Chapter 7
Pelvic Floor Anatomy as It Relates to the Design and Development of Vaginal Mesh Kits

Jonia Alshiek and S. Abbas Shobeiri

Introduction

Pelvic floor disorders, including urinary incontinence (UI), fecal incontinence, and pelvic organ prolapse (POP), represent a major public health issue in the United States [1]. Pelvic floor disorders, including POP and urinary incontinence, are debilitating conditions; 24% of adult women have at least one pelvic floor disorder [2], which results in surgery in one of nine women [3]. In the United States, the National Center for Health Statistics estimates 400,000 operations per year are performed for pelvic floor dysfunction, with 300,000 occurring in the inpatient setting [4]. A study of Australian women found that the lifetime risk of surgery for POP in the general female population was 19% [5]. In an Austrian study, an estimation of the frequency for post-hysterectomy vault prolapse requiring surgical repair was between 6% and 8% [6]. A single vaginal birth has been shown to significantly increase the odds of prolapse (OR 9.73, 95% CI 2.68–35.35). Additional vaginal births were not associated with a significant increase in the odds of prolapse [7].

It is forecast that the number of American women with at least one pelvic floor disorder will increase from 28.1 million in 2010 to 43.8 million in 2050. During this time period, the number of women with UI will increase 55% from 18.3 million to

J. Alshiek
Department of Obstetrics and Gynecology, Gynecologic Subspecialties, Inova Women's Hospital, Virginia Commonwealth University, Falls Church, VA, USA

Tel Aviv Sourasky Medical Center, Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel

S. A. Shobeiri (✉)
INOVA Health System, Falls Church, VA, USA
e-mail: Abbas.shobeiri@inova.org

S. A. Shobeiri (ed.), *The Innovation and Evolution of Medical Devices*,
https://doi.org/10.1007/978-3-319-97073-8_7

28.4 million. For fecal incontinence, the number of affected women will increase 59% from 10.6 to 16.8 million, and the number of women with POP will increase 46% from 3.3 to 4.9 million. The highest projections for 2050 estimate that 58.2 million women in the United States will have at least one pelvic floor disorder, 41.3 million with UI, 25.3 million with fecal incontinence, and 9.2 million with POP. This forecast has important public health implications. Understanding the causes of pelvic floor disorders is in its infancy. But what is known is that prolapse arises because of injuries and deterioration of the muscles, nerves, and connective tissue that support and control normal pelvic function. Conventionally, the surgical repair of pelvic floor disorders including stress urinary incontinence and pelvic organ prolapse was done using the patient's native tissue to restore the injured pelvic structure. However, the failure rate of this conventional treatment was high due to the reduced native connective tissue and musculature integrity, requiring repeated surgery in approximately 40% [3, 8, 9]. Therefore, a better method was required for a higher operative success rate. Both biological and synthetic grafts were introduced in order to provide such a purpose and decrease the failure rate [10].

The idea of using synthetic meshes as a treatment of pelvic floor organ prolapse originates from the general surgery field where they have used meshes for abdominal hernia repair. According to abdominal hernia studies, mesh repair-related outcomes were found to be superior to native tissue repair [11, 12]. Therefore, in the 1970s, gynecologists had assumed that similar outcomes would be achieved using meshes of abdominal hernia repair for abdominal pelvic floor organ prolapse or incontinence repair and had begun cutting hernia meshes and adjusting them for usage in abdominal pelvic organ prolapse repair. In 1990s, hernia meshes were first used for vaginal pelvic floor organ prolapse repair and for stress urinary incontinence repair. There were no specifically designed vaginal meshes, instead, gynecologists were using abdominal hernia meshes after cutting and fitting them to the specific pelvic repair that was required. In 1996 the first mesh for the treatment of SUI was introduced (ProteGen Sling, Boston Scientific Corporation, Marlborough MA, USA). Afterward, tension-free vaginal tape (TVT) followed by synthetic vaginal mesh kits which were introduced for vaginal repair of pelvic floor disorders. Understanding the anatomy will help us in understanding the rationale behind vaginal mesh kits usage and in which disorders it might be indicated. This chapter focuses on the *functional* anatomy of the pelvic floor, and how the anterior, posterior, apical, and lateral compartments are supported, as it relates to the design of vaginal mesh kits in women.

Support of the Pelvic Organs: Conceptual Overview

The pelvic organs rely on (1) their connective tissue attachments to the pelvic walls and (2) support from the levator ani muscles that are under neuronal control from the peripheral and central nervous systems. In this chapter, the term "pelvic floor" is used broadly to include all the structures supporting the pelvic cavity rather than the restricted use of this term to refer to the levator ani group of muscles. The

anterior and posterior vaginal mesh kits were designed to enhance or replace the underlying pubocervical or rectovaginal tissue, respectively. These kits could be used only after identifying precisely the underlying pathology that leads to the prolapsed organ; this will be discussed later in this chapter.

To convey the pelvic floor supportive structures' 3D architecture to the reader, we can use the "room analogy." Using this analogy, the reader can conceptualize the pelvic floor hiatus as the door out of a room (Fig. 7.1). Using this very simplified analogy, if you view the pelvic floor hiatus from where the sacrum is, the door frame for this room is the perineal membrane, the walls and the floor the levator ani muscle, and the ceiling the pubic bone. However, the pelvic floor is artificially separated into three compartments (Fig. 7.2). We arbitrarily call these anterior, middle, posterior, and lateral compartments (Fig. 7.3). The tissue separating the anterior and middle compartments is pubocervical fibromuscularis or pubocervical fascia. The

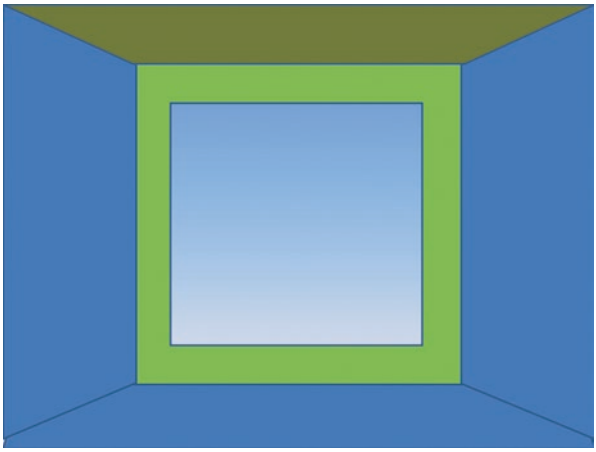

Fig. 7.1 Pelvic floor room analogy. (© Shobeiri 2013)

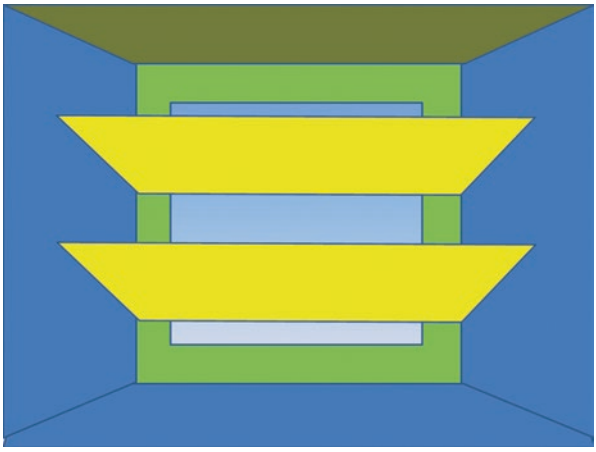

Fig. 7.2 Pelvic floor room analogy with three compartments separated. (© Shobeiri 2013)

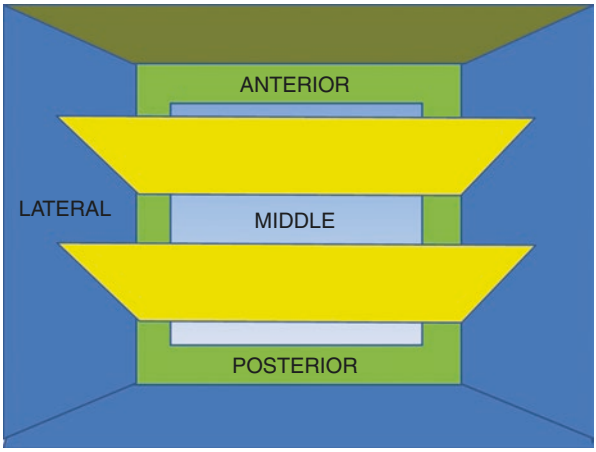

Fig. 7.3 Pelvic floor room analogy with anterior, middle, and posterior compartments and the lateral walls marked. (© Shobeiri 2013)

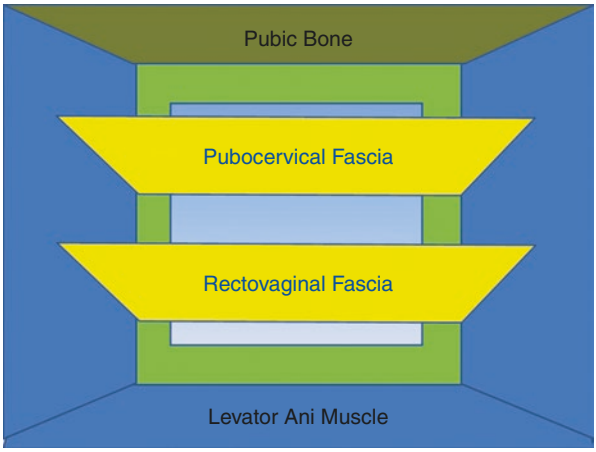

Fig. 7.4 Pelvic floor room analogy: pubocervical fibromuscularis and rectovaginal fascia separating the three compartments. (© Shobeiri 2013)

tissue separating the middle and posterior compartments is rectovaginal fibromuscularis or rectovaginal fascia or septum (Fig. 7.4). The pubocervical fibromuscularis and the rectovaginal septum are attached laterally to the levator ani muscle with thickening of adventitia in this area. Anatomically, the endopelvic fascia refers to the areolar connective tissue that surrounds the vagina. It continues down the length of the vagina as loose areolar tissue surrounding the pelvic viscera. Histologic examination has shown that the vagina is made up of three layers: epithelium, muscularis, and adventitia [13, 14]. The adventitial layer is loose areolar connective tissue made up of collagen and elastin, forming the vaginal tube. Therefore, the tissue that surgeons call fascia at the time of surgery is best described as fibromuscularis, since it is a mixture of muscularis and adventitia.

