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REVIEW ARTICLE

Cystocele and functional anatomy of the pelvic floor: review and update of the various theories

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Abstract
Introduction and hypothesis We updated anatomic theories of pelvic organ support to determine pathophysiology in various forms of cystocele.
Methods PubMed/MEDLINE, ScienceDirect, Cochrane Library, and Web of Science databases were searched using the terms pelvic floor, cystocele, anatomy, connective tissue, endopelvic fascia, and pelvic mobility. We retrieved 612 articles, of which 61 matched our topic and thus were selected. Anatomic structures of bladder support and their roles in cystocele onset were determined on the international anatomic classification; the various anatomic theories of pelvic organ support were reviewed and a synthesis was made of theories of cystocele pathophysiology.
Results Anterior vaginal support structures comprise pubocervical fascia, tendinous arcs, endopelvic fascia, and levator ani muscle. DeLancey's theory was based on anatomic models and, later, magnetic resonance imaging (MRI), establishing a three-level anatomopathologic definition of prolapse. Petros's integral theory demonstrated interdependence between pelvic organ support systems, linking ligament–fascia lesions, and clinical expression. Apical cystocele is induced by failure of the pubocervical fascia and insertion of its cervical ring; lower cystocele is induced by pubocervical fascia (medial cystocele) or endopelvic fascia failure at its arcus tendineus fasciae pelvis attachment (lateral cystocele).
Conclusions Improved anatomic knowledge of vaginal wall support mechanisms will improve understanding of cystocele pathophysiology, diagnosis of the various types, and surgical techniques. The two most relevant theories, DeLancey's and Petros's, are complementary, enriching knowledge of pelvic functional anatomy, but differ in mechanism. Three-dimensional digital models could integrate and assess the mechanical properties of each anatomic structure.

Keywords Pelvic floor · Cystocele · Connective tissue · Anatomy · Endopelvic fascia · Pelvic mobility

Abbreviations

ATFP	Arcus tendineus fasciae pelvis
ATLA	Arcus tendineus levator ani
CL	Cardinal ligament
EF	Endopelvic fascia
LAM	Levator ani muscle
PB	Perineal body
PCF	Pubocervical fascia
USL	Uterosacral ligament

Introduction

Pelvic organs are supported by ligamentous, fascial, and muscular structures [1]. Urogenital prolapse results from anatomic lesions to these support structures. Risk factors are well

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identified, but actual anatomic defects remain poorly analyzed despite the advent of biomechanical models [2–4]. The respective roles of each pelvic floor structure have been the subject of controversy for many years [5, 6], producing numerous anatomic theories [7–11]. These theories sought to account for the transition from normal pelvic anatomy to pathological anatomy of genital prolapse but without objective assessment or proof [7–11]. In this review, we investigated which anatomic defects account for the various anatomic and clinical types of cystocele. The study was founded on a literature review of anatomic structures of pelvic support and theories relating them to clinical prolapse.

Materials and methods

A computerized search of the MEDLINE/PubMed, ScienceDirect, Cochrane Library, and Web of Science databases was performed using the following search terms: pelvic floor, cystocele, connective tissue, anatomy, endopelvic fascia, and pelvic mobility. The search identified 612 studies; 154 studies were selected, and 93 of them did not match the theme of pelvic floor functional anatomy and were excluded; thus, 61 studies were selected for analysis. Anatomic structures were designated using the international anatomic classification *Terminologia Anatomica*. First, all anatomic structures involved in bladder support and their roles in cystocele onset were determined on that classification [12]. The second step updated the various anatomic theories of pelvic organ support. The third step consisted of a synthesis of theories on cystocele pathophysiology: Enhörning's initial theory, Tansatit's theory, Petros's integral theory, and DeLancey's anatomic theory [7–11, 13].

