Review

A practical update on functional and dysfunctional anatomy of the female pelvic floor - Part 2 Dysfunction

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Abstract: Background The Integral Theory System considers pelvic organ prolapse (POP), pain, bladder & bowel dysfunctions to be mainly caused by laxity in up to 5 suspensory ligaments and their vaginal attachments. *Aim* To define the role of ligaments in normal function (Part1), then dysfunction and principles of surgical cure (Part 2). *Methods* The role of pelvic ligaments and muscles in normal bladder & bowel closure, evacuation, central and peripheral neurological control is analysed.
Results Normal function Ligaments stretch minimally, vagina stretches extensively during coughing, straining, squeezing, micturition, defecation. Competent ligaments suspend organs and act as insertion points for 3 striated muscle forces. These act in opposite directions to close and open urethra & anorectum, stretch organs to prevent inappropriate activation of micturition and defecation reflexes. Dysfunction Ligaments must be competent, otherwise the muscles which contract against them lengthen and weaken. A cascade of dysfunctions follow from elongated ligaments: prolapse; muscles cannot close urethral and anal tubes (incontinence) open them (emptying problems) or stretch organs sufficiently to support stretch receptors which may fire off prematurely to activate the micturition reflex (urge incontinence, frequency, nocturia) or defecation reflex (fecal incontinence). Conclusions Part 1 demonstrated that competent ligament insertion points are required for the 3 directional forces which control mechanical closure and evacuation and the neurological feedback mechanisms for defecation and micturition reflexes. Lengthening of the sarcomere due to ligament laxity was considered the ultimate link between loose ligaments and dysfunctions in these organs.

Keywords Loose ligaments; Integral Theory; Stress incontinence; TFS; Chronic pelvic pain; OAB; Nocturia; Fecal incontinence.

INTRODUCTION

Part 1 analysed the crucial role of pelvic suspensory ligaments in normal structure and function of bladder and bowel. Key to normal closure (continence) and opening (emptying) of the urethra and anal tubes were three oppositely acting directional muscle vectors. These contracted against firm suspensory ligaments. The same oppositely acting muscle forces controlled urge incontinence by stretching the organs to provide a firm support for peripheral stretch receptors which control the micturition and defecation reflexes.
The aim of Part 2 is to analyse the anatomical pathways from childbirth, to loose ligaments, to prolapse, to organ dysfunction, to a ligament based diagnostic system and finally, ligament based TFS surgical cure.

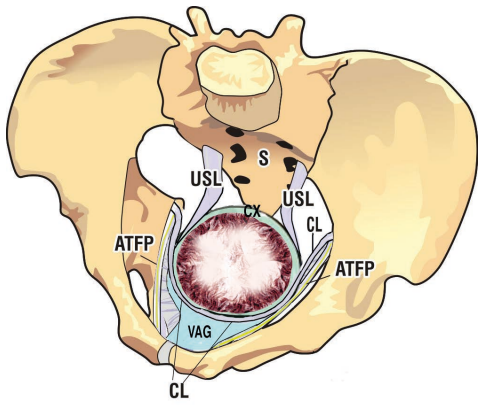

Figure 1. Ligaments are stretched by the head at full dilatation (10 cm). Birth pressure at 10cm dilatation on the cervical ring may overstretch uterosacral ligaments (USL) and cardinal (CL) ligaments. Extension or tearing of these attachments and vagina (vag) from cervix (CX) may cause uterine/apical prolapse, cystocele anteriorly, high rectocele and enterocele posteriorly. The perineal body may be damaged and separated to cause low rectocele (perineocele) and descending perineal syndrome as the head exits the birth canal.

Pathways to ligament elongation and prolapse

Fundamental to the Integral Theory's concept of dysfunction is ligament elongation and weakening mainly from childbirth, age and menopause[1].
Collagen depolymerizes to lose 95% of its strength prior to labour, allowing the cervical ring, ligaments and vagina to stretch considerably during birthing, fig1. The pelvic diameter is only 12-13 cm. Full cervical dilatation is at 10cm, where very considerable tissue stretching occurs: ligaments and vagina stretch, are pushed laterally and may remain stretched post-partum; vaginal attachments may rupture. The end result may be prolapse and dysfunction of bladder/bowel and chronic pain.

