BY THOMAS JULIAN, MD

# Pelvic-support defects: a guide to anatomy and physiology

Due to high postoperative failure rates, the traditional treatment for pelvic-organ prolapse—hysterectomy with anterior and posterior colporrhaphy—is being replaced by procedures that target specific pelvic defects. Thus, a familiarity with pelvic-support mechanisms is crucial to determining corrective measures.

Childbirth, chronic coughing, heavy lifting, or just inherent connective-tissue weakness and aging—each of these can cause pathophysiologic changes in the muscular and fascial structures of the pelvic floor, possibly leading to pelvic-support defects and, over time, pelvic-floor dysfunction. Patients with this condition may experience discomfort, urinary or fecal incontinence, or organ prolapse.

Traditionally, hysterectomy with anterior and posterior colporrhaphy has been the standard treatment for pelvic-organ prolapse. But due to this procedure's high postoperative failure rate,[1-3] a new approach is beginning to emerge. Lately, clinicians in the field of pelvic-floor dysfunction have begun advocating specialized evaluation techniques—among them, site-specific physical examinations, urodynamic testing, anal manometry, and anal sphincter ultrasound—to identify and measure anatomic and physiologic pelvic-support defects. Once the physician has determined the specific cause of the patient's dysfunction, he or she can then direct treatment toward that defect.[4] Therefore, when site-specific examination uncovers a paravaginal defect, for example, or ultrasound demonstrates anal sphincter disruption, a repair can be planned that will include correcting those problems.

Recently, pelvic reconstructive surgeons have focused on developing procedures that restore normal anatomy. Unfortunately, this goal is not achieved in most reparative pelvic operations. Instead, the most common operations reduce vaginal volume, create resistance to prolapse, or suspend an organ from a stable anatomic site—thereby repairing the defect by creating a compensatory defect, rather than by recreating a normal pelvic anatomy.

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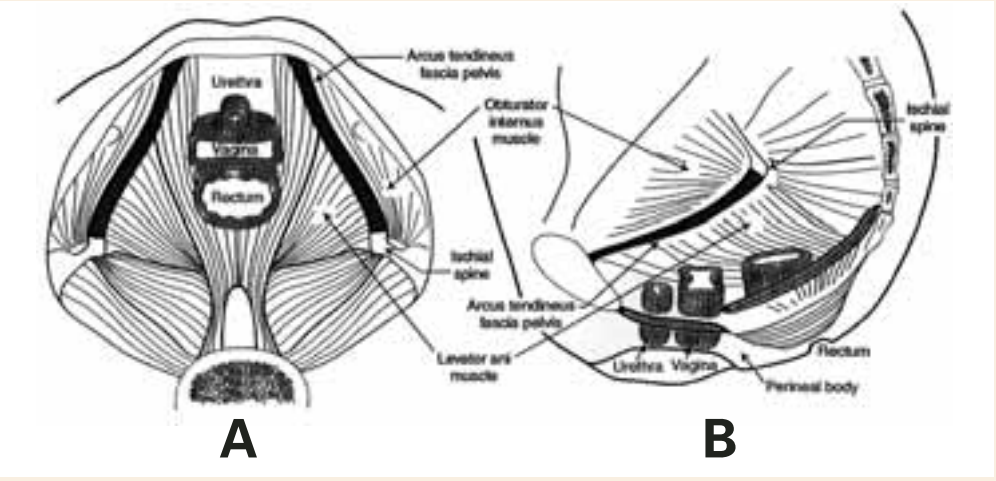
**KEY POINTS**

- The levator complex of muscles and the surrounding connective tissue, vessels, and nerves are often referred to as the pelvic floor.
- Studies have shown damage to muscle bundles of the levator and nerves of the pelvic floor after childbirth, including atrophic and degenerative muscle changes and slow nerve-conduction velocities.
- To maintain urinary continence, there must either be an intrinsic urethral pressure greater than the pressure in the bladder or occlusion of the urethra when intra-abdominal pressure increases.

**FIGURE 1**

**The pelvic musculature**




These are transabdominal (A) and lateral (B) views of the pelvic floor demonstrating the pelvic musculature.



Beyond identifying patients with pelvic-floor dysfunction, the clinician's job is to ascertain the defects causing the problem[5] and determine appropriate diagnostic and therapeutic techniques. A familiarity with the anatomy of the pelvis is, of course, vital to this process. In this article, I detail the anatomic and physiologic support mechanisms of the female pelvis, along with various conditions leading to pelvic-organ prolapse.

## Anatomic support mechanisms

Since malpositioning of the pelvic organs is the main contributor to uterine or vaginal prolapse, special attention must be paid to the positioning and support of the urethra, bladder, vagina, uterus, and perineum. These organs are held in place by an interactive network of bones, connective tissues, muscles, vessels, and adventitial sheaths, all of which create normal pelvic-organ support.

