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Overexpansion of the hiatus causing prolapse and LUTS is a failed concept

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ABSTRACT

Background: A strong association between levator expansion, pelvic organ prolapse (POP) and lower urinary tract symptoms (LUTS).

Aim: To test the hypothesis that anatomical damage at childbirth is the underlying cause for excessive hiatal dilatation, prolapse and LUTS.
Methods: Anatomy and biomechanics of the levator hiatus (LH), pelvic muscles and effects thereof of labour were analysed.

Results: LH is a space between horizontally oriented pubococcygeus muscles containing urethra, vagina, and anorectum. These muscles turn vertically downwards into a "tunnel" surrounded by vertically oriented puborectalis muscle, inserting into anal sphincters and perineum. Hiatal expansion is not necessarily pathogenic. A calculated 45sqcm expansion is required for evacuation of larger stools. Co-occurrence of over-expanded LH, prolapse and LUTS are explained as follows: pre-labour depolymerization of collagen "plasticizes" connective tissues, allowing stretch without rupture. Failure to regain normal length post-delivery may cause nerve damage and extended entheses; both may cause hiatal muscle to sag, resulting in LH expansion; also, causing loose or weakened cardinal/uterosacral ligaments which may cause prolapse and weakened muscle forces which cannot open, close or stretch, leading to bladder/bowel incontinence and evacuation problems. A 79% recurrence of POP after successful surgical confinement of LH by puborectalis mesh sling invalidated the expansion hypothesis. However, data showing 80% cure of OAB, SUI and prolapse by TFS (Tissue Fixation system) minisling ligament repair indicated these ligaments caused the conditions associated with LH expansion, not the expansion per se.

Conclusions: Association is not causation. Muscle, nerve, ligament damage by head descent down the birth canal adequately explains LH/prolapse association. Birth damage simultaneously causes levator hiatus overexpansion, prolapse and LUTS.

Keywords: Cystocele; enterocele; pelvic organ prolapse; rectocele

INTRODUCTION

As its Latin name implies, the levator hiatus (LH) is a space - between the two parts of the pelvic diaphragm. Study after study over the past 10 years has affirmed a strong association between levator expansion, pelvic organ prolapse (POP) and lower urinary tract symptoms (LUTS). In spite of this, such observations remain as hypotheses, as little has been stated about why the hiatus causes LH expansion, and why LH expansion causes POP and LUTS. It is necessary to do so. It is an anatomical incongruity for a space *per se*, to cause POP or LUTS.

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Research Questions: This incongruity, POP and LUTS causation by a space, led to our research questions. What is the structure of the hiatus? What is the function of the muscles around the hiatus? Which anatomical structures dilate LH on straining? How can the "strong association" between LH expansion, prolapse and symptoms, be explained anatomically? How can the LH/prolapse hypothesis be proved or disproved?

Principal Hypothesis: Childbirth is the underlying cause for excessive hiatal dilatation, prolapse and LUTS, by damaging ligaments, muscles or their entheses and nerves.

METHODS

This was a limited review which was confined to anatomical or surgical papers relevant to the hypothesis. Anatomy and biomechanics of the levator hiatus (LH), pelvic muscles and effects thereof of labour were analysed. Particular emphasis was given past major anatomical cadaveric studies of the hiatal muscles.[1-5] More recent studies were also examined [6-12] as were functional anatomical and surgical studies based on the Integral Theory System.[13-22] Traditional nomenclature was used.[1,2]

RESULTS

Levator Hiatus and its Muscles

Shafik[1] Levator Hiatus and its Muscles: Based on 25 cadavers, Shafik[1] described LH, Figure 1, as, "*The levator hiatus occupies the anterior part of the levator plate. It is bounded on either side by the medial portion of the levator plate, which I call the "levator crura" posteriorly by the anococcygeal raphe, and anteriorly by the back of symphysis pubis. It is covered by a fascial membrane which is the continuation of the fascia on the pelvic surface of the levator plate. The membrane is pierced by the intrahiatal structures: Rectal neck and prostate in males, and rectal neck, vagina and urethra in females. Its fibers are condensed at the periphery of the hiatus to form the hiatal ligament.*"

The pubococcygeus muscles originated from the lower end of symphysis, ran horizontally backwards as two crura, became tendinous as they passed behind the rectum, decussated in a crisscross pattern behind the rectum to form the anococcygeal raphe. Laterally, each crus blended with the corresponding iliococcygeus to form the "levator plate" which turned downwards to enclose the intrahiatal structures as a muscular tube descending down to the perineum, "levator tunnel". The "tunnel" was separated from the organs by a fascial membrane, a continuation of fascia from levator plate. The outer surface of the tunnel was formed by puborectalis muscle. At the rectal neck inlet, the levator plate bent sharply downward to form what is now known as the conjoint longitudinal muscle of the anus (Shafik's[1] "levator anal sling"), extending along rectal neck and anal canal proper, to penetrate external anal sphincter and insert into the perianal skin. In adults, anteroposterior diameter of LH ranged from 3 to 4 cm, and transverse LH diameter from