Pathway to uterine prolapse pathogenesis and surgery

Fig. 2 is self -explanatory. Uterine prolapse is clearly caused by weak and elongated cardinal (CL) and uterosacral ligaments (USL), not vagina. The only way uterine prolapse, fig. 2. can be cured is to shorten and strengthen CL and USL. 'Native tissue repair' of the vagina will not strengthen the ligaments, nor will a mesh. The ultimate proof that the vagina is not a structural organ was recently provided by the Lancet Prospect RCT[2], where native tissue repair of the vagina was compared against vaginal mesh repair. Both methods had >80% failure rate at 6 months. In contrast, The

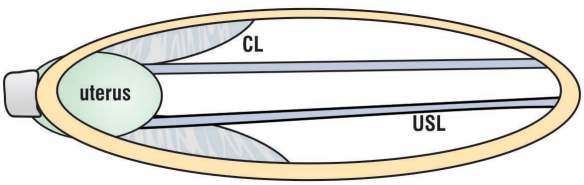

Figure 2. Both CL and USL are elongated in uterine prolapse.
It is self-evident that loose cardinal (CL) and uterosacral (USL) ligaments will elongate to cause uterine prolapse, and that only shortening and strengthening

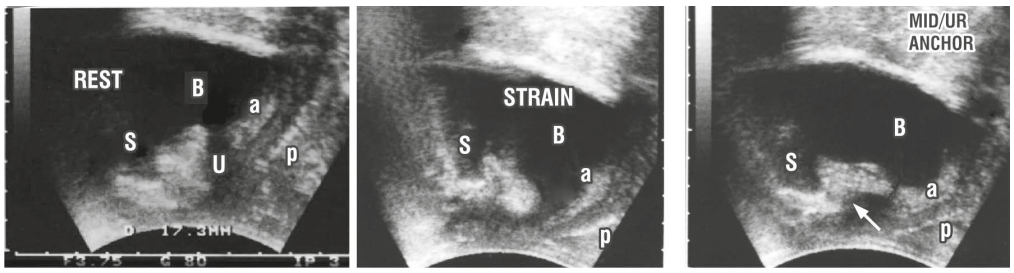

Figure 3. Direct ultrasound proof of Gordon's Law applied to lax pubourethral ligament 'PUL'[10].
Left figure, 'REST' S=symphysis; U=urethra; B=bladder; a=anterior vaginal wall; b=posterior vaginal wall.
Middle figure 'STRAIN' Inability of PUL to support posterior urethra allows the posterior pelvic muscles to stretch the vaginal walls 'a' and 'b' backwards; this action pulls open the posterior urethral wall. The urethra opens out proximally (funnelling) and distally.
Right figure 'MID/UR ANCHOR' When the PUL is shortened by pressing upwards with a hemostat (arrow), the strength of the muscle forces is restored; 'a'&'b' visibly tension; distal and bladder neck closure are restored.
Anatomical pathway to the above events In the middle figure (lax PUL), all 3 directional forces which act on PUL lengthen and lose contractile strength. Once PUL length is restored by the hemostat (white arrow), all 3 muscles can now 'grip' and close the distal urethra and bladder neck (right image). By permission of Prof P Petros.

Kamakura[3] and Yokohama[4] units reported surgical >90% cure rate for 3rd and 4th degree POP at 12 months, falling to 79.0 at 60 months[5] following TFS ligament repair.