**The bony pelvis.** This structure serves as the anchoring site for the dense connective tissue or fascial coverings of the pelvic musculature. The anterior connective tissue attachments, inserting along the pubic bone, consist of the pubo-urethral ligaments and endopelvic fascia. Laterally, the dense connective-tissue covering of the obturator muscle attaches to the inferior pubic ramus from behind the symphysis pubis along the linea terminalis and, posteriorly, to the ischial spines. Posteriorly, the lateral margins of the sacrum serve as the attachment for the cardinal-uterosacral ligament complex.[5]

**The muscles of the pelvic floor.** The bony structures fuse to the pelvic organs through a contiguous network of connective tissue that surrounds the obturator and levator muscles. The obturator muscle is in contact with the lateral pelvic wall. The bowl-shaped levator muscle is joined to the obturator muscle at the arcus tendineus fascia—or "white line"—of the pelvis and consists of 3 parts: iliococcygeus, pubococcygeus, and puborectalis. Within the levator muscle sit the urethra,

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bladder base, vaginal tube, and anorectum. The levator complex of muscles and the surrounding connective tissue, vessels, and nerves are often referred to as the pelvic floor (**FIGURE 1**).[2,5]

**Connective tissue of the pelvis.** These organs are surrounded by what has traditionally been called endopelvic fascia (**FIGURE 2**). For more than 50 years, experts have debated whether this tissue is truly fascia.[6-8] Histologically, it is largely smooth muscle, elastin, and collagen. More recently, it was suggested this be called fibromuscular connective tissue.[5] It serves as a surrounding connective tissue, anchoring the pelvic organs to the muscles, and the muscles to the bony pelvis. Through these tissues traverse the nerves and blood vessels that add support to the tissue and keep it vital.[6-8] The urethra, vagina, and anus open onto the vulva and perineum by passing through a breach in the levator muscle collectively called the genital hiatus.

**A unifying theory of the anatomic vaginal support.** According to DeLancey, the normal anatomic position of the distal vagina in the standing female is a vertical ascent of 1 to 3 cm.[9] From the midvagina to the vaginal apex, the axis of the vagina is more horizontal, forming an approximately 120° angle.

The connective-tissue attachment of the urethra to the pubic bone and laterally to the levator muscle supports the distal vagina (DeLancey Level III). The midvagina is supported by the connective-tissue sheath that surrounds it and its lateral connective-tissue attachments to the levator muscle (DeLancey Level II). The vaginal apex and cervix (DeLancey Level I) are held by the surrounding endopelvic connective tissue—mainly the thickened lateral and posterior portions referred to as the cardinal and uterosacral ligaments (**FIGURE 3**).[9]

## Physiologic support mechanisms

In addition to the anatomic components of female pelvic-organ support, there are physi-

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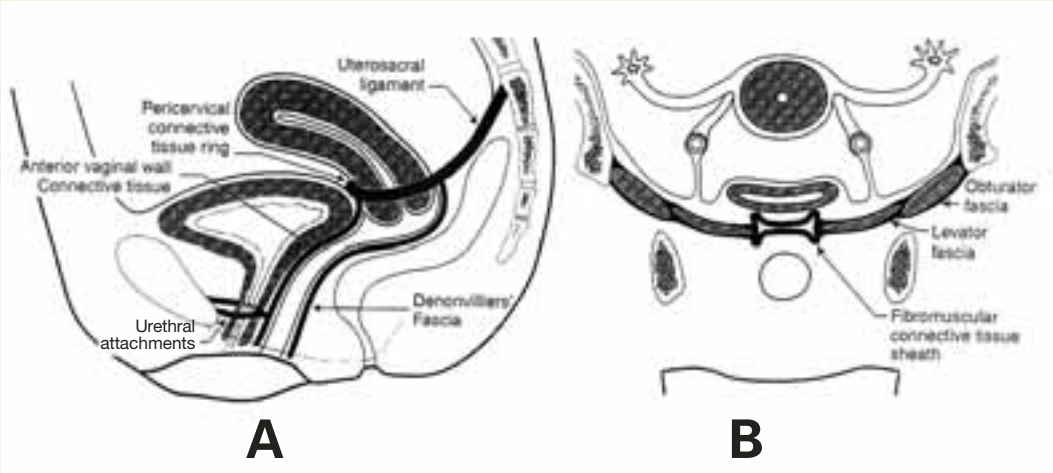
**FIGURE 2**

**Pelvic connective-tissue network**




The heavy black lines in these lateral (A) and coronal (B) views represent the pelvic connective-tissue network known as endopelvic fascia. It demonstrates the continuity of the pelvic connective tissue as it surrounds the pelvic organs and ultimately attaches to the pelvic sidewall.



