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What's new in the functional anatomy of pelvic organ prolapse?

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Abstract

Purpose of Review—Provide an evidence-based review of pelvic floor functional anatomy related to pelvic organ prolapse.

Recent Findings—Pelvic organ support depends on interactions between the levator ani muscle and pelvic connective tissues. Muscle failure exposes the vaginal wall a pressure differential producing abnormal tension on the attachments of the pelvic organs to the pelvic side-wall. Birth-induced injury to the pubococcygeal portion of the levator ani muscle is seen in 55% of women with prolapse and 16% of women with normal support. Failure of the connective tissue attachments between the uterus and vagina to the pelvic wall (cardinal, uterosacral, paravaginal) are strongly related with prolapse (effect sizes ~2.5) and are also highly correlated with one another ($r \sim 0.85$). Small differences exist with prolapse in factors involving the vaginal wall length and width (effect sizes ~1). The primary difference in ligament properties between women with and without prolapse is found in ligament length. Only minor differences in ligament stiffness are seen.

Summary—Pelvic organ prolapse occurs due to injury to the levator ani muscles and failure of the connections between the pelvic organs to the pelvic sidewall. Abnormalities of the vaginal wall fascial tissues may play a minor role.

Keywords

Pelvic organ prolapse; Pathophysiology; Pelvic floor anatomy; Levator ani muscles; Endopelvic fascia

Introduction

“There are in fact two things, science and opinion;

The first begets knowledge, the second ignorance.”

Hippocrates, Law, Book IV, circa 395 B.C.E.

Our understanding of the pathophysiology of pelvic floor disorders is in transition. We are moving from discussing empirically derived hypotheses based on gross anatomy and physical examination to testing mechanistic hypotheses by comparing the structure and function between living women with and without prolapse using MRI, ultrasound and functional pelvic floor testing. This marks the transition from opinion to science. The framework for any such scientific discussion rests in having an overall disease model whose individual elements can be subjected to scientific experiments to determine whether or not they are true.

How do the pelvic floor structures work together in preventing prolapse?

The longest-running debate about the etiology of pelvic organ prolapse concerns whether muscle or connective tissue is the most critical element in pelvic organ support. Modern biomechanical analysis has shown that asking this question is like trying to decide which blade of a pair of scissors is the most important. The muscles and connective tissues involved in maintaining the pelvic organs in their normal locations act together to provide support in much the same way that the ventricles and valves are both needed for the heart to propel blood forward.

Normal pelvic organ support is provided by the interaction between the levator ani muscles and the connective tissues that attach the uterus and vagina to the pelvic sidewalls. The levator ani muscles hold the pelvic floor closed and provide lifting and closing forces to prevent pelvic floor descent [Figure 1].¹ In this situation, the pressures in the anterior and posterior compartments are equal and balanced, canceling each other out. When the muscles are damaged or weakened, the hiatus in the levator can be pushed open and the pelvic organs descend so that one or both of the vaginal walls protrudes below through the levator hiatus. When the vaginal walls (or the uterus) descend below the hymen (the level where the levators can act), it comes to lie between atmospheric and abdominal pressure. The pressure differential that is created acting on the vaginal wall(s) creates a downward force that places abnormal stresses on the tissues that attach the uterus and vagina to the pelvic walls. Conversely, if the connective tissues are too lax to hold the organs in alignment so that they descend below normal levator ani muscles, the same imbalance in pressure can occur.

This pressure differential has important implications for the stresses placed on the support system. The force that results from pressure being applied to the vaginal wall is directly proportional to the amount of vaginal wall that is exposed to atmospheric pressure. There is a strong relationship between exposed vaginal wall length and how far the bladder descends ($R^2=0.91$).² Initial descent while the vaginal wall is still in contact with the posterior vaginal wall does not increase the amount exposed to a pressure differential, but when the bladder descends 4 cm below its normal position, exposed vaginal wall length subjected to the difference in pressure increases significantly, with a 2 cm increase in exposed AVW length for every additional 1 cm of drop bladder location. Exposed vaginal wall length is also highly correlated with hiatus diameter ($R^2=0.85$), suggesting why the status of the levator that is responsible for closing the hiatus is so important in prolapse.