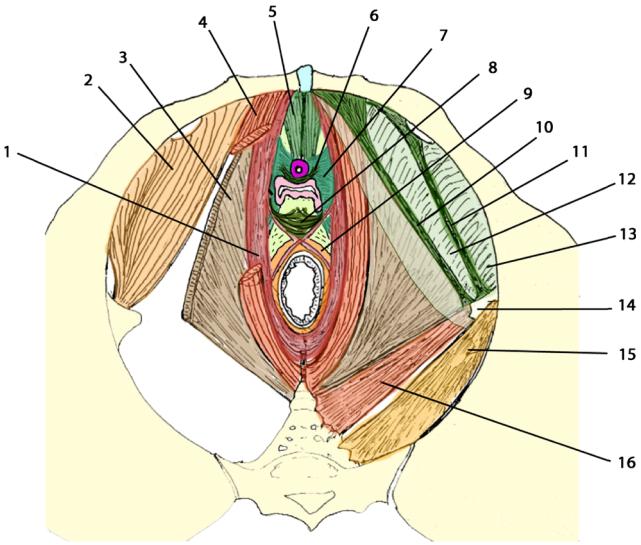
Anatomic structures of bladder support

Anterior vaginal wall support

Anterior vaginal wall support, or perineal sling, plays a major role in maintaining bladder position. The main structures involved are the anterior vaginal wall (AVW) plus the subvesical or pubocervical fascia (PCF), arcus tendineus fasciae pelvis (ATFP), arcus tendineus levator ani (ATLA), endopelvic fascia (EF), and levator ani muscle (LAM) (Figs. 1 and 2).

Anterior vaginal wall

The vagina is a tubular organ between the lower urinary apparatus to the front and the rectoanal apparatus behind. It comprises two segments: The superior segment is composed of mucosa (nonkeratinized, multilayered malpighian epithelium), submucosa (lamina propria or connective lamina), and a



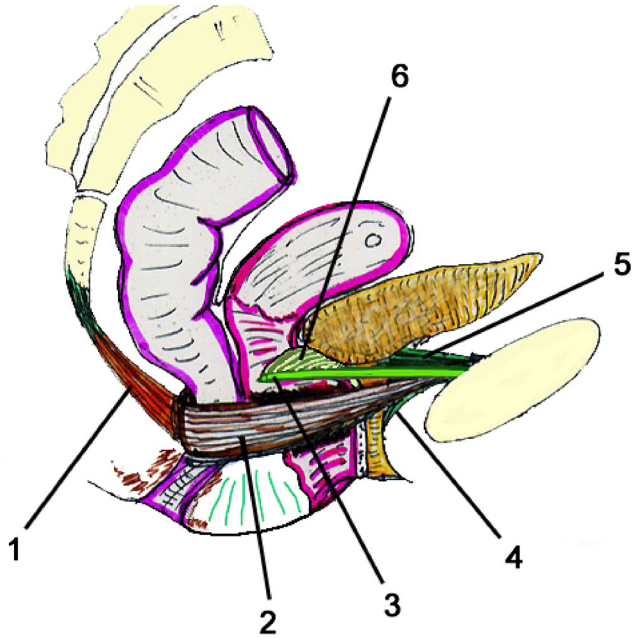
1. puborectal muscle
2. internal obturator muscle
3. iliococcygeus muscle
4. pubococcygeus muscle
5. pubourethral ligament
6. suburethral fascia
7. paravaginal tissue
8. perineal body
9. external anal sphincter
10. arcus tendineus fascia pelvis
11. arcus tendineus levator ani
12. endopelvic fascia
13. internal obturator muscle fascia
14. ischial spine
15. piriform muscle
16. ischiococcygeus muscle

Fig. 1 View of the pelvic floor seen from above (courtesy of Eric Voiglio, with permission)

peripheral muscle layer; on coronal cross-section, it has a transverse long axis extending above the levator plate and oriented by the uterosacral ligaments (USL) toward S3/S4. The inferior introital segment has a sagittal long axis and is composed of mucosa and submucosa interacting with adjacent muscle structures [14]; laterally, the mucosa and submucosa lie directly on the puborectal muscles superiorly and perineal triangle muscles (ischiocavernosus, bulbospongiosus, and pubovaginalis) inferiorly. Anterior vaginal wall composition is a subject of controversy, notably with regard to a possible suburethral fascial layer: some authors reported no specific vaginal fascia, while DeLancey described a suburethral fascia anterior to the AVW, contributing to urethral support [13]. The entire vagina is suspended like a hammock from the PCF and, laterally, the EF and tendinous arcs (Figs. 1 and 2).

