considered to have three distinct parts: the upper, middle and lower rectum. Although not anatomically distinct, the upper, mid, and lower rectal divisions are important when considering surgical treatment of rectal cancer. From the anal verge, the lower rectum is 0–7 cm; middle rectum, 7–12 cm; and upper rectum 12–15 cm [32]. However, the rectum is actually variable in length and may extend beyond 15 cm from the anal verge. During surgery, the upper rectum can be distinguished from the sigmoid colon by the absence of taenia coli and epiploic appendages on the rectum.

The majority of the rectum lies outside of the peritoneal cavity, although anteriorly and laterally the upper rectum is covered by a layer of visceral peritoneum down to the peritoneal reflection. The location of the anterior peritoneal reflection is highly variable and can be significantly altered by disease such as rectal prolapse. One study sought to identify the location of the anterior peritoneal reflection in 50 patients who were undergoing laparotomy [33]. It was found that the anterior peritoneal reflection was located on average 9 cm from the anal verge in females and 9.7 cm from the anal verge in males—there was no statistically significant difference based on gender.

Valves of Houston

The rectum has been classically described to have three distinct, semicircular, inner folds called valves of Houston with the superior and inferior valves located on the left side of the rectum and the more prominent middle rectal valve on the right. However, this is not uniformly the case [34]. In one anatomic study, only 45.5% of patients had the classic three valve rectal anatomy with 32.5% having only two valves; and, 10.25% with four valves.

Mesorectum

The origin of the word "mesorectum" is difficult to identify and may be attributed to Maunsell in 1892 [35], but was certainly later popularized by Heald et al. [36]. Unfortunately, the term mesorectum is a misnomer that is not generally acknowledged in classic texts of anatomy such as the *Nomina Anatomica* [37]. In anatomic terms, the prefix "meso" refers to two layers of peritoneum that suspend an organ and the suffix applied indicates the target organ (e.g. mesocolon). The term "meso", cannot be assigned to the rectum, as it implies a mobile, suspended rectum, which may only be the case in patients with rectal prolapse.

The mesorectum is a term employed by surgeons to describe the fascial envelope of the rectum that is excised during surgical treatment of rectal cancer. Indeed, failure to completely excise this envelope intact has been associated with an increased incidence of local recurrence of rectal cancer [38]. The mesorectum is contained within the fascia propria. The fascia propria is an upward projection of the parietal endopelvic fascia that lines the walls and floor of the pelvis. The fascia propria encloses the perirectal fat, lymphatics, blood vessels and nerves and is not considered a barrier strong enough to prevent the spread of infection or malignancy [39].

Presacral Fascia

The presacral fascia (Fig. 1.5) is a thickened portion of the parietal endopelvic fascia overlying the sacrum that covers the presacral veins and hypogastric nerves. It extends laterally to cover the piriformis and upper coccyx. As the presacral fascia extends laterally, it becomes continuous with the fascia propria and contributes to the lateral ligaments of the rectum. Caudally, this fascia extends to the anorectal junction covering the anococcygeal ligament. During total mesorectal excision, the fascia propria is elevated sharply off the presacral fascia. Leaving the presacral fascia intact eliminates the possibility of causing presacral bleeding.

Retrosacral Fascia

The retrosacral fascia originates at the third and fourth portion [40] of the sacrum and extends anteriorly to the posterior layer of the fascia propria 3–5 cm proximal to the anorectal junction [41]. This tough fascia layer is surgically relevant as it must be sharply incised during total mesorectal excision [39]. The space posterior to the retrosacral fascia is referred to as the supralevator space (Fig. 1.6) and is the location where supralevator abscesses are found.

