1 Anatomy and Physiology of the Pelvic Floor

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1.1 Introduction: The Pelvic Content

The small pelvis in the female contains three reservoirs made of smooth muscle, an inner mucosal layer and a fibro-adipose adventitia externally. The bundles running together with the hypogastric fascia, from the pelvic sidewalls toward the midline, provide blood and nerve supply. The bladder is a true reservoir, since it is closed upward by the vesico-ureteral valves and downward by the urethral smooth- and rhabdosphincter. The vagina is not a proper reservoir, since it has not any biologic fluid or solid to contain, but it is the site of the dramatic accommodation that takes place during childbirth. It communicates upward with the cervix, which is part of the birth canal and has an open bottom that is the introitus. The vagina has not a real sphincter at its introitus, but the perineal membrane and muscles, together with the perineal body can be seen as a closure mechanism. Posteriorly the rectum is a particular kind of reservoir with slow transit, open top and a complex sphincteric system. All the three viscera have a proper morphology, a definite relationship one to each other, and a typical position inside the pelvis. These features are strictly associated with their function, as for storage and emptying phases. The position and mobility of each organ is basically provided by two mechanisms: On one hand the fixation systems to the connective and muscular structures of the pelvis, which are real mesenteries and act as ligaments holding the viscera in place and, on the other hand, the pelvic floor muscles that support the pelvic content, close the genital hiatus and compensate the abdominal rising pressures. In particular the

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muscular sling of the pubococcygeus lifts and moves upward and forward the whole pelvic content, acting as an additional sphincter. The pelvic floor is a dynamic unit that allows the physiology of continence, emptying, sexual function and delivery. Pelvic ligaments and fasciae are the anatomic connections that allow viscera and muscles to work together.

The ligaments of the pelvic regions are usually considered by most anatomists as parts of the thickened retropelvic fascia that run along blood and lymphatic vessels, nerves and connective tissue, while surgeons consider it as a true fascial layer. It becomes denser adjacent to the lateral aspects of uterine cervix and vagina, being usually called broad ligament; it is formed by a thin, double plication of the peritoneum connecting the lateral pelvic structures to the uterus. The corresponding fascia that links the cervix and the vagina to the pelvic wall is called cardinal or Mackenrodt's ligament. The posterior part of this fascia extending from the upper portion of the cervix to the sacral vertebra are indicated as uterosacral ligaments.

1.2 The Bony Pelvis

It is made of the two symmetrical coxal or hip bones (composed of ileum, ischium, and pubis), sacrum and coccyx. The hip bones are fused to the sacrum posteriorly and to each other anteriorly at the pubic symphysis.

The bony pelvis is divided into superior or false or greater pelvis and inferior or true or smaller pelvis by the pelvic brim, whose structures are the sacral promontory, the anterior sacral ala, the arcuate line of the ilium, the pectineal pubic line and the pubic crest that ends in the pubic symphysis.

The inferior pelvic outlet is closed by the pelvic floor. The muscles of this regions have two fundamental functions: they provide support or act as a "floor" for the abdominal viscera including the rectum, and constrictor or continence mechanism to the urethral, anal, and vaginal orifices (in females).

In comparison to males, females have a pelvis with wider diameter and a more circular shape (Fig. 1.1). The wider inlet facilitates the engagement of the fetal head and parturition. On the other hand, this shape predisposes to subsequent pelvic floor weakness. Attachment sites for ligaments, muscles, and fascial layers are provided by a number of projections and contours.

The ischial spine, at the midst of the brim of the ischiatic foramen, is the anchorage of several connective and muscular tissues. Some authors describe the spine and the related ligament complex as a star. The sacrospinous ligament, the coccygeal muscle, the posterior portion of the arcus tendineus fasciae pelvis, and the arcus tendineus levator ani leaving the spine stand like beams around it. The sacrospinous ligament extends from the ischial spines to the lateral margins of the sacrum and coccyx anteriorly to the sacrotuberous ligament. During the dissection it can be seen as a separate structure, just behind the ischiococcigeus muscle, or as a complex with the muscle, superficial or deep. The greater and lesser sciatic foramina are above and below the ligament (Fig. 1.2).

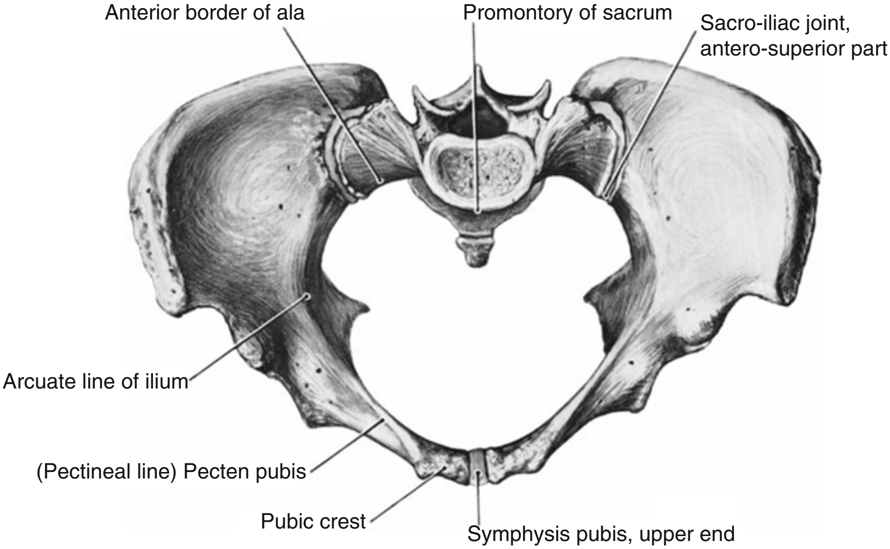

Fig. 1.1 The female bony pelvis

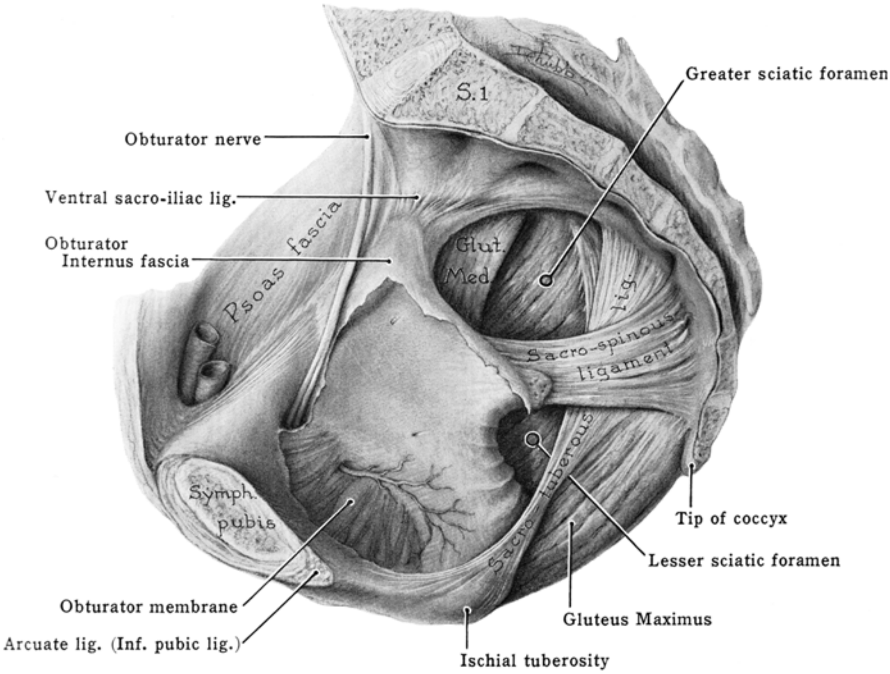

Fig. 1.2 The female bony pelvis with the sacro-spinous and the sacro-tuberous ligaments ant the greater and lesser sciatic foramen (Grant (1962) An atlas of anatomy)

1.3 The Muscular Pelvic Floor

1.3.1 Pelvic Diaphragm

The pelvic diaphragm is a thin and wide muscular structure that forms the inferior limit of the abdominopelvic cavity. It is formed of a tunnel-shaped muscle that extends with its fascia from the symphysis pubis to the coccyx and laterally from one wall of the pelvis to the other. The levator ani and coccygeus muscles that are attached to the internal surface of the minor pelvis form the muscular floor of the pelvis.

They originate from the pubic bone at the level of pectinate line and from the obturator internus fascia and are connected to the coccyx.

The levator ani muscle (LAM) is composed of two major muscles: the pubococcygeus and iliococcygeus muscles from medial to lateral. The medial portion of the LA is the bulky pubococcygeus muscle that takes origin from the middle of inferior pubic rami, the pubic symphysis and anterior portion of its arcus tendineus. The arcus tendineus of the LA is indeed a dense structure made of connective tissue, running from the pubic ramus to the ischial spine along the surface of the obturator internus muscle. The muscle passes behind the rectum in an almost horizontal direction. The inner aspect forms the margin of the urogenital hiatus, which is crossed by the urethra, vagina, and anorectum.