Pubocervical fascia

The PCF is composed of collagen and smooth muscle, constituting the anterior vesical fascia under the bladder base and neck. It is sometimes referred to as Halban's fascia, described as a membrane between the neck of the bladder and the cervical ring



1. pubococcygeus muscle
2. puborectal muscle
3. arcus tendineus fascia pelvis
4. pubourethral ligament
5. pubovesical ligament
6. endopelvic fascia

Fig. 2 Lateral view of the median part of the pelvic floor (adapted from JPH Neidhardt) showing relations of arcus tendineus fasciae pelvis with pubovesical ligament (courtesy of Eric Voiglio, with permission)

[14]. Anteriorly, it is supported by the pubovesical ligaments and posteriorly by the cervical ring (peri-isthmic collagen condensation), as described by Petros; laterally, it is supported by the EF and ATFP (Fig. 3) [8, 10]. According to Petros, given the lack of any transverse ligament in the mid-region, the PCF is the main ventral support structure of the bladder [8, 10].

Tendinous arcs

Tendinous arcs are fibrous reinforcements contributing to anterior vaginal support (Fig. 1). Two collagenous connective condensations are identified histologically as tendinous and ligamentous structures but are much more highly organized than the EF.

Arcus tendineus levator ani The ATLA is a collagenous thickening comprising the lateral insertion of the pelvic aponeurosis and the LAM plane on the internal side of the internal obturator muscle. It runs from the anterior insertion on the posterior pubis to the posterior insertion near the ischial spine (Fig. 1). This type of muscular insertion is unparalleled in the entire organism, as striated muscles generally have bone insertions via tendons or insert directly via fleshy fibers.

Arcus tendineus fasciae pelvis The ATFP is a dense linear collagenous reinforcement constituting the LAM's insertion onto the lateral PCF. [15] It is more inferomedial than the ATLA (Fig. 2). Insertion is on the pubovesical ligament medially and on the ATLA dorsolaterally 1.5±0.4 cm above the summit of the ischial spine [16–18] (Figs. 1 and 2). The two ATFPs act as suspension cables on either side of the vagina and bladder, mooring them to the pelvic wall (Fig. 1). Uni- or bilateral detachment from the tendinous arc induces pelvic imbalance that may lead to lateral cystocele.

Endopelvic fascia The EF is a layer of connective tissue covering the LAM and internal obturator laterally and the pelvic viscera medially (Fig. 1) [9, 13, 19]. The inferior lateral vaginal region, or paravagina, is also known as the paracolpium, constituting the suburethral vaginal wing [9]. The term fascia is not well defined, leading to terminological and functional confusion despite the international anatomical terminology (*Terminologia Anatomica*) [12]. The EF is a loose connective tissue network consisting of a variable layer of collagen–elastin, smooth-muscle cells, and neurovascular pedicles, and is much less organized than skeletal ligamental connective tissue [20, 21]. At the pubic symphysis, it thickens into a true pubourethral ligament in the middle third of the urethra up to the ATFP pubic insertion [10, 22].

Levator ani muscle The LAM is the essential component of active support (Fig. 1). It comprises two distinct parts: anatomical and functional. A puborectal bundle, with a thick, solid, medial sphincteral region of pubic origin, terminates at the external anal sphincter (Fig. 1); it consists of an anteriorly concave muscular strap bordering the urogenital hiatus, playing an essential role in pelvic organ support and hiatus closure [2, 23]. The pubovisceral bundle is a dynamic elevator comprising three muscles (puboperineal, pubovaginal, puboanal), clearly identifiable on magnetic resonance imaging (MRI) [24, 25] and originating in the posterior pubis. The pubovaginal muscle inserts to the lateral inferior vaginal wall at midurethra and is part of the periurethral complex [26, 27]. The lateral part of the pubovisceral bundle joins the coccygeus and iliococcygeus muscle fibers, forming the levator plate around the anorectal angle [28, 29].

Superior vaginal support

The USL and cardinal (CL) ligaments hold the uterus and upper third of the vagina in the pelvic space above the levator plate. USLs are clearly identifiable but CLs less so.