Biomechanical simulation

3D biomechanical simulation has added significant insights by allowing individual components of the support system to be altered in a way not possible in human subjects.^{3–5} This type of analysis has demonstrated definite interactions between connective tissue and levator factors interact [Figure 2].^{6,7} When muscle damage and ligament failure were combined, a larger cystocele formed compared to what happened with impairment of either element alone. Levator ani muscle impairment caused a larger urogenital hiatus size, longer length of the distal vagina exposed to a pressure differential, larger apical descent, and resulted in a larger cystocele size. Interactions between the anterior and posterior compartments have also been demonstrated⁸ that help explain postoperative development of new prolapse in the opposite compartment, despite its support appearing normal prior to the operation.⁹

3D Stress MRI Findings; evaluation of connective tissue failure sites

3D Stress-MRI has provided a technique capable of producing data to document the location of pelvic organs during maximal Valsalva in living women with and without prolapse.^{10,11} This allows hypotheses to be tested about the relative contribution of apical descent, paravaginal defect size, and changes in vaginal width and length that would be associated with the connective tissue fascia of the pelvis.¹² 3D models of the vaginal wall and pelvic bones at maximal Valsalva are made so that vaginal dimensions and location relative to the bony pelvis can be quantified and compared between women with and without prolapse [Figure 3]. Differences in the location of the uterus and lateral vaginal margins reflect changes in the connective tissue attachments (cardinal, uterosacral, paravaginal). Differences in the width and length of the vagina reflect changes in the vaginal wall and its associated fascia.

With this technique, the paravaginal distance between the edge of the vagina and the normal location of the arcus tendineus fascia pelvis is measured at 5 equally spaced locations along the length of the vagina as well as the vaginal width at these locations. The location of the cervix and the length of the vagina are also quantified. Substantial differences exist between each of the 5 paravaginal distances between cases and controls with effect sizes between 2.2 and 2.8 and also in apical support (effect size 1.7) [Figure 4]. There is also a strong correlation between apical descent and paravaginal gap size ($r = 0.77–0.93$) that is greatest in the segments closest to the apex, indicating that apical descent and paravaginal gap are essentially two components of the same phenomenon. This is consistent with clinical observations showing a strong relationship between apical descent and cystocele size.^{13,14} Regarding the assessments of the vaginal wall, vaginal length was 24% longer in women with prolapse as has previously been shown.¹⁵ Vaginal width was only slightly different at just one of the 5 locations. Therefore, the primary differences between women with and without prolapse are seen in the uterovaginal connections to the pelvic sidewalls by cardinal, uterosacral and paravaginal attachments and are highly correlated with one another.

LEVATOR ANI MUSCLE

Levator ani muscle injury associated with prolapse

The hypothesis that levator ani muscle injury is an important cause of pelvic organ prolapse put forth by Halban and Tandler in 1907¹⁶ is now proven. Major levator injury (injury affecting more than half of the muscle bulk) is found in 16% of women with normal pelvic organ support but in between 34 and 55% of women with prolapse, thus proving the association between levator ani muscle injury and pelvic organ prolapse.^{17,18} This injury specifically involves the pubovisceral portion¹ of the muscle but does not involve the puborectal portion¹⁹, a point made somewhat confusing because some authors have used the term puborectal for the injured portion of the muscle before this distinction was clarified.

This levator injury occurs in 13–36% of women who deliver vaginally.^{20–22} It involves the origin of the pubovisceral muscle from the pubic bone.^{23,24} The pubovisceral muscle's attachment to the pubic bone is a thin and transparent aponeurosis that arises tangentially to the pubic periosteum.²⁵ The thin nature of the attachment of the aponeurosis makes it subject to injury when the forces in the fiber direction exceed the strength of the muscle origin.

As one might expect with a muscle injury, injury severity matters. Analysis of 503 subjects from case-control studies of prolapse reveals that only cases where 50% or more of the muscle is missing are associated with prolapse.²⁶ This cutoff has been independently confirmed using ultrasounds.¹⁸ Levator injury involving Major Loss is associated with a 40% reduction in the force that the levator ani muscles can generate during a pelvic muscle contraction.¹⁷

Levator Ani Lines of Action

The action of pelvic floor muscles is determined by the direction of its muscle fiber, the muscle shape, and points of attachment. The angle of each subdivision has been established in living women²⁷ [Figure 5] and forms the basis for understanding the mechanical effect of muscle contraction and tone. The pubovisceral muscle fibers course 41° above the horizontal in the standing posture. By contrast, the puborectal muscles actually act below the horizontal (–19°). The anal sphincter is also below the horizontal in its line of action.