The medial portions of the pubococcygeus has been divided in various subparts to reflect the attachments of the muscle to the urethra, vagina, anus, and rectum [1]. These portions are often called by some investigators as the pubourethralis, pubovaginalis, puboanalis, and puborectalis muscles or, all together, as the puboviseralis muscle, because of their attachment to the pelvic viscera [2].

The puboviseralis definition was originally championed by Lawson and currently supported by a lot of writings of Delancey [3, 4]. While this term is well accepted in the urogynecological literature, it is rarely mentioned in the anatomical or gastroenterology textbooks.

Lawson felt that the portions of the puboviseralis muscle are inserted into the urethra, vagina, anal canal and perineal body and he assigned respectively the names pubouretheralis, pubovaginalis, puboperinealis, and puboanalis muscles to those portions.

The urethral portion of the muscle forms part of the periurethral musculature; parallely the vaginal and anorectal parts insert into the vaginal walls, perineal body, and external anal sphincter (EAS) [5].

Posteriorly the puborectalis portion passes behind the rectal canal and is fused with the muscle of the opposite site to form a sling behind the rectum itself.

Other parts of the pubococcygeus run more posteriorly taking attachment to the coccyx.

The lateral part of the levator ani is rather thin and is called iliococcygeus; it takes origin from the arcus tendineus of the levator ani and reaches the ischial spine and the last two segments of the coccyx. The fibers from the two sides also fuse to form a raphe denominated as the anococcygeal ligament. This median raphe linking the anus and the coccyx is known as the levator plate and is the structure on which the pelvic organs rest. It is formed by the connection of the two iliococcygeus and the posterior fibers of the pubococcygeus. During the standing position, the plate is almost horizontal and supports the pelvic viscera situated just above, that is the rectum and the upper two thirds of vagina. Every event leading to a weakness of the levator ani affects this plate and may cause the levator plate to sag. As a consequence of that, the urogenital hiatus opens and predisposes to pelvic organ prolapse. Women affected by genital prolapse have been shown to have an enlarged urogenital hiatus [6, 7].

The coccygeus muscle that runs from the ischial spine to the coccyx and to the lower portion of the sacrum forms the posterior part of the pelvic diaphragm. It is situated on the anterior surface of the sacrospinous ligament (Fig. 1.3). Using 3D MRI imaging of the pelvic diaphragm, we can clearly verify its peripheral attachments and illustrate the urogenital hiatus [8].

As regards the type of these striated muscle, it has been shown that the majority of the fibers are of the slow-twitch type: they can maintain a constant tone (type I) [9] while an increased presence of fast-twitch (type II) fibers is present in the periurethral and perianal areas. This concept confirms that the normal levator ani maintains its tone in the upright position to support the pelvic viscera. Furthermore, a voluntary contraction of the puborectalis during squeezing may increase the tone to balance an increased intra-abdominal pressure.

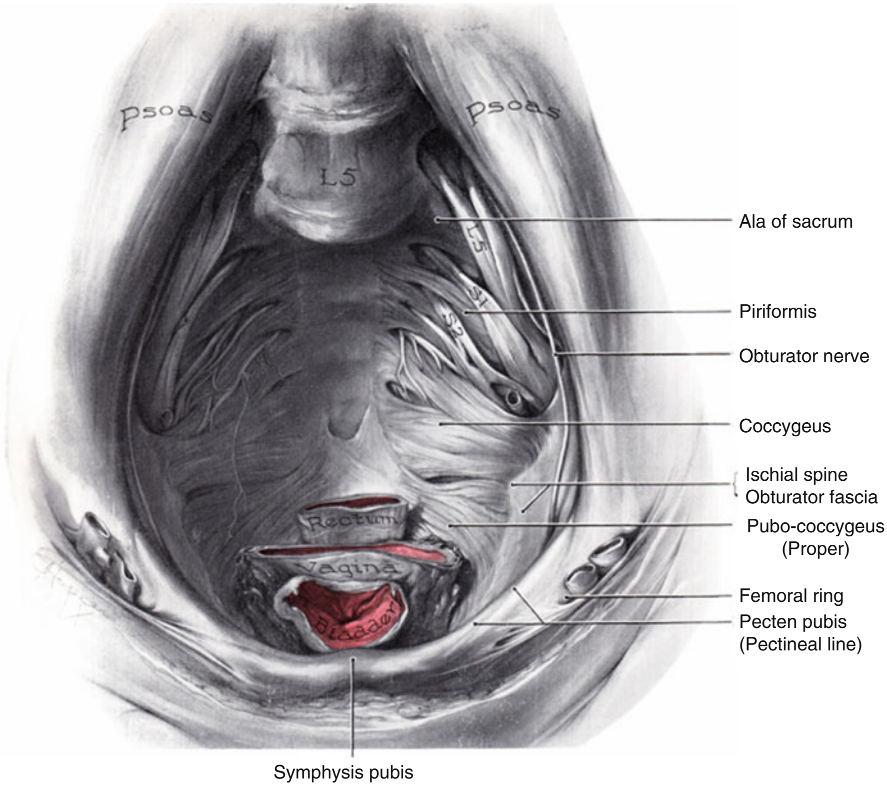

Fig. 1.3 The levator ani and its components

The urogenital diaphragm, also called the triangular ligament, is a strong, muscular membrane that lies in the area between the symphysis pubis and ischial tuberosities being therefore situated in the triangular anterior portion of the pelvic outlet.

The analysis of the urethral structure has led recently to abandon the term "external urethral sphincter" because the urethral rhabdosphincter is not really external to the urethra, but more properly it surrounds the middle of the urethra. The term "urethral rhabdosphincter" has been therefore recommended.

In contrast to the levator ani muscles, the rhabdosphincter and perineal muscles embryologically develop from the cloaca, with a 2-week delay in striated muscular differentiation compared with the LA and other skeletal muscles [10].

We remember that they are completely separated from the LA by connective tissue [11].

Thus, the striated muscles associated with the viscera are quite distinct from the striated skeletal muscle of the pelvic floor.

1.3.2 Anal Sphincters

The prolongation of the circular muscle layer of the rectum, expanding caudally into the anal canal, becomes the internal anal sphincter (IAS), as reported by Varuna Raizada and Ravinder K. Mittal. In a parallel fashion, the longitudinal muscles of the rectum extend into the anal canal and end up as thin septa that penetrate into the puborectalis and external anal sphincter (EAS) muscles [12]. The autonomic nerves, sympathetic (spinal) nerves and parasympathetic (pelvic) nerves supply the IAS [13].

Sympathetic fibers originate from the lower thoracic ganglia and form the superior hypogastric plexus. Parasympathetic fibers originate from the 2nd, 3rd and 4th sacral nerves and form the inferior hypogastric plexus, which gives rise, from up to down to the superior middle and inferior rectal nerves: they innervate the rectum and the anal canal. These nerves synapse with the myenteric plexus of anal canal and the rectum. The majority of muscular tone of the IAS is myogenic, i.e., due to the properties of the smooth muscle itself. Angiotensin 2 and prostaglandin F2α play modulatory roles. Sympathetic nerves mediate IAS contraction through the stimulation of α and relaxation through β1, β2, and β3 adrenergic receptors.

Recent studies show that low affinity β3 receptors are predominant in the IAS. Anal sphincter relaxation is obtained by stimulation of parasympathetic pelvic nerves through nitric oxide containing neurons that are situated in the myenteric plexus. A more limited role is played by other potential inhibitory neurotransmitters like VIP (vasointestinal peptide) and by CO (carbon monoxide). Acetylcholine and substance P are the neuromediators for excitatory motoneurons located in the myenteric plexus of IAS.

The anatomy of external anal sphincter (EAS) has been largely investigated; it is formed by three parts: subcutaneous, superficial and deep ones. However, several investigators have found that the EAS is constituted only by the subcutaneous and superficial muscle bundles [14, 15].

The first is located caudal to the IAS, while the superficial one surrounds the distal part of the IAS. The deep portion is very thin and is often confused with the puborectalis muscle. 3D US and RMN studies have confirmed this anatomical point of view. EAS is attached to the perineal body and to the transverse perineal muscle anteriorly and to the anococcygeal region posteriorly. We must underline that the EAS is not completely circular in all its sections: the posterior part is indeed shorter in the inferior aspect. This should not be misconstrued as a muscle defect in the axial US and MR images of the lower anal canal.

The muscolaris propria of the rectum contains low threshold slowly adapting mechanoreceptors: they are involved in the detection of mechanical deformation of the myenteric ganglia and of the tension of the longitudinal rectal muscle. Mechanical deformations of the anal canal stimulate numerous free and organized nerve endings like Meissner, Krause and Golgi-Mazzoni corpuscles. They are exclusively sensitive and respond to light touch, pain and temperature. Afferent unmyelinated C and larger A fibers are responsible for neural transmission from the rectum and the anal canal to the spinal cord [16].

1.3.3 Urethra

Female urethra presents an outer layer that is formed by the striated muscular structure of the urogenital sphincter: it can be found in the middle three fifths of its length. In its upper two thirds, the sphincter-like fibers are disposed in a circular fashion. In the distal part, the fibers leave the urethra and surround the vaginal wall forming the urethrovaginal sphincter; other parts of the same muscle extend along the inferior pubic rami above the perineal membrane being indicated as the urethral compressor. This muscle is composed mainly by fibers of the slow-twitch type that are especially suited for maintaining a constant tone. When needed, a voluntary muscle activation of this striated muscle can increase the constrictive action on the urethra.