ologic mechanisms that help maintain the positioning and functioning of pelvic organs. The muscles, vessels, and nerves of the pelvic floor function interactively as a compression mechanism in a manner similar to a valve, maintaining positioning of the viscera. The resting tone of the pelvic floor provides a valve-like closure surrounding the urethra, vagina, and rectum. This mechanism helps to close the genital hiatus, thereby preventing descent of the uterus, vagina, and adjacent structures.[10]

**The pelvic axis.** The connective-tissue supports of the vagina anchor it to the pelvic skeleton and hold the vagina in the previously described semihorizontal axis. When this axis is maintained, increased abdominal pressure pushes the vaginal tube into the hollow of the sacrum, maintaining an intra-abdominal position (**FIGURE 4**).[5,10]

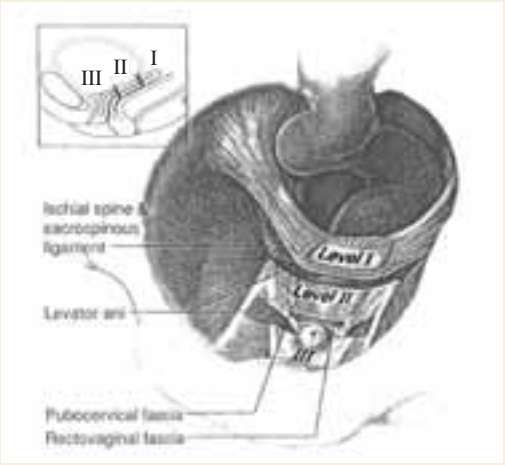
When the connective-tissue supports of the vagina and uterus are broken, the pelvic organs align in a more vertical axis. Increased intra-abdominal pressure causes the pelvic organs to descend through the natural openings in the pelvic floor (genital hiatus). The resulting descent of the uterus and vagina is what we refer to as pelvic organ prolapse. The genital hiatus also may be widened secondary to the trauma of childbirth.[5,9,10]

## Causes of pelvic-support defects

**Childbirth.** Damage to the levator muscle during childbirth has been associated with the beginning of pelvic-organ dysfunction, either as noticeable descent of the pelvic viscera, or bladder or bowel incontinence. Studies have shown damage to the muscle bundles of the levator and to nerves of the pelvic floor after childbirth, including atrophic and degenerative muscle changes and slowed nerve-conduction velocities.[10,11] While there is some evidence of muscle and nerve recovery, these postnatal findings of injury have been generalized to populations at increased risk for pelvic-organ prolapse and fecal and urinary incontinence,[12,13] especially multigravid

**FIGURE 3**

### Anatomic and physiologic support mechanisms




This demonstrates the levels of support described by DeLancey. Level I shows connective-tissue fibers extending both cephalad and dorsally toward the sacrum. Level II shows the lateral attachment to the arcus tendineus fascia of the pelvis. Level III shows the lateral attachment and anterior attachment of connective tissue to the lateral arcus tendineus fascia and posterior pubic symphysis.

Reprinted with permission from DeLancey JO. Anatomic aspects of vaginal eversion after hysterectomy. *Am J Obstet Gynecol*. 1992;166:1717-1728.

patients and patients with disruption of the anal sphincter.

**Age.** Aging also has been shown to decrease the functioning of the pelvic-floor muscles. The microscopic findings here are similar to those after childbirth, demonstrating both a loss of muscle substance and degeneration of the nerve supply. In addition, there appears to be atrophy of the remaining musculature.[14]

**Collagen in the pelvis.** In addition to an age-related loss of the endopelvic fascia, there also may be inherited differences in the ratio of collagen types and in the quality of the collagen ground substance. This is best exemplified by nulliparous patients who demonstrate pelvic-organ prolapse or joint hypermobility and by patients with hereditary hyperelastosis syndromes.[15,16]

**Estrogen and pelvic-floor dysfunction.** The theory that estrogen helps maintain the quality of connective tissue stems from the observation that urinary incontinence and pelvic-organ prolapse seem to begin around the time of menopause. While estrogen may indeed play a role, no prospective study has shown that estrogen replacement has a significant effect on urinary or fecal incontinence.[17-19] Nor have any parallels been found in young women who have experienced surgical castration, chemotherapy, or premature ovarian failure.

## Maintaining continence

**Urinary continence.** To maintain continence, there must be either an intrinsic urethral pressure greater than the pressure in the bladder or occlusion of the urethra when intra-abdominal pressure increases.