Anatomical pathway to cystocele pathogenesis and surgery

The attachment of CL and anterior vaginal wall to the anterior cervical ring (fig1), may be dislocated or torn to cause a 'transverse defect' or 'high cystocele'. In our experience this defect is responsible for 80% of all cystoceles. A cardinal ligament TFS sling simultaneously shortens and reinforces CL and 're-glues' the anterior vaginal wall to the cervical ring[6]. If the cystocele defect is more extensive, for example a central defect due to either a dislocated pubovisceral muscle or from a lax ATFP, the TFS ATFP 'U Sling' can re-attach the muscles and tissues distally to the ATFP insertion point just behind symphysis pubis[7].

Anatomical pathway to rectocele causation and repair

The perineal body 'PB' supports the lower half of the posterior vaginal wall[8]. PB is suspended from the descending ramus by the deep transversus perinei ligaments. If these stretch or are torn, PB stretches and the rectum protrudes. The TFS PB repair shortens and tightens the deep transversus perinei ligaments; the laterally displaced parts of PB are approximated; the rectum is displaced back to its normal position.

Gordon's Law- the ultimate pathway from ligament looseness to muscle dysfunction and symptoms

The theory states that it is mainly loose ligaments which cause chronic pain, bladder & bowel dysfunction. Part 1 demonstrated how three opposite striated muscles contract against the suspensory ligaments to open or close the urethral and anal tubes. These directional muscle actions are co-ordinated by the cortex as part of the neurological reflexes which close urethra and anus (continence) and evacuate them, (micturition and defecation reflexes). A loose ligament effectively lengthens the muscles which contract against it. This weakens the muscle force according to Gordon's Law.
Gordon's Law is the key to understanding the causation of chronic pelvic pain, bladder and bowel dysfunction. It states *"A sarcomere contracts optimally over a short length only. Lengthening or shortening the contractile length results in a rapid loss of contractile strength"*[9].
Because a ligament is the effective insertion point of the muscle, if the ligament is loose, the muscle lengthens accordingly and its contractile strength weakens.

Ligament length is critical for anatomy and function- a practical objective demonstration of Gordon's Law

A hemostat (arrow, right frame, fig. 3) pressed upwards immediately behind the symphysis 'S', effectively shortens PUL, prevents the funneling seen in the middle frame, restores the contractile strength of the muscle vectors to close the urethra and bladder neck, right frame, fig. 3.
Fig. 3 objectively demonstrates the anatomical pathway to continence control by midurethral hemostat pressure demonstrated and predicts cure by the midurethral sling operation.

Muscle or ligament- which is the main cause of symptoms?

A blinded study in 47 patients[11], 46 with histologically proven pubococcygeus muscle damage had a midurethral sling procedure which involved a sling placed at midurethra to reinforce the pubourethral ligament 'PUL'; 89% became continent on the day after the procedure, suggesting that the cause of the problem was ligament rather than muscle related.

The anatomical pathway to urinary stress incontinence, low urethral pressure and fecal incontinence.

Fig. 4 demonstrates the effect of a loose pubourethral ligament 'PUL' when it lengthens from normal length 'E' by 'L'. According to Gordon's Law, if PUL is loose, the intraurethral area 'U' will expand; the force of the closure muscles m.pubococcygeus (PCM) will weaken and the urethral tube 'U' cannot be adequately closed. The rhabdosphincter (which is responsible for urethral pressure measurement) cannot contract sufficiently, so a low maximal urethral closure pressure 'MUCP' or Valslava Leak Point Pressure 'VLPP' may be recorded, as a consequence of enlarged intraurethral area (Pressure = Force/Area). The intra urethral resistance to intra abdominal pressure increase is lowered exponentially, inversely by the 4th power of the radius (Poiseuille's Law) and the patient may lose urine on effort.
We propose that a similar scenario applies for anorectal closure: if PUL or USL are loose, the muscle vectors which contract against them weaken, the anal tube cannot be closed and feces may leak (fecal incontinence).

