Review article

# Anatomical Basis of Pelvic Floor Defects

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## Abstract

Pelvic Floor Disorders (PFDs) are a common problem affecting women of various ages. Pelvic floor defects are one of the entities that belong to pelvic floor disorders. Each pelvic floor defect is caused by specific damage to the anatomical and functional integrity of the pelvic floor. Understanding the underlying pathological anatomy is crucial for the proper therapeutic approach in treating this condition. Pelvic organ prolapse refers to the descent of pelvic organs into or through the vaginal canal. This includes the bladder, uterus, vaginal vault, small intestine, and large intestine. There are three groups of prolapse: anterior, posterior, and apical. Prolapses occur due to damage to the fibromuscular support system known as the endopelvic fascia, which maintains the pelvic organs in their normal position. To understand the normal statics of pelvic organs, the concept of DeLancey's levels of support—comprising three levels—is essential. The first level of support consists of the cardinal and uterosacral ligaments. The second level of support consists of the pubocervical and rectovaginal fascia. The third level of support consists of the connection of the vagina to surrounding structures. Each prolapse occurs due to damage to individual levels of support. Thus, defects of the endopelvic fascia have been recognized as key in the pathophysiology of pelvic organ prolapse. Pelvic floor defects often impair the normal mechanisms of maintaining continence, resulting in urinary incontinence as a common symptom of this condition. Only by repairing such damage and restoring the original anatomical relationships can success in surgical therapy be achieved. The aim of this paper is to describe in detail the anatomy of each defect with the purpose of creating a foundation for diagnostic and therapeutic approaches.

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## Introduction

Pelvic floor defects are a clinical entity that forms part of the diagnosis of pelvic floor disorders (PFDs). In addition to the defects themselves, PFDs include a range of consequences arising from the disruption of pelvic organ support. Damage to the pelvic floor can therefore lead to pelvic organ prolapse (POP), urinary incontinence, anal incontinence, and sexual dysfunction.Epidemiological data suggest that 25% of women will develop some form of pelvic floor dysfunction during their lifetime (1). This indicates that it is a common condition, affecting women of various ages, and significantly impairing quality of life—not only due to physical symptoms, but also through emotional distress and feelings of shame.

The development of pelvic floor defects is closely linked to the loss of stability and the compromised integrity of pelvic support structures. The pelvic floor is a complex unit, composed of many structures, together forming a unique anatomical and functional whole. As such, it requires detailed knowledge of anatomical relationships to enable accurate diagnosis and appropriate surgical correction.

Pelvic floor defects occur as a result of damage to the muscles, nerves, and connective tissues that provide stable support to the pelvic floor (2). Each defect of the pelvic floor is fundamentally associated with specific damage to the endopelvic fascia. Until 1976, the pathoanatomy underlying pelvic floor defects was not fully understood. At that time, Richardson introduced new key concepts in understanding this diagnosis. After a series of anatomical dissections, he concluded that isolated injuries to the endopelvic fascia lead to specific pelvic floor defects. This was later confirmed by additional studies (2–7). From this, it follows that identifying the damage and surgically repairing the endopelvic fascia are crucial for therapeutic success in treating this condition (8).

The endopelvic fascia is a unique fibromuscular support system responsible for maintaining the position of pelvic organs. Damage to this structure leads to pelvic organ prolapse, which is directly associated with urinary and bowel symptoms.

Pelvic organ prolapse refers to the pathological descent of pelvic organs into or through the vaginal canal. This includes the uterus, bladder, vaginal vault, small intestine, and rectum (1,9). Each defect is underpinned by specific injury to the endopelvic fascia. Accordingly, in clinical practice, defects are often referred to as cystocele, cystourethrocele, rectocele, enterocele, and uterine prolapse.

Although these terms are commonly used in medical literature, the National Institutes of Health has identified them as imprecise, as they merely assume which organ is prolapsed rather than confirm it objectively. Therefore, before describing the anatomical changes of the pelvic floor, prolapse should be categorized into three compartments: uterine (apical), anterior, and posterior. This approach established a standardized terminology that is now used among professionals in the field (2).

The aim of this paper is to describe in detail the pathological anatomy of individual pelvic floor defects, with an emphasis on their classification, mechanisms of development, and clinical significance. Given that surgical outcomes are directly related to accurate recognition and understanding of the anatomical changes, clear definition of each defect is essential for a successful treatment strategy.

This paper is based on the assumption that long-term stability and functional restoration of the pelvic floor can only be achieved by restoring its normal anatomical relationships. The goal is to highlight the importance of precise identification of pathological anatomy as a key determinant in both diagnostic and therapeutic approaches.

## Mechanisms of Pelvic Floor Support

There are two principal mechanisms responsible for maintaining the support of the pelvic organs. The first involves active muscular support through the contraction of the levator ani muscle, while the second refers to passive

suspension provided by the endopelvic fascia (10). The endopelvic fascia bilaterally anchors the vagina and cervix to the lateral pelvic walls. The pelvic floor should not be viewed as an isolated muscular system but as a complex, integrated structure comprising components situated between the peritoneum and the vulvar skin. These components include the peritoneum, pelvic organs, endopelvic fascia, levator ani muscle, perineal membrane, and the superficial genital muscles (2). From a biomechanical standpoint, pelvic organs are considered integral to the pelvic floor, owing to their anatomical continuity with adjacent support structures such as the cardinal and uterosacral ligaments (2,11). As early as 1934, Bonney illustrated the relationship between the vagina and the abdominal cavity using the analogy of an inverted finger of a surgical glove—stating that, when pressure increases within the glove, the finger protrudes. Similarly, increased intra-abdominal pressure promotes vaginal protrusion. This process, however, is prevented if the supporting mechanism remains intact (12).

## Endopelvic Fascia and Levels of Vaginal Support (DeLancey's Levels I, II, and III)

In clinical practice, the endopelvic fascia is frequently described as comprising the uterosacral ligaments, cardinal ligaments, pubocervical fascia, and rectovaginal fascia (2,5,13). An alternative anatomical perspective considers this fibromuscular system in relation to the pelvic organs: the portion attaching to the uterus is referred to as the parametrium, whereas the portion anchoring the vagina is termed the paracolpium (3). Through these attachments, the vagina is laterally secured to the pelvic walls, effectively dividing the female pelvic cavity into anterior and posterior compartments (Figure 1). This anatomical arrangement underlies the classification of pelvic organ prolapse into anterior prolapse (descent of the anterior vaginal wall into the introitus), apical prolapse involving the cervix and vaginal apex, and posterior prolapse (descent of the posterior vaginal wall into the introitus) (2,14).

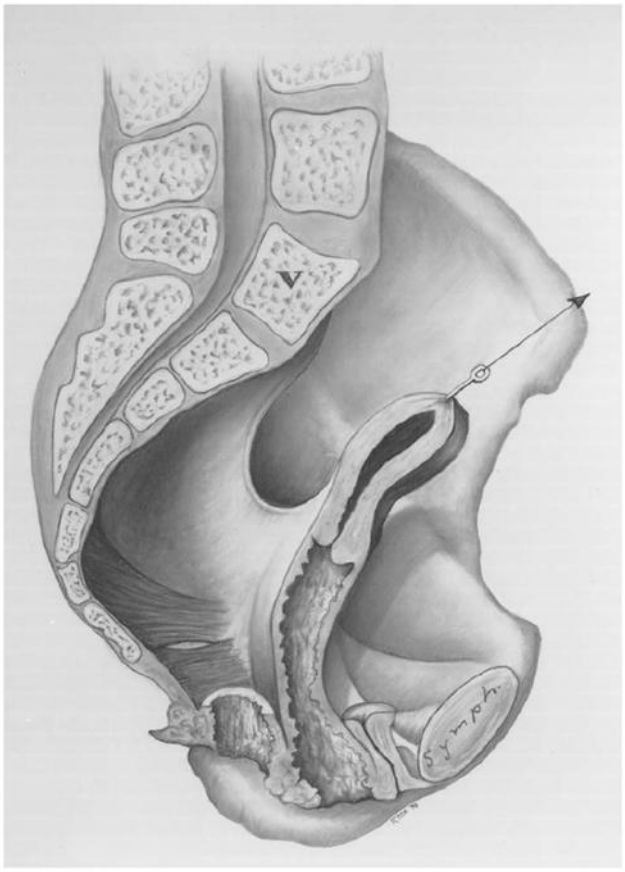


**Figure 1. Anterior and posterior pelvic compartments**

The vagina, laterally connected to the pelvic walls via the endopelvic fascia, divides the pelvis into anterior and posterior compartments. (2)

The structural integrity of the endopelvic fascia is a prerequisite for adequate support of the pelvic organs, particularly the vagina (10). DeLancey proposed a three-level classification of vaginal support within the endopelvic fascia, which remains fundamental to understanding the pathophysiology of pelvic organ prolapse (Figures 2 and 3).