The urethral mucosa extends from the bladder (that presents a transitional epithelium) to the external meatus: its epithelium is primarily a nonkeratinizing squamous one. Among its characteristics we must underline that it is hormonally sensitive undergoing important changes with stimulation. This submucosal tissue is hormonally sensitive too and contains a rich vascular plexus. Groups of a specialized type of arteriovenous anastomoses have been found in it: they are thought to provide a watertight closure of the mucosa, also thanks to an increase in blood flow contemporary to an increase in abdominal vessel pressure. The considerable quantity of connective tissue that is interspersed within the muscle and submucosa contains collagen and elastin fibers: its function is that of a passive addiction to urethral closure. A series of glands, mostly predominant in the middle and lower third of the urethra, are found in the submucosal space, mainly along its vaginal surface. Therefore we can conclude that the admixture of the smooth and striated muscle, connective tissue, mucosa, and submucosa accounts for the urethral functional sphincter. This sphincter needs obviously an intact neural control to provide the watertight closure of the urethral lumen, the compression of the wall around the lumen, and to create a sophisticated means for compensating abdominal pressure changes.

1.3.4 Pelvic Floor Imaging

The anatomy and the functions of pelvic floor muscles has been recently deeply investigated with MRI and 3D US, so that novel insights have been reached and diffused. After that, muscles themselves have been analyzed during contraction and defecation with ultrafast CT scanning [17]. These studies demonstrated that the changes of pelvic floor hiatus are in relationship with puborectalis activity, being smaller during contraction and larger during defecation (Figs. 1.4 and 1.5). The changes in the pelvic floor hiatus size are predominantly related to the puborectalis muscle and they reflect the constrictor function of pelvic floor. On the contrary the other muscles, i.e., pubococcygeus, ischiococcygeus, and ileococcygeus muscles, are mainly responsible for pelvic floor and levator plate ascent and descent. The anorectal angle becomes acute and moves cephalad

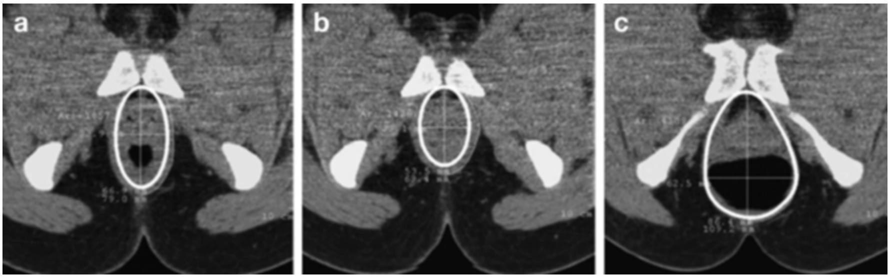

Fig. 1.4 Seated CT defecography, axial images of puborectalis at rest (**a**), squeeze (**b**), and defecation (**c**). Note that the pelvic floor hiatus becomes smaller during squeeze and larger during defecation (Adapted from Li D, Guo M. Morphology of the levator ani muscle. *Dis Colon Rectum*. Nov 2007;50(11):1831–1839, with permission)

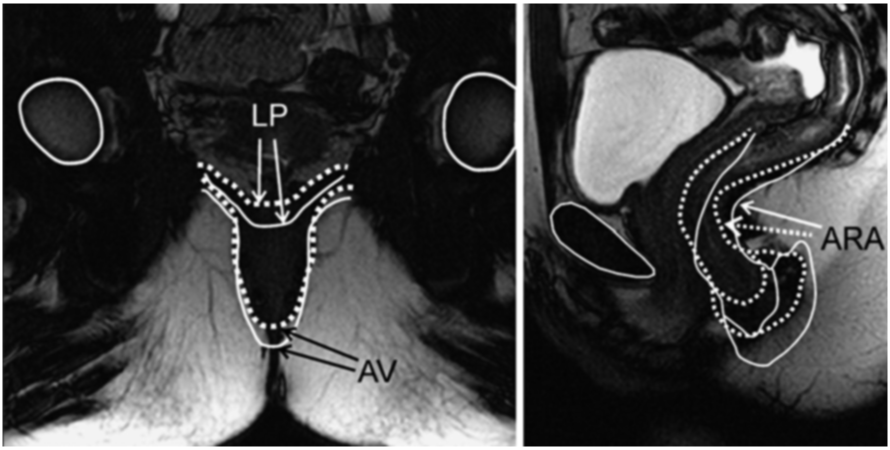

Fig. 1.5 Magnetic Resonance images (MRI) in the mid-sagittal and coronal planes: these images were obtained at rest (solid) and squeeze (dotted) and the images were overlapped to show the movements of various structures during squeeze. *LP* levator plate, *AV* anal verge, *ARA* ano-rectal angle

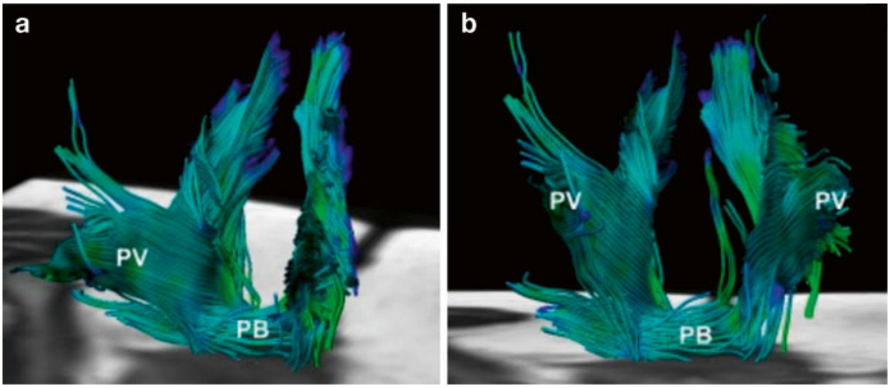

Fig. 1.6 Fiber tractography demonstrates the complex, multidirectional organization of the different pubovisceral (*PV*) muscle components in a 28-year-old female subject in both oblique-anterior (**a**) and anterior-posterior view (**b**). At the bottom of the pelvic floor transverse orientation of the fiber tracts are displayed matching the perineal body (*PB*) [18]

during pelvic floor contraction, while opposite changes occur during relaxation and defecation. Diffusion tension imaging (DTI) with fiber tractography has recently been used to analyze normal pelvic floor anatomy in a three-dimensional fashion (Fig. 1.6) [18].

1.4 Neuroanatomy

1.4.1 Puborectalis and Deep Pelvic Floor

Even if the concept that pelvic floor muscles are innervated from the S2, S3 and S4 sacral roots is commonly recognized, some controversies regard the innervation of levator ani muscle by the pudendal nerve. Varuna Raizada et al. reported that the middle layer of LA (puborectalis m.) is innervated by the pudendal nerve [19], whereas the deeper muscles (pubococcygeus, ileococcygeus and coccygeus) are innervated by the direct branches of sacral roots S3 and S4 [20]. Therefore they affirm that in human the levator ani muscle is innervated by the levator ani nerve [21]. It primarily arises from sacral spinal roots and travels along the intrapelvic face of the levator ani muscle with a high degree of variability in branching patterns. The consequence of this affirmation is that pudendal nerve damage may cause dysfunctions of puborectalis m, urethral and anal rhabdosphincters, responsible for urinary and fecal incontinence.

In humans, there is some controversy concerning whether or not the pudendal nerve also innervates the levator ani muscle [22, 23]. Several observations induce to describe a distinct special somatic motor innervation of the rhabdosphincters by the pudendal nerve vs. a typical skeletal motor innervation of the levator ani muscle by the levator ani nerve.

1.5 Peripheral Innervation of Urethral and Anal Rhabdosphincters

The motor neurons that innervate the striated muscle of the external urethral and the anal sphincters originate from a localized column of cells in the sacral spinal cord called Onuf's nucleus, expanding in humans from the second to third sacral segment (S2–S3) and occasionally into S1. Within Onuf's nucleus there is some spatial separation between motor neurons concerned with the control of the urethral and anal sphincters. Spinal motor neurons for the levator ani group of muscles seem to originate from S3–S5 segments and show some overlap [21]. Extensive studies of the urethral rhabdosphincter, anal rhabdosphincter, bulbocavernosus and ischiocavernosus muscles have shown that these muscles are innervated by the pudendal nerve. Traditionally the pudendal nerve is described as being derived from the S2–S4 anterior rami, but there may be some contribution from S1 and, possibly, little or no contribution from S4.

The nerve runs along the lateral aspect of the internal obturator and coccygeus muscles and through Alcock's canal (Fig. 1.7). In this portion, it branches into the inferior rectal nerve (for the anal rhabdosphincter), the perineal nerve (for the urethral rhabdosphincter, the bulbospongiosus muscle, the ischiocavernosus muscle, the superficial transverse perineal muscle and the labial skin), and the clitoris dorsal nerve. The branches of the perineal nerve are more superficial than the latter, and, in most cases, travel on the superior surface of the perineal musculature.