The anatomical pathway to 'mixed incontinence'

'Mixed incontinence' is co-occurrence of USI and urge incontinence. Urinary stress incontinence (USI) and urge are two entirely different symptoms. USI is caused by PUL laxity. When USI and urge co-occur, if the cause for the urge is inability of the forward vectors to support the stretch re-

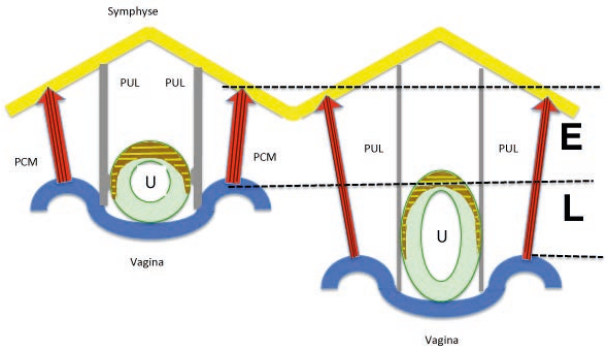

Figure 4. Stress incontinence and ISD interpreted by Gordon's Law. Schematic coronal view at midurethra.
Left figure: continent patient. The pubourethral ligament 'PUL' is of normal length 'E', as is the pubococcygeus muscle (PCM) and rhabdosphincter.
Right figure: patient with urinary stress incontinence PUL is loose, elongated by length 'L'. PCM and rhabdosphincter secondarily elongate to 'L' and lose contractile strength. U= urethral cavity.

ceptors because of PUL laxity, urge will also be cured by a midurethral sling. This explains simultaneous cure of urge in 50-60% of patients having a midurethral sling for USI. If the cause of urge is lax cardinal or uterosacral ligaments constipation (see algorithm, fig. 10), the urge won't be cured simultaneously with USI cure. In that instance, patients will likely have other posterior symptoms such as pain, abnormal emptying, nocturia.

The anatomical pathway to 'double incontinence'
'Double incontinence' is co-occurrence of USI and fecal incontinence. If PUL is loose, the directional closure forces may weaken to cause USI and sometimes fecal incontinence also, because the LP part of the LP/LMA vectors which close the anorectal angle contracts against PUL. Hocking demonstrated cure of both conditions by shortening and reinforcing PUL with a midurethral sling in 90% of cases[12].

The different pathways to retropubic and transobturator midurethral sling closure
Though many studies indicate that the transobturator approach for midurethral sling surgery gives equivalent results for cure of urinary stress incontinence as the retropubic approach, it is generally acknowledged that the retropubic is superior in patients with ISD (intrinsic sphincter defects) or those requiring repeat surgery for USI. A midurethral retropubic tape automatically grips the lateral sides of the urethra before proceeding behind the symphysis (PS), fig5; a retropubic tape will reinforce PUL and the pubovesical ligament (PVL) to restore both distal and bladder neck closure mechanisms as proven by transperineal ultrasound in fig. 3. The bladder is rotated around the precervical arc of Gilvernet by LP/LMA vector forces to effect bladder neck closure at '0-0'.
The TOT provides an anchoring point at the base of PUL for the posterior rotating closure forces LP/LMA, fig5. The TOT relies on an intact PVL and precervical arc for optimal bladder neck closure, fig5. However, a TOT cannot repair a damaged PUL or PVL, nor can it grip the sides of the urethra, potentially diminishing the traction required to fully enact the two closure mechanisms, distal and proximal. As the ultimate pathway to continence is exponentially determined by the internal resistance to flow by Poisseille's Law (inversely by the 4th power of radius narrowed), precise attention to surgical methodology as previously explained[13] may account for the high cure rates for ISD reported by Nakamura et al. Poiseuille's Law can work in reverse. A loose tape and failure to repair the distal urethral closure mechanism may explain suboptimal cure of USI and ISD.