Level I support is provided by the cardinal and uterosacral ligaments as components of the parametrium. The parametrium connects the cervix and uterus to the pelvic walls and transitions caudally into the paracolpium, which secures the vagina to the pelvic walls, thereby

maintaining the continuity of the endopelvic fascia (3,15,16).

Level II support consists of the lower portion of the paracolpium, which attaches laterally to the vagina and stabilizes it in a transverse plane between the bladder and the rectum. This portion of the endopelvic fascia is referred to clinically as the pubocervical and rectovaginal fascia (2,13). Both structures are anchored laterally to the arcus tendineus fasciae pelvis, a key element in maintaining pelvic organ support.

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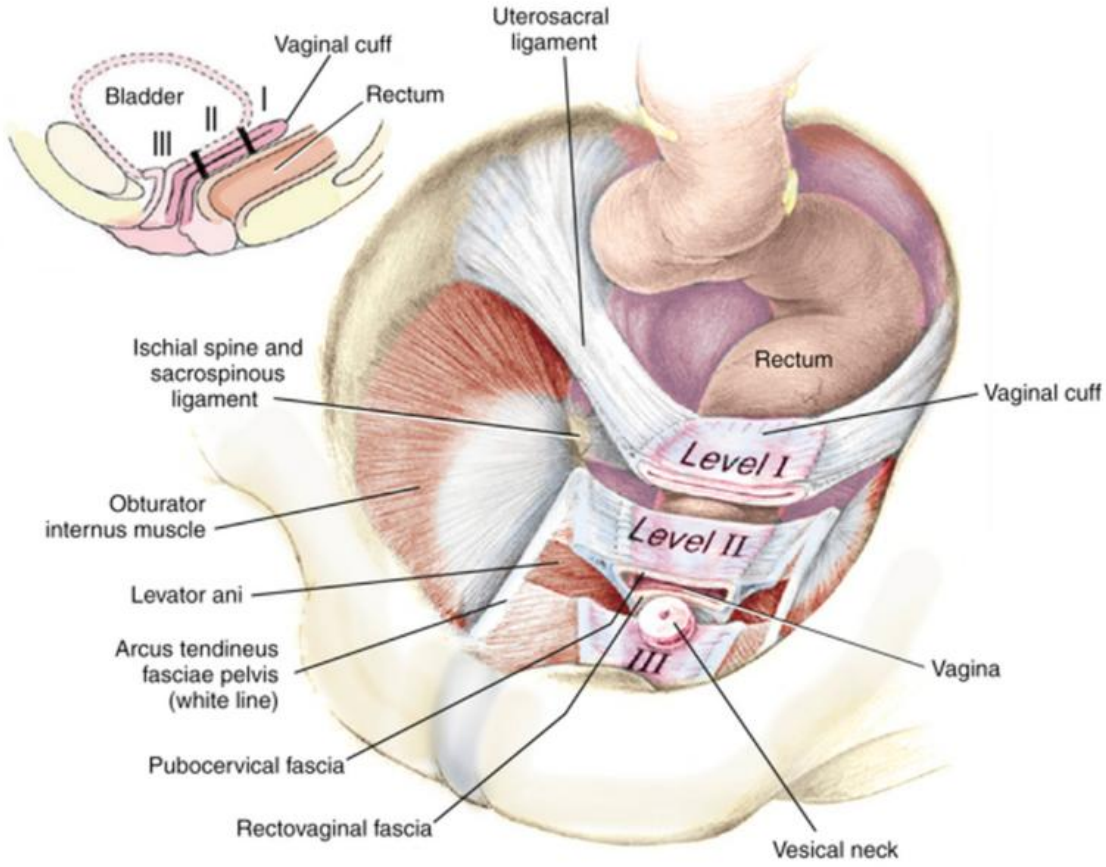


**Figure 2. Levels of vaginal support according to DeLancey (17)**

However, DeLancey emphasized that these fasciae are primarily clinical constructs and, from a strict anatomical standpoint, are not discrete entities. In this context, the pubocervical fascia represents the anterior vaginal wall together with the endopelvic fascia attaching it to the pelvic walls, while the rectovaginal fascia comprises the posterior vaginal wall and the endopelvic fascia whose attachments prevent posterior prolapse (2).

Level III support is distinct in that it is achieved without the involvement of the paracolpium. This level supports the distal vagina, whose walls are directly attached to adjacent structures. Anteriorly, the vagina is connected to the urethra; posteriorly, to the perineal body; and laterally, to the levator ani muscle (2,10)

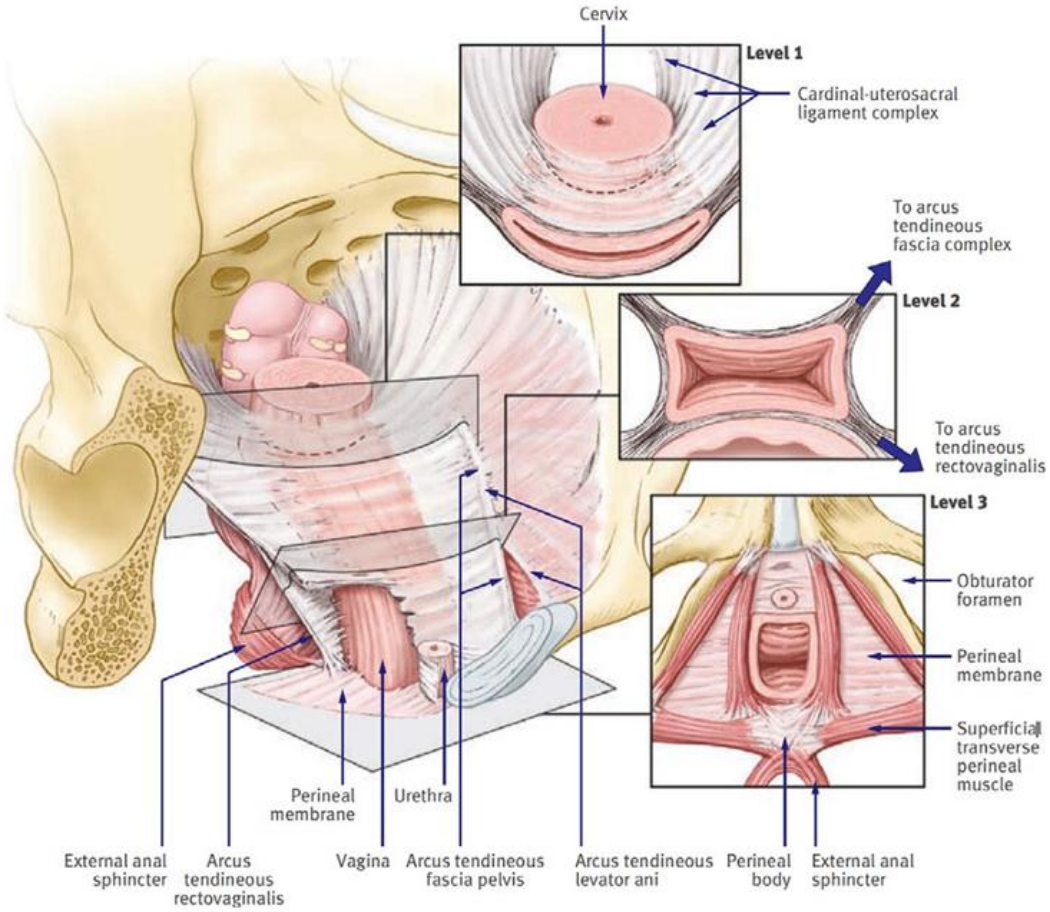


**Figure 3. Three levels of vaginal support according to DeLancey (18)**

## Mechanisms of Urinary Continence Maintenance

Pelvic floor defects often cause urinary incontinence as one of the leading symptoms. This symptom is a common indication for surgical treatment of pelvic floor defects. Urinary continence in women is maintained through the action of several interconnected mechanisms that together prevent the occurrence of stress urinary incontinence (4,19). Although it is a complex physiological process, two key elements are crucial: 1) adequate support to the lower urinary tract, and 2) normal sphincter function.