Therefore, Thor and de Groat [24] conclude that the pudendal nerve does not innervate to a significant degree the major muscles of the pelvic floor, i.e., iliococcygeus, pubococcygeus or coccygeus muscles. As regards morphology, levator ani motor neurons are similar to other skeletal motor neurons, showing large α and small γ neuronal cells distributed in the sacral ventral horn. One distinguishing feature, however, are some projections from levator ani motor neurons into Onuf's nucleus, the location of rhabdosphincter motor neurons: this ought to represent a mechanism of coordination between pelvic floor and rhabdosphincter functions. Unfortunately the position of the LA nerve at the level of the intrapelvic surface of the muscles may expose it to be damaged during the passage of fetal head during the 2nd stage of delivery, thus contributing to the correlations between parity and POP.

1.6 Reflex Activation of Urethral and Anal Rhabdosphincters

The activation of the segmental reflex by pelvic nerve stimulation related to bladder distension stimulates the urethral rhabdosphincter; therefore the afferent inputs from the urinary bladder have been emphasized as being of primary importance. This reflex is often referred to as the guarding reflex or continence reflex.

However, recent studies are placing greater emphasis on urethral afferent fibers [25], so that some researchers are speculating that the guarding reflex is actually activated more vigorously by urethral afferent fibers: if urine inadvertently begins to pass through the bladder neck and into the proximal urethra, a fast closure of the more distal urethral sphincter (against any urine loss) is requested more rapidly as compared with simple bladder distensions or increases in intravesical pressure. Since the pudendal nerve is composed even by some urethral afferent fibers (as well as rectal,

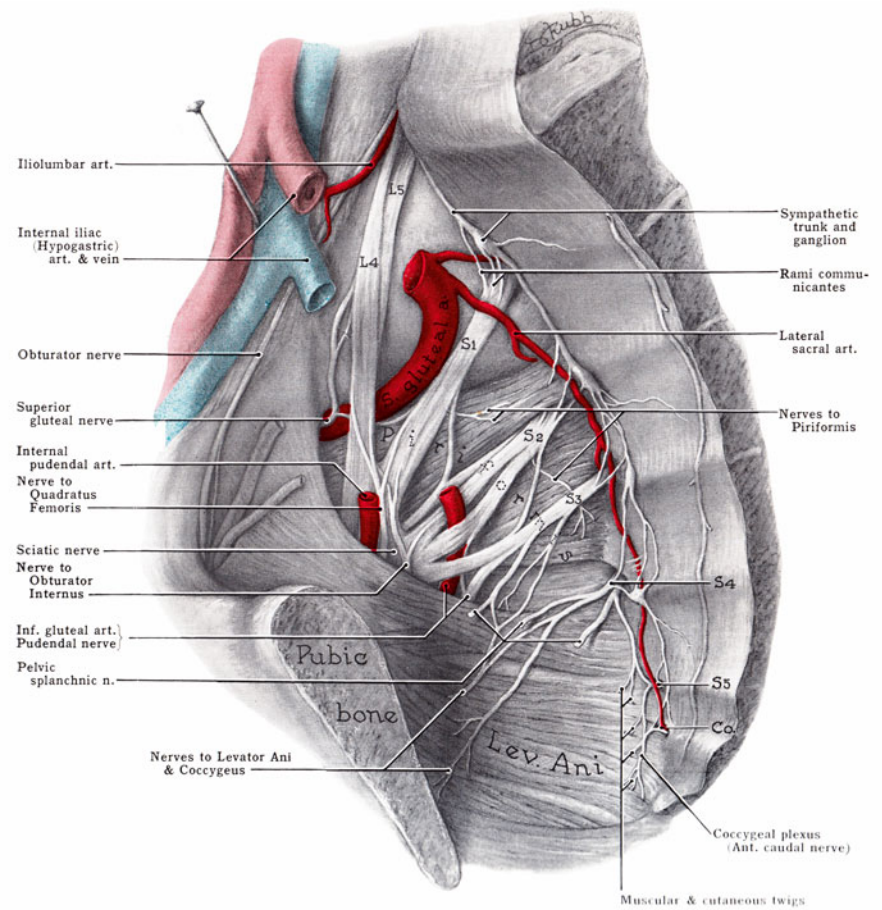

Fig. 1.7 Pelvic floor innervation (Grant (1962) An atlas of anatomy)

genital, and cutaneous ones), we may argue that the spinal urethral sphincter activation by pudendal afferent stimulation is also a manifestation of the guarding reflex.

Internal anal sphincter is innervated by autonomic sympathetic (spinal nerves) and parasympathetic (pelvic nerves) terminations. The former originate from the lower thoracic ganglia and form the superior hypogastric plexus, whereas the latter take origin from the 2nd, 3rd and 4th sacral roots to form the inferior hypogastric plexus, giving rise to the nerves that supply the rectum and the anal canal, that is the superior, middle and inferior rectal nerves.

1.7 Inhibition of Urethral Rhabdosphincter Reflexes During Voiding

Voluntary micturition is a behavior pattern that starts with the relaxation of the rhabdosphincter and the pelvic floor muscles. This can be detected as a disappearance of all EMG activity, which precedes detrusor contraction. The striated anal sphincter relaxes similarly with defecation and with micturition. Voluntary pelvic floor muscles contraction during voiding can lead to a stop of micturition, probably because of collateral connections to detrusor control nuclei. As a matter of fact descending inhibitory pathways for the detrusor have been often demonstrated. Bladder contractions are also inhibited by other reflexes that can be activated by afferent input from the pelvic floor muscles, perineal skin, and anorectum.

In addition to supraspinal inhibitory mechanisms, a spinal, urine storage reflex inhibitory center (SUSRIC) was found: it inhibits the somatic and the sympathetic urine storage reflexes, acting on the urethral rhabdosphincter and smooth muscle, respectively. Activation of SUSRIC by electrical stimulation of the pelvic nerve afferent fibers occurs simultaneously with the activation of the urethral rhabdosphincter reflex itself. Possibly the inhibition of rhabdosphincter activity by distension of the bladder represents a physiological corollary of the high-frequency electrical stimulation of pelvic nerve afferent fibers.

1.8 Levator Ani Innervation Damage in Childbirth and POP (see also Chap. 9)

It is well known that childbirth represents a risk factor for development of POP. Various studies have indicated that the damage to the innervation of the pelvic floor muscles may be the initial pathological cause for pelvic descent and prolapse [26–30]. Many other studies [31, 32] have underlined that levator ani nerve damage follows parturition in about 25 % of women with approximately one-third of those showing a persistent nerve damage at 6 months after parturition. On the contrary, women undergoing elective cesarean section (CS) (i.e., without preceding labor) showed no signs of levator ani nerve damage, while damage occurred in similar proportions of women who had CS after protracted labor as the ones with vaginal delivery, i.e., without CS. Furthermore, changes in function of the urethral rhabdosphincter were also associated with pregnancy itself (i.e., before labor), and these remained evident at 6 months after childbirth. Some experimental studies on squirrel monkeys indicate that, in nulliparous animals, the pelvic floor muscles play a minor role in providing visceral support, thus suggesting that the connective tissue plays the major role. On the other hand, after childbirth and consequent stretching of the pelvic connective tissue, the muscle may play a compensatory role. The levator ani muscle stretching might induce a reflex muscle activity and hypertrophy.

Other lesion mechanism than neurogenic and structural damage, such as muscle ischemia, may also be operative during childbirth. Although muscle weakness may be a common consequence of childbirth injury, it is the strength of muscle contraction that defines its functional integrity. Parous women with stress urinary incontinence (SUI) are subject to significant reduction of duration of motor unit recruitment, unilateral recruitment of reflex response in the pubococcygeal muscle and paradoxical inhibition of continuous firing of motor units in pelvic floor muscles activation on coughing.

Another mechanism through which the pelvic muscular support may facilitate pelvic organ prolapse is the avulsion of the puborectalis muscle from the pubic rami in the multipara. This kind of damage is present in 18–70 % of multiparous women, the majority of them showing POP on clinical examination [33–35].

Although not proven in studies, it is reasonable to assume that motor denervation is accompanied also by sensory denervation of the pelvic floor muscles. In addition to denervation injury there may be some further temporary "inhibitors" of pelvic floor muscles activity, such as periods of pain and discomfort after childbirth, increased by attempted pelvic floor muscles contraction.

All abovementioned factors may lead to a temporary disturbance of pelvic floor muscles neural control after childbirth. This, in combination with a particularly vulnerable pelvic floor neural control, might become persistent, even if the factors originally leading to the problem disappear.

1.8.1 Applied Physiology

Broadly, the pelvic floor muscles can be considered to have two important functions.