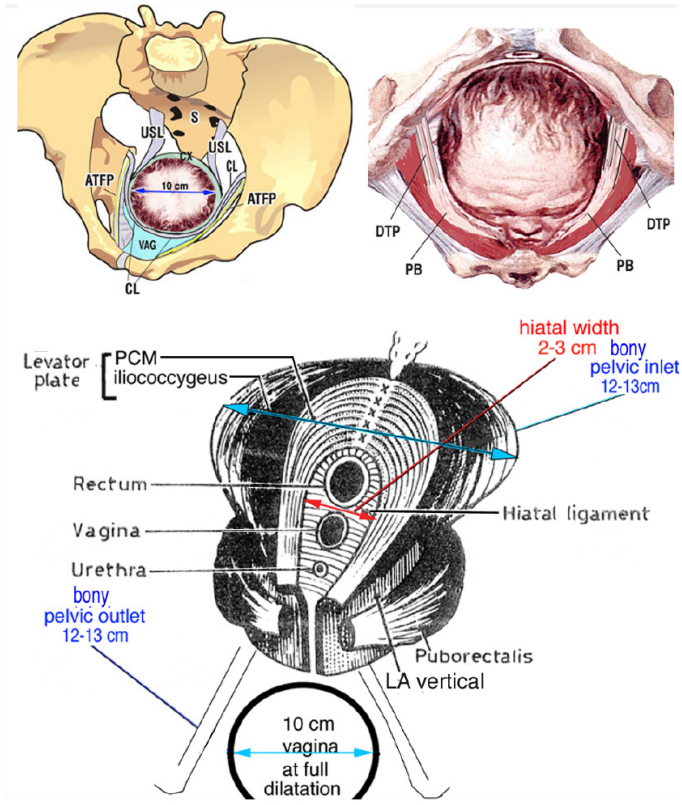

Figure 1. Lower Figure. 3D Schematic view of the levator hiatus, non-pregnant state, behind pubic symphysis, from Shafik[1], by permission. It shows the horizontal pubococcygeus muscles (PCM) bordering the hiatus ("crura") joining with iliococcygeus to form the levator plate and with each other to form the anococcygeal raphe. PCM muscles turn vertically downwards to form a "tunnel" which surrounds the organs. Puborectalis muscle (PRM) forms the outer wall of the tunnel. Below, bounded by the descending rami is the vagina at full dilatation.
Left upper figure: Cervix at full dilatation, 10cm, head entering birth canal. Cardinal (CL) and uterosacral USL) ligaments are severely stretched, as is ATFP attachment to ischial spine. Vaginal attachment to CL and cervix is stretched and often tears to cause a cystocele.
Right upper figure: Vagina at full dilatation, 10 cm. Perineal body (PB) is stretched and displaced downwards and laterally. Deep transversus perinei ligament attachments (DTP) of PB to descending ramus are also stretched. Levator/puborectalis muscle attachments to symphysis may be torn or stretched and dislocated.

2 to 3 cm.[1] Hiatal ligament composition of collagen and elastin[1] explains its ability to expand.

Courtney Conjoint Muscle

Allowing for differences in nomenclature, Courtney's descriptions[2] of pubococcygeus, puborectalis, the surrounding levator muscle tube essentially accord with Shafik's.[1] He described the conjoint muscle (now "LMA") extending inferiorly from the level of the levator fascia to the perianal skin below. The "LMA" was composed of the longitudinal muscle layer of the rectum, fibers from the levator fascia and muscle fibers from both the superior and inferior layers of all three portions of the levator muscle (puborectalis, pubococcygeus and iliococcygeus).[2] This combined layer completely encircled the rectum below the level of the levator fascia, much as described by Shafik.[1]

Zacharin Hiatal Attachments[3]

Figure 2 shows that, at the level of the hiatal ligament, the vagina "V", urethra "U", rectum "R" are tightly bound to each other and to the levator crus "LC" by connective tissue. The uterus "UT" and cervix "CX" are well above the levator crus (LC). Zacharin saw the levators as a contracted floor to retain abdominal contents. He accepted the concept of a widened hiatus causing POP and devised an operation to suture the crura. He reported post-operative urinary retention from this procedure.

Pathogenesis of Cystocele and Uterine Prolapse

Figure 2 (right), shows LH in red circles, and a round fetal head moving down the birth canal. Note the level 1 supports of the uterus, USL and cardinal and their fascial supports are well above LH. So it is impossible, no matter how widely LH is expanded, for LH to cause POP, unless the head has also damaged CL and USL at the same time as it has caused the structural damage leading to pathological LH expansion.