Anteriorly, pubocervical fibromuscularis is attached to the levator ani using arcus tendineus fascia pelvis (Fig. 7.5). Posterior attachment of rectovaginal septum to the levator ani is poorly understood, but we will refer to it as the posterior arcus

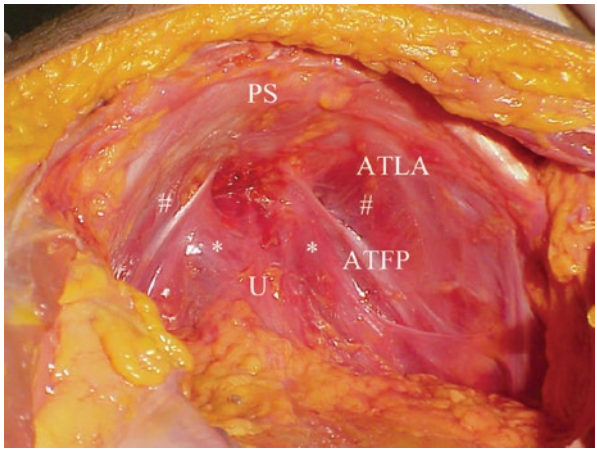

Fig. 7.5 Retropubic anatomy showing points of attachments of the arcus tendineus levator ani and the arcus tendineus fascia pelvis. The urethra sits on the hammock-like pubocervical fibromuscularis. # denotes the levator ani attachment to the obturator internus muscle. (© Shobeiri 2013)

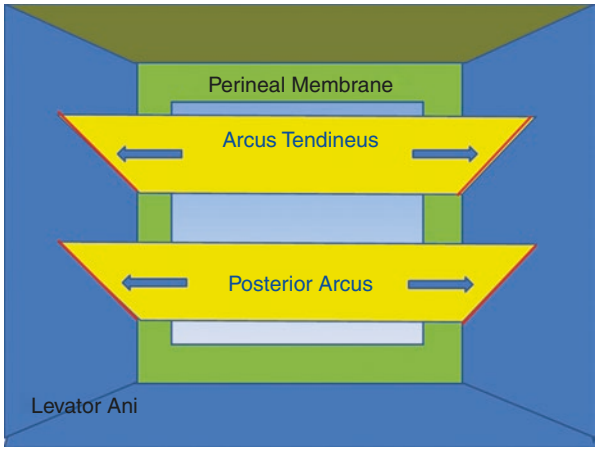

Fig. 7.6 Pelvic floor room analogy: the line of attachment of the pubocervical fascia to the levator ani is arcus tendineus fascia pelvis. The line of attachment of the rectovaginal fascia to the levator ani is the posterior arcus. Both are shown as *red lines*. (© Shobeiri 2013)

(Fig. 7.6) [15]. The anterior compartment is home to the urethra and the lower part of the bladder. The middle compartment is the vagina, and the posterior compartment is home to anorectum (Fig. 7.7). This analogy is not far from reality. When one looks at the pelvic floor structures, the three compartments are clearly separated as described (Fig. 7.8). Compartmentalization of the pelvic floor has led to different medical specialties looking at that specific compartment and paying less attention to the whole pelvic floor (Fig. 7.9).

If one looks at the middle compartment from the side, he or she can appreciate different levels of support as described by DeLancey (Fig. 7.10) [16]. Looking at these supportive structures from the sagittal view exposes the connective tissue elements that keep the room standing. Generally, a "suspension bridge" analogy is useful for describing these structures (Fig. 7.11). Although in the room analogy, the anterior, middle, and posterior compartments house the pelvic organs, in reality, the pelvic organs are part of the pelvic floor and play an important supportive role

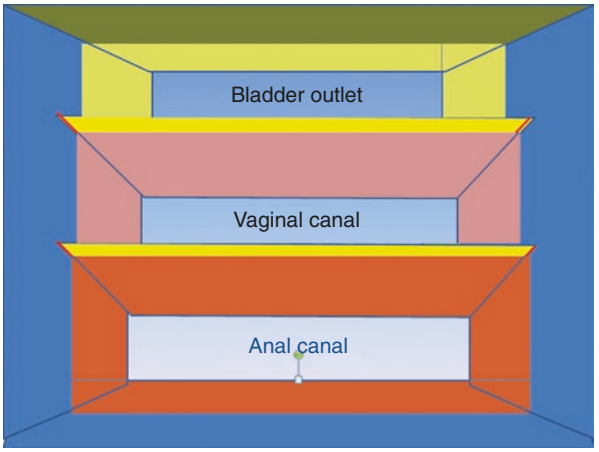

Fig. 7.7 Pelvic floor room analogy: three compartments separated. (© Shobeiri 2013)

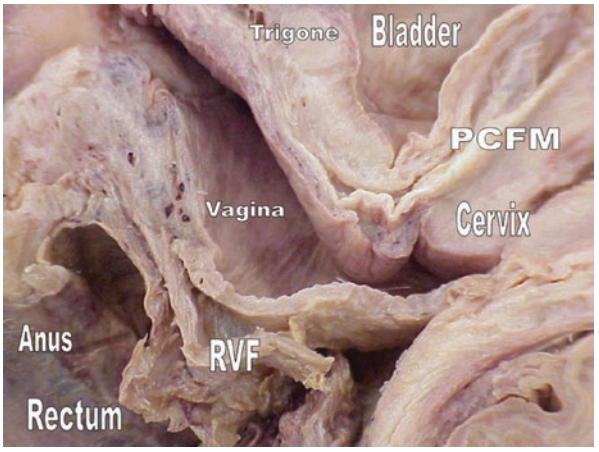

Fig. 7.8 Midsagittal anatomy of an intact cadaveric specimen demonstrating the three different compartments. (© Shobeiri 2013)

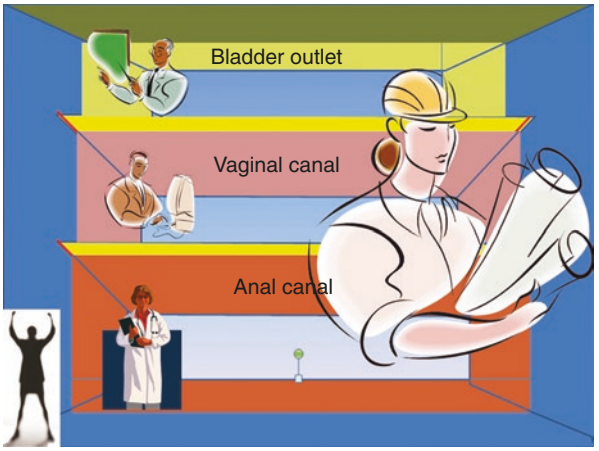

Fig. 7.9 Pelvic floor room analogy: each area or compartment may be managed by a different specialist. There is a great need for one specialty that understands the interaction between different compartments and manages them concurrently as much as possible. (© Shobeiri 2013)

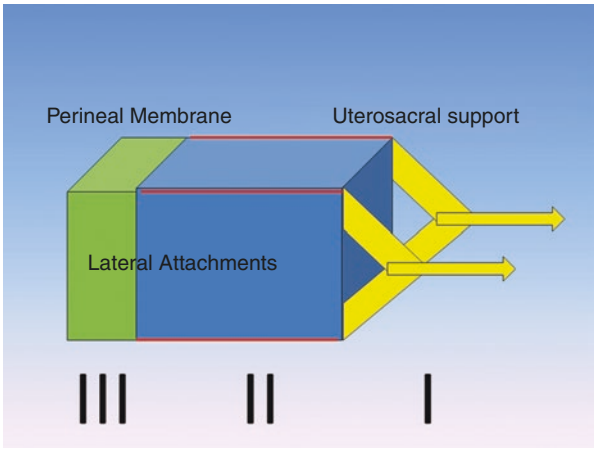

Fig. 7.10 Pelvic floor room analogy: Level 1 supports are provided by the uterosacral-cardinal ligament complex (*yellow arrows*), which keep the "room" upright. Level II supports are provided by the lateral tendineus attachments (*red lines).* The support is provided by perineal membrane (*green area).* (© Shobeiri 2013)

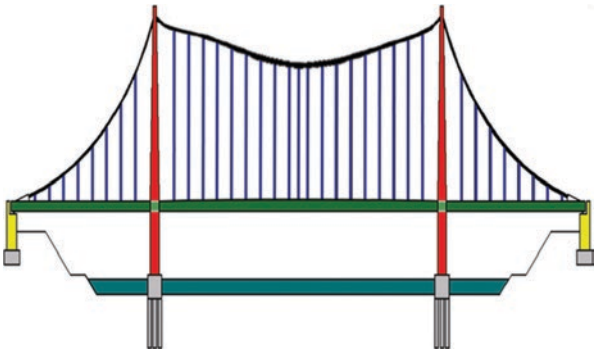

Fig. 7.11 Suspension bridge analogy; the depiction of a normal bridge. (© Shobeiri 2013)

through their connections with structures, such as the cardinal and uterosacral ligaments. Adapting this suspension bridge to the human body and the perineal body and the sacrum become the two anchoring points of the bridge. The perineal membrane (Level III) and the uterosacral and cardinal ligaments (Level I) form the two masts of the suspension bridge (Fig. 7.12). The lateral wires are the levator ani muscles of the lateral wall (Fig. 7.13), and the attachments of the vagina to the levator ani muscles laterally in the mid-part of the vagina form Level II support. The anterior and posterior vaginal mesh kits were designed to enhance or replace this Level II support mainly and Level I slightly. The levator ani muscles and the interconnecting fibromuscular structures support the bladder and urethra anteriorly, the vaginal canal in the middle, and the anorectal structures posteriorly (Fig. 7.14).

Like a room or a suspension bridge, the pelvic floor is subjected to loads that should be appropriate for its design. Should these loads exceed what the pelvic floor is capable of handling, there would be failure in one or multiple supportive elements. The pelvic floor is not a static structure. The levator ani works in concert with the ligamentous structures to withstand intraabdominal pressure that could predispose to POP and urinary or fecal incontinence during daily activities (Fig. 7.15).