They provide (1) support and physiologic position to the pelvic viscera and (2) sphinteric or closure functions to urethra, vagina, and anal canal. In cadavers, the pelvic floor is shaped like a basin, but in living individuals its shape is like a dome [36, 37]. This changes of the pelvic floor during contraction, from a basin to dome shape, is due to the shortening of the three components of LA muscle. At the same time, conversion to the "dome" causes the lift of the pelvic viscera (including the rectum) in the cranial direction and provides mechanical support to all pelvic floor viscera. Therefore, the weakness of these muscles results in perineal descent. The puborectalis is generally considered to be fundamental in maintaining the anorectal angle [38]. Contraction of this muscle results in an acute anorectal angle; during defecation, its relaxation causes this angle to become obtuse. The anorectal angle can be analyzed with contrast defecography or MRI.

The puborectalis muscle is also involved in the anal canal closure mechanism, i.e., in anal canal pressure [39, 40].

In the proximal anal canal the closure pressures are related to the activity of IAS and puborectalis muscle, while, in the middle part, also to the contraction of the EAS, and, in the distal part, to that of the EAS alone.

The consequence of the contraction of the puborectalis muscle is the lifting up of the anal canal in the ventral or anterior direction causing the compression not only of the anal canal itself, but also of the vagina, and the urethra against the rear aspect of pubic symphysis. Therefore there would be a high-pressure zone in the vagina [41]. As a matter of fact the analysis of the vaginal high pressure zone reveals that the anterior and posterior pressures are higher than the lateral one, which suggests that vagina is compressed in the anterior-posterior direction by the contraction of puborectalis.

3D US images show that the dimensions of the pelvic floor hiatus become respectively smaller and larger with the contraction of the puborectalis [42]. During the pudendal nerve block, the pelvic floor hiatus is increased and the vaginal pressure is decreased.

1.9 Anorectal Function and Fecal Incontinence

Feces stored in the colon are transported past the rectosigmoid “physiological sphincter” into the normally empty rectum, which can store up to 300 ml of contents. Rectal distension causes regular contractions of the rectal wall, which is affected by the intrinsic nervous (myenteric) plexus, and prompts the desire to defecate. Stool entering the rectum is also detected by stretch receptors in the rectal wall and pelvic floor muscles; their discharge leads to the urge to defecate. It starts as an intermittent sensation, which becomes more and more constant. Contraction of the pelvic floor muscles may interrupt the process, probably by concomitant inhibitory influences to the defecatory neural “pattern generator,” but also by mechanical insistence on sphincter contraction and the propelling of feces back to the sigmoid colon. The pelvic floor muscles are intimately involved in anorectal function. Apart from the “sensory” role of the pelvic floor muscles and the external anal sphincter function, the puborectalis muscle is thought to maintain the “anorectal angle,” which facilitates continence, and has to be relaxed to allow defecation. Current concepts suggest that defecation requires increased rectal pressure coordinated with relaxation of the anal sphincters and pelvic floor muscles. Pelvic floor relaxation allows opening of the anorectal angle and perineal descent, facilitating fecal expulsion. Puborectalis and external anal sphincter activity during evacuation is generally inhibited. However, observations by EMG and defecography suggest that the puborectalis may not always relax during defecation in healthy subjects.

The etiology of fecal incontinence is nowadays considered multifactorial and can be divided into the following pathophysiologies (1) diarrhea or excessive amount of liquid stools, (2) rectal dysfunction, and (3) anal canal closure impairment. Three distinct anatomical structures such as IAS, EAS, and puborectalis muscle are involved in the physiological anal canal opening and closure. The interaction of these structures is fundamental in maintaining rectal continence under different circumstances and for different contents (solids, liquids, air). The specific role of each muscular component has been deeply investigated in several studies. They demonstrated that patients with fecal incontinence have often more than one defect in muscles responsible for continence, and that the severity of symptoms is related to the amount of defects in multiple muscles, confirming the credence to the “multi-hit hypothesis” of fecal incontinence.

1.10 Pelvic Floor Muscles and Constipation

The etiology of constipation, just like that of incontinence, is multifactorial. Two major types of constipation are commonly described: (1) slow transit type in which the movement of stools through the colon is reduced and (2) outlet obstruction type, in which there are some problems in emptying rectal contents. Outlet obstruction, related to pelvic floor dysfunction, accounts for 50 % or more cases of constipation in adults. Several names, i.e., anismus, pelvic floor dyssynergia, paradoxical puborectalis contraction, paradoxical sphincter contraction, and dyssynergic defecation have been commonly used to define this condition. Normally, during pushing for obtain defecation or in Valsalva maneuver, the respiratory diaphragm and the abdominal wall contract together, resulting in an increase in the intra-abdominal and rectal pressure.

Simultaneously, a relaxation of the pelvic floor and anal sphincter is observed. Analyzing the rectal and anal sphincter pressures, Rao classified dyssynergic defecation disorders into three different types.

Type 1 – increase in rectal pressure but anal sphincter contraction, Type 2 – no increase in the rectal pressure and in anal sphincter contraction, and Type 3 – increase in the rectal pressure but absent or incomplete sphincter relaxation.

Dyssynergic defecation is usually acquired in adult age, but in some cases symptoms begin in childhood: this fact suggests that the individual has never been able to learn correctly the defecation mechanism.

During defecation, pelvic floor muscles must undergo two modifications: (1) pelvic floor descent and (2) pelvic floor hiatus enlargement. The descent of pelvic floor muscles leads the anorectal angle below the pubococcygeal line (an imaginary line going from the lower end of pubic symphysis to the coccyx) and causes a widening of the pelvic floor hiatus with an increase in the anorectal angle. On the contrary, during pelvic floor contraction, the anorectal angle is pushed cranially and ventrally.

1.11 Mechanism of Urinary Continence

Urethral closure pressure must be greater than bladder pressure, both at rest and during increases in abdominal pressure to retain urine in the bladder. The resting tone of the urethral muscles maintains a favorable pressure relative to the bladder when urethral pressure exceeds the bladder pressure. During activities, a dynamic process increases urethral closure pressure to enhance urethral closure and maintain continence.

At rest continence is assured by a competent sphincter mechanism, including not only the striated and smooth muscle sphincter, but also the pelvic floor muscles and an adequate bladder storage function. Normal kinesiological sphincter EMG recordings show continuous activity of motor units at rest (as defined by continuous firing of motor unit potentials), which as a rule increase with increasing bladder fullness. Reflexes mediating excitatory outflow to the sphincters are organized at the spinal level (the guardian reflex).

The L region in the brainstem has also been called the “storage center”. This area is thought to exert a continuous exiting effect on the Onuf’s nucleus and thereby on the striated urinary sphincter during the storage phase; in humans it is probably part of a complex set of “nerve impulse pattern generators” for different coordinated motor activities such as breathing, coughing, straining, etc.

The dominant element in the urethral sphincter is the striated urogenital sphincter muscle, which contains a striated muscle in a circular configuration in the middle of the urethra and strap-like muscles distally. In its sphincteric portion, the urogenital sphincter muscle surrounds two orthogonally arranged smooth muscle layers and a vascular plexus that helps to maintain closure of the urethral lumen.

Anatomically the urethra can be divided longitudinally into percentiles, with the internal meatus representing point zero and the external meatus representing the 100th percentile. The urethra passes through the wall of the bladder at the level of the vesical neck where the detrusor muscle fibers extend below the internal urethra meatus to as far as the 15th percentile. The striated urethral sphincter muscle begins at the termination of the detrusor fibers and extends to the 64th percentile. It is circular in configuration and completely surrounds the smooth muscle of the urethral wall. Starting at the 54th percentile, the striated muscles of the urogenital diaphragm, the compressor urethrae and the urethrovaginal sphincter can be seen. They are continuous with the striated urethral sphincter and extend to the 76th percentile. Their fiber direction is no longer circular. The fibers of the compressor urethrae pass over the urethra to insert into the urogenital diaphragm near the pubic ramus. The urethrovaginal sphincter surrounds both the urethra and the vagina. The distal terminus of the urethra runs adjacent to, but does not connect with, the bulbocavernosus muscle. Functionally, the urethral muscles maintain continence in various ways. The U-shaped loop of the detrusor smooth muscle surrounds the proximal urethra, favoring its closure by constricting the lumen. The striated urethral sphincter is composed mainly of type 1 (slow twitch) fibers, which are well suited to maintaining constant tone as well as allowing voluntary increases in tone to provide additional continence protection. Distally, the recruitment of the striated muscle of the urethrovaginal sphincter and the compressor urethrae compress the lumen. The smooth muscle of the urethra may also play a role in determining stress continence. The lumen is surrounded by a prominent vascular plexus that is believed to contribute to continence by forming a watertight seal via coaptation of the mucosal surfaces. Surrounding this plexus is the inner longitudinal smooth muscle layer. This in turn is surrounded by a circular layer of striated muscle.

The attachments formed by the fascia connect the periurethral tissue and the anterior vaginal wall bilaterally to the arcus tendineus, whereas the muscular structures connect the periurethral tissue to the medial aspect of the levator ani [43]. Urethral support is warranted by a simultaneous action of fascia and muscles working together under the neural control. This musculofascial support forms a hammock against which the urethra is compressed during any increase in abdominal pressure.

The mechanism of continence is furthermore increased by the action of the urethral sphincter. Therefore the failure of one of the support components will not always be followed by a situation of stress incontinence, thanks to the compensatory effect of the other component. This may explain why urethral hypermobility does not produce incontinence in every woman. It may also explain why bulking agents, injected in the urethral wall, can be useful in women with urethral hypermobility, as they may improve urethral sphincter function.