The Biomechanics of Labour Impact All Collagenous Structures to Cause LH Expansion and Prolapse[23,24]

Prior to labour commencing, connective tissue collagen in the birth canal depolymerizes and loses 95% of its strength.[23] Collagen repolymerization "snaps back" by 24 hours.[14] Depolymerization plasticizes all connective tissues, so they can stretch without rupturing. Inability of the collagen to regain its prebirth length and strength is, according to,[13,14] a major causation of pelvic organ prolapse and LUTS. With reference to Figure 2, at 10 cm dilatation of the cervix, Figure 1, USLs may stretch to cause uterine prolapse, the cardinal (CL) ligaments to cause a cystocele, further down, damage to the hiatal ligament, muscles or their insertions to bone, perineal body, a recto-perineocele. The required expansion of LH from 2-3 cm to 10 cm during birth grossly stretches the LH muscles laterally and may place extreme pressure on the entheses at the symphysis or sacrum, extending them to cause widened LH at rest and on straining, as

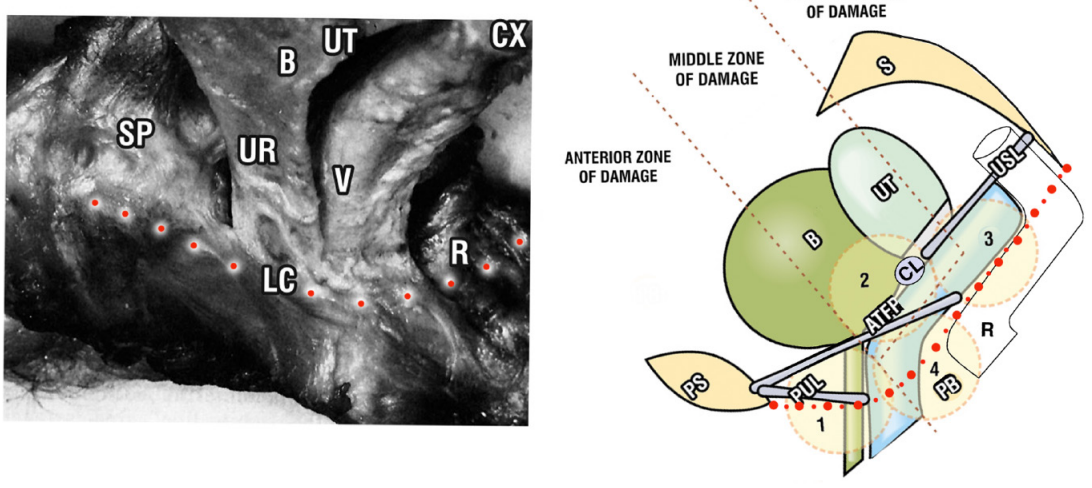

Figure 2. Sites of damage of the head to the levator hiatus and muscles and ligaments as it descends down the birth canal. The red circles represent the pubococcygeus muscle, "levator crus (LC)" which forms the lateral border of the hiatus.
Left figure: Zacharin's[3] dissection showing the attachments of the hiatal ligament. At this level, urethra "U", vagina "V" and rectum "R" are tightly bound to each other and to "LC" by connective tissue. UT: uterus; CX: cervix; SP: symphysis pubis. From Zacharin[3] 1985, by permission.
Right figure: Head descending down birth canal to damage. The red circles represent the levator crura extending from symphysis to cocccyx. "3" USL-uterosacral ligament; "3" CL, cardinal ligament and ATFP attachment to ischial spine; "4" PB, perineal body. "1" PUL pubourethral ligament and pubovisceral muscles.

reported.[6,7] Pathogenesis of rectal prolapse was described,[24] "*The USLs are attached to the lateral wall of the rectum by filamentous ligament-like structures. When the uterus descends, USLs lengthen and invariably splay laterally, to create an enterocele. Lateral USL displacements carry the lateral rectal wall with them; the anterior rectal wall stretches laterally, widening it, so it prolapses inwards to cause intussusception.*"