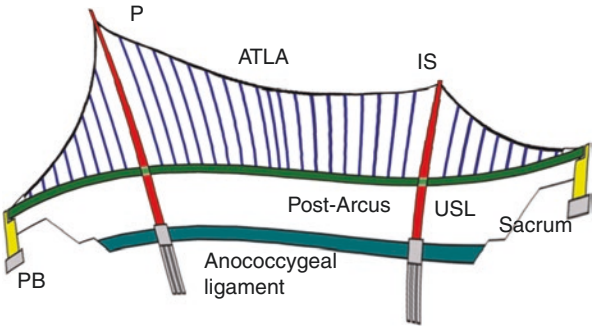

Fig. 7.12 Suspension bridge analogy; the depiction of a suspension bridge adapted to human female pelvic floor structures. The *red masts* are the ischial spine and the pubis. The *blue lines* are the levator ani fibers. The *green line* is the uterosacral ligaments continuous with the posterior arcus line. The anococcygeal ligament provides anchoring point for the posterior structures. (© Shobeiri 2013)

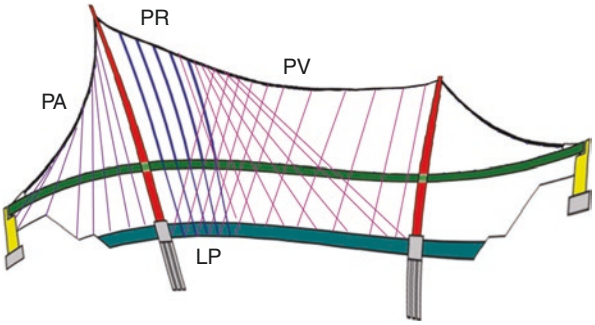

Fig. 7.13 Suspension bridge analogy; the depiction of a suspension bridge adapted to human female pelvic floor structures. The levator ani fibers have intricate and overlapping paths. The puboanalis (PA) and puboperinealis form some of the supportive structures of the perineum. The puborectalis (PR) fibers form the sling behind the rectum. Pubovisceralis (PV) is a collective term we have applied here to the iliococcygeus and pubococcygeus fibers. The levator plate (LP) is formed by overlapping of the iliococcygeus/pubococcygeus (PV) and the puborectalis (PR) fibers. (© Shobeiri 2013)

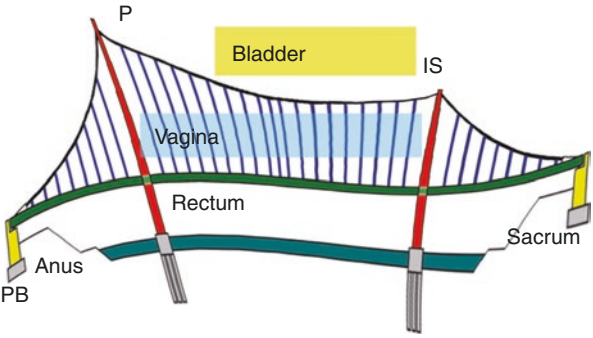

Fig. 7.14 Suspension bridge analogy; the depiction of different compartments of pelvic floor. (© Shobeiri 2013)

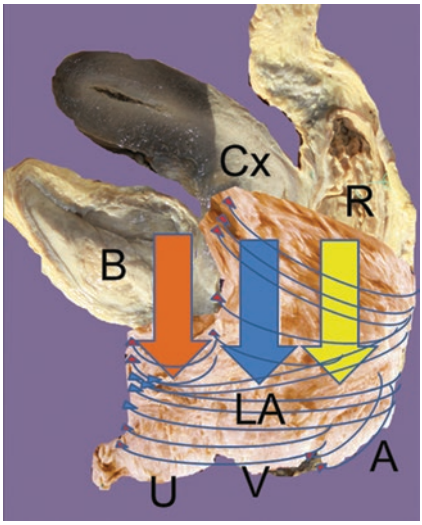

Fig. 7.15 Right lateral standing anatomic depiction of the three compartments exposed to intraabdominal pressure, which results in activation of the muscles to prevent prolapse or urinary and fecal incontinence. Bladder (B), cervix (Cx), rectum (R), levator ani (LA), urethra (U), vagina (V), anus (A). (© Shobeiri 2013)

The lower end of the pelvic floor is held closed by the pelvic floor muscles, preventing prolapse by constricting the base. The spatial relationship of the organs and the pelvic floor are important. Pelvic support is a combination of constriction, suspension, and structural geometry.

The levator ani muscle has puboperinealis, puboanalis, pubovaginalis, puborectalis, pubococcygeus, and iliococcygeus subdivisions (Fig. 7.16) [17]. The pubococcygeus is a functional unit of the iliococcygeus. These two collectively are known as the pubovisceralis muscle in our prior publications. In the older studies that utilized MRI for visualization of the levator ani muscles, pubovisceralis denotes puboperinealis, puboanalis, and pubovaginalis together. Due to the fact that ultrasound can see these subdivisions clearly, and the fact that the newer MRIs can see some of these subdivision better, the term pubovisceralis is falling out of favor. We will be using the exact terms when referring to these subdivisions. The relationship of these muscles to each other is interesting, as they crisscross in different angles to each other (Figs. 7.17 and 7.18).

Practical Anatomy and Prolapse

Overview

Level I support is composed of the uterosacral and cardinal ligaments that form the support of the cervix and upper one third of the vagina by attaching them to the pelvic wall. Stretching and failure of Level I can result in pure apical prolapse of the uterus or an enterocele formation. The cephalad arms of the vaginal mesh kits are meant to recreate the Level I support by attachment to the sacrospinous posteriorly

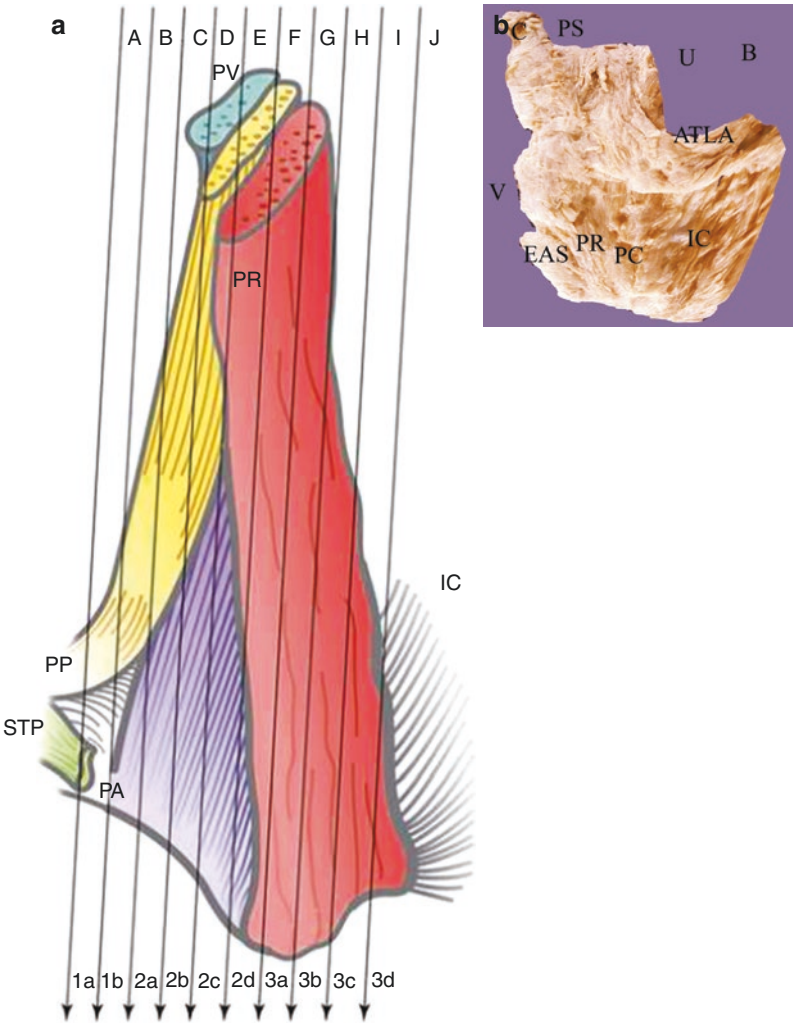

Fig. 7.16 (**a**) The relative position of levator ani subdivisions during ultrasound imaging. Iliococcygeus (IC), puboperinealis (PP), superficial transverse perinei (STP), puboanalis (PA). Illustration: John Yanson. (From Shobeiri et al. [17], with permission). (**b**)The left lateral view of the left hemipelvis. Arcus tendineus levator ani (ATLA), bladder (B), external anal sphincter (EAS), iliococcygeus (IC), pubococcygeus (PC), puborectalis (PR), pubic symphysis (PS), urethra (U). (© Shobeiri 2013)

or attachment to the cephalad aspect of arcus tendineus anteriorly. At Level II, there are direct lateral attachments of the pubocervical fibromuscularis and rectovaginal fibromuscularis to the lateral facial compartments formed by the levator ani muscles. The variations of defect in this level will be described in the following sections. Anterior mesh was designed mainly to restore and enhance Level II. In Level III the

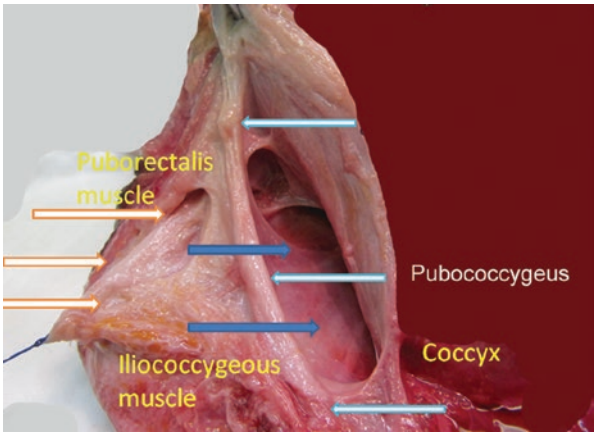

Fig. 7.17 Right hemipelvis of a fresh frozen pelvis showing the overlapping of the levator ani subdivisions fibers. *Orange arrows*, puborectalis; *blue arrows*, iliococcygeus; *white arrows*, pubococcygeus. Note the relationship between the iliococcygeus and pubococcygeus fibers. (© Shobeiri 2013)

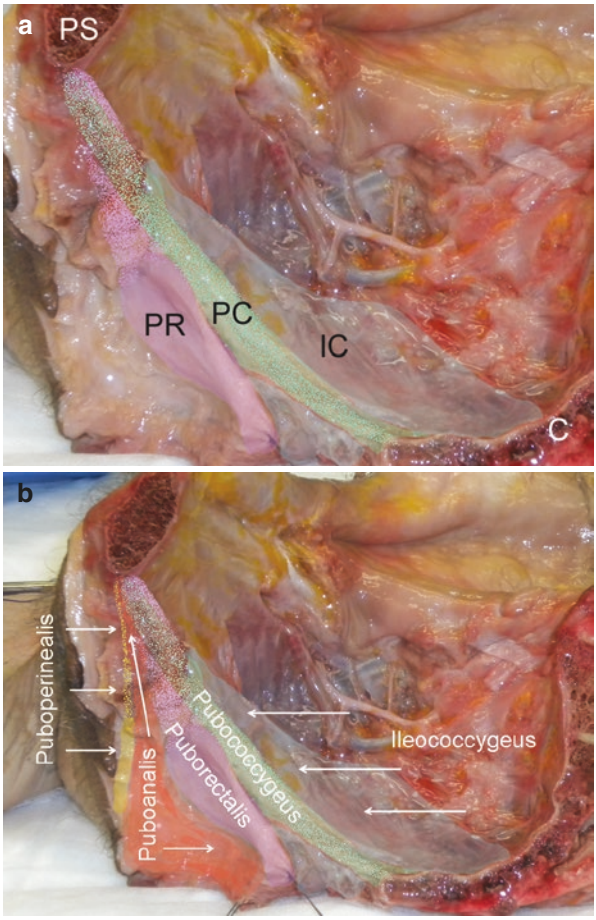

Fig. 7.18 (**a**) Right hemipelvis of a fresh frozen pelvis with the organs removed. The puborectalis (PR), iliococcygeus (IC), and pubococcygeus (PC) form the lateral sidewall. Note the relationship between the iliococcygeus and pubococcygeus fibers. (**b**) The same right hemipelvis of a fresh frozen pelvis with the organs removed. The puboanalis and the puboperinealis are outlined. These fibers are involved in the stabilization of the anus and the perineum, respectively. (© Shobeiri 2013)

vaginal wall is anteriorly fused with the urethra, posteriorly with the perineal body. Levator ani muscles in this area are poorly described but mostly consist of fibrous sheets that envelop the lateral aspects of the vaginal introitus.