The large difference (60°) in mean fiber angle between the pubovisceral and the puborectal shows that they have two different mechanical actions [Figure 5B]. The diagonal vectors of the muscles can be resolved into two physiological vectors. The first, a vertically oriented “lifting” vector, acts against gravity lifting the perineal structures and can be attributed to the pubovisceral muscle. Clinically, descent of the perineal structures is found in women with prolapse and is associated with injury to the pubovisceral portion of the levator either with or without prolapse.^{28,29} Both the puborectal and pubovisceral muscle have a horizontal component. This vector helps to develop a “closing” force, acting in a horizontal direction so as to close the levator hiatus. This horizontal action creates a vaginal high pressure

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¹We have chosen the term pubovisceral coined by Larson rather than the traditional pubococcygeal term, because it more accurately reflects the origins, insertions, and function of the muscle.

zone.^{30,31} The small vertical puborectal component actually acts in a downward (caudal) direction so that it has no “lifting” action.

Connective tissue support

The connective tissues of the pelvis connect the vagina and uterus laterally to the pelvic walls as outlined in Figure 1. They can be divided into three levels (I, II, III), reflecting the changing nature of support³² [Figure 6]. In Level I, the cervix and upper third of the vagina are attached to the pelvic walls by mesenteric structures that suspend these organs (cardinal and uterosacral ligaments). In Level II the middle third of the vagina it is attached laterally to fascial structures (arcus tendineus fascia pelvis, AFTP, or fascial arch and a similar posterior structure).³³ Distally in Level III, the vagina is fused with the surrounding structures, namely the levator ani muscles and the perineal body.

Anatomy

The cardinal ligaments, often called the “transverse cervical ligaments,” are neither transverse nor ligaments.³⁴ There is no band of dense regular connective tissue that connects the uterus laterally to the pelvic walls.³⁵ These structures are bilateral mesenteries going to the pelvic organs and consist of vascular, neural, lymphatic and areolar tissue that consist of fat and a delicate meshwork of connective tissue between different elements.³⁶ Rather than being transverse, they are relatively vertical in orientation in the standing posture.³⁷ The cardinal ligament tends to be more vascular in the cranial portion with more neural components caudally. The vascular section represents the branches of the internal iliac vessel going to the uterus and vagina with their surrounding connective tissues, whereas the neural section contains parts of the inferior hypogastric plexus.

Three-dimensional orientation and lines-of-action—In the standing posture, the cardinal ligaments are in a relatively vertical orientation in the upright posture—a logical direction for them to resist downward forces³⁸ [Figure 7]. The uterosacral ligaments are more dorsally directed towards the sacrum³⁹ in an orientation where it can prevent the uterus and upper vagina from sliding down the inclined plane of the levator plate towards the opening in the levator muscles through which prolapse occurs.

The lines-of-action for the cardinal and uterosacral cardinal ligaments in normal women are 18° from the cranio-caudal body axis with an average length of 5.7 cm. The deep uterosacral ligaments are dorsally directed and 92.5° from the body axis and average 2.7 cm. Using the lines-of-action for the cardinal and uterosacral ligament and the inclination of the levator plate (the portion of the levator ani muscles in the midline behind the rectum), it is possible to make a theoretical calculation of the tensions on the two ligaments for a given 1N unit load. Analysis shows a cardinal ligament load that is 52 % larger than the load on the uterosacral ligament.

Ligament alignment changes with prolapse—The cardinal and uterosacral ligaments change their length and angle in response to the forces applied to the pelvic organs during increases in abdominal pressure.⁴⁰ [Figure 7]. The cardinal ligament is 20% longer at rest in women with prolapse compared to women with normal support (71 mm vs. 59 mm). By

contrast, the deep uterosacral ligament at rest has a similar length in women with prolapse and normal support (38 mm vs. 36 mm, $p=0.8$ respectively). During a maximal Valsalva maneuver, the cardinal ligament lengthens by 30 mm in prolapse as opposed to the 15 mm change in women with normal support. During Valsalva, deep uterosacral ligament elongation was 15 mm vs. 7 ± 4 mm ($p=0.09$). By contrast, the cardinal ligaments remain at roughly the same angle relative to the body axis from rest to strain, while the deep uterosacral ligament angle was significantly different from the cardinal ligament angle change.