Integral System[13-22,24-27] a Holistic "Monocoque" Support System, Figure 3

The Integral Theory's concept of a holistic "monocoque" pelvic structure follows the same structural principle of a wire suspension bridge, Figure 3: Every pelvic part supports every other part, with the final structure greater than the sum of the parts. All elements of the pelvis, muscles, ligaments, fascias, organs, are integrated to create a strong, holistic light efficient structure.[25-27] With reference to Figure 3, like a suspension bridge, the ligamentous attachments of USL to rectum, cervix, sacrum, PUL, PVL to symphysis, urethra, bladder are all structural components. However, pelvic anatomy differs suspension bridge analogies because reflex muscle stretching of tissues and smooth muscles (arrows, Figure 4), creates tensile strength VIDEO1 https://www.youtube.com/watch?v=3vJx2OvUYe0. A 4th muscle, puborectalis (PRM), is part reflex (during anorectal closure and evacuation), and part voluntary, during "squeezing" or interrupting the urinary stream ("Kegel muscle"). PRM does not tension pelvic ligaments/fascias. It contracts only against the pubic bone anteriorly. At the level of the pubococcygeus muscle, Figure 2, rectum, vagina, and urethra are densely connected by dense collagenous tissue. The anterior part of the hiatus, is stabilized by insertion of PUL (pubourethral ligaments) into PCM, symphysis, midurethra and vaginal skin.[27] The pubovesical ligament (PVL) stabilizes the bladder by its attachment to the arc of Gil-Vernet et al[5].[14] The forward vector (VIDEO1), constitutes

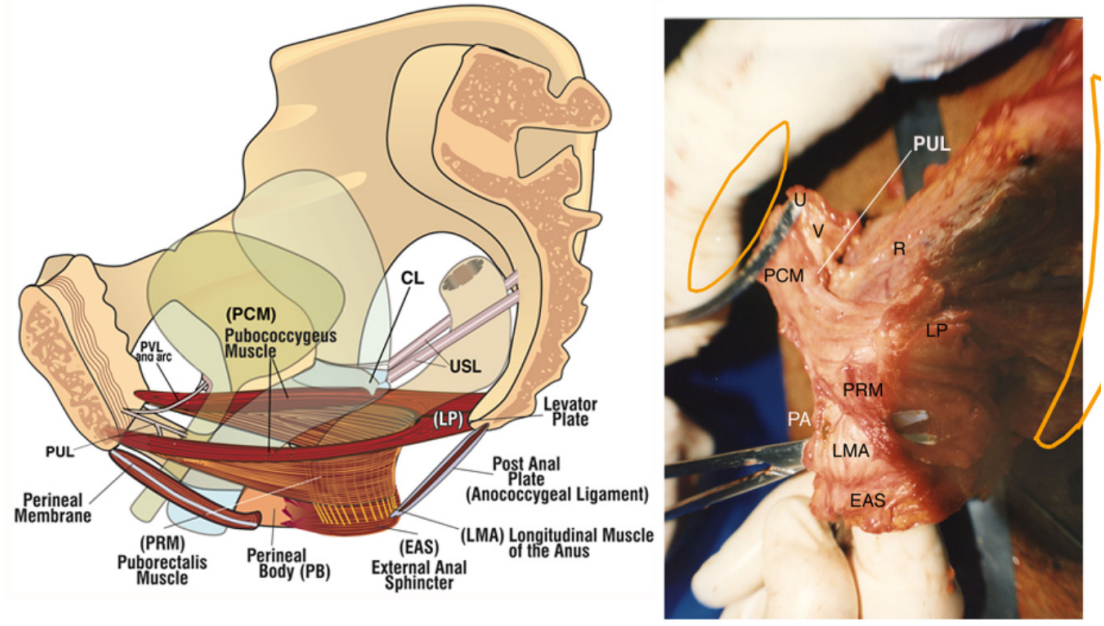

Figure 3. A "monocoque" support system

Right figure: The pelvic muscles. A large knife cut away all pelvic supports and muscles from the pelvic side wall. Vagina and bladder removed at bladder neck level. The horizontal pubococcygeus muscles (PCM) sweep behind the rectum (R) and merge with the contralateral side to form part of the levator plate (LP). The vertical remnant of muscle forward, above and below the label "LP" is the severed iliococcygeus muscle. Below PCM and LP is the vertical puborectalis muscle (PRM) which becomes medial to PCM and attaches directly to the symphysis. The vagina (V) is seen attached to rectum (R) at the level of the hiatus. The conjoint longitudinal muscle of the anus (LMA) is internal to PRM and PCM, but outside the rectum as indicated by the dissecting scissors. LMA inserts into the external anal sphincter (EAS). Puboperinealis (PA) was necessarily cut away along with the perineal body attachments to the descending rami. PUL is the pubourethral attachment to the inner wall of PCM (PP PUL paper).