When intra-abdominal pressure increases, a phenomenon occurs in which the pressure from the abdominal cavity is transmitted to the urethra, preventing the onset of stress urinary incontinence (4). This theory was proposed by Enhöring, who emphasized the importance of the anatomical position of the urethra relative to the pelvic floor. Specifically, when the urethra descends below the level of the pelvic floor, it exits the area of intra-abdominal pressure influence, which prevents effective pressure transmission and can result in stress urinary incontinence. Although significant, this concept alone does not fully explain continence, as it cannot account for why women with marked cystourethroceles may remain continent despite urethral descent (20). Based on such theories, surgical techniques were developed to position the urethra as far above the pelvic floor as possible (4). However, studies have shown that the urethral position is not directly linked to the sphincter mechanism, which is clinically confirmed by the presence of continence in women with cystourethroceles (4,21).

A key anatomical mechanism in maintaining continence is the fact that the urethra rests on a

supportive tissue layer composed of the endopelvic fascia and the anterior vaginal wall (4). This layer provides urethral stability through its lateral attachments to the pelvic walls and the arcus tendineus fasciae pelvis. This creates the so-called "hammock" mechanism of urethral support, enabling the urethra, during increases in intra-abdominal pressure, to be compressed between downward pressure from above and the firm tissue layer beneath it. This results in efficient pressure transmission, pressing the urethra against the supportive tissue and bringing the anterior and posterior urethral walls closer together, thereby closing the lumen (Figures 4 and 5). This means that the continuity of the endopelvic fascia, vaginal wall, arcus tendineus fasciae pelvis, and the levator ani muscle together form the described "hammock" mechanism.

During straining, the proximal (mobile) urethra shifts, tightening the connective tissue and the arcus tendineus fasciae pelvis, which provides firm support to the urethra (2,4).

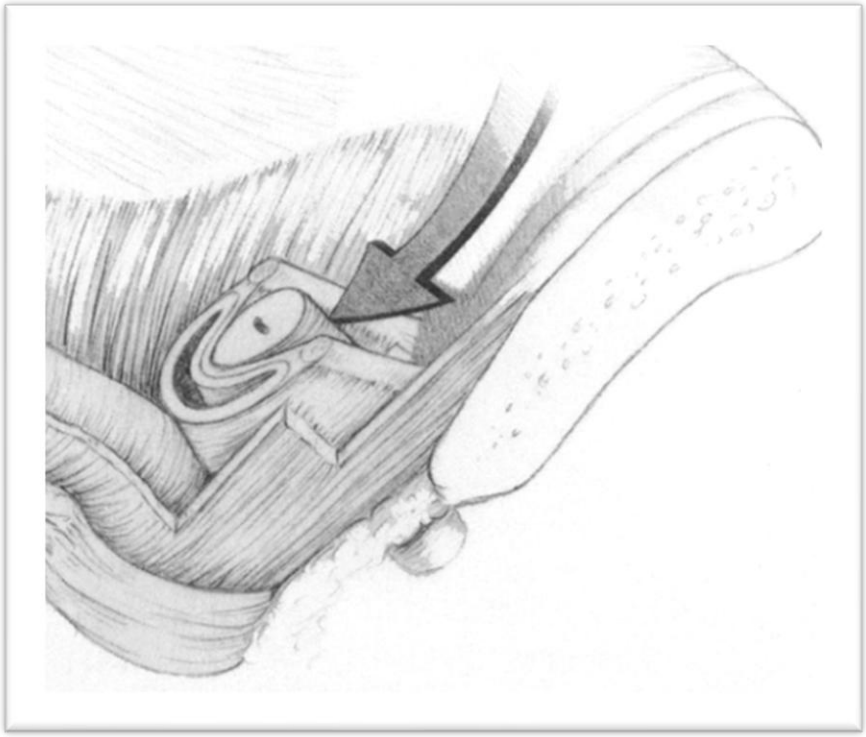

**Figure 4. Illustration of urethral compression by preserved suburethral tissue – the "hammock" mechanism. The arrow indicates the direction of intra-abdominal pressure (4).**

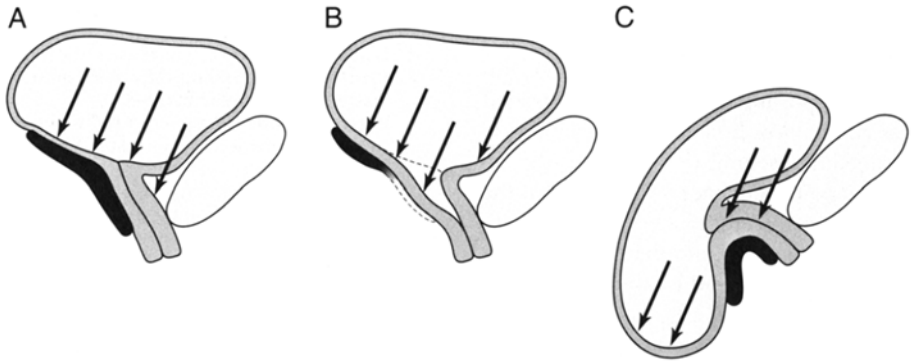


**Figure 5. Illustration of the effect of intra-abdominal pressure on the urethra and pelvic floor.**

A) Preserved suburethral support allows normal transmission of intra-abdominal pressure (arrows) that compresses the urethra against the suburethral tissue.

B) Damaged suburethral support cannot serve as a layer against which the urethra is compressed.

C) Presence of cystourethrocele and low urethral position. The support system is preserved, and no incontinence is present (4).

Research findings indicate that stress urinary incontinence may persist even after surgical interventions such as urethral suspension, despite the bladder neck being positioned high. This confirms that the strength of the supportive layer beneath the urethra is more important for continence preservation than the absolute height of the urethra and bladder neck relative to the pelvic floor (22).

It is important to emphasize that the proximal part of the urethra is mobile, whereas the distal segment is stabilized and fixed within a firm tissue structure, connected to the pubic bones via the perineal membrane. The urethra is positioned obliquely relative to the horizontal plane, with the retropubic space (space of Retzius) located anteriorly and cranially, and the endopelvic fascia and vaginal wall posteriorly and caudally (2,4).

For the maintenance of continence, it is crucial that urethral pressure remains higher than intravesical pressure, even during maximal exertion (13). The most common cause of stress urinary incontinence is associated with weakened support of the proximal urethral segment and bladder neck (19).

The ligamentous system of the urethra is an important component in maintaining continence. This system is also part of the endopelvic fascia and consists of the pubocervical fascia, pubourethral ligaments, and urethropelvic ligaments. The pubocervical fascia plays a key role in maintaining the normal anatomical position of the bladder. The pubourethral ligaments are located anterior to the pubocervical fascia, surround the distal urethra, and attach behind the symphysis (13). The connection between the distal paraurethral tissues and the pubic bones is achieved through the pubourethral ligaments, which continue into the perineal membrane. The urethropelvic ligaments provide stability to the bladder neck (13,19).