During physical stress the urethral and anal sphincters may not be sufficient to passively withhold the pressures arising in the abdominal cavity, and hence within the bladder and lower rectum. Activation of the pelvic floor muscles is mandatory and may be perceived as occurring in two steps by two different activation processes. Coughing and sneezing are thought to be generated by individual pattern generators within the brainstem, and thus activation of pelvic floor muscles is a preset coactivation and not primarily a "reflex" reaction to increased intra-abdominal pressure. In addition there may be an additional reflex pelvic floor muscle response to increased abdominal pressure due to distension of muscles spindle within pelvic floor muscles.

The pelvic floor muscles can of course also be voluntarily activated anticipating an increase in abdominal pressure. Such timed voluntary activity may be learned.

Functionally, the levator ani muscle and the endopelvic fascia play an interactive role in maintaining continence. Impairments usually become evident when the system is stressed. The increase in abdominal pressure acts transversally across the urethra so that the anterior wall is deformed toward the posterior wall, and the lateral walls are deformed toward one another, thereby helping to close the urethral lumen and prevent leakage due to the concomitant increase in intravesical pressure. If there are breaks in the continuity of the endopelvic fascia or if the levator ani muscle is damaged, the supportive layer under the urethra will be more compliant and will require a smaller pressure increment to displace a given distance. During a cough, the levator ani muscle contracts simultaneously with the diaphragm and abdominal wall muscles to build abdominal pressure. This levator ani contraction helps to tense the suburethral fascial layer, thereby enhancing urethral compression. It also protects the connective tissue from undue stresses.

1.11.1 Urogenital Diaphragm (Perineal Membrane) and Perineal Body

The urogenital diaphragm is a strong, muscular membrane running between the symphysis pubis and ischial tuberosities across the triangular anterior part of the pelvic outlet. It lies external and inferior to the pelvic diaphragm. It contains three contiguous striated muscles: urethral compressor, urethral sphincter and urethrovaginalis sphincter; its inferior fascia is called the perineal membrane [44].

The perineal membrane is a complex three-dimensional structure composed by two distinct different regions, respectively dorsal and ventral; it is not indeed a simple trilaminar sheet perforated by pelvic viscera.

The more superficial ischiocavernosus and bulbocavernosus muscles, in conjunction with the thin layer of the superficial transverse perineal m. [45], complete the inferior aspect of the urogenital diaphragm. The structure fills the space between the inferior pubic rami and the perineal body, thus closing the urogenital (levator) hiatus; it supports and has a sphincter-like effect at the level of the distal vagina; moreover, thanks to its attachment to the periurethral striated muscles, it contributes to continence, providing also a structural support for the distal urethra. The posterior triangle around the anus does not present a corresponding diaphragm or a similar membrane.

The perineal body is a pyramidal fibromuscular structure with a cephalad apex situated in the midline between the anus and vagina, linked cranially to the rectovaginal septum. Below this, muscles and fascia converge and interlace medially. Attached to the perineal body are situated in the rectum, the vagina and the anal sphincter; it also contains smooth muscles, elastic fibers and nerve endings. During childbirth, the perineal body is heavily distended. Acquired weakness of the perineal body may predispose to defects such as rectocele and enterocele [46].

Using an MRI scan, three distinct perineal body regions are visible: (1) a superficial region situated at the level of the vestibular bulb, (2) a mid-region corresponding to the proximal end of the superficial transverse perineal muscle, and (3) a deeper region at the level of the mid-urethra and of the puborectalis muscle [47] (Fig. 1.8).

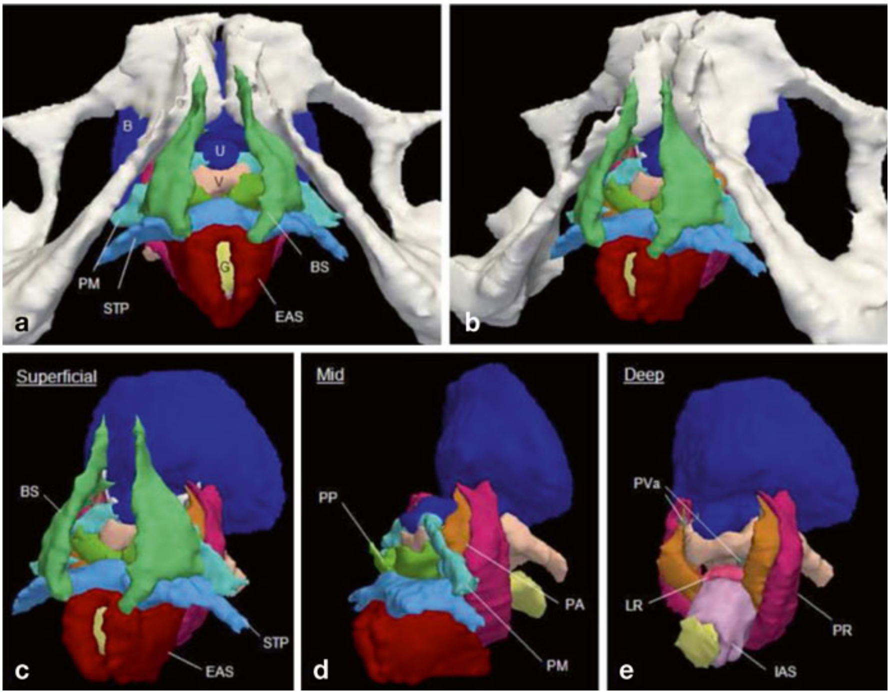

Fig. 1.8 (**a**) Dorsal lithotomy view and (**b**) left lateral aspect of same image. With removal of pelvic bones able to better appreciate superficial region (**c**) with BS, STP and EAS. (**d**) Lateral view with BS removed to illustrate mid region at proximal STP with PP, PM and PA. With removal of EAS, STP, PP, and PM can visualize deep region (**e**) with LR, PVa, PA. IAS visible with EAS removed. PVa muscle is barely visible lateral to the vagina in this image. PR-puborectalis, B-bladder, U-urethra, V-vagina, G-GI tract. © DeLancey 2010. Kindra A. Larson, et al. Am J Obstet Gynecol. ;203(5):494.e15-494.e21

1.12 Endopelvic Fascia and Connective Tissue Supports

This structure is situated just beneath the peritoneum and presents various thickenings in different areas. The endopelvic fascia, often called subserous fascia from an anatomical point of view, is continuous with the visceral one, which provides a sort of capsule containing pelvic organs and allows their displacements and possible changes in size. The distinct regions of this structure are indicated with different names, both ligaments and fascia. So we can distinguish, e.g., the pubourethral ligaments, the broad ligaments, the cardinal or Mackenrodt's ligaments, the uterosacral ligaments, and the arcus tendineus of the pelvic fascia.

1.12.1 Anterior Compartment Supports

The connective tissue supporting the urethra, bladder, and vagina takes origin from the arcus tendineus of the pelvic fascia [48, 49]. The pubourethral ligaments are situated anteriorly and consist of connective tissue structures that extend from the urethra to the pubic bone.

Because this represents an attachment of the lower third of the urethra to the pubis, it is postulated that exist two separate structures – one supporting the mid- or distal urethra and the other adjacent to the bladder neck – that must open it during voiding. The distal support is described as a connective tissue that links the vaginal wall and the periurethral tissue to the arcus tendineus of the pelvic fascia and to the levator ani muscles.

Urethropelvic ligaments running from the suburethral fascia at the level of bladder neck and proximal urethra to the levators and to arcus tendineus have been demonstrated using MRI imaging. DeLancey demonstrated the presence of a fascial layer situated suburethrally on the anterior vaginal wall. On the other hand, there is little actual endopelvic fascia at the level of the bladder base between the bladder and vaginal wall, so that the support is warranted by the lateral attachment of the vagina to the arcus tendineus of the pelvic fascia.

The pubocervical fascia has been described as a structure extending from the symphysis along the anterior vaginal wall to blend with the fascia surrounding the cervix. It is continuous with the pubococcygeus muscle laterally and it is suspended to the arcus tendineus too.

1.12.2 Apical Supports

The paracolpium and parametrium are the segments of connective tissues surrounding respectively the vagina and the uterus. In the mid-vagina, the paracolpium fuses laterally with the pelvic [28]. The cardinal ligaments (also called the ligaments of Mackenrodt) extend from the lateral aspects of the cervix and upper third of vagina to the lateral pelvic walls. They are connected to a large surface coming from the greater sciatic foramen over the piriformis muscles and from the region of the sacroiliac joint to the lateral side of sacrum. They represent a condensation of the lower parts of the broad ligaments. Laterally, they are also continuous with the connective tissue surrounding the hypogastric vessels. Medially, they are continuous with the paracolpium and parametrium and also with the connective tissue of the anterior vaginal wall, that is the pubocervical fascia.

Posterolaterally we found the uterosacral ligaments that are attached to the cervix and upper vaginal fornices. They attach to the presacral fascia in front of the sacroiliac joint. The connective tissue of the latter is continuous around the cervix with that of the cardinals. This complex of ligaments, i.e., cardinal and uterosacral ligaments, have the responsibility of keeping the uterus and upper vagina in their proper place over the levator plate.

