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The Achilles' Heel of Pelvic Reconstructive Surgery

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As our population ages, pelvic organ prolapse is becoming more common. A woman's lifetime risk of surgery for pelvic organ prolapse is at least 11.1%, and almost one in three will need repair for recurrent prolapse [1]. Traditional repair for anterior vaginal wall prolapse has a high failure/recurrence rate of 40% to 60% and these techniques are based on surgical knowledge from a century ago. Back then the goal of pelvic organ prolapse surgery was to reduce the bulge, not correct the cause of prolapse. According to a questionnaire by the American Urogynecology Society, 80% of urogynecologists still perform this 100 year-old technique with its dismal current rates.

Why is anterior vaginal wall prolapse repair so difficult? Perhaps midline defects and paravaginal defect repairs are not the real cause of anterior vaginal wall prolapsed? If not, why the high failure rates of midline plication and paravaginal repairs? Of the 300,000- 400,000 pelvic organ prolapse surgeries performed annually in the United States, 30% to 40% are for recurrences and 60% occur at the same site [2]. This is the Achilles' heel.

This also brings up some questions: (1) Is traditional anterior colporrhaphy (midline plication) the "gold standard" for repair of the prolapsed anterior vaginal wall as some academics propose? (2) Is prolapse of the anterior vaginal wall a true hernia as Richardson suggested? (3) How and where does vaginal birth cause vaginal defects?

Dr. Richardson did not ascribe to the theory of stretching or thinning of the pubocervical supportive tissues assumed by Kelly in 1913 [2]. Instead, Richardson described several defects [3]. A midline defect is, "an anterioposterior separation of the fascia that occurs between the vagina and the overlying bladder and or urethra. It results in a cystocele and urethrocele". He further suggested "this defect is one of the most easily repaired and excellent results can be expected with Kelly-Kennedy type procedures." However, over time this repair has unacceptable failure rates.

Modern gynecologists displeased with the recurrence rates of Kelly, White, and Richardson procedures turned to mesh kits [3,4]. Unfortunately, these kits ignored reconstruction of the defects and instead accepted a new "industry" concept to construct a mesh bridge for the centrally prolapsed bladder. The focus of mesh kits changed from reconstruction using long recognized and accepted theories of vaginal defects to simply reconstructive surgery using a mesh bridge promoted by industry but basically untested in the pelvis. With these kits, supposedly identification of the fascial defects was unnecessary; instead, the surgeon just opened the vaginal epithelium, laid down the bolster, and closed the epithelium. The concept that industry sold to surgeons was that a permanent repair required a permanent biomaterial. When the complications from mesh became more prevalent, industry removed them from the market, and gynecologic surgeons had no choice but to return to traditional midline placation, regardless of its dismal recurrence rates, to avoid erosions, pain, dyspareunia, and potential legal issues caused by vaginally inserted mesh.

Richardson also described the lateral or paravaginal defect as "a defect found in the fascia laterally at or near its attachments to the levator insertion in the lower margin of the superior pubic ramus." He further suggested "that it usually results in a mild or moderate cystourethrocele, a loss of the urethrovescial angle, and significant stress urinary incontinence which could be unilateral or bilateral." This defect occurs distal to the connection between the arcus tendentious fascia and the arcus rectovaginalis, and he believed it to cause stress urinary incontinence. However many researchers do not document this belief. Instead, surgeons adopted the concept that a paravaginal defect extended to the ischial spine and was the cause of cystoceles. This was never described by Richardson.

Richardson identified a defect that was "a transverse separation that occurred in the pubocervical fascia from its insertion into the pericervical ring of connective tissue." He further stated, "It usually results in a large cystocele in which the bladder herniates beneath the mucosa of the anterior vaginal fornix. The urethra remains well supported with a good urethrovesical angle and there is rarely, if ever, stress urinary incontinence."

Are cystoceles hernias as Richardson suggested? Some reconstructive surgeons propose that reconstruction of anterior and posterior vaginal wall prolapse is really about hernia surgery. If this is true, the protrusion of the bladder or rectum through the tissues designed to contain them must be documented as true hernias. The only hernia that Richardson identified was with a transverse defect. Did he not describe hernias with midline or paravaginal defects because he and others have not observed the bladder actually herniating through a midline or paravaginal defect during surgery? Unless the bladder can be identified protruding through a proposed defect, are such repairs warranted?