Left figure: The hiatal muscles, PCM, LP, LMA, PRM are the central point of the "monococque" support system. However, the bladder, uterus and rectum by means of their ligamentous attachments, *well above the hiatus*, are important stabilizers, laterally, cardinal ligaments (CL); posteriorly, USLs; anteriorly PUL (pubourethral ligamenst), PVL (pubovesical ligaments) and insertions of PVL to the anterior bladder wall (arc of Gilvernet). The inferior attachments of rectum and vagina to PB and its attachments to the descending ramus by the transverse perinei ligaments (see Figure 1), bulbo and ischio cavernosus, stabilize the organs inferiorly. From Petros PE.[17,18]

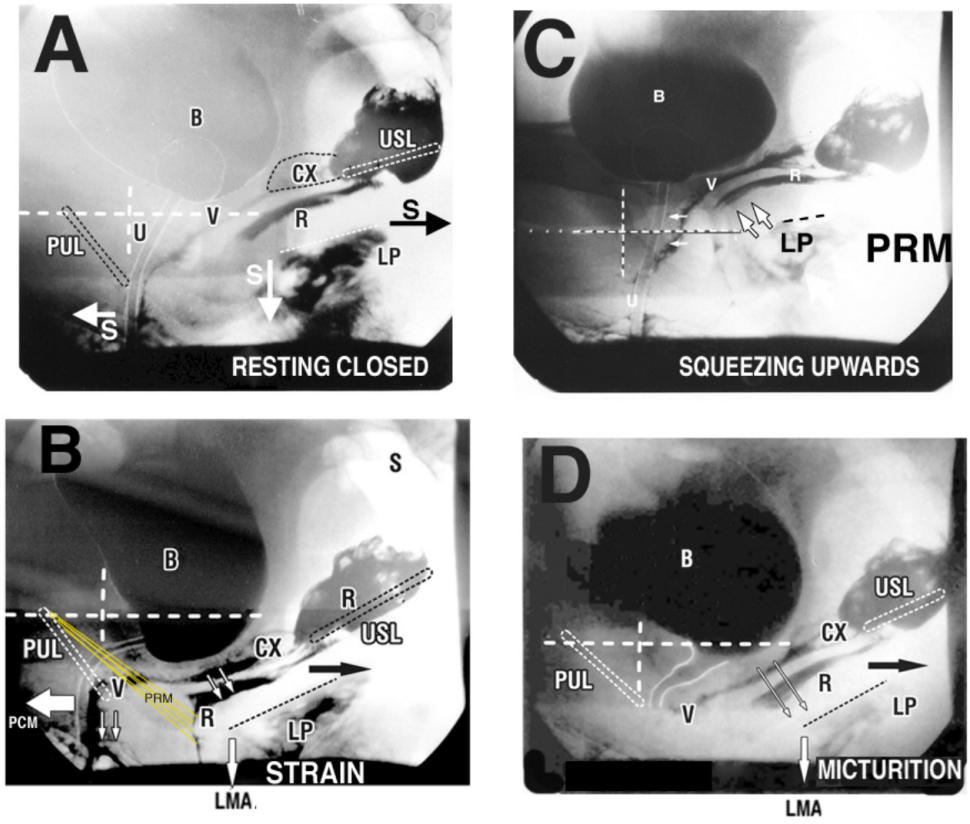

Figure 4. Role of the levator plate in bladder/anorectal closure and micturition. Sitting X-rays Vertical and horizontal lines are bony co-ordinates; A: RESTING CLOSED note S= directional slow twitch muscles as in B. radio-opaque dye inserted into bladder (B), rectum (R), vagina (V) and levator plate (LP). B: STRAIN; note 3 fast-twitch opposite directional forces (arrows) activated by strain; indentation of rectum by PRM; downward angulation of LP by LMA rotates bladder "B" around PUL and rectum "R" around PRM for closure; D: MICTURITION note 2 directional forces for micturition (forward force relaxed), angulation of LP by LMA pulls open posterior urethral wall; C: SQUEEZING UPWARDS: Note LP lifted upwards and forwards from below (by puborectalis PRM). PCM=pubococcygeus; LMA: conjoint longitudinal muscle of the anus. PCM: pubococcygeus muscle; PUL: pubourethral ligaments; USL: uterosacral ligaments. CX: cervix.
From Petros PE [ref].

the medial part of PCM.[25] Its forward action against PUL further stabilizes the anterior part of LH, while the lateral part of PCM, also acting against PUL, Figure 3, sweeps backwards to join with liococcygeus muscle (ICM) to form levator plate (LP), insert into posterior wall of rectum to stabilize the posterior part of LH and the rectum itself. The bladder is attached to the lower end of the anterior cervical ring by the vesicovaginal ligament[14] which must be severed prior to any hysterectomy. Posterior stabilization is by insertion of levator plate (LP), to the lower end of sacrum and coccyx, and by USLs from sacrum, into lateral walls of rectum and cervix, Figure 3. Important lateral stabilization of the monococque system is provided by cardinal ligament (CL), liococcygeus muscle and ATFP into the side wall of the pelvis.