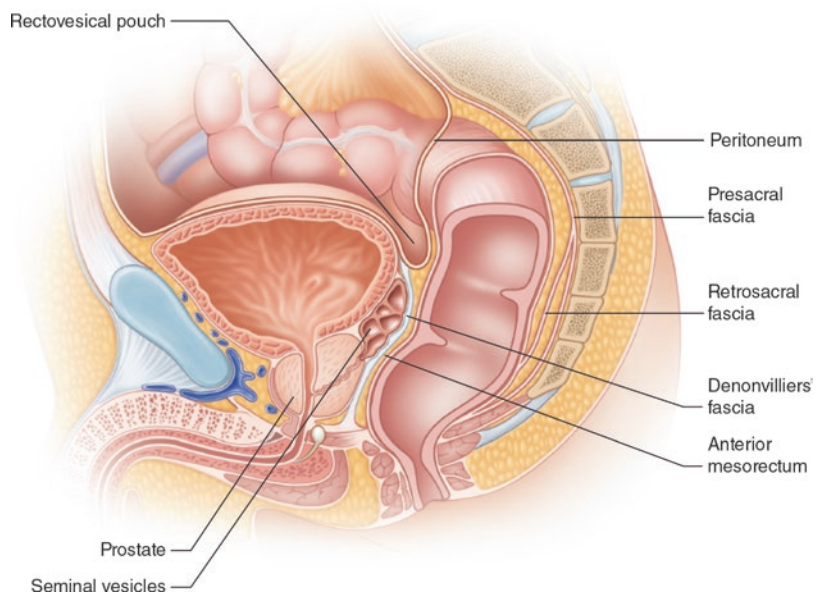

Fig. 1.5 Fascial relationships of the rectum. From [3]. With permission © 2016 Springer

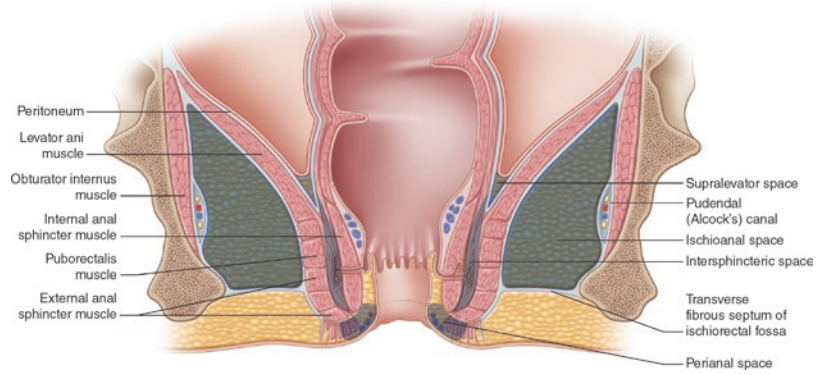

Fig. 1.6 Perianal and perirectal spaces, coronal view. From [3]. With permission © 2016 Springer

Waldeyer's Fascia

There is significant confusion about what Waldeyer's fascia represents as the eponym has been used to describe the presacral fascia, the retrosacral fascia or all fascia posterior to the rectum. In Waldeyer's original description of pelvic fascia, there was no particular emphasis on the presacral component [39, 41]. While the debate continues regarding Waldeyer's fascia, it is important to simply understand that can have the potential to mean presacral fascia, retrorectal fascia or both [42].

Denonvilliers' Fascia

Denonvilliers' fascia arises from the fusion of the two walls of the embryological peritoneal cul-de-sac and extends from the deepest point of the rectovesical pouch to the pelvic floor [43]. Originally described by Denonvilliers in 1836 as a 'prostatoperitoneal' membranous layer between the rectum and seminal vesicles, Denonvilliers fascia is also present in females as part of the rectovaginal septum and is sometimes referred to as rectovaginal fascia. It is found immediately beneath the vaginal mucosa and is clearly what

most would consider as part of the vaginal wall. It merges superiorly with the cardinal/uterosacral complex in females or the rectovesical pouch in males. It merges laterally with the endopelvic fascia overlying the levator muscle and distally with the perineal body. It contains collagen, some strands of smooth muscle, and heavy elastin fibers. Rectoceles represent a defect in this layer that allows the rectum to bulge anteriorly [44].

Microscopically, the Denonvilliers' fascia has two layers; however, it is not possible to discern two layers during pelvic dissection [43]. In the anterior rectal plane, the mesorectum is contained by the fascia propria which lies dorsal to Denonvilliers' fascia. The cavernous nerves run in neurovascular bundles at the anterolateral border of Denonvilliers' fascia.

Anorectal Spaces

It is important to acknowledge and understand the anorectal spaces created by the various myofascial relationships in the pelvis as these spaces help us understand how anorectal sepsis can spread throughout the pelvis.

Perianal Space

The perianal space (Fig. 1.6) contains external hemorrhoid cushions, the subcutaneous external anal sphincter and the distal internal anal sphincter. The perianal space is in communication with the intersphincteric space. The perianal space has it's cephalad boundary at the dentate line and laterally to the subcutaneous fat of the buttocks or is contained by fibers extending from the conjoined longitudinal muscle often referred to as *corrugator cutis ani* muscle fibers. Otherwise, the perianal space is contained by anoderm.

Intersphincteric Space

The intersphincteric space is the potential space that lies between the internal and external anal sphincter and is continuous with the perianal space. Like the other anorectal spaces, it is important to understand that this space communicates circumferentially around the anorectum (Fig. 1.7). This space is of clinical importance as cryptoglandular infections tend to begin in this area and expand elsewhere to create anal fistula [6].