1.12.3 Posterior Compartment Supports

The posterior vaginal wall, below the cardinals, is supported from both sides by the paracolpium, which is attached to the endopelvic fascia (referred to as rectovaginal fascia) and pelvic diaphragm. In this region there is only one attachment to the vagina, and there are not two distinct systems for the anterior and posterior vaginal sides. Therefore, when an increase of abdominal pressure pushes the vaginal downward, its attachments to the levator muscles prevent this downward movement.

Posteriorly, a rectovaginal septum, consisting of fibromuscular and elastic tissue and extending from the peritoneum to the perineal body has been described. We must remember that the peritoneal cavity extends to the cranial part of the perineal body during fetal life, but it is rapidly closed in early life. Its layers (known as the Denonvillier's fascia) probably become part of the rectovaginal septum that appears to be adherent to the surface of the posterior vaginal wall. This fascia forms the anterior margin of another potential space, the rectovaginal one. The rectovaginal septum, when intact, allows independent mobility of the rectum and the vagina. The mid-posterior vaginal wall is connected to the levator ani by the endopelvic fascia. These connections prevent the vagina from moving forward during the increases of abdominal pressure. The medial aspect of these parallel sheets is referred to as the rectal pillars.

In the distal vagina, 2–3 cm above the hymeneal ring, the vaginal wall is connected to surrounding structures without any intervening paracolpium. Anteriorly, the vagina is fused with the urethra and the connective tissue of the perineal structures of the urogenital diaphragm. It blends with the levator ani muscles laterally, while it fuses with the perineal body posteriorly. The rectovaginal fascia is thick [50] and therefore the vagina has no mobility in this area [51]. If the attachments of the perineal membrane to the perineal body is lacking as a consequence of fascial disruption, the bowel protrudes downward creating a posterior vaginal wall prolapse. The fascial supports for the rectum, that is the lateral rectal ligaments, extend from the posterolateral pelvic side wall (about at the level of the third sacral vertebra) to the rectum.

As outlined before, the cranial part of the vagina is suspended by the uterosacral ligaments. The compartment is rather wide at this point and there is not a direct contact between the genital tract and the pelvic walls.

In the mid-portion, the shape of the posterior compartment becomes smaller with the posterior vagina forming the ventral boundary, while the lateral margins are

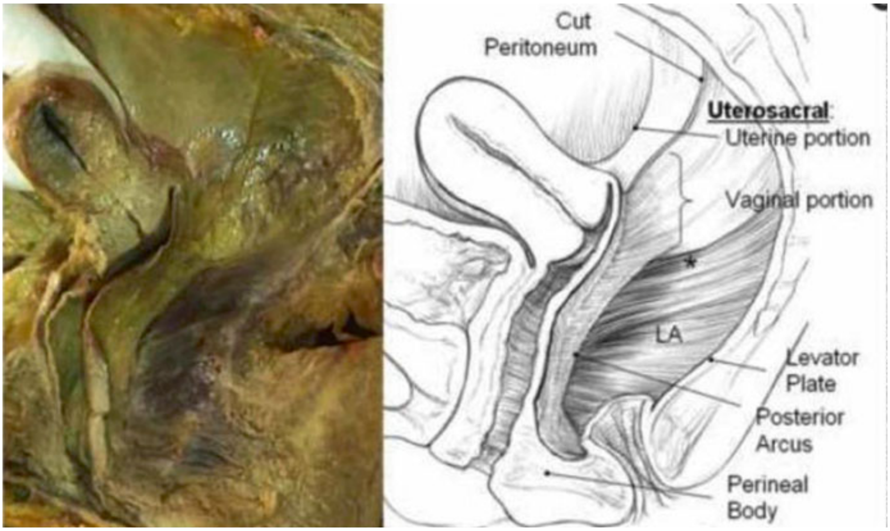

Fig. 1.9 Cadaver dissection (left) and illustration (right) of posterior compartment of a 56 year old multipararous female showing structural relationships after the rectum has been removed. Note the apical connections of the upper posterior vagina to the inside of the pelvic wall in a retroperitoneal position. These lie below the peritoneum and are dorsal and caudal to what is traditionally referred to as the utero-sacral ligament. These structures are continuous with the posterior arcus tendineus fascia pelvis. At the distal end of the vagina the wall merges into the top of the perineal body. The lateral and dorsal margins of the compartment are formed by the levator ani muscles (LA) and the levator plate. The asterisk (*) denotes the region of the sacrospinous ligament overlain by the coccygeus muscle. © DeLancey 2007

formed by the pubococcygeus muscle and the dorsal aspect is formed by the puborectalis. The lateral margins of the vagina form a visible line indicated as the posterior arcus tendineus of pelvic fascia (Fig. 1.9).

Although the vagina in this portion is closer to the fibers of the levator ani, these structures are separated by a perivascular halo. In the lower portion of the posterior region, the perivascular halo is lost as the vagina appears fused to the adjacent levator ani, perineal body, and anus. The bottom of this compartment is marked where the subdivisions of the external anal sphincter and the perineal body can be seen [52].

1.13 Relationship Between the Pelvic Floor Muscles and the Fascial Support System

The interaction between the pelvic floor muscles and the supportive ligaments is critical to pelvic organ support. According to Ashton-Miller and DeLancey, as long as the levator ani muscles function to properly maintain closure of the genital hiatus, the ligaments and fascial structures supporting the pelvic organs are under minimal tension. The fasciae simply act to stabilize the organs in their position above the levator ani muscles. The constant tone maintained by the pelvic muscles relieves the tension placed on the endopelvic fascia. If the nerves to the levator ani muscles are damaged (such as during childbirth), the denervated muscles would atrophy and leave the responsibility of pelvic organ support to the endopelvic fascia alone. When the pelvic floor muscles relax or are damaged, the pelvic floor opens and the vagina lies between the zones of high abdominal pressure and low atmospheric pressure outside the body. In this situation it must be held in place by the suspensory ligaments. Although the ligaments can sustain these loads for short periods of time, if the pelvic floor muscles do not close the pelvic floor then the connective tissue will eventually fail and these ligaments gradually stretch under the constant load. This viscoelastic behavior leads to the development of pelvic organ prolapse. The attachment of the levator ani muscles into the perineal body is important and damage to this part of the levator ani muscle during delivery is one of the irreparable injuries to pelvic floor. It has been shown that up to 20 % of primiparous women have a visible defect in the levator ani muscle on MRI.

1.14 Summary and Conclusions

Pelvic floor muscles have two fundamental functions: they must provide (1) a physical support to the pelvic viscera and (2) the constrictor mechanism to the anal canal, vagina, and urethra. Newer imaging and physiological studies strongly suggest that these two functions are quite distinct and are likely related to different components of the pelvic floor muscles.

The pubococcygeus, iliococcygeus, and ischiococcygeus muscles are referred as providing the physical support or act as a “floor” for the pelvic viscera. On the other hand, the puborectalis muscle provides the closure mechanism for the anal canal, vagina, and urethra.

The urethra and anal canal have two separate sphincters of their own. As regards the anal canal these are the IAS and EAS, while for the urethra they are represented by the smooth sphincter situated at the bladder neck, and by the striated external urethral sphincter. Looking to the physiological studies, it appears that the puborectalis muscle may be considered as the 3rd constrictor, with a sphinteric function for the anal canal. The puborectalis serves also as a constrictor for the urethra itself. On the other hand, the vagina has only one closure mechanism, provided by the puborectalis portion or the pelvic floor muscle. We believe therefore that puborectalis muscle represents the real point of linkage among gastroenterologists, colorectal surgeons, urologists, and urogynecologists: all of them are the subspecialists taking care of patients with pelvic floor problems or dysfunctions. Recently this figure may be represented by a kind of new hyperspecialist named perineologist.