The 4 main pelvic muscles, pubococcygeus (PCM), levator plate (LP), conjoint longitudinal muscle of the anus (LMA) and puborectalis (PRM), Figures 1&3, act together in different combinations in bladder/bowel closure and evacuation.[13,16-18]

Figure 4A RESTING CLOSED X-ray myograms show the opposite directional movements of the 3 reflex muscle forces, slow-twitch at rest "S". Figure 4B STRAIN, fast-twitch movements during coughing/straining: PCM contracts forwards against pubourethral (PUL) ligament to close distal urethra; LP contracts backwards to tension vagina and rectum in preparation for the downward vector LMA to contract against uterosacral ligaments (USL) to rotate bladder around PUL for urethral closure, and around a contracted PRM for anorectal closure. Figure 4C SQUEEZING UPWARDS. It is clear from Figures 1 and 3, that PRM is sited below LP and is lifting it upwards above the horizontal co-ordinate during squeezing (See "squeezing segment VIDEO3, defecation). Figure 4D MICTURITION PCM relaxes, LP/LMA unrestrictedly pull open the posterior urethral wall to exponentially lower resistance to urine expulsion the 4th power of radius (Poiseuille's Law)[21], micturition VIDEO2 https://www.youtube.com/watch?v=eiF4G1mk6EA&feature=youtu.be

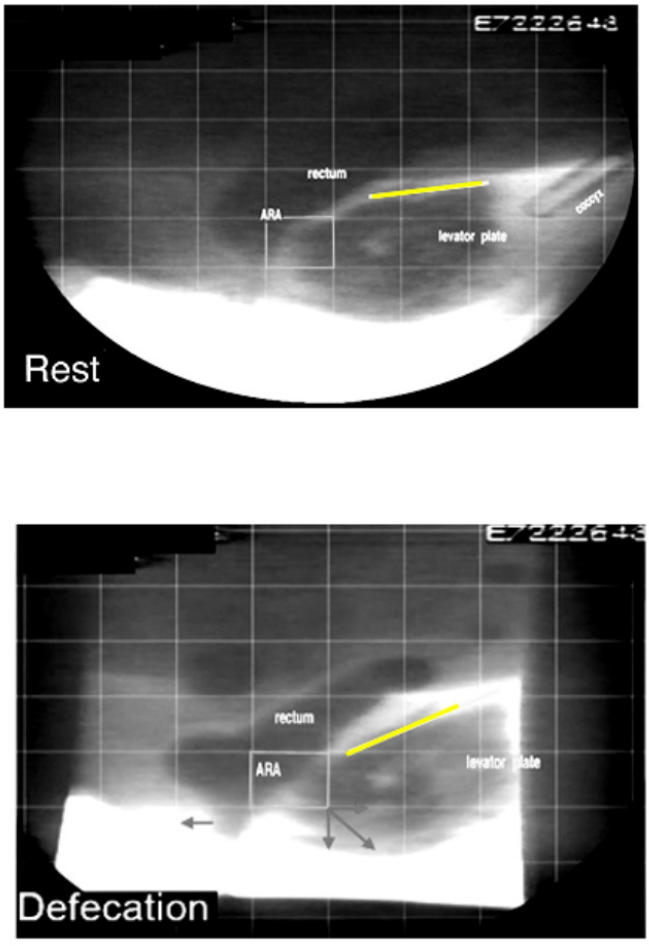

Figure 5. Rest: ARA is the anorectal angle at rest. The yellow line marks the superior border of levator plate.
Defecation: Note attachment of levator plate (backward vector) to rectum just above ARA as in the anatomical dissection, Figure 3. Note angulation downwards of the anterior part of levator plate (yellow line) by the downward vector LMA (conjoint longitudinal muscle of the anus, Figure 3), to open out the posterior wall of the rectum, effectively doubling outlet tube diameter.

VIDEO4. Note lateral expansion of the rectum. By permission Dr Ilario Froehner Jnr.

Defecation

Figure 5 Puborectalis muscle (PRM) relaxes. This allows the backward/downward vectors LP/LMA to pull open the posterior rectal wall. External opening of the anorectum exponentially lowers the resistance to expulsion of feces by the 3rd power of the radius.[22] See defecation VIDEO3 https://youtu.be/MS82AZoWn7U