Apical Segment

While Level I cardinal and uterosacral ligaments can be surgically identified supporting the cervix and the upper third of the vagina [18, 19], as they fan out toward the sacrum and laterally, they become a mixture of connective tissue, blood vessels, nerves, lymphatics, smooth muscle, and adipose tissue. The uterosacral ligaments act like rubber bands in that they may lengthen with initial Valsalva but resist any further lengthening at a critical point in which they have to return to their comfortable length or break (Fig. 7.19). Level I and levator ani muscles are interdependent. Intact levator ani muscles moderate the tension placed on the Level I support structures, and intact Level I support lessens the pressure imposed from above on the pelvic floor.

Anterior Compartment

Anterior compartment support depends on the integrity of vaginal muscularis and adventitia and their connections to the arcus tendineus fascia pelvis. The arcus tendineus fascia pelvis is at one end connected to the lower sixth of the pubic bone, 1–2 cm lateral to the midline, and at the other end to the ischial spine. A simple case of a distension cystocele could result from a defect in pubocervical fibromuscularis (Fig. 7.20). This disorder is repaired by suturing the defect and plication of the torn fascia. Such a repair could be performed by general ob/gyn surgeon, without the need of anterior mesh.

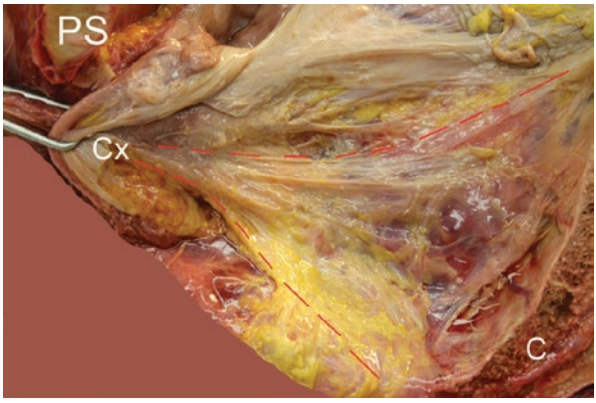

Fig. 7.19 Right hemipelvis of a fresh frozen pelvis showing the uterosacral fibers. The borders of the ligament are shown in *dotted line*. Cervix (Cx), coccyx (C), pubic symphysis (PS). (© Shobeiri 2013)

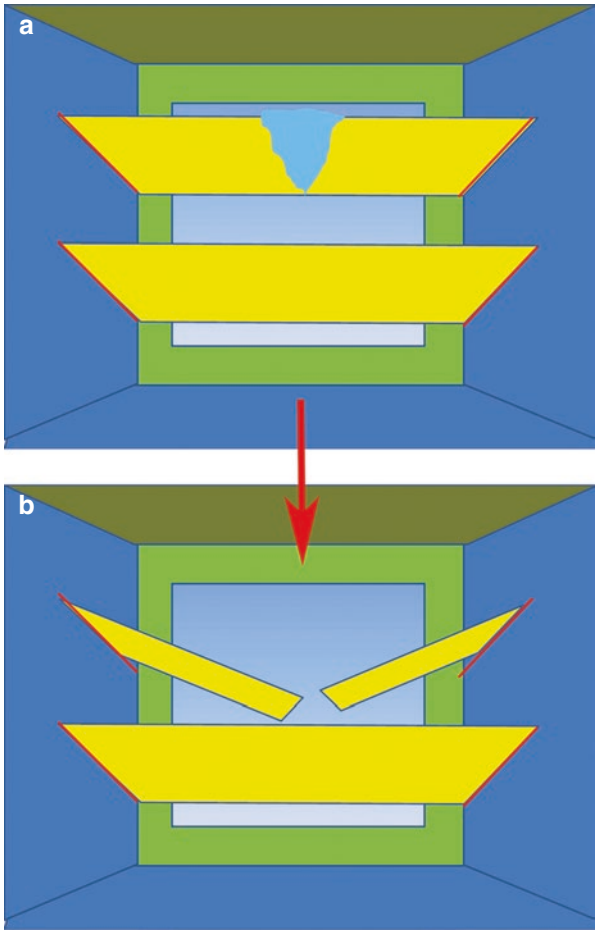

Fig. 7.20 Pelvic floor room analogy: (**a**) an occult pubocervical fibromuscularis defect can result in an overt cystocele (**b**). (© Shobeiri 2013)

The anterior wall fascial attachments to the arcus tendineus fascia pelvis have been called the paravaginal fascial attachments by Richardson et al. [20]. Detachment of arcus tendineus from the levator ani is associated with stress incontinence and anterior prolapse. The detachment can be unilateral (Fig. 7.21) or bilateral (Fig. 7.22), causing a displacement cystocele. In addition, the defect can be complete or incomplete. The surgeon who performs a traditional anterior repair in reality worsens the underlying disease process. In this case, a subspecialist surgeon should perform the surgical repair of the paravaginal detachment defect laparoscopically or vaginally. In this type of disorder, the primary repair shouldn't be anterior mesh repair. Moreover, using anterior mesh and suturing it to the detached arcus tendineus will worsen the patient's condition and might result in complications, including shrinkage of mesh, as the lateral side in not intact (see Figs. 7.21b and 7.22b).

The cephalad portions of the anterior vaginal wall can prolapse due to lack of Level I support and failure of uterosacral-cardinal complex. Over time this failure may lead to increased load in the paravaginal area and failure of Level II paravaginal

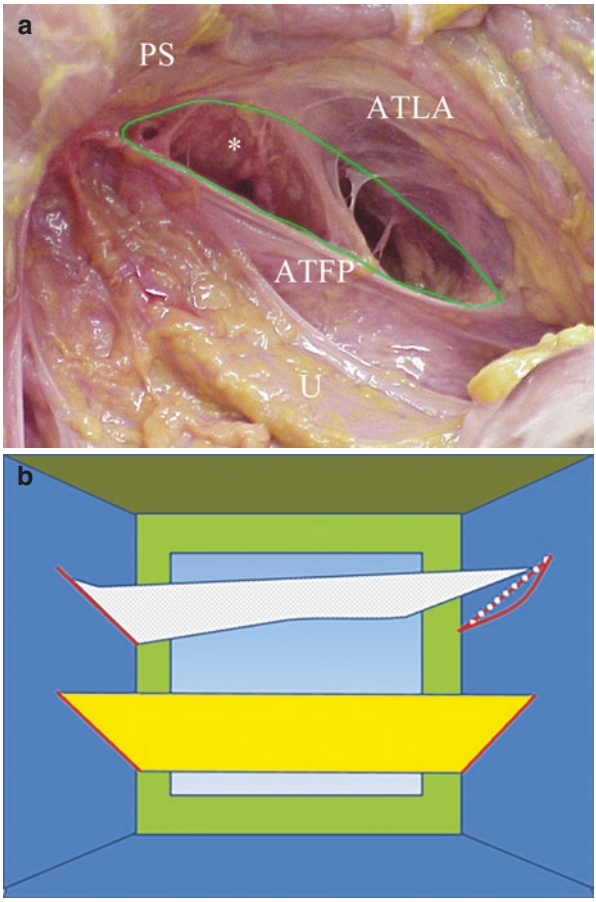

Fig. 7.21 (**a**) Right hemipelvis of a fresh frozen pelvis showing a paravaginal defect repair *outlined in green.* © Shobeiri 2013 (**b**) Pelvic floor room analogy: Left side is an intact lateral tendinous compartment, while a paravaginal defect (pubocervical fascia detachment) is illustrated in the right side of the patient (*red convex line*). The first step in this case should be repairing of this detachment by re-approximation of the detached tissues. Mesh usage without appropriate lateral repair will lead to complications in this case. (© Shobeiri 2013)

support. A study of 71 women with anterior compartment prolapse has shown that paravaginal defect usually results from a detachment of the arcus tendineus fascia pelvis from the ischial spine and rarely from the pubic bone [21]. Resuspension of the vaginal apex at the time of surgery, in addition to paravaginal or anterior colporrhaphy, may help to return the anterior wall to a more normal position or at least to prevent future failures. Another scenario that the surgeon faces is the lack of any tangible fibromuscular tissue in the anterior compartment (Fig. 7.23a). Plication of the available tissue may cause vaginal narrowing and dyspareunia. The knowledge of this condition is essential, as it will require bridging of the anterior compartment with biologic graft mostly by autologous fascia lata graft [22] or synthetic anterior mesh. The commercially available biologic tissue has had high failure rates for the anterior compartment and no improvement in the posterior compartment. The anterior mesh and vaginal mesh kits may have been an ideal product for this scenario as long as the levator ani muscles are intact and the pubocervical fibromuscularis is lacking (Fig. 7.23b). Yet, if the patient doesn't have muscles, the anterior mesh won't have lateral walls for appropriate attachment, and the mesh arms of the

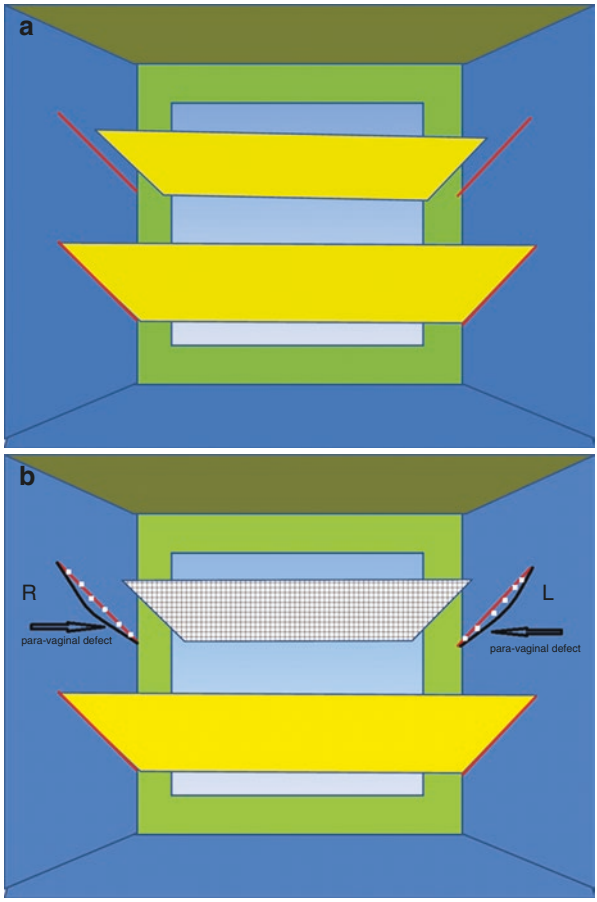

Fig. 7.22 (**a**) Pelvic floor room analogy: bilateral detachment of the pubocervical fibromuscularis can result in a cystocele. (**b**) Pelvic floor room analogy: Bilateral defect of tendinous compartments, a paravaginal defect (pubocervical fascia detachment), is illustrated in both left and right sides of the patient (black convex line). The first step in this case should be repairing of this detachment by re-approximation of the detached tissues in both sides. Mesh usage without appropriate bilateral repair will lead to complications in this case including shrinkage of mesh. (© Shobeiri 2013)

anterior mesh kits won't have an anchoring point, and if the muscles are intact, the mesh arms may cause pain traversing through muscles that move constantly.

