A practical update on functional and dysfunctional anatomy of the female pelvic floor - Part 1 Function

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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 & 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 defection 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 defection 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 defection 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: TFS; Integral Theory; Bladder function; Bowel function; Pelvic ligament; Sphincter; Vagina; Stress incontinence; Chronic pelvic pain; OAB; Nocturia; Fecal incontinence.

INTRODUCTION

“Precise, almost mathematical knowledge of anatomy is a highly fertile source of surgical applications, suggesting new techniques and helping to perfect and simplify existing surgical methods, making them less mutilating and more benign and, in short, raising surgery to the rank of true science.”1

It is a fundamental truth that an accurate knowledge of anatomy is a pre-requisite for any surgery to any part of the human body. In conceptualizing this paper, we have been guided by the writings of the great Spanish Anatomist/Urologist Salvador Gil-Vernet (1892-1987)1 and our own practical experience of application of the Integral Theory System over a collective period of more than 60 years.

STRUCTURE

Five main ligaments suspend the organs from the pelvic girdle. They are pubourethral (PUL), arcus tendineus fascia pelvis (ATFP), cardinal (CL), uterosacral (USL) and the deep transversus perinei portion of the perineal body (PB), figs 1, 2. A 6th ligament, external urethral ligament (EUL) attaches the external urethral meatus to the anterior surface of the pubic symphysis. It mainly controls urethral mucosal sealing.2

It is the ligaments which provide suspensory strength. The main structural component of ligaments is cross-bonded collagen 1. According to Yamada1, the breaking strain of ligaments is approximately 300 mg/mm2. Suspensory ligaments do not stretch significantly during the effort of closure and micturition. This is evident on simple inspection of PUL and USL in the dynamic x-rays, fig. 3.

The vagina is a weak elastic organ, breaking strain 60 mg/mm² 3. Its elasticity permits the independent function of the 3 opposite muscle forces (arrows), fig. 3. The vagina’s elastic function is self-evident on simple inspection of fig. 3: the vagina is stretched significantly during urethral closure (straining) and micturition. Though the vagina helps to support the bladder, fig. 3, its role cannot be primarily structural as its breaking strain is only 60 mg/mm², a consequence of the predominance of collagen 3, a more elastic, but weak collagen. Structurally, the vagina acts like an (elastic) plaster board of a domestic ceiling, with cardinal ligaments, ATFP and cervical ring acting as the joists.

Figure 1. The relationship of ligaments, muscles and function. The 5 ligaments which suspend the organs PUL= pubourethral ligament; CL=cardinal ligament; USL= uterosacral ligament; PB= perineal body. ATFP = arcus tendineus fascia pelvis); EUL= external urethral ligament is a 6th ligament which attaches the external meatus to the anterior surface of pubic symphysis (PS); Forward acting muscles: m.pubicoccygeus (PCM), m.puborectalis (PRM), PCM contracts against the pubourethral ligament (PUL). PRM contracts only against symphysis pubis. Backward acting muscles:levator plate (LP) contracts backwards against PUL anteriorly; LMA contracts solely downwards against USLs. N=bladder stretch receptors; R=rectum; EAS=external anal sphincter; PS=pubic symphysis; S=sacrum.
The muscles
The vagina and rectum rest on the pelvic floor muscles. There are four directional striated muscles vectors, fig.1; two are forward acting: m.pubococcygeus (PCM), m.puborectalis (PRM). PCM is attached to distal vagina and contracts forwards against the pubourethral ligament (PUL). PRM contracts only against symphysis pubis. There are two posterior vectors (arrows, fig.1): levator plate (LP) and conjoint longitudinal muscle of the pelvis (LMA). LP is attached to the posterior wall of rectum; it contracts backwards against PUL; LMA inserts into the anterior portion of LP proximally and the external anal sphincter distally; it contracts solely downwards against USLs, figs 1,3.

Muscle function
The opposite directional forces, figs 2&3, have three main functions.
1. They create shape and strength of the organs.
2. They close the urethral and anal tubes (continence) and help to empty them during micturition and defecation.
3. They stretch the vagina to support the bladder stretch receptors ‘N’, and tension USLs to support the nerve ganglions in the uterosacral ligaments.