In addition to the supporting role of the ligaments, the position of the proximal urethra and the bladder neck is of critical importance. These two structures are both mobile and subject to voluntary control. The bladder neck lies superior to the level of the pubourethral ligaments, and its position is further regulated by the activity of the m. levator ani (19). Through its anatomical connections with the endopelvic fascia, this muscle governs the positioning of the bladder neck.Relaxation of the m. levator ani at the onset of micturition permits downward rotation of the bladder neck to the limit allowed by the elasticity of its fascial attachment. Subsequent contraction at the completion of micturition restores the bladder neck to its original position (2,17). Furthermore, histological studies indicate that the m. levator ani consists predominantly of slow twitch (type I) muscle fibers, which are adapted for sustained activity. This morphological characteristic explains the ability of the muscle to maintain the bladder neck in a consistently elevated position without conscious awareness of ongoing contraction (23).Correspondingly, relaxation of the muscle occurs exclusively during micturition and defecation (24).

Several lines of evidence support the role of the m. levator ani in maintaining urinary continence. First, as previously noted, the muscle's structural composition inherently implies continuous activity. Second, denervation of this muscle has been identified in some female patients with stress urinary incontinence. The interaction between muscular and connective tissues is also of critical importance: in the absence of muscular activity, connective tissue can temporarily assume a supportive function; however, without adequate muscle performance, structural failure occurs rapidly.Moreover, injury to the m. levator ani disrupts the transmission of intra abdominal

pressure to the urethra, thereby compromising the continence mechanism. The clinical relevance of the m. levator ani in preserving continence is further supported by the therapeutic efficacy of pelvic floor muscle training, which has been shown to alleviate symptoms of stress urinary incontinence (4,19).The m. levator ani actively participates in the process of micturition. Its activity explains the phenomenon whereby voluntary increases in intra abdominal pressure enhance urinary flow, and increases in intra abdominal pressure during coughing in healthy women trigger the continence mechanism, i.e., urine retention. This dual function can be attributed to two coordinated actions of the m. levator ani: contraction to sustain continence and relaxation to facilitate micturition.The muscle alters the position of the bladder neck via the pubovesical ligament, a thickened portion of the endopelvic fascia located anterior to the bladder neck. At the onset of micturition, the bladder neck and proximal urethra—both mobile structures—descend caudally, causing the pubovesical ligaments to stretch and pull the bladder neck forward, thereby promoting its opening. Conversely, during contraction of the m. levator ani, the bladder neck is compressed against the pubovesical ligament, contributing to its closure.Taken together, these findings underscore the necessity of restoring native anatomical relationships for the effective treatment of stress urinary incontinence (19,25,26).

It is important to emphasize that urethral support is not the sole mechanism for maintaining urinary continence, but rather one component within the complex interplay of micturition physiology and the anatomical prerequisites for continence (4, 19). Research has shown that the greatest increase in urethral pressure does not occur in the proximal urethra—where intra-abdominal pressure is directly applied—but in its distal segment (27). Moreover, for continence to be maintained, urethral pressure must exceed both bladder pressure and intra-abdominal pressure during maximal exertion (13).These observations highlight the critical role of sphincter function, since the muscle sphincters m. compressor urethrae and m. sphincter urethrovaginalis—located in the distal urethra where pressure peaks—are fundamental to continence (28–30). Clinically, the urethral sphincter mechanism is divided into two segments: the internal sphincter (bladder neck) and the external sphincter (extending along most of the urethra). The internal sphincter comprises the proximal 20% of the urethral lumen, whereas the external sphincter spans 20–80% of its length (19).The term "bladder neck" often denotes a functional unit of the bladder base through which the urethral lumen passes within the detrusor musculature, rather than a single discrete anatomical structure (2). The internal sphincter is frequently modulated by a detrusor muscle loop that functions independently of the detrusor itself, owing to regional $\alpha$ -adrenergic innervation (19). Between this detrusor loop and the urethral lumen lies a ring of smooth muscle—known as the trigonal ring—which may also contribute to bladder neck closure (19, 31). In this region, the previously described mechanism of bladder neck compression against the pubovesical ligament is likewise operative.

Urethral support and the proximal sphincter act together to prevent urinary incontinence. However, it has been demonstrated that in 50 % of continent women, urine enters the urethra during coughing. In such cases, the distal sphincter is the key determinant of continence. Its importance is particularly evident in women who develop stress urinary incontinence following radical vulvectomy (32).

Urethral sphincter function results from the interplay of three tissue components: striated muscle, smooth muscle, and the submucosal vascular plexus. Each component contributes one third of urethral closing pressure at rest (2, 19, 33). The striated muscle constitutes the external urethral sphincter and is primarily circular in orientation. Distally, it takes a divergent course, extending beyond the urethral wall into the vaginal wall as the urethrovaginal sphincter or into the region above the perineal membrane as the m. compressor urethrae (2, 19, 30). Both muscles are composed of slow-contracting (type I) fibers that maintain tonic

contraction for continence and allow voluntary augmentation of urethral pressure when needed (2, 19). The smooth urethral muscle comprises two layers—an outer circular layer and an inner longitudinal layer. The longitudinal layer is more prominent, although its precise function remains to be fully elucidated (2). The submucosal vascular plexus of the urethra is highly developed relative to the organ's size. It consists of specialized arteriovenous anastomoses that form a barrier contributing to urethral closing pressure. This plexus engorges with blood during coughing, aiding urethral closure and continence. Interrupting arterial flow to this plexus disrupts urethral closing pressure (2, 19).All three mechanisms—urethral support, the internal sphincter, and the external sphincter—work in concert to maintain continence. Dysfunction of any one component may, but does not necessarily, be compensated for by the others (19).

## Prolapse of uterus and vaginal apex

Prevention of any pelvic organ prolapse depends on the interplay between the endopelvic fascia and the pelvic floor muscles. When the levator ani muscle is damaged, the burden of support shifts to the connective tissue of the endopelvic fascia (34).

The biomechanical properties of connective tissue allow it to withstand increased load only for a short period; beyond that, it yields and prolapse ensues. For this reason, sustained tonic activity of the levator ani is essential. This muscle works in concert with other muscles throughout the body. Without such coordination, the striated fibers of the levator ani would not contract rapidly enough in response to sudden rises in intra-abdominal pressure (e.g., coughing), leading to stress urinary incontinence. Thus, contraction of the pelvic floor muscles is synchronized with the respiratory diaphragm, intercostal muscles, and abdominal wall muscles to maintain continence (19).

The apical compartment of prolapse includes uterine prolapse, vaginal vault prolapse, and enterocele (35). In this form of prolapse, the support provided by the cardinal and uterosacral ligaments is compromised (13). Such damage corresponds to a disruption of DeLancey's level I support (2).Descent of the uterus to the hymenal ring is termed descensus, whereas descent through the hymenal ring is classified as total or subtotal prolapse. This group of defects is often associated with rectocele or cystocele (13).Enterocele refers to the descent of the cranial portion of the posterior vaginal wall resulting from damage to the rectovaginal septum. In this situation, small bowel loops herniate into that segment of the vaginal wall. The International Continence Society (ICS) notes that, in the presence of an enterocele, peristaltic waves of small intestine loops may be observed bulging through the posterior vaginal wall (13,36).

The cranial 2–3 cm of the vagina are stabilized by level I support. At this location, the fibers of the endopelvic fascia run both horizontally and vertically, so that the upper vaginal segment is suspended from the pelvic sidewalls and the sacrum (3).

Cadaveric studies on pelvic sections have shown that when a downward force is applied to the vaginal apex in an attempt to provoke vaginal eversion, the tension of the corresponding paracolpium prevents such an event. Transection of the paracolpium at the level I suspension induces vaginal eversion, confirming the role of level I support (Figure 6). In a similar fashion, the importance of the parametria in suspending the uterus and cervix has been demonstrated (2, 3). The parametria permit a certain degree of cervical descent during surgery, after which they tighten and prevent further displacement (2, 37).