Uterosacral ligaments USLs are mesenchymatous structures composed of connective tissue, smooth muscle, and an autonomic nervous system originating in the inferior hypogastric plexus [30]. It is generally agreed that they originate from the

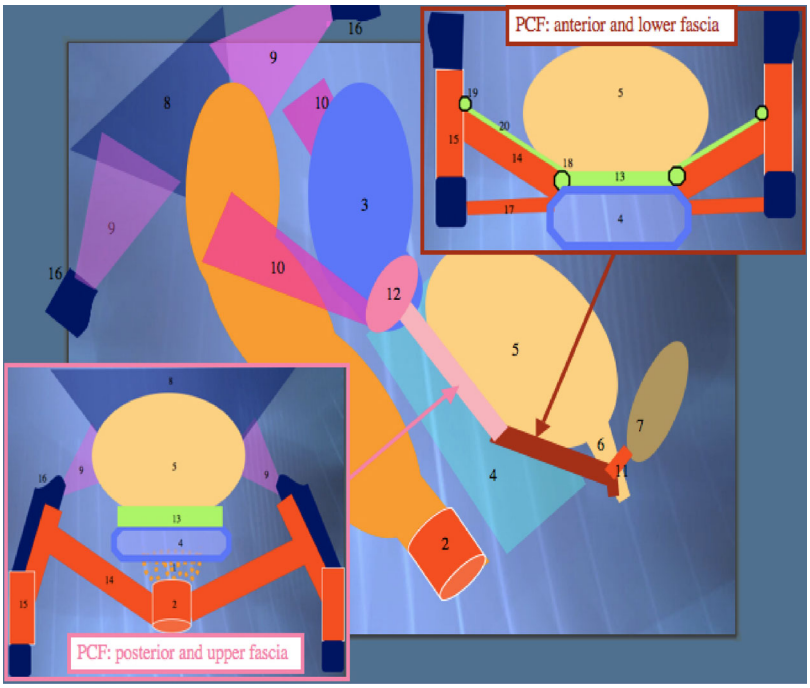


Fig. 3 Simplified diagram of bladder-support structures (pubocervical fascia; anterior and posterior segment)

1: Rectum, 2: Anal canal, 3: Uterus, 4: Vagina, 5: Bladder, 6: Urethra, 7: Pubic bone, 8: Coccygium, 9: Sacrospinous ligament, 10: Uterosacral ligament, 11: Pubourethral ligament, 12: Cervical ring, 13: Pubocervical fascia, 14: Levator ani muscle, 15: Internal obturator muscle, 16: Ischial spine, 17: Perineal membrane, 18: ATFP, 19: ATLA, 20: Endopelvic fascia

presacral fascia adjacent to S2-S3-S4 but without direct bone insertion. Insertion is to the cervical ring and posterosuperior vaginal fornix angles at the cervical orifice, constituting a front-to-back fan-shaped architecture. Mean length is 8.7 cm [95% confidence interval (CI) 7.5–10.0]; the right USL is thicker than the left due to the mesosigmoid [31].

Cardinal ligaments CLs are "mesenchymatous-like" fascial structures sheathing the internal iliac pedicles, with distal insertion to the anterosuperior cervical neck and PCF. [32] In mid-vagina, they are in continuity with the AVW and PCF. Histologically, Range et al. found no structures resembling skeletal ligaments but, rather, areolated connective tissue with blood vessels and nerve plexus better defined at the lateral edges of the cervix and vagina than toward the pelvic floor [33]. According to Salman et al., alterations in CLs include loosely arranged connective tissue fibers and less-dense extracellular matrix, with sparsely distributed fibroblasts. Under electron microscopy, collagen fibers were more sparsely distributed and thicker in women with prolapse [34].