Submucous Space

This space lies between the medial boarder of the internal anal sphincter and the anal mucosa proximal to the dentate line. It is continuous with the submucosa of the rectum. This area contains internal hemorrhoid vascular cushions.

Ischioanal/Ischiorectal Space

The ischioanal (also referred to as ischiorectal) space is the largest anorectal space. It has been described as a pyramid shape with its apex at the levator muscle insertion into the obturator fascia. The medial boarder is thus the levator ani muscle and external anal sphincter. The obturator internus muscle and obturator fascia make up the

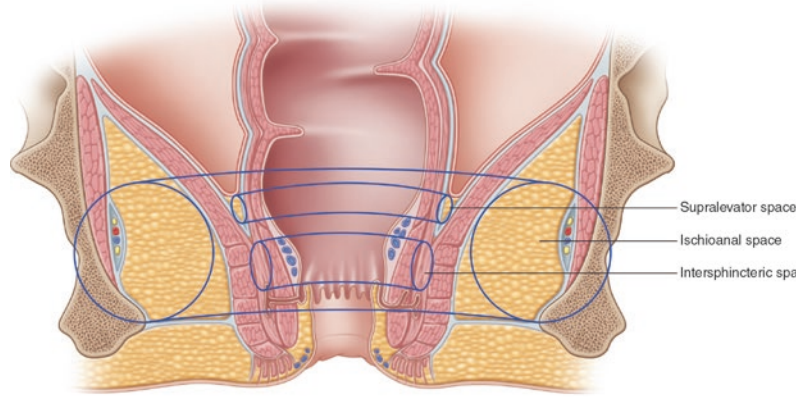

Fig. 1.7 Communication of the anorectal spaces. From [3]. With permission © 2016 Springer

lateral boarder of the ischioanal space. The posterior boundary is formed by the lower border of the gluteus maximus muscle and the sacrotuberous ligament. The space is has an anterior boundary formed by the superficial and deep transverse perineal muscles. The caudal boundary is skin of the perineum. The ischioanal fossa contains adipose tissue, pudendal nerve branches and superficial branches of the internal pudendal vessels. The right and left ischioanal space communicate posteriorly through the deep postanal space between the levator ani muscle and anococcygeal ligament [45]. When the ischioanal and perianal spaces are regarded as a single space, it is referred to as the ischioanal fossa [42].

Supralevator Space

The upper boundary of the supralevator space is the peritoneum, the lateral boundary is the pelvic wall, the medial boundary is the rectum and the inferior boarder is the levator ani muscle (Fig. 1.8).

Superficial and Deep Postanal Spaces

These spaces are located posterior to the anus and inferior to the levator muscle. The superficial postanal space is more caudal and is located between the anococcygeal ligament and the skin. The superficial postanal space allows communication of perianal space sepsis.

The deep postanal space (retrosphincteric space of Courtney) [46] is located between the levator ani muscle and the anococcygeal raphe. This space allows ischioanal sepsis to track from one side to the other resulting in the so called "horseshoe" abscess.

Retrorectal Space

The retrorectal space is found between the presacral fascia and fascia propria. It contains no major blood vessels or nerves. It is limited laterally by the lateral ligaments of the piriformis fascia and inferiorly by the retrosacral fascia. The fascia propria and presacral fascia come together at the apex of this space [39].

Lateral Ligaments

The lateral ligaments of the rectum are a point of controversy [47]. First, some argue that the lateral ligaments do not exist at all. Second, there is considerable controversy about what they contain if they in fact do exist. Miles referred to division of the lateral ligaments of the rectum in his seminal description of a performing abdominoperineal resection in 1908. Specifically, he notes "In these structures the middle haemorrhoidal arteries are found but seldom require a ligature" [48]. It is interesting to note that at least one modern cadav-