An experimental upward force applied to the vaginal apex did not mobilize level I support and was limited solely by the elasticity of the vaginal wall. In this context, the interaction between the pelvic floor muscles and the endopelvic fascia is crucial: as long as the levator ani muscle remains functional and intact, minimal load is transferred to the connective tissue (2, 3).

Prolapse involves not only anatomical alterations but also histological changes that primarily affect the vaginal wall and pelvic

ligaments. Studies examining these alterations have demonstrated a 20 % reduction in smooth muscle content within the vaginal wall of women with prolapse. Concurrently, the proportion of connective tissue in the vaginal wall increases, indicating active remodeling in prolapsed tissues (38,39).

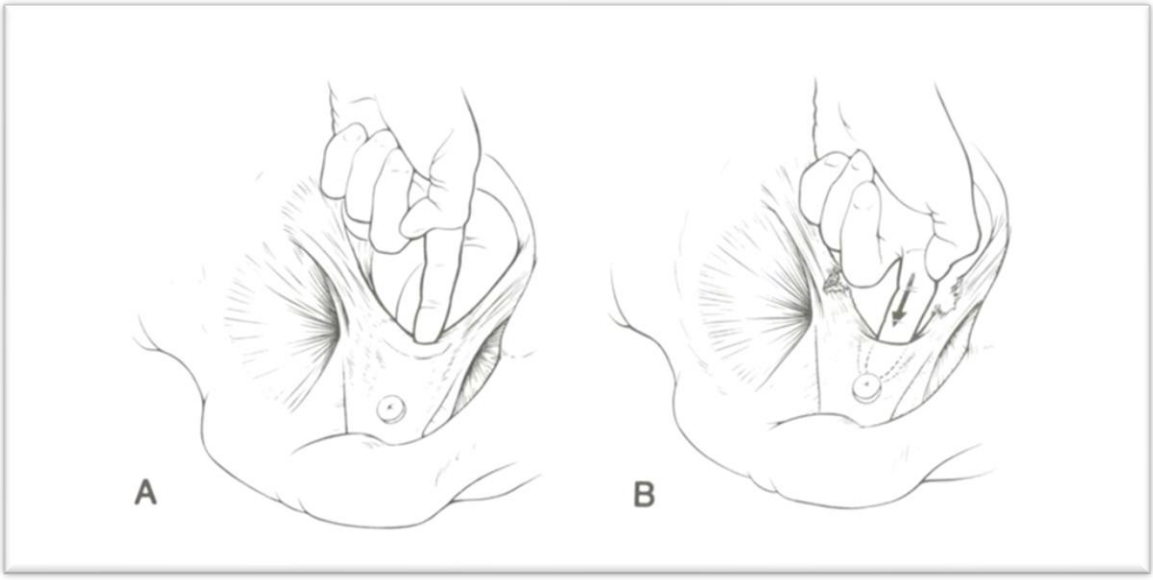


**Figure 6.**
**A) When a downward-directed force is applied to the vaginal apex, eversion of the vagina is prevented by Level I support.**
**B) Disruption of Level I support allows vaginal eversion to occur (3).**

Although apical vaginal prolapse in women post-hysterectomy often occurs in combination with cystocele or rectocele—reflecting damage to the level II support system—vaginal eversion does not develop unless the level I suspensory fibers are also disrupted (3). Different forms of prolapse may present concurrently in the same patient, producing varied clinical manifestations; however, the specific defect always corresponds to the support level that is compromised (2,3,8).This understanding carries significant clinical implications. If the level II support apparatus remains intact, only vaginal eversion will occur, and all symptoms can be resolved by surgically suspending the vaginal apex (3). Accordingly, post-hysterectomy vaginal eversion is prevented by anchoring the cardinal and uterosacral ligaments to the vaginal cuff. An intrafascial hysterectomy—avoiding dissection of the paracolpium from the vagina—can also be performed (3,40).The benefits of this surgical technique are manifold: it ensures postoperative vaginal stability, reduces the risk of infection, and minimizes injury to the bladder and ureters (41). Whether performed via abdominal or vaginal approach, intrafascial hysterectomy also preserves vaginal length, axis, and overall configuration (40).

## Anterior vaginal wall prolapse

The anterior vaginal wall is the segment most commonly involved in prolapse (42). This defect encompasses bladder descent (cystocele), urethral prolapse (urethrocele), and combined bladder-urethral prolapse (cystourethrocele) (13,35).

The term cystocele denotes pathological herniation of the anterior vaginal wall together with the bladder base. Urethrocele refers to prolapse of the distal anterior vaginal wall involving only the urethra (13). Anterior vaginal wall prolapse arises from impairment of level II and III support structures (2).

Support of the anterior vaginal wall depends on the continuity of the vagina and periurethral tissues with the pelvic musculature and bony walls, rather than on direct urethral attachments. At level III support, this continuity is robust because the distal vagina is anchored to the medial fibers of the levator ani muscle (Figure 7). This region's primary role is to facilitate levator ani activity in closing the distal vagina, although injuries to urethral support and the perineal body in this area remain clinically significant (3).

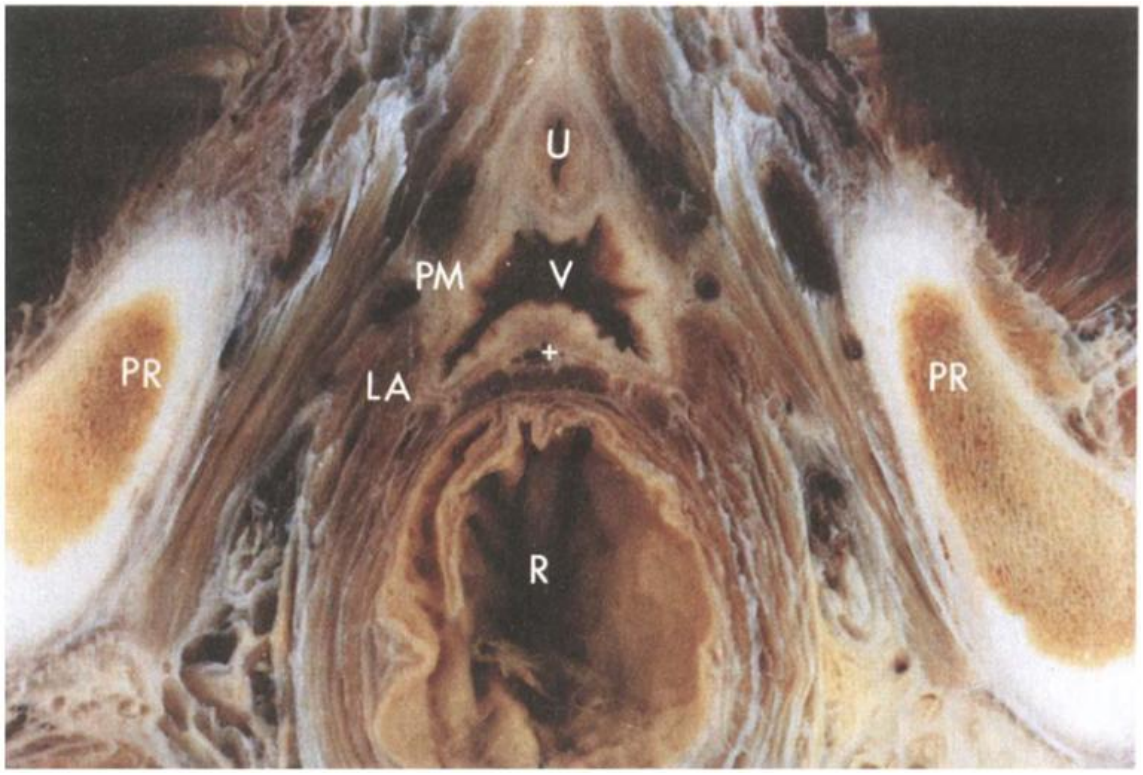


**Figure 7. Transverse cadaveric section showing Level III. The connection of the vagina (V) with the urethra (U), the perineal membrane (PM), and the m. levator ani (LA) is shown. PR (pubic ramus), R (rectum) (3).**

A key structure in pelvic organ support is the arcus tendineus fasciae pelvis, which serves as one of the anchoring sites for the endopelvic fascia. It is a band of connective tissue on each side of the pelvis that attaches anteriorly to the pubic bone and posteriorly to the ischial spine (19).