Various grading systems such as Pelvic Organ Prolapse Quantification (POP-Q) system [23] used to describe prolapse do not take into account the underlying cause of the prolapse. Different clinical- and imaging-based modalities have been used to pinpoint the location of defect, and pelvic floor ultrasound has become valuable in the skilled hands to diagnose levator ani defects.

Perineal Membrane (Urogenital Diaphragm)

A critical but perhaps underappreciated part of pelvic floor support is the perineal membrane as it forms the Level III support (Fig. 7.24) [16] and one of the anchoring points in the suspension bridge analogy. On the anterior part caudad to the levator ani muscles, there is a dense triangular membrane called the urogenital diaphragm.

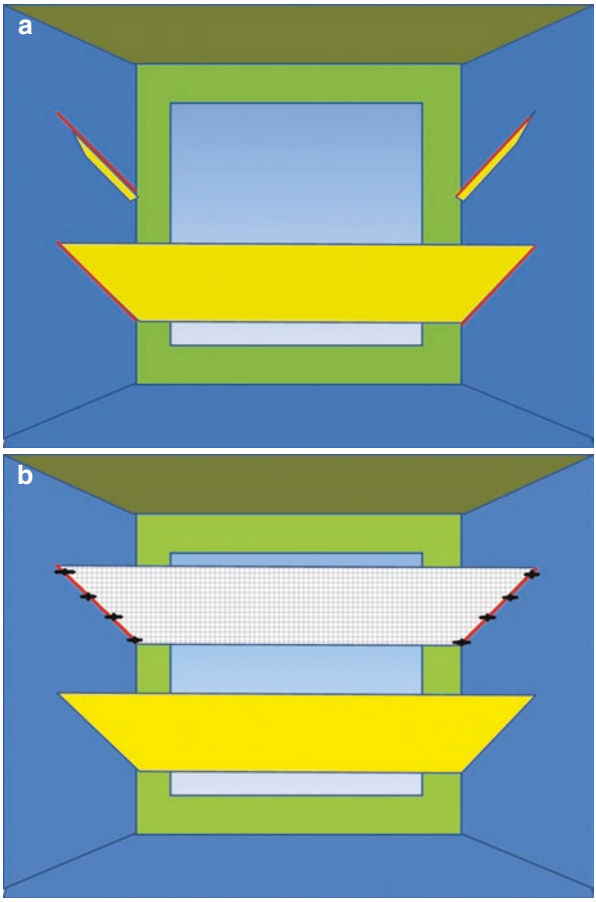

Fig. 7.23 (**a**) Pelvic floor room analogy: absence or severe deficiency of the pubocervical fibromuscularis can result in a cystocele. (**b**) Pelvic floor room analogy: the ideal scenario for synthetic mesh placement when there is total lack of the pubocervical fascia. Anterior mesh replaces the fascia and is sutured to intact lateral walls (arcus tendinous) as shown in red line. (© Shobeiri 2013)

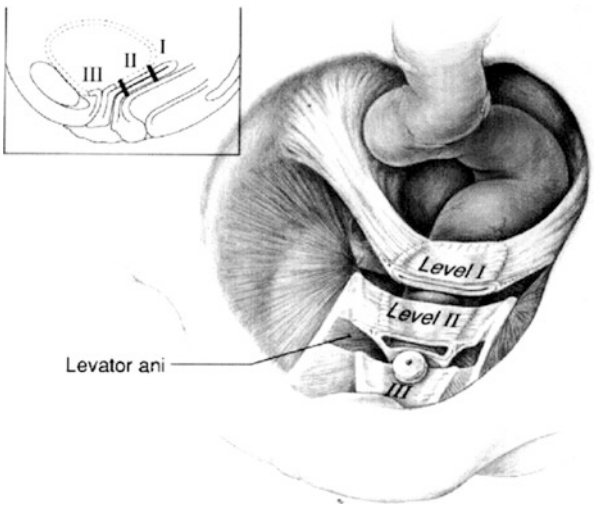

Fig. 7.24 Three levels of support. (From DeLancey [16], with permission Elsevier)

However, this layer is not a single muscle layer with a double layer of fascia ("diaphragm"), but rather a set of connective tissues that surround the urethra; the term perineal membrane has been used more recently to reflect its true nature [24]. The perineal membrane is a single connective tissue membrane, with muscle lying immediately above. The perineal membrane lies at the level of the hymen and attaches the urethra, vagina, and perineal body to the ischiopubic rami.

Posterior Compartment and Perineal Membrane

The use of mesh for the repair of rectoceles has been discredited. As such in this section, we describe the thought process on how the use of mesh may have been plausible from an anatomic perspective.

The posterior compartment is bound to perineal body and the perineal membrane caudad (Level III), paracolpium and the uterosacral ligaments cephalad (Level I), and the posterior arcus connected to the levator ani laterally (Level II). As in the anterior compartment, a simple defect in rectovaginal fibromuscularis (Fig. 7.25) can cause a distention rectocele. This type of disorder (similar to the anterior

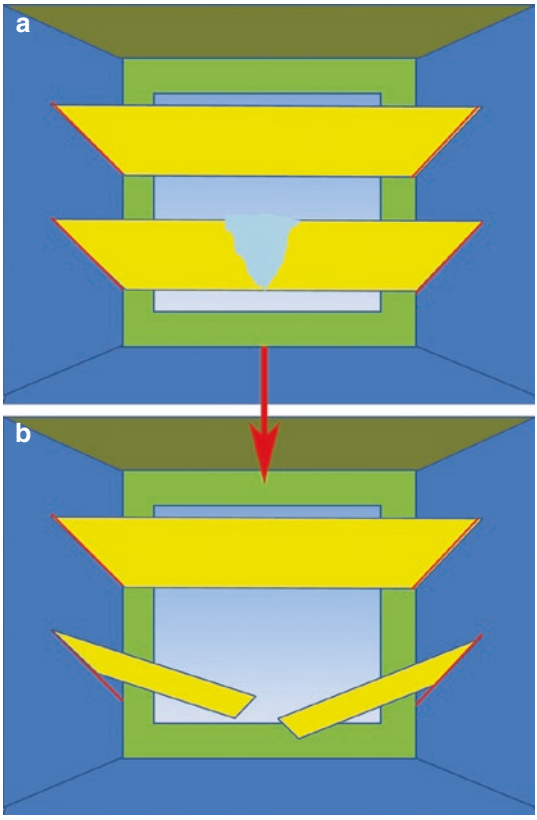

Fig. 7.25 Pelvic floor room analogy: (**a**) an occult rectovaginal defect can result in an overt rectocele (**b**). (© Shobeiri 2013)

distention cystocele) requires a plication and stitching of the rectovaginal fascia, with no need of the use of posterior mesh. A defect in the posterior arcus also called arcus tendineus rectovaginalis (ATRV) is associated with a pararectal defect that can be unilateral (Fig. 7.26) or bilateral (Fig. 7.27). This defect requires a pararectal repair rather than plication of the rectovaginal fascia which might worsen the rectocele stage. Furthermore, in this scenario, the use of posterior mesh is not

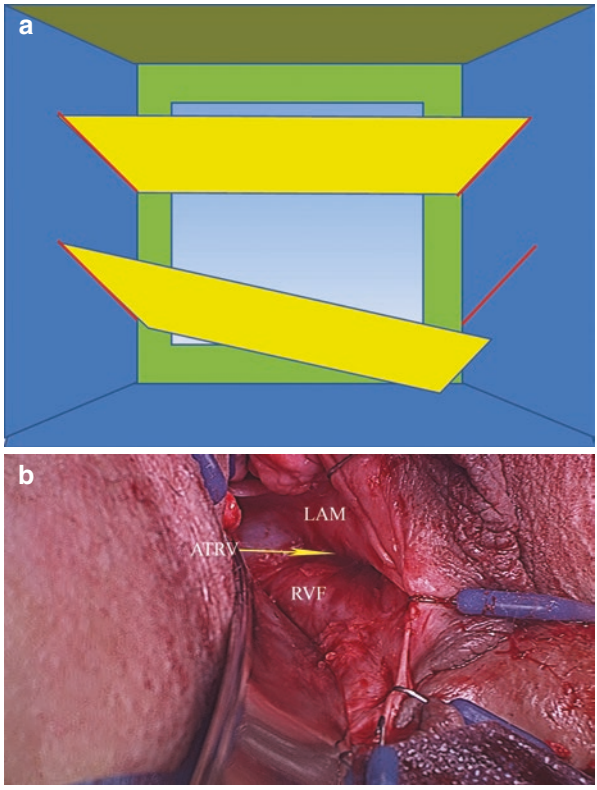

Fig. 7.26 (**a**) Pelvic floor room analogy: right lateral detachment of the rectovaginal septum can result in a rectocele. (**b**) The surgical view of the posterior compartment showing the relationship between the levator ani muscle (LAM), the rectovaginal fibromuscularis (RVF), and the arcus tendineus fasciae rectovaginalis (ATRV). (© Shobeiri 2013)

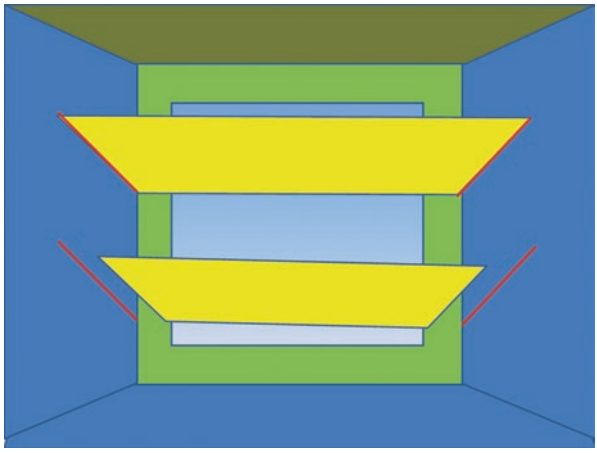

Fig. 7.27 Pelvic floor room analogy: bilateral detachment of the rectovaginal septum can result in a rectocele. (© Shobeiri 2013)

recommended, and since there is no intact lateral wall for the attachment of mesh or for the anchoring of the posterior kits, placing mesh will result in complications as contraction of mesh, pain or erosion. Such defects need to be differentiated from total loss of rectovaginal fibromuscularis (Fig. 7.28a), which was perceived as the ideal scenario in which one should perform an augmentation of the posterior