Posteroinferior vaginal support

Perineal body The perineal body (PB) is a dense, medial, pyramid-shaped fibromuscular mass lying between the internal anal sphincter and the inferior third of the posterior vaginal wall. Depending on the report, it is 15- to 30-mm long anteroposteriorly [35, 36]. *Gray's Anatomy* pointed out that "central nucleus of the perineum" is an inappropriate term, as the PB is neither central nor tendinous [35]. It is composed of connective tissue, elastin, and smooth muscle, distributed irregularly within the body but becoming almost horizontal toward the rectovaginal septum [36]. The PB is thus interconnected with DeLancey's level 1 and the pericervical ring through the rectovaginal septum [9]. In a series of 46 dissections, Shafik described a superior plane comprising external anal sphincter and puborectalis, and an inferior plane comprising superficial and deep transverse muscles [37]. Contraction of the external anal sphincter lowers the ligamentous–fascial hammock, moving the pelvic organs downward [36]. Soga et al., in a histotopographic study, described a lateral PB extension, LEX, with connections to the internal anal sphincter and vaginal smooth muscles [36], composed of fibromuscular tissue and smooth muscle in amounts larger than in the PB itself, and they change with age. Soga's LEX may thus be the level 3 perineal membrane described by DeLancey [9, 36].

Biomechanical alterations

Several connective tissue biomechanical properties have been reported to contribute to anterior vaginal support in prolapse

[38–42]. Uniaxial traction tests on pelvic tissue determined mechanical behavior [42], which differed between vaginal tissue and pelvic organs: rigidity differed between anterior and posterior vaginal walls, whereas the bladder showed anisotropic behavior [42]. The inferior AVW, in contact with the bladder, is subject to strong pressures, in line with viscoelastic behavior [42–45]. The entire AVW represents a hyperelastic system supporting the bladder–urethra apparatus [42, 43]. Pelvic ligaments also differ in behavior, but it is now established that USLs are the most rigid, accounting for their prime role in supporting the vaginal apex, with consequent apex prolapse in case of USL failure [3, 43, 46].

Analysis of various anatomic theories

Various anatomic theories of pelvic organ support have been described. The earliest was by Enhörning, which explained urinary incontinence as the urethra sliding out of the abdominopelvic pressure enclosure; this theory, devoid of anatomic foundation, has been refuted [7]. A literature review retrieved three main theories of prolapse: Petros's integral theory, DeLancey's anatomic theory, and the more recent proposal by Tansatit et al. [8, 9, 11].

Petros's integral theory

Petros et al. described a fixed suburethral point as the keystone to stress urinary incontinence and basis for the development of suburethral slings [8]. Their integral theory explains pelvic symptoms mainly by laxity of vaginal-wall connective tissue and ligamentous–fascial structures. The theory supposes a sagittal ligamentous–fascial support extending from the posterior pubis in front to the S3/4 sacral concavity behind, comprising pubourethral ligaments and the PCF and inserting into the cervical ring. This creates a vast sagittal hammock on which, from front to back, lie the urethra, bladder, uterus, and upper rectum between the two USLs. To this is added the pelvic muscular complex, a dynamic pelvic organ support forming the "wings" of the hammock [8, 10]. Pelvic–perineal musculature efficacy depends on system integrity, and according to Petros, ligament–fascia failure has two consequences: destabilizing pelvic–perineal musculature and weakening pelvic organ support; together, this induces organ prolapse and dysfunction, with loss of ligament–fascia and muscle support. Petros explained pelvic organ function by the interaction of organ displacement secondary to muscle-group contraction and demonstrated that muscle force acting on the vagina substantially contributes to cystocele onset [8, 10]. He distinguished two vaginal regions with nine indispensable anatomic structures and three muscle forces underlying pelvic organ suspension. In this theory, all pelvic symptoms are due to anatomic lesions. Thus, USL hyperlaxity destabilizes the pubourethral ligaments and PCF, with risk of both cystocele and urinary incontinence: a visceral symptom may be due to ligamentous–fascial disorder remote from the symptomatic organ.