Anatomical Perspective-some Explanatory Comments

There are many difficulties in reporting any anatomical studies. Anatomical dissections give highly variable, complex findings, very different for every dissection. In the hiatus alone, Shafik[1] noted three distinct pubococcygeus crural patterns, direct insertion,[18] crossover and "scissors.[3] In spite of attempts to "standardize" nomenclature, major differences remain. Purported functions are at best hypotheses. Some traditional names are admittedly inaccurate, e.g., "anococcygeal raphe"[1] or insufficient, "puboccygeus" as it should include iliococcygeus.[2,7] Shafik[1] hypothesized bladder/bowel function.[1] Petros implicated ligaments in normal function and damaged ligaments in causation of dysfunction.[13-19] Shafik[1] defined the levator hiatus as a 3D structure, which he called "levator tunnel", enclosed by levator muscles, which he called "levator sling", both surrounded by puborectalis muscle Figure 1. Courtney also described these structures, but named them differently. Shafik's[1] "anal sling", a conjoint striated/smooth was described by Courtney[2] as the posterior "longitudinal muscle of the rectum" and by Petros as the "longitudinal muscle of the anus (LMA)" to distinguish it from the longitudinal smooth muscle of the rectum which joins the LMA as the conjoint muscle. Shafik[1] and Courtney[2], described structures equivalent to puboperineus and puboanalis, as offshoots from PCM or PRM. They described in detail, the complex insertions of PCM, PRM, LMA into the 3 anal sphincters, coccyx, lower sacrum and intermuscular connections, fibres extending to the urethral rhabdosphincter and bulbocavernosus. Whether extensions or actual muscles, all must have an important role in function, as the pelvic floor functions holistically. For example, MRI observations of anterior wall of the anorectum being pulled forwards to open out the anus during defecation,[20] were attributed wholly to forward contraction of PCM. Specific prolongations as described to the anal fascia and inter-sphincteric fascial structures ("puboanalis") would more directly assist this action. A direct insertion into the perineal tendon, as described by Courtney[2] (now "puboperineus") would play an important role in stabilizing the pelvic structures and may further explain the downward movement of the distal urethra seen on straining.[17,18] Cadaveric dissections, dynamic ultrasound, Berglas and Rubin's[4] X-ray myogram methods, Figures 4, 5, (VIDEOS 1-3) were used to correlate organ displacements during bladder/bowel closure and evacuation, with pelvic muscle actions.[13-22] More recent innovative anatomical studies, using high definition 3D/4D ultrasound and MRI studies, many linked to cadaveric or mathematical models,[6-12] promise to take interpretations of pelvic muscle anatomy to yet another, higher, level of understanding.

DISCUSSION

The research questions are discussed seriatim.

Research Question 1: "What is the structure of the hiatus?" LH is a variable space 2-3 cm in diameter[1], bound by a collagenous hiatal ligament, Figure 2. LH is usually in a contracted state,[4] as it is important to minimize its diameter to support the abdominal contents. A fine collagen/elastin connective tissue layer across the LH,[1] confirmed by live dissection of the anterior part of LH by the author,[27] prevents abdominal contents slipping through.

Research Questions 2-4: "What is the function of the muscles around the hiatus? Which anatomical structures dilate LH on straining?" Expansion of the LH laterally is necessary for childbirth and to open out the anorectum prior to defecation, Figure 5, VIDEOS 3&4. LH expansion is not pathogenic as implied by many authors. Figure 1 shows the magnitude of distension required for childbirth, 10 cm. As regards defecation, an LH diameter as little as 2 cm,[1] has to expand considerably to allow evacuation of large Bristol type 2 stools.[28] The horizontal attachments of iliococcygeus muscle to levator crura, and crisscross pattern of the "anococcygeal raphe" behind the rectum Figure 1, allow the hiatus to expand LH laterally to enable expulsion of large fecal stools. Using an LH area at rest of approximately 12 sq cm and observations of the rectal diameter doubling during defecation, fig5, a "normal" woman with no prolapse would need to dilate her LH to a minimal 45 sq cm to allow expulsion of feces. This is far beyond the arbitrary 25 sq cm cut-off limit for pathogenic expansion suggested by some experts. Rectal expansion exponentially decreases resistance to fecal evacuation[20,22] enabling defecation without obstruction.

A Hypothesis for a Resting Expanded LH by a Sagging Hiatus:

In 1956, Berglas and Rubin[4] explained that, as a pelvic floor sags, the crura lengthen, and the hiatus enlarges. Sagging of levator muscles may be caused by nerve damage, as demonstrated with EMG studies[29] and by computer simulation,[30] muscle damage[31] elongated entheses.[32] Figure 2 graphically shows how a head moving down the birth canal can damage the ligamentous parts of the holistic "monocoque" organ support system.

Is it Anatomically Possible for a Widening Levator Hiatus to Cause POP?

In both the cadaveric and schematic images, Figure 2, the uterus, and therefore, its level 1 ligament supports (CL and USL), are well above LH, so it is impossible for an LH to cause POP, *unless the head has also damaged CL and USL at the same time*.

Research Question 5: How can the "strong association" between clinical signs of prolapse and symptoms, be explained anatomically?