FUNCTION
A summary of the role of the suspensory ligaments in bladder and anorectal function and dysfunction
The Integral Theory (IT) states that pelvic organ prolapse, symptoms of chronic pelvic pain, bladder and bowel dysfunction are mainly caused by laxity in 5 suspensory ligaments. The ligaments have a dual function: they suspend the organs and act as insertion points for three oppositely acting muscle forces. Lax ligaments weaken these muscle forces so they cannot adequately close the urethral or anal tubes (incontinence), evacuate them (constipation, bladder emptying), or tense the bladder and rectum sufficiently to prevent inappropriate activation of the micturition and defecation reflexes by their stretch receptors (urge incontinence of bladder & bowel). Up to 80% cure/improvement for the above conditions has been achieved following repair of one or more damaged ligaments using precisely positioned TFS tensioned tapes, Tables 1&2. The Integral Theory states “Repair the structure (ligaments) and you will restore the function”.

Normal bladder function
The bladder is a storage container for urine. Continence and evacuation are via the urethral tube. The bladder has 3 modes of function, fig. 3.
1. Resting closed mode, fig. 3, middle figure B. Urethral closure is maintained by vaginal elasticity, urethral elasticity/smooth muscle and slow twitch striated muscle contractions against PUL, ‘S’ acting forwards, backwards, downwards.
2. Effort closed mode fig. 3, left figure A. On effort, fast-twitch forward and backward forces act against PUL in opposite directions (arrows) to close urethra distally and proximally. A 3rd downward force (arrow) pulls down LP. This action rotates the bladder downwards to close (kink) bladder neck.
3. Open (micturition) mode (fig. 3, right figure C). The forward arrow, S1, fig B, relaxes. Two fast-twitch directional forces* pull the posterior wall of urethra backwards. Bladder contracts to empty. Note relative immobility of PUL and USL in fig. 3 and very significant stretching of the vagina in both closure and micturition.

Normal anorectal function

Bladder and bowel have similar closure and opening mechanisms.\(^{14}\)

The rectum is a storage container for feces. Continence and evacuation are via the anal tube. The anorectum has 3 modes similar to those of the bladder in fig. 3.\(^{14}\)

1. Resting closed mode, is maintained by slow twitch striated muscle contraction, organ elasticity/ smooth muscle (figs. 3&4).

2. Effort closed mode is activated by 3 fast-twitch directional forces during straining or coughing (fig. 3) (left frame). The uterosacral ligaments have a key role in anorectal closure. The USLs are attached to the lateral rectal walls by thin ligamentous attachments. LP stretches the rectum backwards against PUL to tension it prior to LMA contraction, fig4. LMA contracts against USL to pull down the anterior part of LP. This downward angulation rotates the rectum around a contracted pubopectalis (PRM) to ‘kink’ the rectum and close the anorectal angle.\(^ {14}\) If either PUL or USL are loose, closure may not occur (fecal incontinence ‘FI’). Hocking reported cure of double incontinence USI and FI with repair only of PUL with a midurethral sling.\(^ {15}\) The Kamakura group reported cure of FI with a cardinal/uterosacral sling operation Table 1

3. Open (defecation) mode is an active process.\(^ {14}\) Defecation is activated by 3 fast-twitch directional forces, LP/LMA posteriorly, fig4 and a forward force possibly PCM or puboanals acting on the anterior anal wall, fig. 5, forward arrow. With reference to fig. 5, the anterior border of the levator plate has been pulled downwards apparently by a downward vector. Levator plate ‘LP’ is clearly shown attaching to the posterior wall of rectum. It contracts backwards towards the coccyx. The posterior wall of the rectum has been stretched downwards and backwards (red arrows) apparently by the resultant of these two vectors. The anorectal angle ‘ARA’ descends into the light green rectangle at 45°. The opening extends all the way down the posterior wall of the anus. The anterior wall of rectum has been pulled forwards to further open out the anal canal. The USLs have a key role in defecation. The USLs are attached to the lateral walls of rectum; the downward vector (arrows) contract against USL to open out the posterior wall of urethra, thus vastly reducing the internal resistance to flow.

Figure 3 Normal bladder/bowel function Video xray, sitting position. Radio-opaque dye has been injected into the bladder, vagina, rectum, levator plate.

Middle figure B At rest, asymptomatic patient. Slow twitch directional forces ‘S’ (arrows) stretch the organs bidirectionally against pubourethral ligaments ‘PUL’ anteriorly and uterosacral ligament ‘USL’ posteriorly to close urethra and anus and to support the bladder base and rectal stretch receptors, preventing activation of the micturition and defecation reflexes. LP=levator plate; U=urethra; B=bladder; V=vagina; CX=cervix.