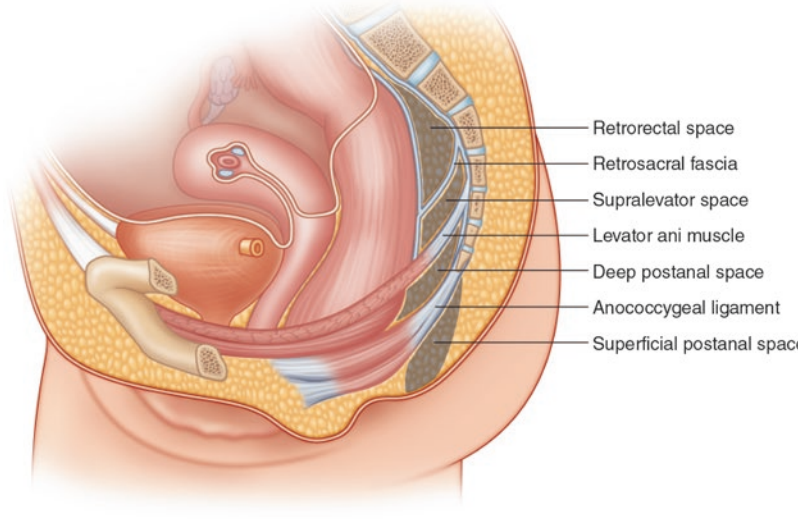

Fig. 1.8 Perianal and perirectal spaces, lateral view. From [3]. With permission © 2016 Springer

eric dissection study identified the presence of a middle rectal artery in only 22% of specimens [40] which could be a contributing factor as to why Miles saw no significant bleeding in this area.

Total mesorectal excision, as popularized by Heald involves sharp dissection along the fascia propria circumferentially to the pelvic floor. While acknowledging that the middle rectal vessels are "divided as far from the carcinoma as possible" Heald does not mention, "lateral ligaments" of the rectum at all [49].

In an extensive review of the anatomy of the lateral ligament, Church notes that it is a common misconception that the lateral ligaments contain the middle rectal artery at all. It appears that the lateral ligaments comprise "primarily nerves and connective tissue" and their division without bleeding attests to the absence of a "significant accessory rectal artery in this location in the majority of patients" [39].

In a separate cadaveric study, the lateral ligaments of the rectum were identified as trapezoid structures originating from mesorectum and anchored to the endopelvic fascia at the level of the midrectum. It was recommended that, as lateral extensions of the mesorectum, the ligaments must be cut and included in the total mesorectal excision (TME) specimen. It was further noted that the lateral ligaments did not contain middle rectal arteries or nerve structures of importance. The urogenital bundle runs just above the lateral ligament at its point of insertion on the endopelvic fascia, the middle rectal artery (if present) runs posterior to the lateral ligament and the nervi recti fibers (which originate from the inferior hypogastric plexus) course transversely under the lateral ligament to the rectal wall [50]. Other modern cadaveric investigations note the rarity of middle rectal arteries and the absence of clinically relevant neurovascular structures in the lateral ligaments [51].

Rectal Blood Supply

The rectum is supplied by the superior, middle, and inferior rectal (hemorrhoidal) arteries. Both the middle and inferior hemorrhoidal vessels are paired arteries and the superior rectal artery is not (Fig. 1.9).

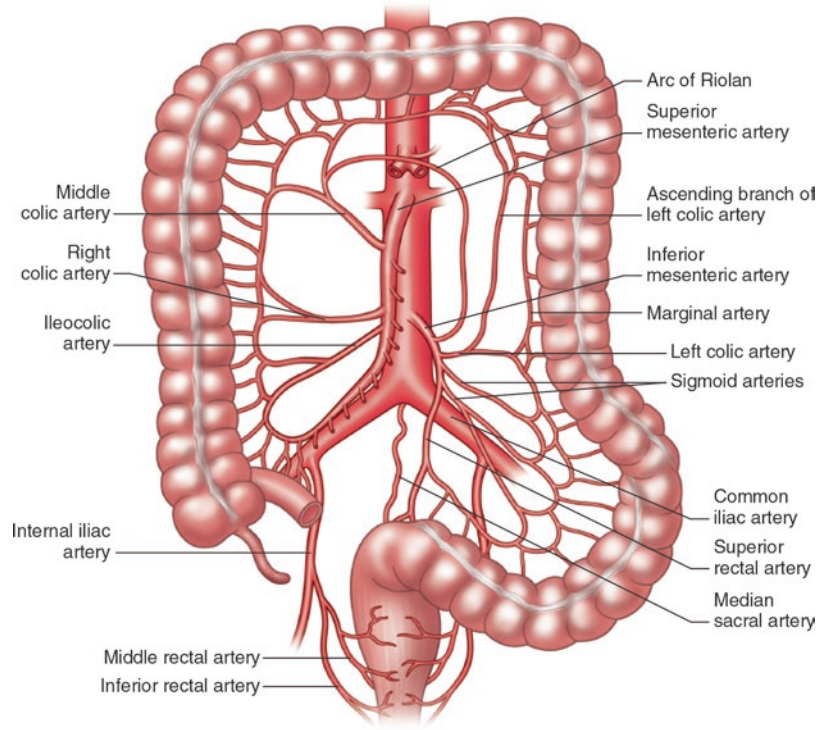

Fig. 1.9 Arterial anatomy of the colon and rectum. From [3]. With permission © 2016 Springer

Superior Rectal Artery

The superior rectal artery (SRA) is the continuation of the inferior mesenteric artery and is so named after the inferior mesenteric artery crosses the left iliac vessels. The SRA gives off a recto-sigmoid branch, an upper rectal branch, and then bifurcates into a right and left terminal branches in 80% [52] of cases as it descends caudally in the mesorectum. On average, eight terminal branches of the SRA have been identified in the distal rectal wall [53].