DeLancey's theory

DeLancey laid the foundations of his anatomic "hammock theory" in 1992 [9]. His understanding of pelvic–perineal pathophysiology derived from anatomic dissection and, later, MRI and biomechanical modeling [47]. The theory is based on "paravaginal support," comprising fascias and ligaments supporting the mid-region of the vaginal wall at the pelvic wall [9]. He described AVW staging in three levels of pelvic support on a transverse plane [9, 13]. Level 1 is described as supporting the vagina and cervix by posterior sagittal USL suspension: here, paracolpium fibers are almost vertical, leading back toward the sacrum. Level 2 comprises the vagina's lateral connections to the ATFP, supporting the mid-third of the vagina: the vagina is moored laterally to the ATFP and superior ATLA and maintained transversally between bladder and rectum. Level 3 comprises connections between the perineal membrane and PB fascias, supporting the lower third of the vagina and playing an essential role in pelvic statics [9]. These three levels are seen in recent MRI studies [39]. Defects in one or more levels may induce cystocele. The fascial attachments connect periurethral tissue and AVW to the ATFP, while the muscular attachments connect periurethral tissue to the pubovaginal muscle. This musculofascial support constitutes a hammock—formed by vaginal wall and EF connected to the ATFP—on which the urethra lies and is compressed under abdominopelvic pressure [46]. According to DeLancey's theory, the suburethral support system contributes to urinary continence [13].

Tansatit's theory

The third, most recent, theory is based on two mechanical structures: a support system, mainly comprising LAM, ATFP, and an adipose cushion filling the ischioanal fossa and ensuring pelvic organ functional interdependence and pelvic stability; and a system of EF suspension on the ATFP, playing a major role in support [11]. The anatomophysiological role of these pelvic spaces filled by connective tissue is not clearly established [11].

Anatomic mechanisms of types of cystocele

We inventoried the above bladder support structures according to Petros's and DeLancey's anatomic/functional theories (Table 1) [8, 9], distinguishing three types of cystocele: apical, medial, and lateral or paravaginal; a combined mediolateral defect is also possible. Each type can be identified on clinical examination.

Table 1 Anatomic structures involved in the mechanisms of medial, apical, and lateral cystocele based on the theories of Petros and of DeLancey

	Medial cystocele		Apical cystocele		Lateral cystocele	
Anatomic structures	PCF	Endopelvic fascia	Cervical ring	Uterosacral ligament	ATFP	ATLA
Petros	Yes	No	Yes	No	Yes	No
Delancey	Yes	Yes	Yes	Yes [1]	Yes	Yes

PCF pubocervical fascia, *Cervical ring* cardinal ligament insertion, *ATFP* arcus tendineus fasciae pelvis, *ATLA* arcus tendineus levator ani,
[1] Origin of uterosacral ligaments

Apical cystocele

Apical cystocele relates to anatomic defect in the superior third of the vagina. The main elements involved are EF and DeLancey's level 1 ligament complex (Table 1). CLs and USLs suspend the uterus and superior vagina like a suspension bridge [8, 10]. This ligament complex directly impacts cervical statics and vaginal-dome suspension, but CLs seem unlikely to play a major role in support: they consist of a collagenous layer corresponding to less-well-defined EF structures, the support function of which is controversial [8, 10]. In Petros's theory, apical cystocele is basically caused by CL defect and complete PCF detachment from the cervical ring [8, 10]; in DeLancey's theory, it is due to CL detachment from the cervical ring and of USL origin [48]. The pubovaginal muscle is also involved in vaginal apex prolapse [9, 13, 48, 49].

Medial cystocele

The bladder lies on the PCF of the AVW. Medial or posterosuperior cystocele relates to a sagittal suspension system defect in the upper part, at the cervical ring. The defect may be confined to the PCF or, often, associate sagittal USL defect. PCF tear or thinning induces cystocele, giving the vaginal epithelium a shiny aspect [8, 9]. DeLancey, comparing cystocele assessed on the Pelvic Organ Prolapse Quantification system (POP-Q) and anatomic lesions assessed on MRI, found a correlation between high-grade cystocele and cervical-ring defect: 75% of high-grade cystoceles were due to defects in USLs and the PCF at its uterine isthmus insertion [50]. Thus, the more severe the cystocele, the larger the number of severe apical suspension lesions [50]. DeLancey's theory adds a further anatomic element to the mechanism of medial cystocele, presuming a major impact of EF (Table 1): degradation of not only the PCF but also EF leads to medial cystocele. For Petros, the pelvic symptoms induced by medial cystocele imply that PCF lengthening at the cervical ring is the key factor responsible [8, 10]. Petros claims a causal relationship between bladder-base lengthening due to medial cystocele and urge incontinence induced by detrusor urinae baroreceptor stimulation [10].