The tissue component serving as urethral support has two attachment sites: a fascial insertion on the arcus tendineus fasciae pelvis and a muscular insertion on the levator ani (4). The fascial insertion connects the paraurethral tissue and anterior vaginal wall to the arcus tendineus fasciae pelvis, known as the paravaginal fascial insertion (2,43). Disruption of this paravaginal fascial insertion from the pelvic sidewall leads to anterior prolapse and stress urinary incontinence (2).

The muscular insertion plays a crucial role in force transmission, allowing pelvic floor muscle tone to contribute to bladder neck stabilization (19). Through this interaction, the muscular and fascial insertions operate as a functionally integrated unit, providing support to the proximal urethra and bladder neck (2).

The levator ani muscle maintains constant tonic activity that enables closure of the distal vagina and supports pelvic organs. Physiological studies have shown that this muscle consists almost exclusively of slow-twitch (type I) fibers, which facilitate steady tonic contraction and give the muscle its characteristic pale color (19). Its

tone varies in response to changes in intra-abdominal pressure (44,45).

Further research has demonstrated that the levator ani also contains a proportion of smooth muscle fibers, enabling dynamic coordination between skeletal and smooth muscle components. The smooth fibers help maintain basal muscle tone, while the skeletal fibers participate in voluntary control of micturition and defecation (45).

Position and mobility of the anterior vaginal wall, bladder, and urethra have a direct impact on the development of urinary incontinence and anterior prolapse. Fluoroscopy demonstrates that the proximal urethra and bladder neck are mobile structures, whereas the distal urethra remains fixed (2).

Anterior vaginal wall prolapse may develop by one of two mechanisms (2). The first involves lateral detachment of the pubocervical fascia insertion from the pelvic sidewall—specifically the arcus tendineus fasciae pelvis—resulting in a "displacement" cystocele (lateral cystocele) (2,13). This defect is also known as a paravaginal cystocele (14). Clinically, a lateral cystocele is identified by preserved vaginal rugae and an intact mucosal light reflex on examination (13,14).

The second mechanism entails central disruption of the vaginal wall, that is, a midline rupture of the pubocervical fascia, which leads to a distension or central cystocele (2,13). On clinical inspection, a central cystocele presents with reduced mucosal rugae and loss of the light reflex over the vaginal mucosa (13).

## Posterior vaginal wall prolapse

Posterior vaginal wall prolapse (rectocele) is the pathological herniation of the distal posterior vaginal wall encompassing the rectum (13). In this form of defect, herniation of the rectum into the posterior vaginal wall manifests as bulging of the posterior vaginal wall (46). Disruption of level II and III support leads to posterior wall prolapse. At level III, the distal vagina joins the perineal body, which is connected to the perineal membranes on both sides (2). As previously described, at this support level the vaginal wall is directly attached to surrounding structures without an interposed paracolpium (10). Studies indicate that among women with documented prolapse, 76 % have a posterior compartment support defect resulting in rectocele (47). The distal rectum rests on the dense connective tissue of the perineal body at level III (5).

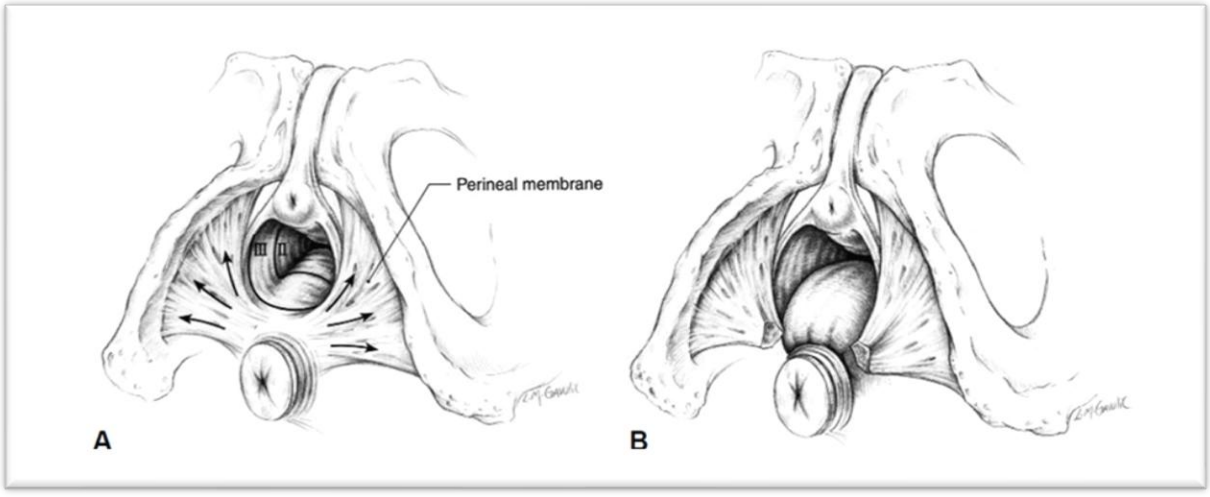


**Figure 8. A. The attachment of the perineal membrane to the pubic and ischial bones is shown, along with the direction of fiber tension that occurs when supporting pelvic structures at level III support. A characteristic (normal) U-shape of the posterior vaginal wall at level III support is observed. B. The attachments of the perineal membrane to the perineal body are severed, resulting in loss of support to the distal part of the rectum. A bulging of the posterior vaginal wall is observed (5).**

The perineal body represents the central convergence of the two perineal membranes, which originate from the inferior pubic and ischial rami and function to resist caudally directed forces on the rectum, thereby preventing rectal prolapse (Figure 8) (5). Level III support structures can provide stable support to pelvic organs only if their continuity with level II support is preserved. The mid-portion of the posterior vaginal wall (level II vaginal support) is connected to the internal aspect of the levator ani muscle via sheets of endopelvic fascia (2).

Due to the continuity between level II and III support, caudally directed forces on the perineal body are resisted not only by the perineal membrane but also by the preserved continuum of all support levels, which maintains the perineal body in its proper position (5). It is important to note that at level I there are no separate supports for the anterior and posterior vaginal walls (2).

Disruption of the integrity of the rectovaginal septum is responsible for the development of posterior vaginal wall prolapse (48). The rectovaginal septum is described as a continuous layer of tissue extending from the sacrum cranially to the perineal body caudally (49).

Denonvilliers originally described a dense tissue layer between the bladder and the rectum in men and termed it the rectovesical septum (6). The tissue layer between the vagina and the rectum is considered analogous to the rectovesical septum in men and is therefore often referred to as Denonvilliers' fascia (46).The majority of fibers in this portion of the endopelvic fascia attach to the lateral aspects of the posterior vaginal wall, preventing ventral displacement of the posterior wall, while only a smaller portion of fibers crosses to the contralateral side (Figure 9) (5). Transection of the fibers that attach to the lateral portions of the posterior vaginal wall leads to significant destabilization of the posterior vagina at level II support. However, cutting the fibers that cross to the opposite side results in less destabilization (5). The posterior vaginal wall attaches caudally to the perineal body (3).From the foregoing, it follows that rectocele arises either from stretching of the rectovaginal fascia or from its tears and separations. Such defects most commonly occur as a result of childbirth or as a consequence of episiotomy (46).