Middle Rectal Artery

The middle rectal artery (MRA) has been variably noted in many studies. It may be found on one or both sides of the rectum and has been noted to be present 12–28% of the time [51, 54]. At least one study reported the presence of the middle rectal artery in at least 91% of cadaveric specimens [50]. The MRA originates from the anterior division of the internal iliac or pudendal arteries. Please see the "Lateral Ligament" discussion above for more review on the anatomic course of the middle rectal artery.

Inferior Rectal Artery

The inferior rectal arteries (IRA) are paired vessels that originate as branches of the internal pudendal artery, which receives its blood supply from the internal iliac artery. The artery originates in the pudendal canal and is entirely extrapelvic (caudal to the levator ani) in its distribution. The IRA traverses the obturator fascia, the ischiorectal fossa and pierces the wall of the anal canal in the region of the external anal sphincter [39].

Venous and Lymphatic Drainage of the Rectum and Anus

Venous drainage from the rectum and anus occurs via both the portal and systemic systems. Middle and inferior rectal veins drain to the systemic systems via the internal iliac vein while the superior rectal vein drains the rectum and upper anal canal into the portal system via the inferior mesenteric vein (Fig. 1.10).

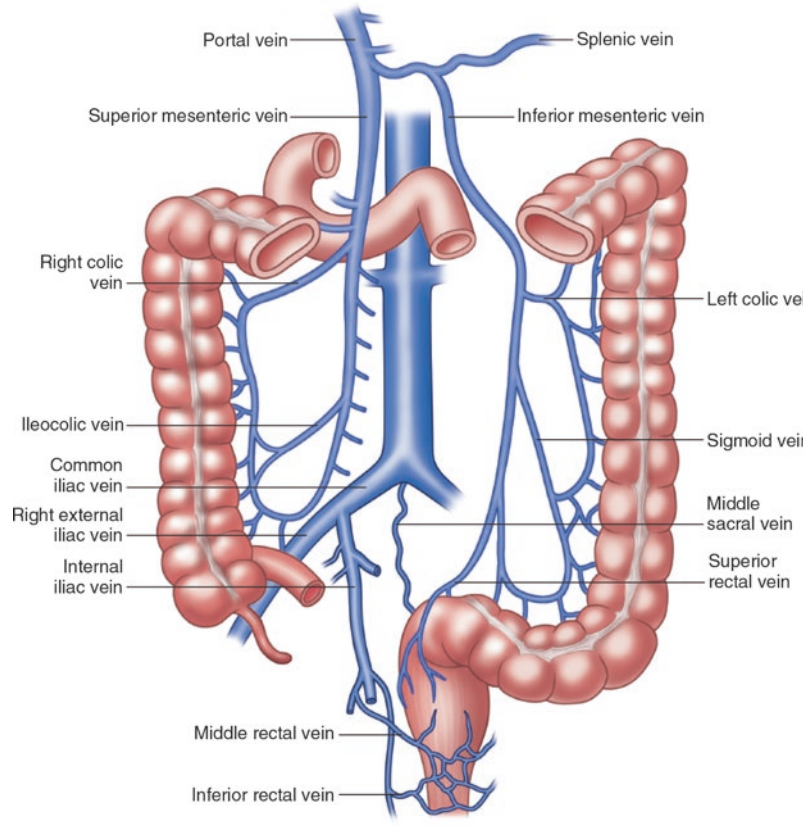

Fig. 1.10 Venous anatomy of the colon and rectum. From [3]. With permission © 2016 Springer

Lymphatics from the upper two-thirds of the rectum drain to the inferior mesenteric lymph nodes and then to the para-aortic lymph nodes. Lymphatic drainage from the lower third of the rectum occurs along the superior rectal artery and laterally along the middle rectal artery to the internal iliac lymph nodes. In the anal canal, lymphatic above the dentate drain to the inferior mesenteric and internal iliac lymph nodes. Below the dentate line lymphatics drain along the inferior rectal lymphatics to the superficial inguinal nodes.

