EDITORIAL

Hiatal expansion and pelvic organ prolapse – the association is not causation

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Citation: Petros P. Hiatal expansion and pelvic organ prolapse – the association is not causation. Cent European J Urol. 2023; doi: 10.5173/ceju.2023.272 [Epub ahead of print]

Article history
Submitted: Nov. 5, 2023
Accepted: Dec. 3, 2023
Published online: Jan. 6, 2024

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Key Words: collateral collagen damage ‹› levator avulsion ‹› hiatal expansion ‹› POP

Pelvic organ prolapse (POP) accounts for more than 300,000 surgical procedures annually in the United States alone [1]. The levator hiatus (LH) is the space between the two parts of the pelvic diaphragm. Recent findings of high cumulative stepwise probabilities linking LH size with POP occurrence has added to the plethora of articles which now seem to suggest cause (LH expansion) and effect (POP). A major ultrasound analysis of 2513 women recently concluded, 'The effect of partial avulsion on POP and prolapse symptoms is explained fully by its effect on hiatal area' [2]. Increasing acceptance of such beliefs has a necessary corollary: LH-related POP is not curable by traditional Fothergill-type operations, which could lead to unforeseen effects such as avoidance of surgery for POP, or even medicolegal actions in women with failed POP surgery.

The aim is to use known anatomy of LH [3, 4] and known biomechanics of labour [5] to test LH causation hypotheses for POP against traditional connective tissue causation for POP which underlie traditional repair methods such as the Fothergill operation.

Shafik's histological finding, that the hiatal ligament was composed of collagen and elastin, [3] is key to the hypothesis that the association of LH expansion and POP is coincident damage from the head to muscles and the 3 levels of the suspensory ligaments (circles, figure1) as the head descends down the birth canal, not cause/effect.

Massive depolymerization of collagen occurs 48 hours prior to delivery; collagen loses 90% of its strength [5]. This process plasticizes the collagenous structures of the pelvis allowing them to be stretched by the descending head, circles, figure 1, without rupture: at the birth canal entrance, uterosacral ligaments (USL) and cardinal ligaments (CL); at the level of levator hiatus, the collagen lining of the pubococcygeus muscles (PCM) [3]; further down, perineal body (PB), and on the way out, pubourethral ligaments (PUL) and insertion of the levator muscles. After the menopause, collagen breaks down, and is excreted as hydroxyproline, a breakdown which continues to the end of the woman's life [6]. Collagen breakdown explains higher incidence of prolapse after the menopause, even in nulliparous women.

Women with prolapse in one compartment, for example, cystocele, usually have lax tissues in other compartments, often without obvious prolapse. As collagen continues breaking down in women after the menopause [6], so does the collagenous lining of LH, plus the fascia and ligaments of all 3 levels of organ support (Figure 1). When the structural

collagen in the hiatal lining and pelvic ligaments falls below critical mass, hiatal expansion and POP may co-occur; the LH collagen lining weakens to over-expand LH, and the supporting ligaments weaken to cause prolapse.

Why cystocele is so often prominent with LH expansion. The cardinal ligament (CL) reflection onto the anterior surface of the cervix is the principal support of the pubocervical fascia of the vagina (PCF) (Figure 2). CL is the most vulnerable ligament for damage by the head due to its direct proximity to the head, and its transverse orientation. Fracture ('r', Figure 2) of CL allows PCF to rotate down and to carry the bladder down with it as a cystocele. This process explains the strong relationship between LH expansion and cystocele [1, 2].

Direct falsification of the LH expansion hypothesis for POP causation was by two experimental studies [7,8] where a puborectalis sling reduced LH expansion by up to 30%, but POP recurred in 78 and 79% of women by 12 months [7, 8]. The question arises why an RCT was performed in 2017 when the original trial in 2010 had a 78% recurrence rate.