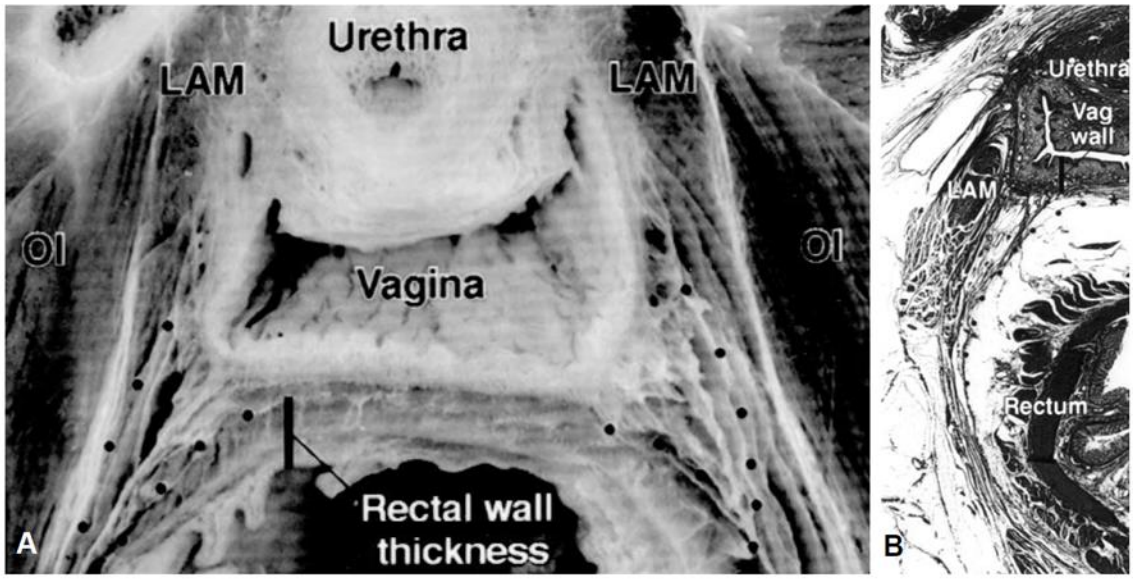


**Figure 9. A. A cross-sectional view in the transverse plane shows that most fibers of the endopelvic fascia (indicated by dots) attach to the lateral sulci of the posterior vaginal wall, while only a smaller number cross to the opposite side. B. The origin of the endopelvic fascia fibers from the superior fascia of the levator ani muscle (LAM) is observed. OI, obturator internus muscle (5).**

The analysis of posterior pelvic compartment dynamics underscores the critical role of the puborectalis muscle. Its sustained contraction maintains constant apposition of the anterior and posterior vaginal walls, creating a balance of forces between the anterior and posterior pelvic compartments and thus imposing minimal load on level II support (Figure 10) (5). The levator ani muscle prevents excessive strain on connective tissue and facilitates the change in vaginal axis from a horizontal to a vertical orientation (2,44). Posterior vaginal wall prolapse typically occurs at or below the levator plate, along the vertical vaginal segment (46). When the levator ani is compromised, the vaginal canal opens and the load shifts to level II support (5). This phenomenon further illustrates the importance of synergistic interaction between muscular and fascial support components in maintaining pelvic-floor integrity (2,5,19).

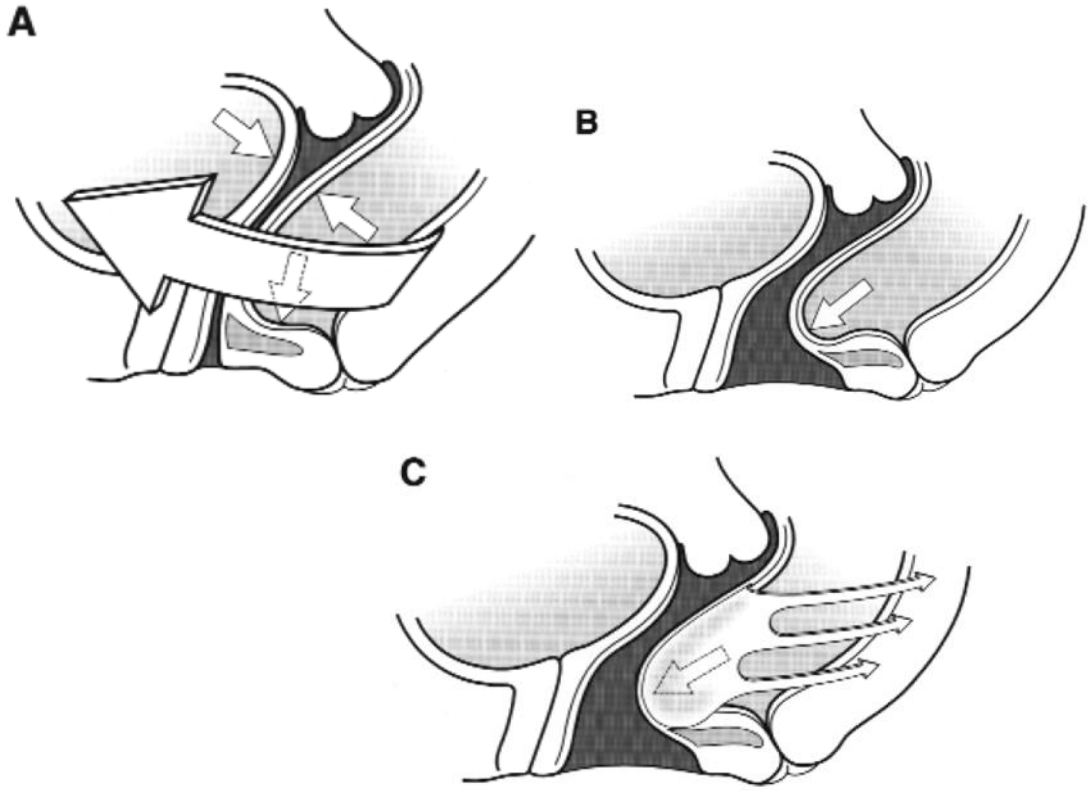


**Figure 10. Mechanism of support in the posterior pelvic compartment.**

A. Closure of the pelvic floor is shown through the activity of the puborectalis muscle, which keeps the anterior and posterior vaginal walls in contact. With an increase in intra-abdominal pressure, the force is evenly distributed to both the anterior and posterior vaginal walls. Below the level of the puborectalis muscle, there is no force to counterbalance intra-abdominal pressure, so in this area, the perineal membrane resists the force. B. Absence of puborectalis muscle activity creates a resultant force on the posterior vaginal wall. C. Lack of puborectalis muscle activity places level II support under load (interaction of the muscular and fascial components of support) (5).

In patients with posterior vaginal wall prolapse, specific morphological changes become evident during the Valsalva maneuver. One hallmark finding is bulging of the vaginal wall, which on MRI takes on a "kneeling posture" appearance—a bending of the wall reminiscent of the body's motion at the onset of kneeling. This feature is considered diagnostically significant for all forms of rectocele (50). Additionally, MRI demonstrates inferior displacement of the upper two thirds of the vagina in every case of rectocele. In a subset of

patients, further changes such as perineal descent and widening of the distal third of the vagina are also observed (Figures 11 and 12)(50).