Innervation of the Rectum and Anus

Sympathetic fibers arise from L1, L2, and L3 and pass through the sympathetic chains and join the preaortic plexus (Fig. 1.11). From there, they run adjacent and dorsal to the inferior mesenteric artery as the mesenteric plexus and innervate the upper rectum. The lower rectum is innervated by the presacral nerves from the hypogastric plexus. Two main hypogastric nerves, on either side of the rectum, carry sympathetic information from the hypogastric plexus to the pelvic plexus. The pelvic plexus lies on the lateral side of the pelvis at the level of the lower third of the rectum adjacent to the lateral stalks (please see discussion of lateral stalks above).

Parasympathetic fibers to the rectum and anal canal originate from S2, S3 and S4 to penetrate through the sacral foramen and are called the nervi erigentes. These nerves course laterally and anterior to join the sympathetic hypogastric nerves and form the pelvic plexus on the pelvic sidewall. From here, postganglionic mixed parasympathetic and sympathetic nerve fibers supply the rectum, genital organs and anal canal. The periprostatic plexus is considered a subdivision of the pelvic plexus and supplies the prostate, seminal vesicles, corpora cavernosa, vas deferens, urethra, ejaculatory ducts, and bulbourethral glands.

The internal anal sphincter is innervated by sympathetic (L5) and parasympathetic (S2, S3 and S4) nerves following the same route as the nerves to the rectum as noted above. The external anal sphincter is innervated on each side by the inferior rectal branch (Fig. 1.3) of the internal

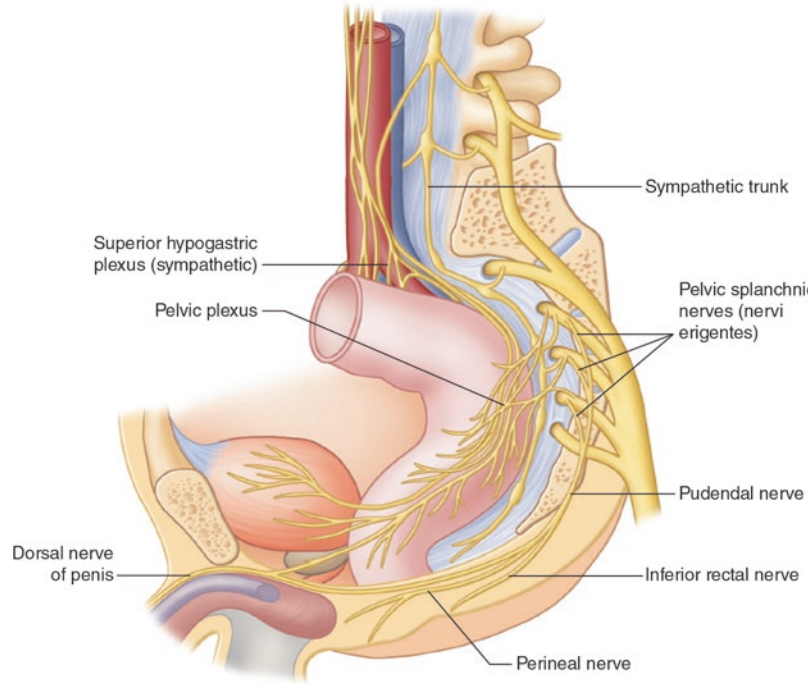

Fig. 1.11 Nerves of the rectum. From [3]. With permission © 2016 Springer

pudendal nerve (S2 and S3) and by the perineal branch of S4. The pudendal nerve mediates conscious external sphincter contraction, but does not play a role in unconscious anal sphincter contraction [55]. The pudendal nerve supplies afferent sensory pathways from the skin of the anal canal and perineum [56].

Physiology

Normal physiology of defecation results in the voluntarily controlled evacuation of stool at a frequency and with an effort, which the individual does not find distressing. While somewhat in the eye of the beholder, normal frequency of defecation in the great majority of people is between three times a day and once every 2 days [57]. Bowel habits outside of that range however are not necessarily pathologic, and if acceptable to the individual are acceptable to the physician if unaccompanied by other symptoms or signs of disease. Stool frequency within that range should still be characterized as abnormal if excessive time and effort are required to eliminate. Incontinence is easier to define, and because continence mechanisms work best with a solid or semisolid stool consistency, fecal elimination is a process dependent on the proper functioning of the entire gastrointestinal tract, but particularly of the colon, rectum, and anal sphincter complex.