Ligament properties (stiffness, length, and viscoelasticity)

The testing techniques used to study the mechanical properties of the apical ligaments are profoundly affected by how they are tested. Classical *in vitro* experiments on small excised ligament pieces suggest that it would take 26 N (or 5.8 lbs.) to stretch the uterosacral ligament by 1mm.⁴¹ In vivo studies of the ligaments in living women, by contrast, reveal that 1lb of force moves the uterus 12 mm.⁴² Using these data, it can be estimated that vivo stiffness is 650 times less than seen in traditional testing.⁴³ Preconditioning is performed during traditional material properties testing wherein the tissue is tensioned a few times before testing is measured.⁴⁴ Since the cardinal ligaments have a lattice-like arrangement³⁶, it seems likely that this preconditioning straightens the fibers so that they are all aligned, making the material properties much stiffer than would be seen when loaded in vivo, where the lattice work is open as it is during life. In addition, after death, smooth muscle tone is lost and so reduces the tissues resiliency.

Studies of ligament properties that evaluate *in vivo* ligament properties in women with and without prolapse reveal that stiffness only explained 19 % of cervix descent as evaluated clinically as POP-Q point C during maximal Valsalva.⁴⁵ Ligament length as judged by cervix location at rest and during maximal traction were much stronger predictors of POP-Q, with 38% and 46% prediction respectively. This suggests a long ligament, rather than a lax ligament, is the primary factor.

The relationship between these mechanical tests and the normal physiological location of the cervix can be studied by comparing what happens during Valsalva maneuver in MRI of known Valsalva pressure with measurements made in the OR under known traction. Only a small traction force (~3 oz. [90 g]), the weight of a large chicken egg, was required in the OR to reproduce the physiologic uterine movement seen during MRI. This is consistent with clinical data that show that the cervix location is, on average, 3.5 cm lower in the OR under traction vs POP-Q exam in clinic during maximal Valsalva.⁴⁶ The large forces used by gynecologists that average 8 lbs,⁴⁷ is a traction force 40 times higher than the force required in the OR to achieve maximal physiologic uterine descent that is seen during maximal Valsalva in the MRI.

Viscoelasticity—These tissues also exhibit the property of hysteresis due to viscoelasticity. This property in which repetitive loading can result in material property changes, in which an elongation under a constant load is resisted less over time, indicates that the types of repetitive loading over time can result in increasing descent. A more

complete understanding of ligament mechanics depends on recognizing that the ligaments are not elastic structures. These properties have been demonstrated in both the cardinal and uterosacral ligaments in animals in vitro⁴⁸ and in human in vivo.⁴⁹

These findings have implications for the repetitive loading that would occur during daily activities and may help to explain changes over time. These findings suggest a much richer and more complex situation than the classical cable and load mode.

Conclusion

Pelvic organ prolapse is caused by combined failure of pelvic floor muscle and connective tissue. Injury to the pubococcygeal portion of the levator ani muscle is seen 40% more often in women with prolapse. Connective tissue failure occurs in the attachments of the vaginal wall to the pelvic sidewall. The actual vaginal wall and its fascia have only minor differences in women with prolapse. The exact nature of each anatomical component of the support mechanism is now being clarified so that the specific lines of action, tissue properties and interactions can be documented and compared between women with and without prolapse.

Acknowledgments

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Key Points

- Women with pelvic organ prolapse are 3 times more likely to have levator ani muscle damage than women with normal supports
- Connections between the uterus and vagina and the pelvic sidewalls (Cardinal, uterosacral, and paravaginal) are the primary sites of connective tissue failure.
- Apical and paravaginal failures are highly correlated with one-another suggesting that they reflect two aspects of a single phenomenon.
- There are not major differences in the vaginal wall and its associated fascia between women with and without prolapse.
- Interactions between the levator ani muscle and connective tissue attachments are critical to normal pelvic organ support.