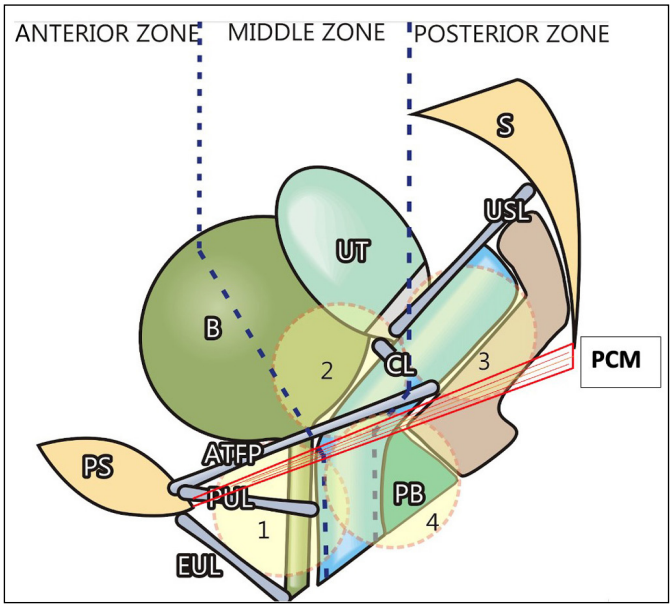

Figure 1. *Descent of the fetal head down the birth canal may damage collagenous and muscle structures in all 3 levels of organ support. At the upper end of the birth canal, figure1, overstretching by the head may cause uterine prolapse (circles '3') and cystocele (circle '2'); further down the head may stretch the collagen/elastin fibrils lining LH (Shafik's 'hiatal 'ligament') to cause LH distension[3] ; further down, the perineal body may be damaged (circle '4'); finally, as the head exits, it may damage the collagenous insertion of pubococcygeus muscle (PCM) to the symphysis (circle '1'), and the pubourethral ligament 'PUL', (circle '1') to cause SUI.*

Supporting the collateral ligament damage hypothesis, figure 1. Surgical cure over the 120 years of the Fothergill (Manchester) operation by cardinal/uterosacral ligament repair [9], supports ligament damage as cause for POP. With reference to figure 1, the anatomical pathway for the Manchester repair, was plication of damaged cardinal ligaments (CL), uterosacral ligaments (USL) and perineal body (PB) [9]. More recently, Inoue et al. reported a 10-year study of 960 women, mean age 70 years, who had 3100 minislings inserted into CL,USL,PUL (pubourethral ligaments) and PB to repair all three levels of POP, as per figure1 [10]. The mechanics of their surgery was, to all intents and purposes, identical to the Fothergill repair, albeit a little more evolved: CL,USL,PB ligaments (and also PUL) were shortened (instead of plicated) with a tensioned minisling, which also created new collagen, a similar principle to the well-known midurethral sling. The anatomical cure rate at 12 months was 90% for each prolapse [10] as against the 11–12% cure of the hiatal sling repair [7, 8].

Collagen is the key structural component of ligaments and vagina. It can be weakened by childbirth and old age. Association, however often it is confirmed, is not causation. A space is not a structural component and as such cannot cause prolapse.

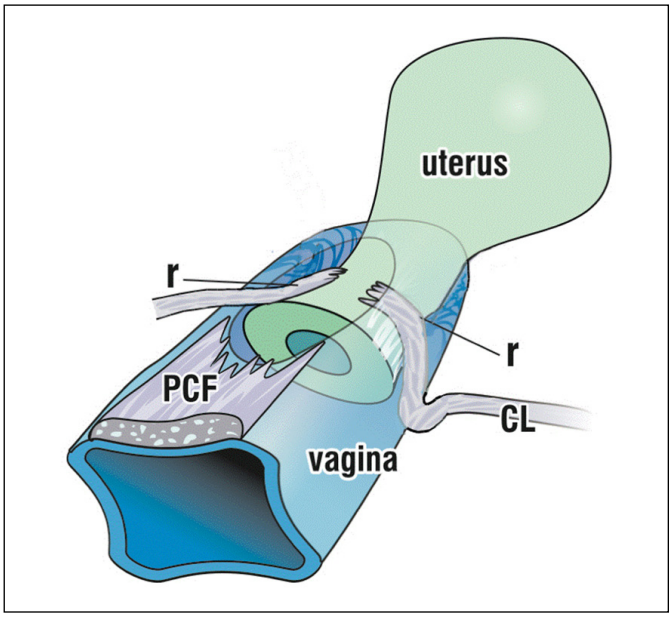

Figure 2. *Pathogenesis of transverse defect cystocele. The cardinal ligaments (CL) reflect onto the anterior part of cervix. The fascial layer of vagina 'PCF' (pubocervical fascia) inserts onto CL. If CLs are ruptured ('r') by the head as it descends down the birth canal, the PCF attachment is also ruptured, and the anterior vaginal wall it prolapses downwards like a trapdoor, as a cystocele.*

An expanded LH space is necessarily a secondary manifestation of an anatomical structural cause. The descending head hypothesis appears to satisfactorily explain associations of prolapse (especially cystocele), LH expansion and levator avulsion. There is no need to stipulate cause (LH expansion) and effect (POP).

CONFLICTS OF INTEREST

The author declare no conflict of interest.

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