Left figure Straining A-anorectal closure. The ligaments do not stretch. They anchor the urethra, distal vagina and rectum. USL is angulated downwards. Fast twitch muscles stretch the distal vagina forwards (arrow) to close distal urethra; backward/downward vectors (arrows) contract against PUL and USL to stretch and rotate the proximal urethra, proximal vagina and rectum, around PUL to effect bladder neck closure.

Left figure Straining A-anorectal closure Pubopectalis muscle (PRM, yellow) contracts. The same posterior vector forces (arrows) contract against PUL and USL, to stretch rectum ‘R’ around a contracted PRM and perineal body (PB) to close the anorectal angle and effect anorectal closure.

Right figure C Micturition The ligaments do not stretch. USL is angulated downwards. There is absence of a forward vector which has been relaxed by the micturition reflex. The vagina and rectum are stretched backwards and downwards. Fast twitch backward/downward vectors (arrows) contract against USL to open out the posterior wall of urethra, thus vastly reducing the internal resistance to flow.
In the normal patient, the stretch receptors ‘N’ sense bladder fullness and send afferent signals to the cortex (green afferent arrows); afferent signals are controlled centrally and peripherally by opposite stretching of the vaginal membrane by muscle forces (arrows); the stretched vagina supports the hydrostatic pressure exerted by the urine column. At a critical point, the afferent signals (green arrows) activate the micturition reflex which causes the forward muscles to relax; the posterior muscles contract to open out the posterior urethral wall; the detrusor contracts to empty. A similar feedback control system, opposite stretching of the posterior vectors and puborectalis supports the anorectal stretch receptors (continence). Increased afferent impulses (green arrows) activate the defecation reflex, relax puborectalis, contract the posterior vectors to open out the anorectal angle, contract the rectum to empty.

Figure 6. The trampoline analogy.

At rest The anorectal angle ‘ARA’ to the left of the green square is angled. The anus is closed. The superior surface of the levator plate muscle is almost horizontal.

Defecation mode ARA is opened out by backward and downward vectors LP/LMA (arrows). The anterior wall of anus is pulled forwards (arrow) further opening out the anal canal. Note the insertion of levator plate into the posterior wall of rectum. The resultant vector action (arrow) seems to be opening out ARA. The downward angulation of the anterior border of levator plate is identical with what happens during micturition; conjoint longitudinal muscle of the anus (LMA) pulling down the anterior margin of levator plate (LP).

Control of the micturition, defecation reflexes and pain - According to the Integral Theory, urge incontinence, nocturia (OAB) even urodynamic ‘detrusor overactivity’ are expressions of a prematurely activated but otherwise normal micturition reflex. This concept was uro dynamically validated in 1993: it was demonstrated that the events which occur during micturition and the ‘unstable bladder’ (‘OAB’, ‘DO’) are identical: 1 Sensation of urge; 2. Fall in proximal urethral pressure; 3. Rise in detrusor pressure; 4. Urine loss. The bidirectional stretching of the vagina (fig. 6), acts like a trampoline to support the stretch receptors ‘N’ at bladder base; as the hydrostatic pressure of urine rises, spindle cells in the oppositely acting muscles automatically cause the muscles to stretch the vagina to support ‘N’, thus preventing activation of the micturition reflex. A similar feedback system applies to the anorectum.

It is hypothesized that the nerve plexuses in the distal parts of the USLs, the Frankenhauser and Sacral plexuses, are similarly controlled: the opposite muscle stretching tensions the USLs to support the nerves preventing them from firing off.

Gordon’s Law - the ultimate pathway for understanding how ligament looseness may cause muscle dysfunction and symptoms.

Gordon’s Law (fig. 7) is the key to understanding the causation of bladder and bowel function and dysfunction. It states “A striated muscle contracts optimally over a short length only (‘E’, fig. 7). If the ligaments against which the 3 vector muscles contract are firm, the muscles contract efficiently over a length ‘E’, fig. 7. However, lengthening the contractile length results in a rapid loss of contractile strength.”

If the ligaments against which the three vector muscles contract lengthen by ‘L”, the muscles lengthen accordingly and their contractile strength weakens, from a nominal 80% to 30%, fig. 7.

Figure 7. Gordon’s Law.

A striated muscle contracts optimally over a short length only, ‘E’, red square. Lengthening the muscle ‘L’, results in a rapid loss of contractile force, black rectangle.
CONCLUSIONS
Part 1 examined the key role of competent ligament insertion points for the 3 directional forces which control normal bladder, bowel & nerve function. Part 2 builds on Part 1 to explain which ligaments may be causing the dysfunctions and how ligament shortening can reverse the cascade of events which led to the dysfunction.

DISCLOSURES
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