Anatomical pathways to pain, bladder, bowel dysfunction from loose cardinal/uterosacral ligaments (CL/USL).
It is our view that the cardinal/uterosacral ligaments (CL/USL) are the most important ligaments in the pelvis. CL/USL are the main supports of the uterus and vaginal apex; they are the anchoring point for the backward/downward vectors which are critical for control of bladder & bowel function. Laxity in CL/USL will cause uterine prolapse. Because the control mechanisms for fluid flow are exponentially determined (Poiseuille's Law), even minor prolapse, fig. 6, may weaken the directional vector forces which pull against CL/USL and may affect the following functions of CL/USL
- *Control of bladder and bowel urgency and frequency.*
- *Control of nocturia.*
- *Control of bladder and bowel evacuation (obstructive defecation 'ODS').*
- *Control of anorectal closure (continence).*
- *Control of chronic of pelvic pain.*

The pathways to these dysfunctions will be examined in turn.

Anatomical pathways to 'obstructive micturition or defecation' (organ emptying problems)
X-ray video studies, Part1, demonstrated an external striated muscle opening mechanism for both bladder and anorectum. It is known that the resistance within a tube is inversely related to the radius (Poiseille's Law). A loose USL (fig. 6), may result in weakening of the urethral or anorectal LP/LMA opening forces. The bladder detrusor or rectum have to contract against an unopened tube. This is perceived by the patient as 'obstructed micturition' or 'obstructed defecation', with symptoms such as 'feeling bladder has not emptied, 'stopping and starting', multiple emptying, post-micturition dribble, raised residual urine[14] and for bowel, constipation

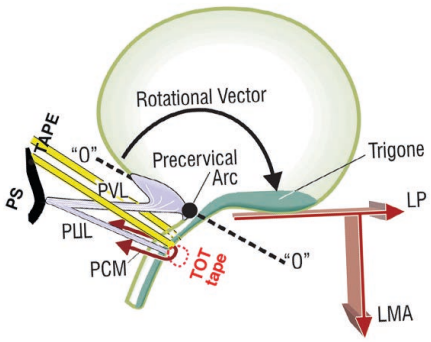

Figure 5. The anatomical difference between retropubic and TOT USI operations.
Patient in sitting position. The TOT tape rests in the horizontal position below midurethra to provide a firm rotation point for the posterior rotational vector forces which close bladder neck, m. levator plate (LP) and the conjoint longitudinal muscle of the anus (LMA); likewise with the retropubic operation; the retropubic tape (yellow) proceeds behind the symphysis (PS) to reinforce the pubourethral (PUL) and pubovesical ligaments (PVL) if they are loose. The reinforced PUL restores two urethral closure mechanisms, proximal and distal, as in the ultrasound fig. 3. PUL provides a firm anchoring point for the posterior rotating vectors which effect bladder neck closure; it anchors the anterior vector m.pubococcygeus (PCM) which stretches the suburethral vagina forwards to close the distal urethra.

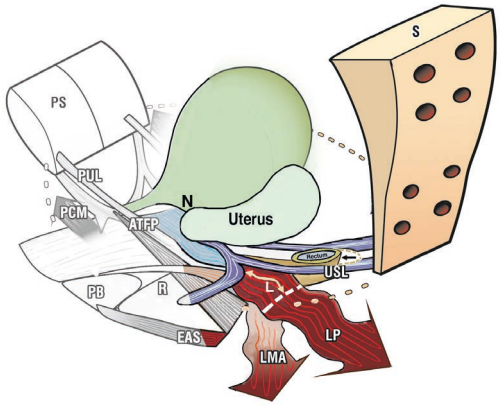

Figure 6. Potential consequences of loose uterosacral ligaments as interpreted by Gordon's Law.
View from above. The uterus has prolapsed to 1st degree. The USLs have elongated by 'L', as have LP and LMA. The rectum also has descended, by virtue of its attachments laterally to the elongated USL as indicated by the very small arrow behind rectum. The large wavy arrows signify diminished contractile strength of LP/LMA.