Lateral cystocele

Lateral cystocele results from both ligamentous–fascial and pelviperineal muscular defect [9, 13, 49]. The ligamentous–fascial defect involves the inferior and anterior PCF elements constituting a transverse hammock: from outside to inside, the ATLA, the pelvic aponeurosis covering the LAM, and the ATFP. Degradation of any of these results in bladder-support defect, manifesting clinically as lateral cystocele [51, 52]. Richardson et al., analyzing high-grade cystocele, attributed >90% of paravaginal defects to detachment of the PCF and vaginal wall at the ATFP [53]. Lateral cystocele can be accounted for by anatomic imbalance between AVW and ATFP, which seems to be the essential lateral ligamentous support structure [8–10, 53, 54]. For DeLancey, the ATLA also contributes to lateral cystocele (Table 1). On clinical examination, the PCF is thickened, with transverse folds, indicating uni- or bilateral paravaginal defect [54]. Pelvic–perineal muscular-floor defect results in hiatus genitalis, depriving the fascial structures of inferior support during abdominal effort [2, 9, 49].

Synthesis of various theories to determine anatomical lesions specific to each clinical type of cystocele

There are no precise data in the literature assessing anatomic theories of urogenital prolapse. It is difficult to compare Petros's and DeLancey's theories, as they are more complementary than contrasting. Moreover, they are in constant evolution, with advances in clinical experience, surgical correction, and—above all—dynamic MRI [38, 49, 55, 56]. DeLancey first based his theory on anatomic models; progress in MRI techniques shed new light on the anatomopathological causes of cystocele and the variations in the pelvic structures involved [38, 49]. On MRI, sagittal AVW lengthening brings the lowest end of the cystocele under the hymen line and PB [38, 49]. Under atmospheric pressure, this AVW segment causes the discomfort associated with cystocele.

According to DeLancey, the pelvic–perineal muscular floor supports the AVW and is essential in the etiopathogenesis of cystocele. The pubovisceral bundle elevates and closes the urogenital hiatus, exerting an anterior force. LAM avulsion

verticalizes the levator plate, enlarges the hiatus, reduces AVW support, and induces cystocele [38, 57]. LAM lesions are more frequent in cases of prolapse [58]. For DeLancey, the ligamentous–fascial defect is real, but the prime mover is the vulvar gap, which increases AVW exposure to atmospheric pressure. Thus, severity and functional impact of cystocele depend on PCF lengthening by ligamentous–fascial degradation and on the hiatus genitalis induced by degradation of the pelviperineal muscular floor supporting the PCF.

DeLancey's theory is based on transverse, level-by-level compartments, with a certain interdependence between levels 1 and 2: level 1 is a posterior sagittal suspension while level 2 stretches to the pelvic side wall (ATFP), and these two levels are linked through the cervical ring belonging to the apical suspension. There is continuity between level 3 connective tissue and level 2 via the EF. Petros's theory is based on global sagittal and lateral suspension by the ATFP and anterior subvesical fascial base, but it is not founded on anatomic reality. According to Petros, bladder support is provided sagittally by the pubourethral ligaments, the PCF, and the cervical ring. An anatomic defect in Petros's anteroposterior trampoline would impact all pelvic organs [8, 10]. This integral theory is based on functional symptoms, integrating the pathophysiological mechanisms of muscle and ligament movement [8, 10]. Petros proposes a direct relationship between anatomic lesion and pelvic symptoms, which may be at different levels; the three muscle forces require solid pubourethral ligament and USL insertions, and degradation of this ligamentous–fascial system induces cystocele [8, 10].

Recent clinical and radiological studies revealed the importance of apical support [59, 60]. Clinically, there was a strong correlation between the anterior compartment (point Ba in the POP-Q classification) and apex (point C) ($r=0.86$; $p<0.0005$), especially in high-grade cystocele [59–61]. Dynamic MRI confirmed this strong correlation between anterior and medial prolapse ($r=0.73$) [62]. These findings are relevant to surgical treatment of cystocele accompanied by uterine prolapse or associated with primary degradation of anterior support structures. Petros demonstrated that vaginal-dome repair can resolve cystocele temporarily, but with a tendency for medial-compartment recurrence if the PCF is damaged [8, 10]. The PCF, supporting the bladder, should be considered as a link between anatomic structures in order to have a clear understanding of apical, lateral, and medial cystocele. It includes a sagittally supported posterosuperior segment and laterally supported anteroinferior segment (Fig. 2). The anteroinferior segment is located at the level of the ATFP and the lateral PCF insertion at the pelvic aponeurosis. The posterosuperior segment is mainly supported by the USLs, constituting the essential support of the posterosuperior vesical base. In parallel with clinical findings, surgical USL sectioning leads to descent of the uterus, cervical ring, and posterosuperior vesical base. PCF degradation may induce not only medial but also apical cystocele by avulsion from the cervical ring [8, 10].