Figure 1.

Diagrammatic representation of interactions between levator ani muscle, anterior vaginal wall prolapse, and cardinal/uterosacral ligament suspension. With normal levator function (a), the vaginal walls are in apposition, and anterior and posterior pressures are balanced. Levator damage (b) results in hiatal opening, and the vagina becomes exposed to a pressure differential between abdominal and atmospheric pressures. This pressure differential (c) creates a traction force on the cardinal ligament (CL) and uterosacral ligament (USL).

Modified from DeLancey 2012. © DeLancey

Figure 2. 2D biomechanical model created from MRI scan. Left panels show model development; (a) mid-sagittal MR image and modeled element traced or projected on mid-sagittal MR image and (b) the lumped parameter biomechanical model. Pubovisceral muscle (PVM) is modeled as a spring in parallel with an active force generator. PS, pubic symphysis; SAC, sacrum; PM, perineal membrane; LA plate, levator plate; R, rectum; V, vagina; CL, cardinal ligament spring; US, uterosacral ligament spring; B, bladder; UT, uterus. Right Panels show simulated deformation of the model anterior vaginal wall, and its support system, under maximal Valsalva with various degrees of pubovisceral muscle (PVM) and cardinal and uterosacral ligament impairment (indicated in percent). The value for D, which presents the size of prolapse measured as the decent of the most dependent point of vaginal wall from end of perineal membrane is provided. Modified from Chen 2006. © Biomechanics Research Lab, University of Michigan, 2006

Figure 3.

Panel a shows 3D reconstruction of the pelvic organs and bones (semitransparent) from MRI at rest with the line of the arcus tendineus fascia pelvis (ATFP) indicated. Anterior vaginal wall, AVW; bladder, B; external anal sphincter, EAS; levator plate, LP; rectum, R; urethra, U; uterus, Ut). Panel b shows the organs at rest with the measuring lines shown. Vaginal wall parameters are shown with white lines; vaginal width is measured at 5 locations and length in the midline. Green lines indicate apical support as the distance from the uterus to the top of the greater sciatic foramen where the cardinal ligament is fixed to the pelvic wall. Paravaginal distance indicated by yellow line (shown for one of five sampling locations). Panel c shows the same distances at maximal Valsalva. © DeLancey

Figure 4.

Data from the comparison of women with anterior vaginal prolapse and controls with normal support. Top panel compares apical and paravaginal distances from normal and bottom panel, vaginal width. Note that “1” represents the apex at the cervico-vaginal junction (CVJ) and position “5,” the urethrovaginal junction (UVJ) at the distal end. Asterisks mark statistically significant differences ($p < 0.05$). Note that right and left paravaginal distance means have been combined to one overall mean. Standard deviation shown. Larson 2012. © DeLancey

Figure 6.

The left panel shows a view of the pelvic organs from above looking over the pubic symphysis, showing structures of the pelvic sidewall in relationship to the vagina (outlined by dotted line) after removal of the bladder and uterine corpus. Right panel shows different levels of support in post-hysterectomy cadaver.

Figure 7.

Left side panels show 3D models in the standing posture with (upper panel) and without (lower panel) the display of the pelvis showing. Pubic bone, PB; Sacrum, Sac; Uterus, Ut; Vaginal, Vag; Cardinal ligaments, CL; and deep uterosacral ligament, US. The middle panels show normal and prolapse ligament comparison under resting and maximal Valsalva. Shown are 3D models of one healthy individual at rest (a) and at maximal Valsalva (b); with their relationship to the normalized arcus tendineus fascia pelvis, ATRP) extending from the public symphysis, P) to the ischial spines, yellow squares). Below are shown a subject with prolapse at rest (c) and at maximal Valsalva (d). “Uterus, Ut; vagina, V; cardinal ligament, CL; deep uterosacral ligament, USLd. Ut uterus, V vagina, CL cardinal ligament, USLd deep uterosacral ligament. Right panel: Average straight length and angle of cardinal ligament (CL) and deep uterosacral ligament (USLd) are shown based on one average, mid-sagittal image from a healthy woman, with outline for uterus, vagina, pubic symphysis, and sacrum. From Luo 2013. © DeLancey