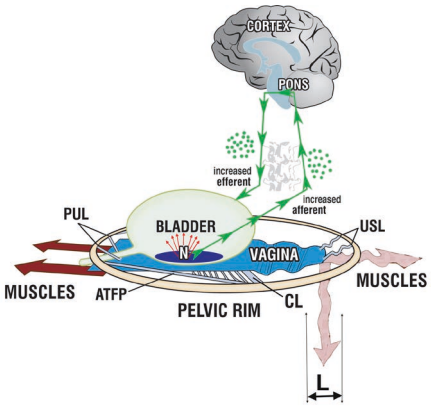

Figure 7. Urge incontinence as interpreted by Gordon's Law
The uterosacral ligaments (USL) lengthen 'L' and are unable to suspend the vagina adequately. The downward/backward muscle forces (arrows)* lengthen by 'L' and weaken. The vagina cannot be stretched sufficiently to support the stretch receptors "N". "N" fire off increased afferent impulses at a low bladder volume and this is perceived by the cortex as urgency. If the quantum of afferents is sufficient to activate the micturition reflex, the efferents are activated; the forward muscles relax; the backward muscles open out urethra; bladder contracts; the patient may uncontrollably lose urine ("urge incontinence").
*the wavy form and pink colour of the arrows denote weakened muscle contractile force.

or obstructive defecation (ODS). Shortening and reinforcing CL/USL by TFS restores prolapse and the external opening mechanism with symptom and residual urine improvement for bladder[14] and bowel[15].

Anatomical pathway to symptoms of 'overactive bladder'- urge, frequency, nocturia. Inability of the weakened LP/LMA muscles (fig. 6), to stretch the vagina or anorectum sufficiently to support the stretch receptors 'N' may cause them to fire off at a low volume to activate the micturition reflex. The cortex perceives this as urinary 'urgency', frequency (fig. 7) and at night, nocturia (fig. 8).

Anatomical pathway to chronic pelvic pain
The role of USLs in producing chronic pelvic pain was described in detail by Heinrich Martius in 1938[16] and was re-discovered independently by Petros[17].
Chronic pelvic pain is perceived in the various nerve distributions,T12-L1, S 2-4 (fig. 9): lower abdomen, groin, lower sacrum[18], introitus[19], paraurethral[20], interstitial cystitis[21], deep dyspareunia[21].
Inability of the weakened muscles to tension the uterosacral ligaments may cause unsupported nerve plexuses within the USLs to fire off (fig. 9). Objective proof of USLs as the pathway to chronic pelvic pain origin was obtained by different types of 'simulated operations**.
Wu et al. reported relief of pelvic pain and suburethral tenderness by insertion of the lower part of a bivalve speculum to support the posterior fornix[20]. Bornstein relieved vulvodynia pain by local anesthetic injection into the cervical part of the ULSs[19] as did Petros in 3 patients with Interstitial Cystitis, abdominal pain and suburethral tenderness[21]. Gunnemann reversed anterior rectal wall intussusception with a cyslindrical vaginal pessary, inserted under ultrasound control[22]. Another method, used to confirm the role of USL in nocturia causation, is to place a large tampon in the posterior vaginal fornix overnight.
** Mechanically supporting ligaments as in fig. 10 and observing change in symptoms.

Pathway from dysfunction to diagnosis- the Pictorial Algorithm.
The sum total of the pathways to prolapse and symptoms is summarized by the Pictorial Algorithm (fig. 10). The Algorithm relates the damaged ligaments to actual symptoms. Thus symptoms marked by 'X' in fig. 10 can indicate which ligaments are damaged. For example, urinary stress incontinence indicates it is PUL which is damaged. The presumptive ligament damage based on these symptoms (fig. 10), is confirmed by vaginal examination: each of the damaged ligaments has specific anatomical criteria detailed in the textbook "The Female Pelvic Floor"[24]. 'Simulated operations' apply mechanical support for specific ligaments[25] and the results are observed, either clinically by improvement in USI urge, objectively by transperineal ultrasound (fig. 3), or by 'interventional' urodynamics (raised intraurethral pressure)[24]. There is no correlation between degree of prolapse and symptom severity[26]. Fig10 is an actual case example with positive symptoms marked in.