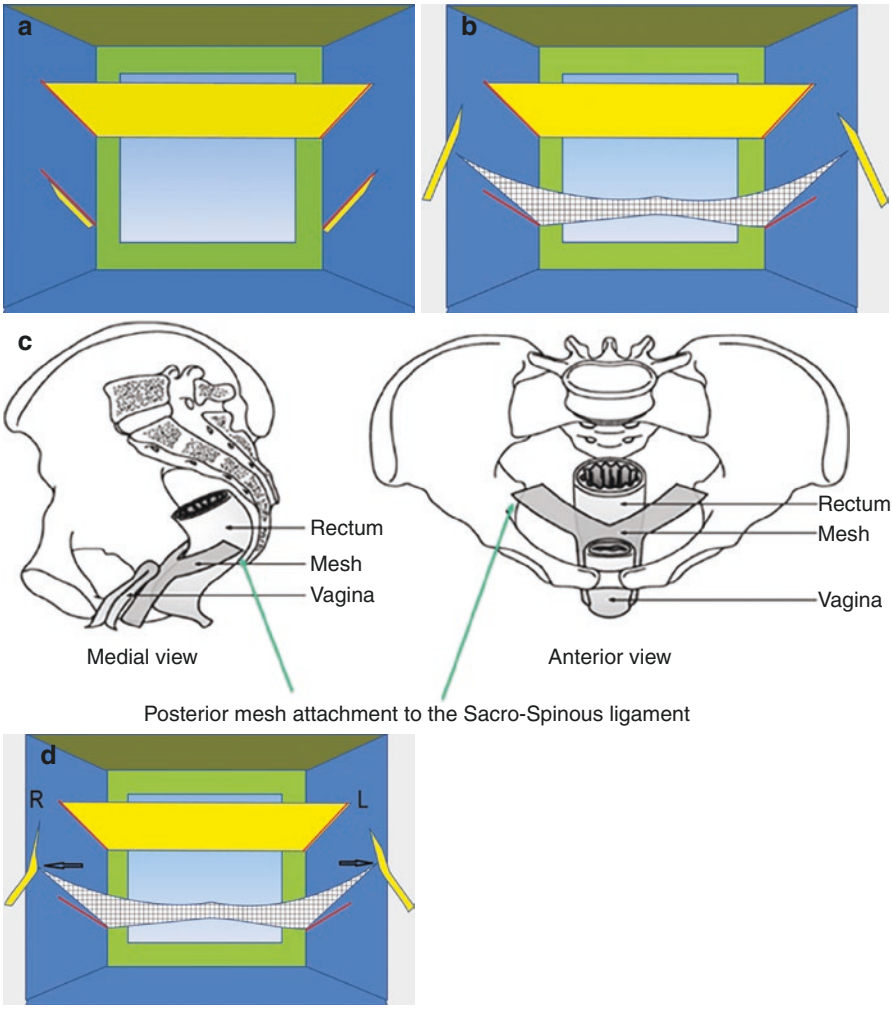

Fig. 7.28 (**a**) Pelvic floor room analogy: absence or severe deficiency of rectovaginal fascia can result in a rectocele. (**b**) Pelvic floor room analogy: This room analogy illustrates a loss of rectovaginal fibromuscularis, which was perceived as the ideal scenario for performing an augmentation of the posterior compartment with mesh. The yellow lines denote the sacrospinous ligaments as the apical attachment point of mesh kit anchors. (**c**) The posterior mesh replaces the rectovaginal fascia. (From Dwyer and O'Reilly [25], with permission John Wiley). (**d**) Pelvic floor room analogy: Bilateral defect of posterior tendinous compartments, a pararectal defect (rectovaginal fascia detachment or levator ani muscle injury), is illustrated in both left and right sides. The first step in this case should be repairing of this detachment by re-approximation of the detached tissues in both sides. Mesh usage without appropriate bilateral repair will lead to contraction of mesh due to the lack of anchoring points. (© Shobeiri 2013)

compartment with autologous, cadaveric tissue, or use posterior mesh/vaginal mesh kits. The posterior compartment mesh kits are mostly designed to be anchored to the sacrospinous ligaments cephalad and go through the perineal membrane and muscles distally (Fig. 7.28b,c) [25]. Most often, the separation of the posterior arcus may be apical and may require reattachment of the posterior arcus to the uterosacral ligament or the iliococcygeal muscle prior to placing posterior mesh. Analogous to the anterior compartment, if the patient has a unilateral or bilateral detached arcus tendinous or doesn't have a levator ani muscle on one or both sides, the mesh arms will not have attachment's walls or anchoring points (Fig. 7.28d).

The fibers of the perineal membrane connect through the perineal body, thereby providing a layer that resists downward descent of the rectum. In the room analogy used here, the perineal membrane is analogous to the door frame. If the bottom of the door frame is missing (see Fig. 7.28a), then the resistance to downward descent is lost, and a perineocele develops. This situation can be elusive, as the clinical diagnosis is made by realizing the patient's need to splint very close to the vaginal opening in order to have a bowel movement, and the physical examination may reveal an elongated or "empty" perineal body. Reattachment of the separated structures during perineorrhaphy corrects this defect and is a mainstay of reconstructive surgery. Because the puboperinealis muscles are intimately connected with the cranial surface of the perineal membranes, this reattachment also restores the muscles to a more normal position under the pelvic organs in a location where they can provide support.

The muscle fibers from the puboanalis portion of the levator ani become fibroelastic as they extend caudally to merge with the conjoined longitudinal layer also known as the longitudinal muscle (CLL) that is inserted between the EAS and IAS (Figs. 7.29 and 7.30) [26]. The CLL fibers and the puboanalis fibers cannot be palpated clinically. However, the puboperinealis fibers, which are medially located, can be palpated as a distinct band of fibers joining the perineal body (Fig. 7.31) (and see Fig. 7.29). The posterior vaginal mesh kits were supposed to attach to the perineal body, but as the mesh shrunk, the mesh was generally pulled cephalad creating either a perineal defect or a low rectocele.

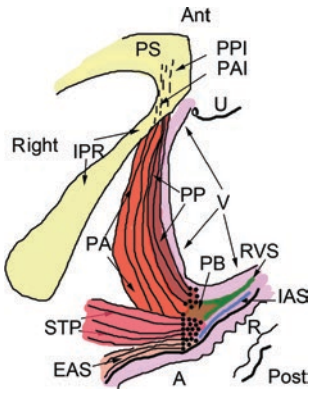

Fig. 7.29 This drawing demonstrates the right sagittal hemipelvis view of the perineal support structures. The perineum, a small seemingly insignificant part of the female body, is packed with muscles and fascial layers that interconnect in an intricate manner. External anal sphincter (EAS), internal anal sphincter (IAS), ischiopubic rami (IPR), puboanalis (PA), puboanalis insertion (PAI), perineal body (PB), puboperinealis (PP), puboperineal insertion (PPI), pubic symphysis (PS), rectum (R), rectovaginal septum, (RVS), superficial transverse perinei (STP), urethra (U), vagina (V). (© Shobeiri 2013)

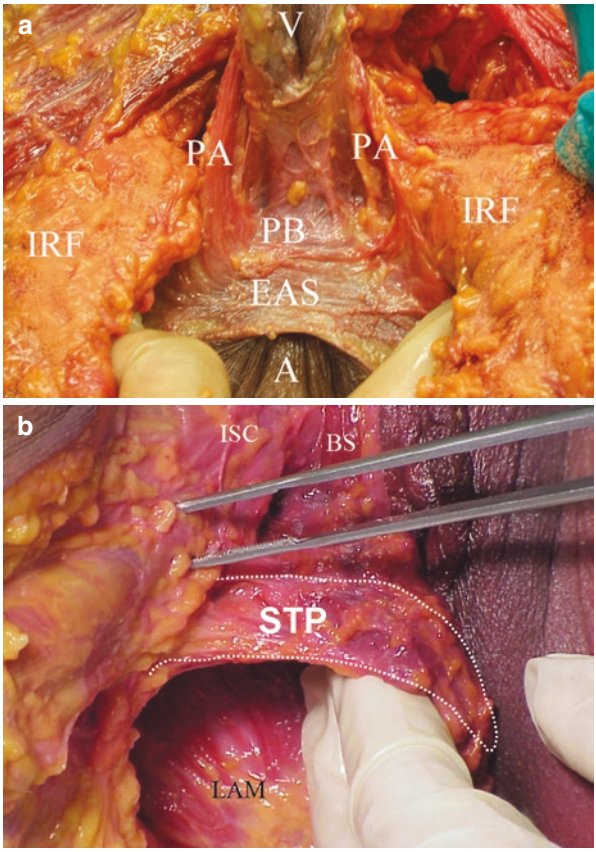

Fig. 7.30 (**a**) Perineal dissection in a fresh frozen pelvis shows the relationship of the external anal sphincter (EAS) to the perineal body (PB) and the puboanalis/puboperinealis complex. Ischiorectal fat (IRF). (**b**) Perineal dissection in a fresh frozen pelvis shows the relationship of the superficial transverse perinei (STP) to the other puboanalis fibers that start inserting at the perineal level at Fig 7.30a and then wrap around the anal canal (LAM). The ischiocavernosus (ISC) and the bulbospongiosus muscle (BS) are depicted here. (© Shobeiri 2013)

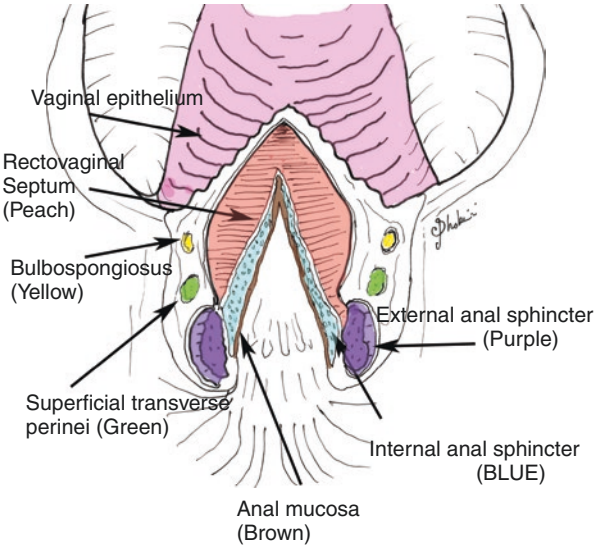

Fig. 7.31 Drawing of perineal region as may be seen after a clean midline episiotomy. The drawing depicts the relationship of muscles to the rectovaginal septum. (© Shobeiri 2013)

Lateral Compartment and the Levator Ani Muscles

It is generally accepted that the levator ani muscles and the associated fascial layer surround pelvic organs like a funnel to form the pelvic diaphragm [27]. Given that we employ concepts such as pelvic floor spasm, levator spasm, and pelvic floor weakness, understanding the basic concepts of pelvic floor musculature is essential to formulate a clinical opinion. The area posterior to the pubic bone is dense with bands of intertwined levator ani muscles; this defies conventional description of the levator ani as comprising the puborectalis, pubococcygeus, and iliococcygeus. The anatomy of distal subdivisions of the levator ani muscle was further described in a study by Kearney et al. [28]. The origins and insertions of these muscles as well as their characteristic anatomical relations are shown in Table 7.1 and Fig. 7.16. Using a nomenclature based on the attachment points, the lesser known subdivisions of the levator ani muscles, the muscles posterior to the pubic bone are identified as pubovaginalis, puboanalis, and puboperinealis. The pubovaginalis is poorly described but may be analogous to the urethrovaginal ligaments. The puboanalis originates from behind the pubic bone as a thin band and inserts around the anus into the longitudinal ligaments. The puboperinealis, which is most often 0.5 cm in diameter, originates from the pubic bone and inserts into the perineal body. The four major components of the levator ani muscle are the iliococcygeus, which forms a thin, relatively flat, horizontal shelf that spans the potential gap from one pelvic sidewall to the other; the pubococcygeus muscle, which travels from the tip of the coccyx to the pubic bone (see Fig. 7.17); the puborectalis muscle, originating from the anterior portion of the perineal membrane and the pubic bone to form a sling behind the rectum; and the puboperinealis and puboanalis, which are thin broad fibromuscular poorly described structures that attach to the perineal body and anus to stabilize the perineal region.