Colonic Role in Normal Defecation

Colonic Absorption

The colon absorbs water, sodium and chloride and secretes potassium and bicarbonate. In healthy individuals, colonic absorption of water reduces the 1000–1500 mL of fluid which enters the colon each day to about 100–150 mL [58]. Absorption of fluid is greatest in the right and transverse colon, though compensatory mechanisms enhance absorption in the remaining colon following a right colon resection, as demonstrated in animal models and as is seen clinically in the normalization of bowel function following that operation [59].

Failure of colonic absorption due either to rapid transit or mucosal absorptive failure results in a liquid stool, which when emptied rapidly into the rectum results in great stress on the sphincters and, even in normal subjects, may occasionally produce urgency and incontinence. The mean values for normal total colonic transit time is approximately 32 and 41 h for men and women respectively, though can be as long as 72 h in adults. The mean segmental transit times are 12, 14 and 11 h for right colon, left colon and rectosigmoid, respectively [60]. Colonic absorption is mostly likely assisted by non-propagated contractions of the muscular wall of the colon. These contractions, which are not thought to move the fecal bolus a significant distance in either direction in a coordinated way, mostly likely serve to expose various aspects of the stool to the colonic absorptive surface to maximize fluid retention [61]. This may explain the relatively long 30–40 h transit time of the colon, compared to the much shorter transit times associated with small bowel.

Colonic Motility

The colon is capable of sustained and powerful contractile force when distended, which all experienced endoscopists have witnessed. Propagated contractions are so named for their ability to move the fecal bolus downstream towards the rectum and anus. They tend to be low pressure with an amplitude under 50 mm Hg, or high pressure with an amplitude over 100 mm Hg [62]. The manometric presence of High Amplitude Propagated Contractions (HAPC's) corresponds to radiographically seen movement of stool in a coordinated way in an aboral direction. These high-pressure waves can begin anywhere in the colon, and tend to terminate in the sigmoid and rectum. They are present on awakening and tend to be absent during the night. They also occur within minutes of initiating a meal, and are responsible for the gastrocolic reflex [63]. Significant psychological and physiologic stress can also trigger HAPC's, and distance runners often note the urge to defecate after completion of a long event. Although their frequency is highly variable, they occur on average five to six times a day, and are

probably less frequent in patients with constipation. Similarly, the proportion of retrograde contractions may be higher in patients with infrequent defecation. A propagated contraction generally precedes the urge to defecate in normal controls, and the contraction is usually high amplitude. Low Amplitude Propagated Contractions (LAPCs) are much more frequent than HAPCs, occurring 40–120 times a day, and may also be less frequent in constipated patients compared to normal controls [64], and may also be less well linked from the proximal colon-to-distal [65].

Colonic contractions are mediated by Auerbach's plexus, which lies between the circular and longitudinal muscle layers, and Meissner's plexus, which lies in the submucosa. The general rate of contraction can be modulated by inhibitory impulses from the sympathetic nervous system and stimulatory impulses from the parasympathetic nervous system. Coordination of intrinsic colonic motility is coordinated the pacemaker like interstitial cells of Cajal.

Role of the Rectosigmoid Junction

It is tempting to consider the abdominal colon as a monofunctional tube whose sole purpose is to deliver stool to the capacious rectum, which when adequately distended signals the need to defecate. Several observations run counter to this notion, including the tendency of constipated patients to have an empty rectum on physical exam, and the generally empty nature of the rectum in normal controls between bowel movements. The sigmoid and the rectosigmoid junction, although still a matter of substantial controversy, may play a role in fecal continence. The sigmoid may act as a reservoir, which when distended leads to relaxation of a physiologic sphincter-like apparatus at the rectosigmoid junction, which then leads to rapid filling of the rectum and the subsequent need to defecate. Accordingly, an electrically hyperactive segment has been found at the rectosigmoid junction, particularly in constipated patients; moreover, a high pressure zone has been noted in at least 50% of the normal population [66].

Rectal Function

Filling of the rectum results in the need to defecate. Balloon distension of the rectum causes urgency, while the same maneuver in the abdominal colon causes only pain [67]. The non-diseased rectum has both viscous and elastic properties which allow it to maintain a low intraluminal pressure while being filled in order to preserve continence, and these properties can to some extent be appreciated surgically in the way the rectum responds to manipulation and division with greater elasticity than the intra-abdominal colon. When rectal compliance deteriorates, smaller volumes of feces will result in higher intraluminal pressures causing urgency and frequency. This phenomenon is observed in patients with ulcerative colitis [68] and radiation proctitis [69]. The preservation of compliance associated with ileal and colonic J-pouches explains the greater functionality associated with those surgical strategies compared to straight anastomoses [70].