Proof that POP and symptoms were related was surgically demonstrated by Inoue et al.[33] with 5 year and 10 year[34] data. Inoue used the TFS tensioned minisling for holistic ligament repair (mean 3.2 per patient).[33,34] Sixty-eight women had multiple POP defects cystocele (n=61), uterine prolapse (n=59), rectocele (n=35), plus SUI (n=28), with at least one stage 3 or 4 defect per patient.[33] The change in cure rates of symptoms and prolapse is summarized in Table 1.[33]

Research Question 6: How can the LH/prolapse hypothesis be proved or disproved? The acid test for this question was provided by some of its most passionate proponents for causation,[35-37] who directly tested the hypothesis with a 3x25 cm mesh sling around the puborectalis muscle to limit PRM expansion. The sling was performed as a 2nd operation on women immediately after they had undergone a primary operation for prolapse. A pilot study, 115 women between 2010-2,[36] and an RCT, 221 women, reported in 2021[37] demonstrated convincing decrease in LH expansion, but it did not prevent POP recurrence: 66% at 2 years for the pilot study[36] and 79% for the RCT.[37] Despite the two operations performed for prolapse, the improvement in POP and LUTS recorded was low,[36,37] far inferior to the ligament repair method.[33,34] Five year data,[33] Table 1, and 10 year data for 960 patients, 3100 TFS implants,[34] using TFS ligament repair methods based on the 3 level pelvic organ ligament support system,[34] validate the hypothesis that POP and LUTS were caused by ligaments, not hiatal distension.

CONCLUSIONS

Association is not causation. Overexpansion of the hiatus causing prolapse and LUTS is a failed concept. This was irrefutably demonstrated surgically: A confining 3 cm by 25 cm mesh placed around the puborectalis muscles prevented hiatal expansion on straining, but registered a 79% prolapse recurrence at two years with a level one RCT study.[37]

The relationship between hiatal expansion, prolapse and LUTS causation was anatomically explained by a holistic ligament-based "monocoque" structural system tensioned by reflex muscle forces. Immediately prior to birth, the collagen of the hiatal and pelvic ligaments depolymerizes to lose 95% of its strength; the head stretches the intrahiatal organs and structures as it descends down the birth canal. Failure to regain their original length and strength explains expanded LH, prolapse and LUTS.

Table 1. Lower und upper 95%-confidence intervals for the observed relative frequencies of Prolapse, Urgency, Nocturia, Day time frequency, Dragging pain and Fecal incontinence after certain time intervals with the tests results by testing Ho: $p \leq p_0$ vs. H1: $p > p_0$.
"*", "#" and "/" means significant p-values when p_0 is setting equal to 0.80, 0.75 and 0.60, respectively. With other words these symbols depict that the observed cure rates are significantly higher than 0.80, 0.75 and 0.60 respectively ($p<0.05$; Binomial tests)

Time after TFS	Cure of prolapse		Cure of urgency		Cure of nocturia		Cure of day time frequency		Cure of dragging pain		Cure of dysuria		Cure of fecal incontinence	
12 mths	62/68		30/31		17/18		30/32		13/14		35/38		16/18	
Observed cure rate (in %)	91.2%		96.8%		94.4%		93.8%		92.9%		92.1%		88.9%	
95% (lower CI; upper CI)	0.877	0.946	0.936	0.999	0.890	0.998	0.895	0.980	0.860	0.997	0.877	0.965	0.815	0.963
Test results of Ho: p≤0.80 vs. H1: p>0.80	*		*		#		*		§		*		§	
24 mths	57/65		25/30		11/17		26/29		14/15		26/28		12/15	
Observed cure rate (in %)	87.7%		82.3%		64.7%		89.7%		93.3%		92.9%		80%	
95% (lower CI; upper CI)	0.836	0.918	0.765	0.901	0.531	0.763	0.840	0.953	0.869	0.998	0.880	0.977	0.697	0.903
Test results of Ho: p≤0.80 vs. H1: p>0.80	*		§		ns		§		§		#		ns	
36 mths	48/58		23/24		14/23		27/30		9/10		25/26		6/7	
Observed cure rate (in %)	82.7%		95.8%		60.9%		0,900		0,900		96.2%		85.7%	
95% (lower CI; upper CI)	0.778	0.877	0.918	0.999	0.507	0.710	0.845	0.955	0.805	0.995	0.924	0.999	0.725	0.989
Test results of Ho: p≤0.80 vs. H1: p>0.80	§		*		ns		#		§		*		ns (?)	
48 mths	42/50		18/20		8/17		13/19		6/6		22/23		5/5	
Observed cure rate (in %)	0.84		90.0%		47.1%		68.4%		1,000		95/6%		100%	
95% (lower CI; upper CI)	0.788	0.892	0.833	0.967	0.350	0.592	0.578	0.791	1.000	1.000	0.914	0.999	1.000	1.000
Test results of Ho: p≤0.80 vs. H1: p>0.80	§		§		ns		ns		§ (?)		*		ns (?)	

Yellow marked fields in table 2 means, that we have to pay attention in the interpretation of significances, because for these fields the corresponding sample sizes are too small. From Inoue[32] by permission. CI: confidence interval

ETHICS

Peer-review: Internally peer-reviewed.

DISCLOSURES

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