The shortest distance between the pubic symphysis and the levator plate is the minimal levator hiatus. This is different from the urogenital hiatus, which is bounded anteriorly by the pubic bones, laterally by levator ani muscles, and posteriorly by the perineal body and EAS. The baseline tonic activity of the levator ani muscle keeps the minimal levator hiatus closed by compressing the urethra, vagina, and rectum against the pubic bone as they exit through this opening [29]. The levator ani

Table 7.1 Divisions of the levator ani muscles – international standardized terminology

Levator ani muscles	Origin/insertion
Puboperinealis (PP)	Pubis/perineal body
Pubovaginalis (PV)	Pubis/vaginal wall at the level of the mid-urethra
Puboanalis (PA)	Pubis/intersphincteric groove between internal and external anal sphincter to end in the anal skin
Puborectalis (PR)	Pubis/forms sling behind the rectum
Iliococcygeus (IC)	Tendinous arch of the levator ani/the two sides fuse in the iliococcygeal raphe
Pubococcygeus (PC)	Pubic symphysis to superficial part of anococcygeal ligament

fibers converge behind the rectum to form the levator plate. With contraction, the levator plate elevates to form a horizontal shelf over which pelvic organs rest. The deficiency of any portion of the levator ani results in weakening of the levator plate and descensus of pelvic organs [30].

Endopelvic Fascia and Levator Ani Interactions

We mention the levator ani muscle here because although the vaginal mesh kits were designed to replace and support deficient anterior or posterior fascia, they did not take into consideration the levator ani muscle function. The levator ani muscles and the endopelvic fascia work as a unit to provide pelvic organ support. If the muscles maintain normal tone, the ligaments of the endopelvic fascia will have little tension on them even with increases in abdominal pressure (Fig. 7.32). If the muscles are damaged by a tear or complete separation from their attachments, the pelvic floor sags downward over time and the organs are pushed through the urogenital hiatus (Fig. 7.33). In such cases the ligaments and the endopelvic fascia will assume the majority of the pelvic floor load until they fail as well. Different varieties of levator ani injury can cause different interesting types of clinical defects. A partial defect and separation of the pubococcygeus muscles will result in a displacement cystocele (Fig. 7.34). However, the clinician may not be able to distinguish if this is a displacement cystocele due to paravaginal defect and arcus tendineus separation or due to muscle loss. The consequences of this lack of recognition can be that the surgeon may elect to do an anterior repair and, by placating the pubocervical

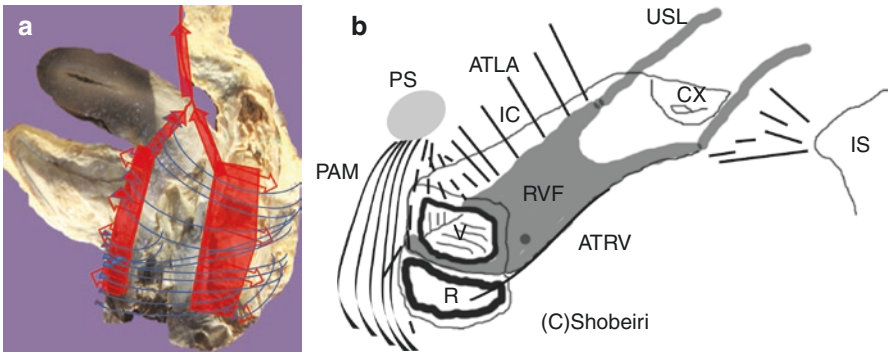

Fig. 7.32 (**a**) Right lateral standing anatomic depiction of the levator ani muscle and uterosacral-cardinal complex interaction. (**b**) Drawing of the interaction between the rectovaginal fibromuscularis and the uterosacral ligaments. The levator ani muscle and uterosacral-cardinal complex give cephalad static support, while the iliococcygeal fibers give lateral support to the posterior compartment. The puboanalis and the puboperinealis muscles stabilize the perineum while the puborectalis closes the levator hiatus. Arcus tendineus levator ani (ATLA), arcus tendineus fascia rectovaginalis (ATRV), cervix (CX), iliococcygeus (IC), ischial spine (IS), pubic symphysis (PS), rectum (R), rectovaginal fibromuscularis (RVF), uterosacral ligament (USL), vagina (V). (© Shobeiri 2013)

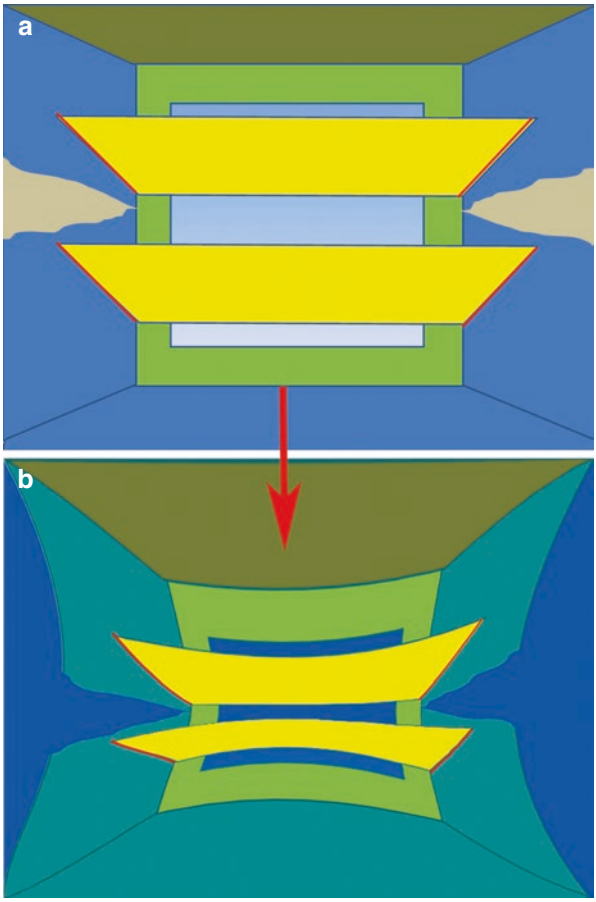

Fig. 7.33 Pelvic floor room analogy: bilateral levator ani tears may or may not result in prolapse or incontinence initially, but over time the other supportive structures will decompensate resulting in pelvic floor laxity. (© Shobeiri 2013)

fibromuscularis, make the lateral defect worse. The lack of basic information about the levator ani status may account for varied results in the anterior repair studies and failure of many vaginal mesh kits. Additionally, in an attempted paravaginal or vaginal mesh kit repair, the surgeon may realize that there is no muscle to attach the arcus tendineus or mesh to, leading to complications.

Previous studies have shown that women with POP have a decreased muscle fiber number and function, and a higher apoptosis pace, and disorganization of smooth muscular fibers. Implanting a mesh into unhealthy muscular tissue may result in a maladaptive remodeling response, which increases the risk for mesh complications, including mesh erosion, extrusion, or contraction. The same is true in implanting anterior vaginal mesh in an individual with unilateral or bilateral levator tear/avulsion (Fig. 7.35) [8, 31, 32].

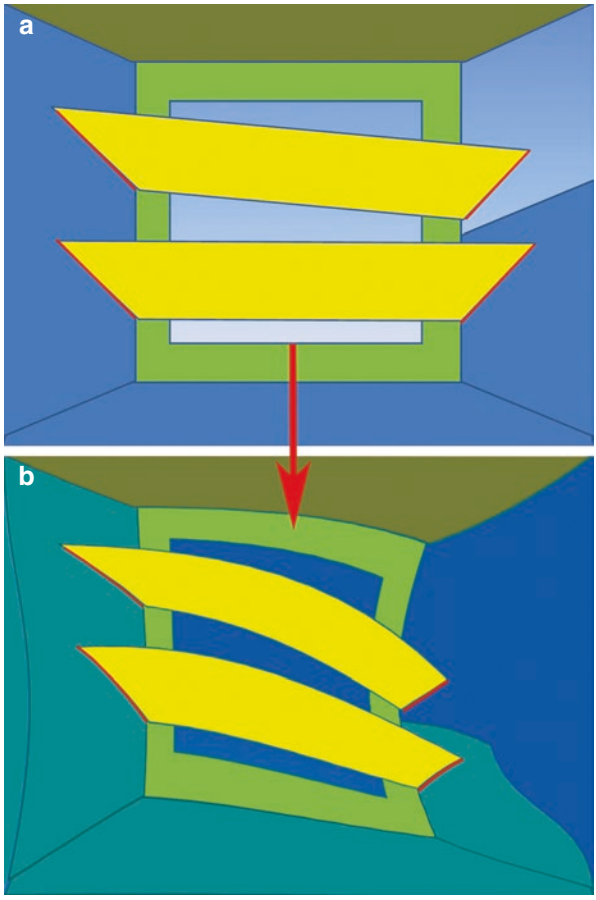

Fig. 7.34 Pelvic floor room analogy: (**a**) unilateral levator ani tears may or may not result in prolapse or incontinence initially, but over time the other supportive structures will decompensate resulting in pelvic floor laxity (**b**). (© Shobeiri 2013)

A partial defect (see Fig. 7.34a) is subjected to excessive forces and may progress over time to involve the apical and posterior compartments as well (see Fig. 7.34b). How fast this occurs depends on the strength of the patient's connective tissue. One woman with injured muscles may have strong connective tissue that compensates and never develops prolapse, while another woman with even less muscle injury but weaker connective tissue may develop prolapse with aging. There are instances of catastrophic injury during childbirth during which complete muscle loss occurs and the patient presents with a displacement cystocele, rectocele, and varied types of incontinence (Fig. 7.35). This scenario is different with patients who have a defect in pubocervical and rectovaginal fibromuscularis (Fig. 7.36), which develops into a distention cystocele and rectocele over time. A cystocele and rectocele repair that can be used for the latter case will worsen the condition of the first patient with levator damage.