Although rectal distension is a crucial signal for impending defecation, the sensory nerves responsible for communication this distension lie mostly outside the rectum [71]. Perhaps the most compelling evidence for the extrarectal location for signaling is the preserved sense of the need to stool in patients having had complete removal of the rectum with an ileoanal or coloanal anastomosis. Also, stimulation of the stretch receptors in the pelvic floor or puborectalis results in urgency [72]. Anesthetizing the rectum does alter sensory aspects of defecation suggesting the rectum itself is also responsible for some aspect of signaling and likely explains the imperfect bowel function seen in patients having sphincter sparing rectal surgery.

Failure of the sensory nature of the rectum and pelvic floor can lead to both incontinence and failure to evacuate. Even if the innervation of the rectum is intact, excessively high thresholds required to activate the afferent neural cascade can also lead to excessive rectal filling. Similarly, excessive rectal compliance can also contribute to a failure on the patient's part to detect rectal distension [73].

During defecation, the contribution of rectal contraction to evacuation is not well described. It may be that different individuals rely on rectal smooth muscle contractions to varying degrees, with some relying solely on an increase in intra-abdominal pressure through the Valsalva maneuver and the like, while others depend on a substantial contribution from the rectum itself.

The Pelvic Floor

The pelvic floor musculature (Fig. 1.2), consisting of the levator ani muscles (iliococcygeus, pubococcygeus, puborectalis), is generally in a state of tonic contracture, which support the abdominal and pelvic organs. Not surprisingly the muscles of the pelvic floor are composed primarily of Type 1 fibers, which are associated throughout the body with tonic contracture [74]. The pelvic floor also creates an acute angle between the rectum and the anal canal, which assists in continence and fecal storage. Of the pelvic floor muscles, the puborectalis plays the largest role in creating this angle and is innervated by the S3 and particularly the S4 nerve roots [75]. A preserved cutaneous-anal reflex suggests intact S4 sensory and motor nerve roots. This is skeletal muscle and can be voluntarily contracted to stave off imminent defecation. Increased abdominal pressure can result in reflex contracture of the pelvic floor as with a cough, or in reflex relaxation when defecation occurs. How these different reflex actions are mediated is not clear. Relaxation results in straightening and inferior movement anorectal angle, which facilitates defecation. Failure of the pelvic floor to relax can sometimes be seen on defecography and is a potential cause of obstructed defecation [76]. Hip flexion also straightens the anorectal angle, emphasizing the importance of posture, be it sitting or squatting, in facilitating a bowel movement [77].

The Anal Sphincter Complex

Internal Anal Sphincter (IAS)

As a smooth muscle, the IAS is in a state of continuous maximum contraction. This is due to both intrinsic myogenic and extrinsic autonomic neurogenic properties. The IAS represents a natural barrier to the involuntary loss of stool. The mean anal canal resting tone in healthy adults is generally in the range of 50–70 mm Hg, and tends to decrease in women and in the elderly [78]. The IAS is responsible for 50–85% of the composition of the resting tone, the EAS accounts for 25–30%, and the remaining 15% is attributed to expansion of the anal cushions [79].

A gradual increase in pressures is noted from proximal to distal in the anal canal; the highest resting pressures are usually recorded 1–2 cm cephalad to the anal verge. This high pressure zone or functional anal canal length corresponds anatomically to the condensation of the smooth muscle fibers of the internal anal sphincter and is shorter in women (2–3 cm) compared to men (2.5–3.5 cm) [78]. Interestingly, although parity may contribute to this difference, nulliparous women still have a significantly shorter functional anal canal than men [80].

Rectal distension causes reflexive transient relaxation of the internal sphincter and the subsequent descent of rectal contents into the proximal anal canal. This rectoanal inhibitory reflex (RAIR) can be recreated through balloon distension of the rectum with simultaneous measurement of a decrease in anal resting pressure. The exposure of the highly innervated anal canal to rectal contents allows discrimination between the various potential consistencies of rectal contents, and facilitates passing of gas without stool. This sampling reflex is mediated by the enteric nervous system, which is why the RAIR is manometrically absent in Hirschsprung's disease, and why the reflex persists following denervation of the rectum and anus.

Although the IAS relaxes in response to rectal distension, it gradually reacquires its tone as the rectum accommodates to the distension. Pronounced impairment of IAS function has been noted in 25% of patients with idiopathic fecal incontinence. Spontaneous relaxation of the IAS without a compensatory increase in EAS activity may be an important factor leading to fecal incontinence [81].