References

1. Strohbehn K (1998) Normal pelvic floor anatomy. Obstet Gynecol Clin North Am 25:683–705
2. Lawson JO (1974) Pelvic anatomy I: pelvic floor muscles. Ann R Coll Surg Engl 54:244–252
3. DeLancey JO, Delmas V (2004) Groos anatomy and functional anatomy of the pelvic floor. Elsevier Saunders, Philadelphia
4. Kearney R, Sawhney R, DeLancey JO (2004) Levator ani muscle anatomy evaluated by origin-insertion pairs. Obstet Gynecol 104:168–173

5. Salmons S et al (1995) Muscle. In: Williams PL, Bannister LH, Berry MM et al (eds) Gray's anatomy, 38th edn. Churchill Livingstone, New York, pp 737–900
6. DeLancey JO, Hurd WW (1998) Size of the urogenital hiatus in the levator ani muscles in normal women and women with pelvic organ prolapse. Obstet Gynecol 91:364–368
7. Dietz HP, Franco AV, Shek KL, Kirby A (2013) Avulsion injury and levator hiatal ballooning: two independent risk factors for prolapse? An observational study. Am J Obstet Gynecol 208(2):148.e1–148.e7. doi:10.1016/j.ajog.2012.12.006. Epub 2012 Dec 5
8. Fielding JR, Dumanli H, Schreyer AG et al (2000) MR-based three-dimensional modeling of the normal female pelvic floor in women: quantification of muscle mass. AJR Am J Roentgenol 174:657–660
9. Gilpin SA, Gosling JA, Smith AR, Warrell DW (1989) The pathogenesis of genitourinary prolapse and stress urinary incontinence of urine: a histological and histochemical study. Br J Obstet Gynaecol 96:15–23
10. Sebe P, Fritsch H, Oswald J, Schwentner C, Lunacek A, Bartsch G, Radmayr C (2005) Fetal development of the female external urinary sphincter complex: an anatomical and histological study. J Urol 173:1738–1742
11. Dorschner W, Biesold M, Schmidt F, Stolzenburg JU (1999) The dispute about the external sphincter and the urogenital diaphragm. J Urol 162:1942–1945
12. Raizada V, Mittal RK (2008) Pelvic floor anatomy and applied physiology. Gastroenterol Clin North Am 37(3):493–vii
13. Fritsch H, Brenner E, Lienemann A, Ludwikowski B (2002) Anal sphincter complex: reinterpreted morphology and its clinical relevance. Dis Colon Rectum 45(2):188–194 [PubMed]
14. Bogduk N (1996) Issues in anatomy: the external anal sphincter revisited. Aust N Z J Surg 66:626–629
15. Peschers UM, DeLancey JO, Fritsch H, Quint LE, Prince MR (1997) Cross-sectional imaging anatomy of the anal sphincters. Obstet Gynecol 90(5):839–844
16. Bharucha AE (2006) Pelvic floor: anatomy and function. Neurogastroenterol Motil 18(7):507–519
17. Li D, Guo M (2007) Morphology of the levator ani muscle. Dis Colon Rectum 50:1831–1839
18. Zijta FM, Froeling M, van der Paardt MP, Lakeman MME, Bipat S, Montauban van Swijndregt AD, Strijkers GJ, Nederveen AJ, Stoker J (2011) Feasibility of diffusion tensor imaging (DTI) with fibre tractography of the normal female pelvic floor. Eur Radiol 21(6):1243–1249
19. Guaderrama NM, Liu J, Nager CW et al (2005) Evidence for the innervation of pelvic floor muscles by the pudendal nerve. Obstet Gynecol 106:774–781
20. Dyck PJ, Thomas PK (2005) Autonomic and somatic systems to the anorectum and pelvic floor, 4th edn. Elsevier Saunders, Philadelphia
21. Barber MD, Bremer RE, Thor KB, Dolber PC, Kuehl TJ, Coates KW (2002) Innervation of the female levator ani muscles. Am J Obstet Gynecol 187:64–71
22. Grigorescu BA, Lazarou G, Olson TR, Downie SA, Powers K, Greston WM, Mikhail MS (2008) Innervation of the levator ani muscles: description of the nerve branches to the pubococcygeus, iliococcygeus, and puborectalis muscles. Int Urogynecol J Pelvic Floor Dysfunct 19:107–116
23. Wallner C, van Wissen J, Maas CP, Dabhoiwala NF, Deruiter MC, Lamers WH (2007) The contribution of the levator ani nerve and the pudendal nerve to the innervation of the levator ani muscles; a study in human fetuses. Eur Urol 54:1143–1144
24. Thor KB, de Groat WC (2010) Neural control of the female urethral and anal rhabdosphincters and pelvic floor muscles. Am J Physiol Regul Integr Comp Physiol 299(2):R416–R438
25. Hurtado EA, Smith PP, Smith CP, Boone TB, Somogyi GT (2008) Urethral afferent signaling leads to activation of the external urethral sphincter and abdominal wall muscles. Neurourol Urodyn 27:105
26. Allen RE, Hosker GL, Smith AR, Warrell DW (1990) Pelvic floor damage and childbirth: a neurophysiological study. Br J Obstet Gynaecol 97:770–779
27. Smith AR, Hosker GL, Warrell DW (1989) The role of partial denervation of the pelvic floor in the aetiology of genitourinary prolapse and stress incontinence of urine. A neurophysiological study. Br J Obstet Gynaecol 96:24–28
28. Snooks SJ, Swash M, Mathers SE, Henry MM (1990) Effect of vaginal delivery on the pelvic floor: a 5-year follow-up. Br J Surg 77:1358–1360

29. South MM, Stinnett SS, Sanders DB, Weidner AC (2009) Levator ani denervation and reinnervation 6 months after childbirth. Am J Obstet Gynecol 200:519.e1–519.e7
30. Weidner AC, Jamison MG, Branham V, South MM, Borawski KM, Romero AA (2006) Neuropathic injury to the levator ani occurs in 1 in 4 primiparous women. Am J Obstet Gynecol 195:1851–1856
31. Weidner AC, Sanders DB, Nandedkar SD, Bump RC (2000) Quantitative electromyographic analysis of levator ani and external anal sphincter muscles of nulliparous women. Am J Obstet Gynecol 183:1249–1256
32. Weidner AC, South MM, Sanders DB, Stinnett SS (2009) Change in urethral sphincter neuromuscular function during pregnancy persists after delivery. Am J Obstet Gynecol 201:529.e1–529.e6
33. Abdool Z, Shek KL, Dietz HP (2009) The effect of levator avulsion on hiatal dimension and function. Am J Obstet Gynecol 201(1):89.e1–89.e5
34. Dietz HP, Franco AV, Shek KL, Kirby A (2012) Avulsion injury and levator hiatal ballooning: two independent risk factors for prolapse? An observational study. Acta Obstet Gynecol Scand 91(2):211–214
35. Lammers K, Fütterer JJ, Inthout J, Prokop M, Vierhout ME, Kluivers KB (2013) Correlating signs and symptoms with pubovisceral muscle avulsions on magnetic resonance imaging. Am J Obstet Gynecol 208(2):148.e1–148.e7
36. Guo M, Li D (2007) Pelvic floor images: anatomy of the levator ani muscle. Dis Colon Rectum 50:1647–1655
37. Hjartardottir S, Nilsson J, Petersen C, Lingman G (1997) The female pelvic floor: a dome–not a basin. Acta Obstet Gynecol Scand 76:567–571
38. Bo K, Lilleas F, Talseth T, Hedland H (2001) Dynamic MRI of the pelvic floor muscles in an upright sitting position. Neurourol Urodyn 20:167–174
39. Jung SA, Pretorius DH, Weinstein M, Nager CW, Den-Boer D, Mittal RK (2008) Closure mechanism of the anal canal in women: assessed by three-dimensional ultrasound imaging. Dis Colon Rectum 51:932–939
40. Liu J, Guaderrama N, Nager CW, Pretorius DH, Master S, Mittal RK (2006) Functional correlates of anal canal anatomy: puborectalis muscle and anal canal pressure. Am J Gastroenterol 101:1092–1097
41. Guaderrama NM, Nager CW, Liu J, Pretorius DH, Mittal RK (2005) The vaginal pressure profile. Neurourol Urodyn 24:243–247
42. Weinstein MM, Jung SA, Pretorius DH, Nager CW, den Boer DJ, Mittal RK (2007) The reliability of puborectalis muscle measurements with 3-dimensional ultrasound imaging. Am J Obstet Gynecol 197(68):e61–e66
43. DeLancey JOL (1989) Pubovesical ligament: a separate structure from the urethral supports ('pubourethral ligaments'). Neurourol Urodyn 8:53–61
44. Stein TA, DeLancey JOL (2008) Structure of the perineal membrane in females: gross and microscopic anatomy. Obstet Gynecol 111(3):686–693
45. DeLancey JOL (1999) Structural anatomy of the posterior compartment as it relates to rectocele. Am J Obstet Gynecol 80:815–823
46. Nichols DH (1997) Central compartment defects. In: Rock JA, Thompson JD (eds) Te Linde's operative gynecology, 8th edn. Lippincott-Raven Publishers, Philadelphia, pp 1006–1030
47. Larson KA, Yousuf A, DeLancey JOL (2010) Perineal body anatomy in living women: 3-D analysis using thin-slice magnetic resonance imaging. Am J Obstet Gynecol 203(5):494.e15–494.e21
48. DeLancey JO (1994) Structural support of the urethra as it relates to stress urinary incontinence: the hammock hypothesis. Am J Obstet Gynecol 170:1713–1723
49. Weber AM, Walters MD (1997) Anterior vaginal prolapse: review of anatomy and techniques of surgical repair. Obstet Gynecol 89:311–318
50. Reiffenstuhl G (1993) Practical pelvic anatomy for the gynecologic surgeon. In: Nichols DH (ed) Gynecologic and obstetric surgery. Mosby, St. Louis, pp 26–71
51. DeLancey JOL (1992) Anatomic aspects of vaginal eversion after hysterectomy. Am J Obstet Gynecol 166:1717–1728
52. Yvonne HSU, Lewicky-Gaupp C, De Lancey JOL (2008) Posterior compartment anatomy as seen in MRI and 3-D reconstruction from asymptomatic nulliparas. Am J Obstet Gynecol 198(6):651.e1–651.e7