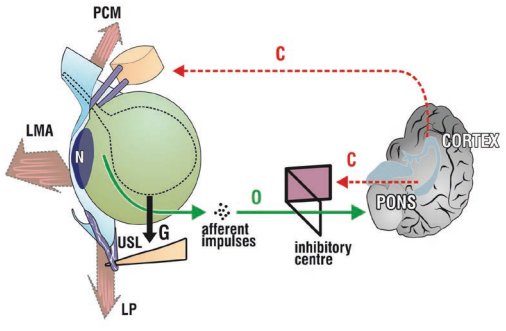

Figure 8. Mechanical origin of nocturia (patient asleep).
Pelvic muscles (large arrows) are relaxed. As the bladder (broken line outline) fills, it is distended downwards by gravity 'G'. If the uterosacral ligaments (USL) are weak, the bladder base continues to descend; at a critical point, the cortical closure reflex 'C' is overcome; the stretch receptors 'N' now activate the micturition reflex: the patient is awakened by a feeling of urgency (nocturia); PCM is actively relaxed by the cortex. If the micturition reflex is not adequately controlled, LP/LMA contract to open the urethra and the detrusor contracts; the patient may lose urine on the way to the toilet.

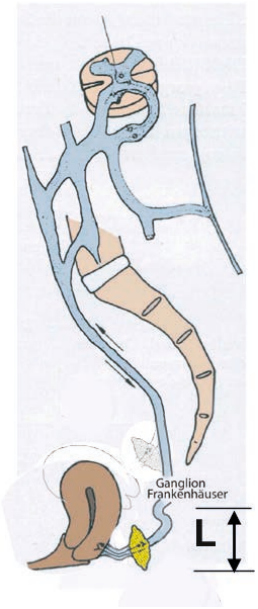

Figure 9. Pathogenesis of chronic pelvic pain.
The Ganglions of the Frankenhauser and the Sacral Plexuses are supported by uterosacral ligaments (USL) at their uterine end. 'L' indicates ligament laxity as per Gordon[9]. The posterior directional forces are weakened and cannot stretch the USLs sufficiently for them to support the nerves. The nerves may be stimulated by gravity or by the prolapse or by intercourse to fire off and be perceived as pain by the cortex.

Consequences of different strength of ligaments and vagina for pelvic surgery technique

Interpretation of the life-long experimental work of Yamada on tissues[27] is the key to understanding the ligament-based rules of surgical reconstruction according to the Integral Theory System[1]. Yamada demonstrated that the breaking strain of ligaments was approximately 300 mg/mm^2 , that of vagina, 60 mg/mm^2 [27]. This means ligaments are primarily structural, the vagina not. Xray video studies[28] confirmed that ligaments do not stretch significantly during effort or evacuation. It follows that any reconstructive surgery has to reinforce the structural part of the ligaments, collagen 1. Only an implanted tape can do this[25]. The same xray video studies indicated that the vagina has a minimal structural role. The vagina is an elastic organ which plays an important role in transmitting the vector forces to close and open the bladder[24]. As elasticity cannot be surgically reproduced, it must be conserved. Any excision of vagina reduces the quantum of elastin and collagen 3 available for function. Finally, the uterus is the direct or indirect insertion point for all the ligaments. Hysterectomy requires severing of the descending uterine artery, which is the main blood supply of the proximal ends of the cardinal and uterosacral ligaments. We have strictly followed the 3 rules of surgery which evolve from the above in our TFS surgery:

1. A loose ligament must be shortened and reinforced with thin strips of tensioned tape to create a collagenous neoligament[25]. This was the surgical principle of the original midurethral sling .
2. The vagina must be conserved, not excised.
3. The uterus must not be removed without good cause.

We have found that following these rules vastly diminishes post-operative pain and urinary retention and allows the TFS to be performed as a day or local anesthetic procedure.