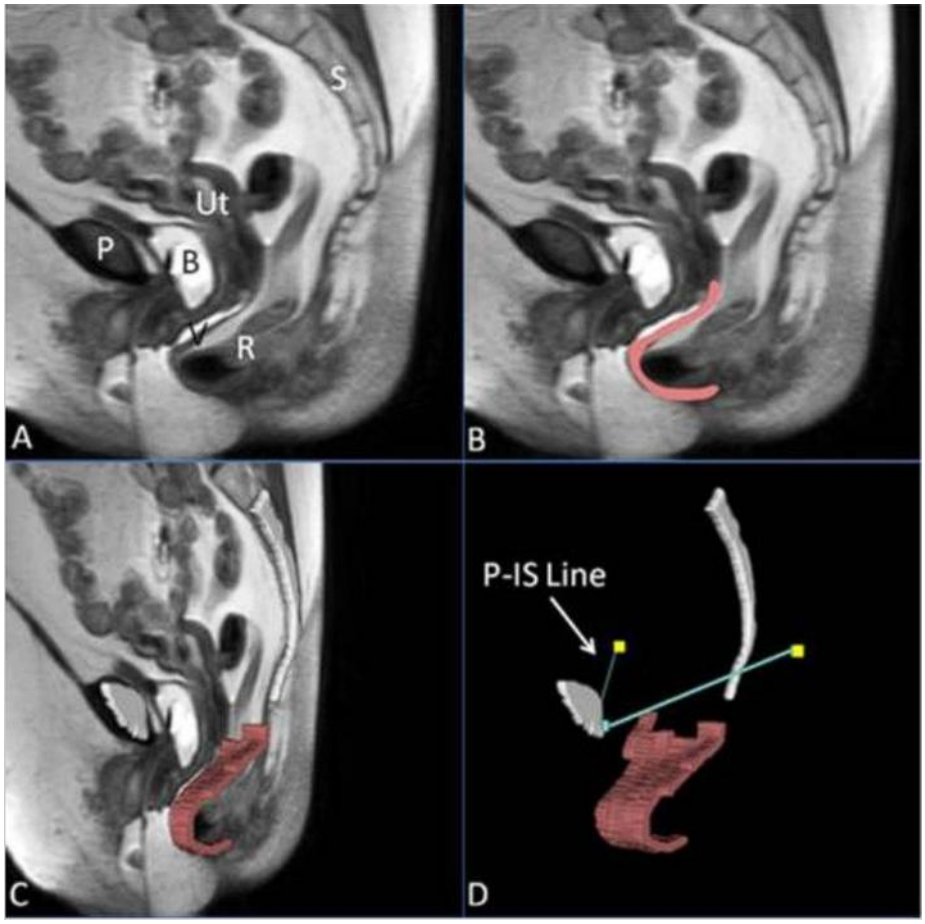


**Figure 11. A. Median sagittal MRI scan of a patient with posterior prolapse.**

B. Prolapse marked in red. C. 3D model of the prolapse showing the median sagittal portion of the pubic bones (marked in white) in an oblique MRI scan. D. Relationship of the posterior vaginal wall to the ATFP, which is shown as a line from the pubic symphysis to the ischial spines (marked in yellow) (50).

The levator ani muscle is directly attached to the superior surface of the perineal membrane. When the perineal membrane splits, the left and right portions of the levator ani separate as well. Repairing the perineal membrane defect restores the muscles to their anatomically correct position (5). With normal levator ani tone, a balance of forces is maintained between the anterior and posterior pelvic compartments, resulting in minimal loading of level II support (24). The distal vagina, which lies outside the influence of the puborectalis portion of the levator ani, lacks this balancing mechanism. Consequently, the dense connective tissue at the junction of the perineal membranes within the perineal body becomes the primary load bearer (5).

Rectoceles may present proximally (high rectocele), centrally, or distally (low rectocele) (46). Each rectocele arises from a specific anatomical defect and therefore requires a tailored therapeutic approach (51). For instance, damage to the level II support system can produce a midvaginal posterior wall rectocele even when levator ani function and perineal body integrity remain intact (5). In the distal region, a perineal rectocele may develop; its treatment does not involve repairing the rectovaginal fascia but rather approximating and

suturing the separated fibers of the perineal body (5).

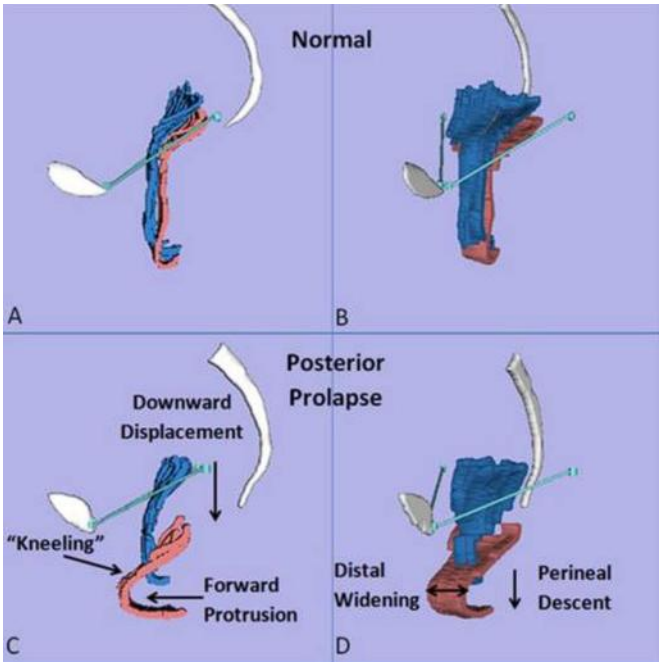


**Figure 12. Characteristics of posterior prolapse (50).**

## Conclusion

Pelvic floor defects are common conditions affecting women of all ages. The aim of this paper was to delineate the fundamental concepts underlying individual pelvic floor defects. Before the fibromuscular endopelvic fascial system and continence-preserving theories were described, precise surgical correction was not feasible. Each defect corresponds to a specific structural lesion, and its identification is essential to enable targeted surgical repair. The fibromuscular apparatus works in concert with active muscle function to maintain pelvic floor integrity; without this dynamic interaction, the biomechanical properties of connective tissue alone are insufficient to preserve the normal position of pelvic organs. Restoring the balance between muscular and connective-tissue elements is a prerequisite for normal pelvic organ support. A single patient may present with multiple concurrent defects, and detailed anatomical knowledge is therefore critical for managing such complex, combined lesions. Different patients exhibit distinct risk factors that produce specific anatomical and histological defects at defined levels of support. DeLancey's classification of vaginal support levels greatly facilitates therapeutic planning and provides a unified framework for categorizing pelvic floor defects. This approach will also pave the way for the introduction of novel diagnostic assessments and therapeutic strategies for this condition.

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Guarantor of the study: Branimir K, Borna K, VLK, IMP, MP

# Anatomski temelj defekata dna zdjelice

**Sažetak**

Defekti dna zdjelice (engl. Pelvic Floor Disorders) čest su problem koji zahvaća žene različite dobi. Jedan su od entiteta koji pripada poremećajima zdjeličnog dna. Svaki defekt dna zdjelice uzrokovan je specifičnim oštećenjem anatomskog i funkcionalnog integriteta dna zdjelice. Razumijevanje patološke anatomije u podlozi ključno je za ispravan terapijski pristup u liječenju ovog stanja. Prolaps organa male zdjelice označava spuštanje zdjeličnih organa u ili kroz vaginalni kanal. To uključuje mokraćni mjehur, maternicu, vaginalnu zaraslicu, tanko i debelo crijevo. Postoje tri skupine prolapsa: prednji, stražnji i apikalni. Prolapsi nastaju zbog oštećenja fibromuslukarnog potpornog sustava koji se naziva endopelvična fascija. Ona održava zdjelične organe u normalnom položaju. Za razumijevanje normalne statike zdjeličnih organa ključan je koncept DeLanceyevih razina potpore koji se sastoji od tri razine. Prvu razinu potpore čine kardinalni i uterosakralni ligamenti. Potpora se druge razine sastoji od pubocervikalne i rektovaginalne fascije. Treću razinu potpore čini spoj rodnice s okolnim strukutrama. Svaki prolaps nastaje zbog oštećenja pojedinih razina potpore. Tako su defekti endopelvične fascije prepoznati kao ključni u patofiziologiji nastanka prolapsa zdjeličnih organa. Defekti zdjeličnog dna često naruše i normalne mehanizme održavanja kontinencije čime nastaje urinarna inkontinencija kao čest simptom ovog stanja. Samo se popravkom takvih oštećenja i vraćanjem izvornih anatomskih odnosa može postići uspjeh u kirurškoj terapiji. Cilj je ovog rada detaljno opisati anatomiju svakog defekta sa svrhom stvaranja temelja za dijagnostički i terapijski pristup.

**Ključne riječi:** prolaps zdjeličnih organa, dno zdjelice, poremećaji zdjeličnog dna