Pelvic support defects are thought to occur, or are at least exacerbated, during the process of childbirth. Gynecologists have long believed that trauma to the supporting structures of the bladder leading to vaginal prolapse are associated with vaginal birth. With clinical observation, there is little doubt that childbirth contributes to the likelihood that clinically symptomatic prolapse will occur. However, a major shortcoming of our profession is that the effects of labor and delivery to the female pelvis resulting in vaginal prolapse have never been fully studied or understood. Also, little effort has been made to analyze the forces of childbirth, and thus there is also little proof as to how and when, in the course of labor, the effects of childbirth occur as they relate to specific damage patterns found in the endopelvic supportive connective tissue [5]. Did the tears to the supportive tissues of the bladder during delivery really occur in a vertical (midline) or lateral (paravaginal) direction as most gynecologists have been lead to believe?

Dr. Richardson must receive credit for considering the bony pelvic outlet and the pelvic supportive structures in mechanical terms [3]. These structures should be considered like a bridge; so when it fails

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the questions, where does it fail and why does it fail must be asked. I strongly believe that it does not fail in either the midline or laterally. After many years of studying this problem, and with the assistance of biomechanical engineers, we theorized that during childbirth descent of the fetal head to the level of the pericervical ring causes significant tensile and sheer strain on the endopelvic fascia (pubocervical fascia) attached to the pericervical ring [6]. As the birth canal narrows at the level of the ischial spine, the narrowest diameter of the pelvis, stress and strain are significantly concentrated because the tissues the fetus passes through must undergo even greater deformation in order to accommodate the fetal head. Internal rotation of the fetal head occurs in order to present the optimal diameter of the fetal head to the bony pelvis. Movement from the rotation of the fetus induces transverse shearing forces onto the pubocervical fascia, already under high-loading strain caused by fetal descent. The strained and shearing forces can exceed the strength of the pubocervical fascia attached the pericervical ring, resulting in soft tissue tears that affect the supportive tissues to the bladder. The stress and strain to the pubocervical fascia occur in both a superior and inferior direction, and the internal rotation of the fetal head results in trauma separating the pubocervical fascia from the pericervical ring in a transverse direction with the bladder herniating in between.

This biomechanical modeling theory strongly suggest that tears to the pubocervical fascia during childbirth are more likely to occur as transverse tears to the pubocervical fascia from the pericervical ring rather than in the vertical direction in the midline or laterally (paravaginal tears). Could transverse tears of the supporting tissues of the bladder be the most likely, and perhaps the only cause, of anterior vaginal wall prolapse?

The validity of this theory was substantiated during cadaver dissections and in the operating theater. Patients who were clinically diagnosed with midline and paravaginal defects preoperatively were studied with MRIs that did not document the bladder protruding through either a midline or paravaginal defect. This was also confirmed in the operating theater where the defect documented and found on each occasion was a transverse defect where the separation of the pubocervical from the pericervical ring was noted, thus causing the bladder to herniate between the separation. The reasons for poor result from midline and paravaginal repairs became obvious. Surgeons have been simply operating in the wrong place for many years. This theory represents a radical, but anatomically correct concept contrary to what gynecologic surgeons have been taught and believed for almost ten decades.

It can be argued that this new biomechanical modeling theory is the most likely cause of anterior vaginal wall prolapse. Further results of the surgical correction using this concept of restoring the pubocervical fascia to the pericervical ring for anterior vaginal wall prolapse are "in press". Two hundred and seventy-six patients have undergone this new technique with 24 month follow-up and success rates of 95%, which demonstrate long-term success rates beyond midline and paravaginal repairs [6]. Current multi-centered studies and time will tell if this new theory and operation to correct the transverse defect will replace traditional repairs and become a higher standard of care for the future. It is easy to recommend this theory and operation to correct the transverse defect because this repair returns the actual distorted anatomy to normal while providing sufficient apical support.

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