The surgical pathway from ligament repair to restoration of structure (prolapse) and symptom cure by TFS

TFS Surgery precisely tensions the ligaments by using tactile feedback by the operator during tightening to sense return of muscle strength. Exactly the same surgical technique is applied to each of the 5 ligaments PUL,ATFP,CL,USL,PB: stretch the uterus downwards to locate the ligament vaginally; make a tunnel through the ligament with dissecting scissors; insert anchor and tape; repeat on the contralateral side; tension the tape. As the laxity 'L' (figs 4-6, 7-9) is removed by the tensioning, the surgeon feels a gradually increasing resistance against the tensioning. This signifies return of muscle strength as defined by Gordon's Law[9]. At this point, the surgeon stops the tensioning.
Once the ligaments have been shortened and reinforced, the directional muscle forces act immediately to restore all the functions dependent on the competent ligaments.

DISCUSSION

TFS ligament surgery shortens and reinforces the ligaments. The tape creates a linear deposition of collagen along the length of the ligament[25]. Shortening and reinforcing the ligament reverses the cascade of dysfunctions described in this work, usually by the day after surgery, with high cure rates for POP and symptoms [3-5, 13, 29-33], as noted in Tables 1&2, Part 1.

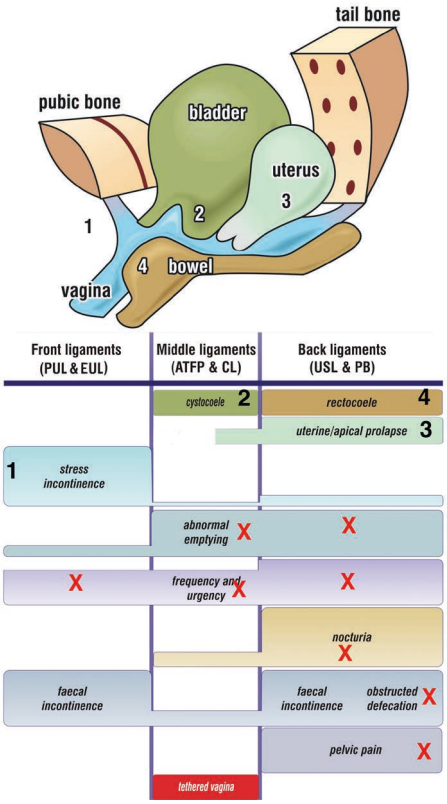

Figure 10. The Pictorial Algorithm
It is a summary guide to causation and management of pelvic floor conditions. The marks 'X' accurately indicate which ligaments are damaged. Because symptoms such as urgency and abnormal emptying may be caused by ligament damage in more than one zone, all rectangles for that symptom should be marked. The area of the symptom rectangles indicates the estimated frequency of symptom causation occurring in each zone. The main ligaments causing the symptoms and prolapse in each zone are indicated in capital letters, two in each zone: PUL 'pubourethral ligament', EUL 'external urethral ligament' (*Anterior ligaments);* ATFP 'arcus tendineus fascia pelvis', CL 'cardinal ligament complex' (*Middle ligaments)*; USL 'uterosacral ligament,' PB 'perineal body'(*Posterior ligaments)*. The numbers in the figure correlate with structural damage and with ligament damage: ; 1: USI; 2: cystocele; 3 uterine prolapse; 4:rectocele. The diagnosis for this patient is cardinal /uterosacral damage and perineal body ligament damage. Thm. NOTE Major symptoms may occur with minimal ligament damage.

CONCLUSIONS

Symptoms may occur as a consequence of minor, barely detectable, anatomical–abnormalities. Jeffcoate, in his 1962 textbook observed that some patients with gross degrees of prolapse had no symptoms at all, while others with minor degrees of prolapse "complained bitterly" of symptoms such as pelvic pain[26]. The explanation for this, as we see it, is the exponential nature of the control mechanisms and patient to patient variation in the sensitivity of the peripheral receptors

DISCLOSURES

Conflicts of Interest: None
Ethics: Not required
Contributions: Conceptualization, writing, revision: all authors

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