Biomechanically, DeLancey's team showed that LAM degradation plays a major role in high-grade cystocele [2, 49]. A theoretic analysis explained cystocele amplitude by combined degradation of pubovisceral muscles and apical support [2, 4, 49]: the more severe the cystocele, the greater the number of severe apical suspension system lesions. Pelvic organ support requires dynamic interaction between the LAM and the connective tissue fixing the organs to the pelvic wall [2, 49]. On this 3D biomechanical model, degradation of 80 % of the pubovisceral muscles combined with degradation of 90 % of the ligamentous complex leads to more severe cystocele, with >2 cm exteriorization from the hymen ring [2, 50]. Petros's theory also integrates a biomechanical dimension: in case of cystocele, vaginal-wall distension increases tension in the vaginal tissue between the two ATFPs. The lower density of the degraded tissue combined with increased pressure increases vaginal mobility in lateral cystocele [8, 10].

There are some limitations to the review presented here due to the multiple theories and controversial data available in the literature [8, 10]. Nevertheless, according to the anatomic theories, it may be hypothesized that anteroinferior bladder-base suspension is transverse and posterosuperior PCF support is longitudinal.

In summation, three types of cystocele are to be distinguished according to the anatomic structures involved: apical, involving lesion of the posterosuperior PCF support structures (cervical ring, USL, and, in some cases CL, corresponding to DeLancey's level 1); lateral, involving lesion of lateral PCF support structures (transverse hammock failure, DeLancey's level 2); and medial, involving isolated PCF lesion [8–10, 13, 49, 60]. DeLancey's decisive contribution promises improved understanding of the mechanisms underlying cystocele according to the anatomic lesions seen on MRI [50]. The strong correlation between high-grade cystocele and cervical-ring defect established by DeLancey can explain the efficacy of apical repair in isolated level 1 defect or apical repair associated with paravaginal repair for level 1 and 2 lesions. On MRI, the specific lesion structure underlying each type of cystocele can be detected in detail, opening the way to an improved treatment strategy with refined indications and surgical repair techniques.

Perspectives

Better knowledge of AVW support mechanisms should improve diagnosis of the different types of cystocele and enable a better approach to the principles of treatment. In the near future, it would be interesting to integrate the mechanical properties of each anatomic structure involved in each type of cystocele in a 3D digital model. Such an anatomic model

could help explain the pathophysiological mechanisms involved in each anatomic theory.

Conclusions

Improved anatomic knowledge of the pelvic system improves understanding the pathophysiological mechanisms of cystocele. This review article updates the various current anatomic theories of pelvic organ support. These theories are actually quite close and complementary to each other but differ in mechanisms. Petros's theory drew up the main contours of the anatomic organization of pelvic support structures, with a direct relation between ligamentous–fascial lesions and pelvic symptoms. DeLancey initially founded his theory on anatomic models then demonstrated the interest of comparative anatomopathologic analysis on MRI. It would thus seem that anatomic study on pelvic–perineal MRI and comparison with clinical examination is currently the most effective means of precisely exploring anatomic lesions associated with cystocele. According to these two theories, there are three types of cystocele: apical, involving lesion of the posterosuperior PCF support structures; medial, due to isolated PCF lesion; and lateral, due to transverse hammock failure. Modeling pelvic organ support structures would both improve knowledge of pelvic statics and provide a teaching aid. To be valid, however, the model must integrate variations in anatomic structures according to location and mechanical properties. An improved understanding of pelvic functional anatomy provided by modeling should provide a tool for better understanding of physiological genital movement, thus refining the surgical correction of cystocele.

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