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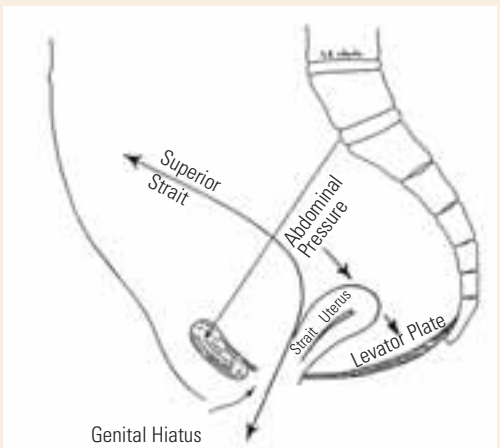
**FIGURE 4**

### Connective-tissue supports




This figure demonstrates how the uterus is held over the levator plate. In this position, increases in intra-abdominal pressure push the uterus caudally into the levator plate rather than through the genital hiatus.

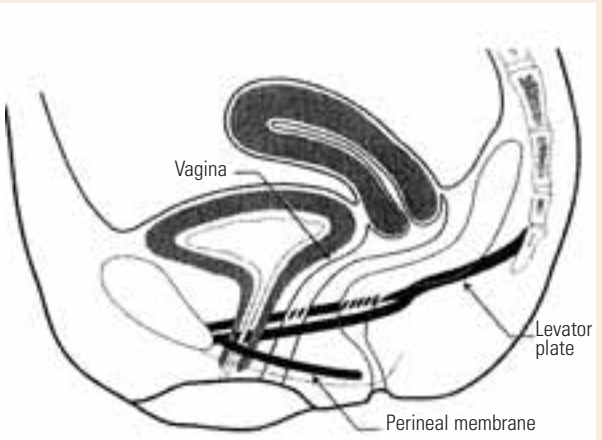
Reprinted with permission from the American College of Obstetricians and Gynecologists (*Obstetrics and Gynecology*, 1960, Volume 15, pages 711-726)

When the anatomy of the pelvis is intact, the proximal urethra and bladder base are held above the perineal membrane (**FIGURE 5**). Some experts believe that an intra-abdominal urethral position at or above the perineal membrane, within the so-called continence zone, maintains female continence. This is because increases in intra-abdominal pressure are equally transmitted to the urethra and bladder, when the proximal urethra remains in the continence zone; the intrinsically higher urethral pressure maintains closure without leakage of urine.[20]

The hammock theory of urinary incontinence, meanwhile, proposes that the midurethra is held closed by suburethral support. Increases in intra-abdominal pressure push the urethra down onto these supporting tissues and functionally obstruct the urethra to maintain continence.[21] As long as there is adequate support below the urethra, this theory holds, continence is maintained. Both mechanisms—intra-abdominal urethral positioning and suburethral support—may contribute to urinary continence.

**FIGURE 5**

**Perineal membrane and urethra**




When the anatomy of the pelvis is intact, the proximal urethra and bladder base are held above the perineal membrane. This intra-abdominal position (at or above the perineal membrane) keeps the urethra within the continence zone.



Continence also is maintained neurologically, by learned cortical mechanisms and conditioned reflexes that inhibit bladder emptying under social controls, and by neurologic coordination of local reflexes that allow the urethra to relax and the bladder to contract when we voluntarily void. Similar mechanisms govern defecation, requiring the same cortical and locally reflexive learned behaviors.[22,23]

**Rectal continence.** The sphincter mechanism of the anal canal consists of 2 separate anatomic structures: the internal and external sphincters.

The internal sphincter is involuntary smooth muscle in the rectal wall. It provides continuous involuntary tone. The external sphincter is composed of a triple-loop system. The top loop is the puborectalis portion of the levator ani muscle. It encircles the rectal neck, suspends the anal canal forward toward the symphysis pubis at a near right angle, and maintains continence by closing the rectal inlet. The intermediate loop of the external sphincter pulls in the opposite direction. It originates at the coccyx as a tendon, encircles the middle segment of the anal canal, and inserts back on the coccyx. The base loop is the smallest and lies beneath the anal mucosa, encircling the anus. It is most commonly referred to as the anal sphincter.

The levator plate is attached to the posterior rectum by the hiatal ligament, an extension of the endopelvic fascia. The hiatal ligament, by contraction or relaxation of the levator plate, provides the mechanism by which

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the rectal inlet is opened and closed. During defecation, the puborectalis contracts and the levator plate relaxes to open the rectal neck.[23]

## Conclusion

Over the past 25 years, new ways of looking at the anatomy and physiology of pelvic-support defects have evolved, demonstrating the need for changes in diagnostic and therapeutic techniques. Specific physical-examination and technical tests allow clinicians to identify abnormalities of both form and function of the pelvic organs. Once identified, pelvic-support defects can be studied more closely, and treatment methods can be developed. Now that there is a subspecialty in pelvic reconstructive surgery, greater emphasis should be placed on female pelvic anatomy and physiology.[1-5,11-14,20,24-26] ■

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