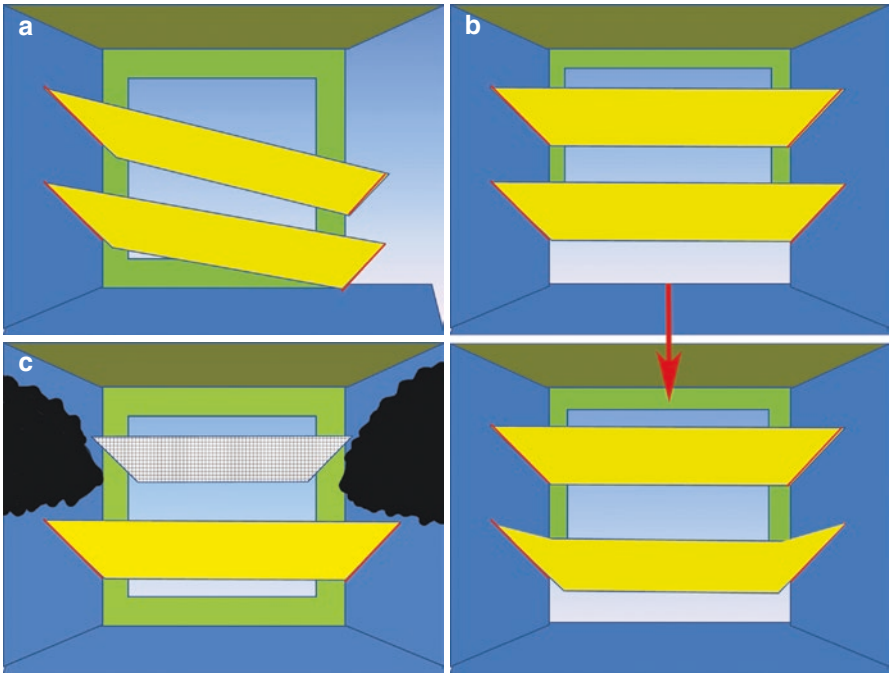

Fig. 7.35 Pelvic floor room analogy: obstetric injuries can be catastrophic or subtle. To the left is a complete right unilateral levator ani detachment (avulsion). To the right is injury to the perineal support (the missing green part of the door frame) (**a**), which may result in sliding of the rectovaginal fascia and a clinical perineocele (**b**). **(c)** Pelvic floor room analogy: anterior mesh displacement /contraction due to the lack of adequate levator ani muscle attachment points. (© Shobeiri 2013)

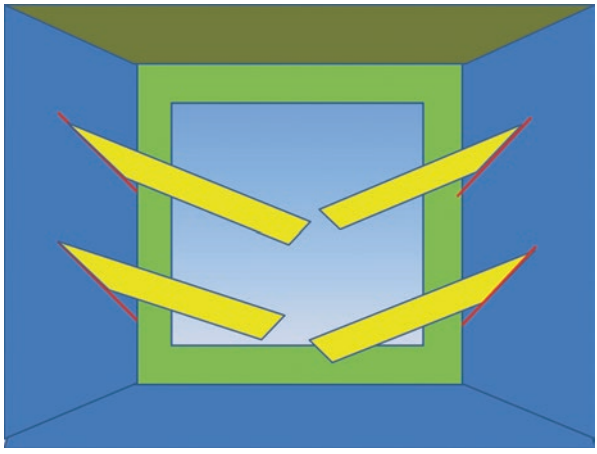

Fig. 7.36 Pelvic floor room analogy: multicompartmental defect – pubocervical fibromuscularis and rectovaginal septum defects. (© Shobeiri 2013)

The Pudendal Nerve

The pudendal nerve is mentioned here because the injury to this nerve caused much irreversible misery for the patients. It supplies the urethral and anal sphincters and the perineal muscles. The pudendal nerve originates from S2 to S4 foramina and runs through the Alcock canal, which is caudal to the levator ani muscles. The pudendal nerve has three branches: the clitoral, perineal, and inferior hemorrhoidal, which innervate the clitoris, the perineal musculature, inner perineal skin, and the EAS, respectively [26]. The blockade of the pudendal nerve decreases resting and squeeze pressures in the vagina and rectum, increases the length of the urogenital hiatus, and decreases electromyography activity of the puborectalis muscle [33]. The pudendal nerve injury due to vaginal mesh kit trocar placement close to or through this nerve is discussed elsewhere, but it is one of the important design flaws of the vaginal mesh kits that resulted in life altering and debilitating pain in many women.

Summary

The knowledge of pelvic floor anatomy, function, and biomechanics is essential for understanding of pelvic floor pathologies and reaching the precise diagnosis and treatment. Moreover, biomechanics studies help to understand part of mesh complications and failures in terms of POP. Models can be used to simulate pelvic floor dysfunction, vaginal childbirth, vaginal meshes, and novel devices for POP treatment and hold significant potential for patient specific diagnostics and surgical planning. The pelvic floor in many ways is a functional unit like the human mouth. Some vaginal mesh kits inhibited vaginal movement and distention because of their stiffness; others inhibited function because their points of attachments were either too firm or too unstable. A good augmentative product takes into account human factors and mechanical properties of pelvic structures and the functionality required for the vaginal tissue.

References

1. NIH state-of-the science conference statement on prevention of fecal and urinary incontinence in adults. NIH Consens State Sci Statements. 2007;24(1):1–37.
2. Nygaard I, Barber MD, Burgio KL, Kenton K, Meikle S, Schaffer J, et al. Prevalence of symptomatic pelvic floor disorders in US women. JAMA. 2008;300(11):1311–6.
3. Olsen AL, Smith VJ, Bergstrom JO, Colling JC, Clark AL. Epidemiology of surgically managed pelvic organ prolapse and urinary incontinence. Obstet Gynecol. 1997;89(4):501–6.

4. Boyles SH, Weber AM, Meyn L. Procedures for pelvic organ prolapse in the United States, 1979–1997. Am J Obstet Gynecol. 2003;188(1):108–15.
5. Smith FJ, Holman CD, Moorin RE, Tsokos N. Lifetime risk of undergoing surgery for pelvic organ prolapse. Obstet Gynecol. 2010;116(5):1096–100.
6. Aigmueller T, Dungl A, Hinterholzer S, Geiss I, Riss P. An estimation of the frequency of surgery for posthysterectomy vault prolapse. Int Urogynecol J. 2010;21(3):299–302.
7. Quiroz LH, Munoz A, Shippey SH, Gutman RE, Handa VL. Vaginal parity and pelvic organ prolapse. J Reprod Med. 2010;55(3–4):93–8.
8. Boreham MK, Wai CY, Miller RT, Schaffer JI, Word RA. Morphometric analysis of smooth muscle in the anterior vaginal wall of women with pelvic organ prolapse. Am J Obstet Gynecol. 2002;187(1):56–63.
9. Morgan DM, Rogers MA, Huebner M, Wei JT, Delancey JO. Heterogeneity in anatomic outcome of sacrospinous ligament fixation for prolapse: a systematic review. Obstet Gynecol. 2007;109(6):1424–33.
10. Jakus SM, Shapiro A, Hall CD. Biologic and synthetic graft use in pelvic surgery: a review. Obstet Gynecol Surv. 2008;63(4):253–66.
11. Burger JW, Luijendijk RW, Hop WC, Halm JA, Verdaasdonk EG, Jeekel J. Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg. 2004;240(4):578–83. discussion 583-5
12. Luijendijk RW, Hop WC, van den Tol MP, de Lange DC, Braaksma MM, Ijzermans JN, et al. A comparison of suture repair with mesh repair for incisional hernia. N Engl J Med. 2000;343(6):392–8.
13. Ricci JV, Thom CH. The myth of a surgically useful fascia in vaginal plastic reconstructions. Q Rev Surg Obstet Gynecol. 1954;11(4):253–61.
14. Gitsch E, Palmrich AH. Operative anatomie. Berlin: De Gruyter; 1977.
15. Albright T, Gehrich A, Davis G, Sabi F, Buller J. Arcus tendineus fascia pelvis: a further understanding. Am J Obstet Gynecol. 2005;193(3):677–81.
16. DeLancey JO. Anatomic aspects of vaginal eversion after hysterectomy. Am J Obstet Gynecol. 1992;166(6 Pt 1):1717–24. discussion 1724-8
17. Shobeiri SA, Leclaire E, Nihira MA, Quiroz LH, O'Donoghue D. Appearance of the levator ani muscle subdivisions in endovaginal three-dimensional ultrasonography. Obstet Gynecol. 2009;114(1):66–72.
18. Campbell RM. The anatomy and histology of the sacrouterine ligaments. Am J Obstet Gynecol. 1950;59(1):1–12.
19. Range RL, Woodburne RT. The gross and microscopic anatomy of the transverse cervical ligaments. Am J Obstet Gynecol. 1964;90:460–7.
20. Richardson AC, Edmonds PB, Williams NL. Treatment of stress urinary incontinence due to paravaginal fascial defect. Obstet Gynecol. 1981;57(3):357–62.
21. DeLancey J. Fascial and muscular abnormalities in women with urethral hypermobility and anterior vaginal wall prolapse. Am J Obstet Gynecol. 2002;187(1):93–8.
22. Chesson RR, Schlossberg SM, Elkins TE, Menefee S, McCammon K, Franco N, et al. The use of fascia lata graft for correction of severe or recurrent anterior vaginal wall defects. J Pelvic Surg. 1999;5(2):96–103.
23. Bump RC, Mattiasson A, Bo K, Brubaker LP, DeLancey JO, Klarskov P, et al. The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. Am J Obstet Gynecol. 1996;175(1):10–7.
24. Oelrich T. The striated urogenital sphincter muscle in the female. Anat Rec. 1983; 205(2):223–32.
25. Dwyer PL, O'Reilly BA. Transvaginal repair of anterior and posterior compartment prolapse with Atrium polypropylene mesh. Br J Obstet Gynaecol. 2004;111(8):831–6.
26. Shobeiri SA, Chesson RR, Gasser RF. The internal innervation and morphology of the human female levator ani muscle. Am J Obstet Gynecol. 2008;199(6):686.e1–6.
27. Lawson JO. Pelvic anatomy. I. Pelvic floor muscles. Ann R Coll Surg Engl. 1974;54(5):244–52.

28. Kearney R, Sawhney R, DeLancey JOL. Levator ani muscle anatomy evaluated by origin-insertion pairs. Obstet Gynecol. 2004;104(1):168–73.
29. Taverner D, Smiddy FG. An electromyographic study of the normal function of the external anal sphincter and pelvic diaphragm. Dis Colon Rectum. 1959;2(2):153–60.
30. Nichols DH, Milley PS, Randall CL. Significance of restoration of normal vaginal depth and axis. Obstet Gynecol. 1970;36(2):251–6.
31. Boreham MK, Wai CY, Miller RT, Schaffer JI, Word RA. Morphometric properties of the posterior vaginal wall in women with pelvic organ prolapse. Am J Obstet Gynecol. 2002;187(6):1501–8; discussion 1508–9.
32. Takac P, Gualtieri M, Nassiri M, Candiotti K, Medina CA. Vaginal smooth muscle cell apoptosis is increased in women with pelvic organ prolapse. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(11):1559–64.
33. Guaderrama NM, Liu J, Nager CW, Pretorius DH, Sheean G, Kassab G, et al. Evidence for the innervation of pelvic floor muscles by the pudendal nerve. Obstet Gynecol. 2005;106(4):774–81.