2 On the anatomical relationship between bladder and bowel function and dysfunction

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Email: pp@kvinno.com
Key words: OAB; faecal and urinary incontinence; posterior fornix syndrome; obstructed defaecation syndrome; urinary retention; chronic pelvic pain
Word count: Abstract 250, Text 4212
Conflicts: None for either author
Funding: Nil

Abstract: We review the emerging evidence supporting our hypothesis that control of bladder and bowel continence and evacuation depends on the same pelvic muscles and the ligaments against which these muscles act. Dysfunction is mainly a consequence of damage to ligaments, connective tissues and muscles and their innervation. We show how a ligament-supportive approach to diagnosis and treatment can bring together the apparently disparate syndromes now treated by 3 separate specialties, urology, gynaecology and coloproctology.

Simplistically, the three female pelvic organs, bladder, uterus and bowel, are storage receptacles connected to the exterior by urethra, vagina or anus. For continence, urethra and anus must be closed at all times. The micturition and defecation reflexes are activated to empty them only when it is socially convenient. These functions are controlled by the central nervous system and applied locally by 3 oppositely-acting muscle forces contracting against competent suspensory ligaments. There are basically 3 clusters of bladder and bowel symptoms deriving from anatomical defects in these local intra-pelvic control systems'; i. e., 1. Inability to maintain closure of the urethral/anal systems causing

leakage of urine or faeces; 2. Inability to evacuate bladder or bowel adequately; 3. Inability to modulate the micturition and defecation reflexes causing bladder or bowel urge symptoms. In addition, there are several pain syndromes associated with structural damage, occurring in adults from childbirth or aging, and in children from structural immaturity. These concepts be used in clinical practice to understand the pathogenesis of bladder and bowel syndromes and to direct their best management.

Introduction

The International Consultation on Incontinence has concluded that, other than stress urinary incontinence, the pathogenesis, inter-relationships, and effective management of disorders of bladder and bowel continence and evacuation are not properly understood (1). Indeed, existing treatments for these pelvic floor dysfunctions are delivered by three different surgical disciplines; urology, gynaecology, and coloproctology (Table1). The treatment strategies used vary widely within each discipline, even for individual symptoms, suggesting they are not consistently effective. In this review we show that, at least in women, the functional disorders listed in table 1 largely share a common pathogenesis and that detailed assessment of the functional deficit should determine treatment (2). Our current understanding of these disorders represents a development of the Integral Theory of female incontinence described in 1990 by Petros and Ulmsten (3), that took into account new concepts of muscular and ligamentous integrity within the pelvic floor in relation to evidence for direct muscular and ligamentous injury and, more recently, recognition of neurogenic muscle damage during childbirth due to stretch injury (4-6), associated with intra-pelvic ligamentous laxity (7). This neurogenic muscle weakness is often progressive (8). We have termed this the 'musculo-elastic hypothesis for pelvic floor damage', stressing that the only remedial action that has been shown to be effective is minimally invasive, surgical reinforcement, by ligament plication or slings. It is possible that exercise-based physiotherapy might also induce neocollagen formation. The importance of ligamentous laxity in uterine prolapse, first suggested by Rivington in 1885, was rejected by his contemporaries (9).

Table 1. Pelvic symptoms

Urologist	Gynecologist	Coloproctologist
BLADDER	UTERUS/VAGINA	UTERUS/VAGINA
• Urge • Frecuency • Nocturia • Painful bladder • Paraurethral tenderness • Interstitial cystitis • Hunner´s ulcer • Stress urinary incontinence • Bladder infection	• Cystocele • Rectocele • Perineocele • Uterine prolapse • Apical prolapse • Apical prolapse • Enterolece • Abdominal pain • Low sacral backache • Vulvodynia • Dyspareunia • Urine loss with intercourse	• Cystocele • Rectocele • Perineocele • Uterine prolapse • Apical prolapse • Enterocele • Abdominal pain • Low sacral backache • Vulvodynia • Dyspareunia • Urine loss with intercourse

Pelvic floor disorders are currently assessed and treated independently by three different specialties, in response to referral based on the predominant symptomatology, using specialty-specific methods, but not always with predictable, satisfactory results. We believe that a more unified approach is needed.

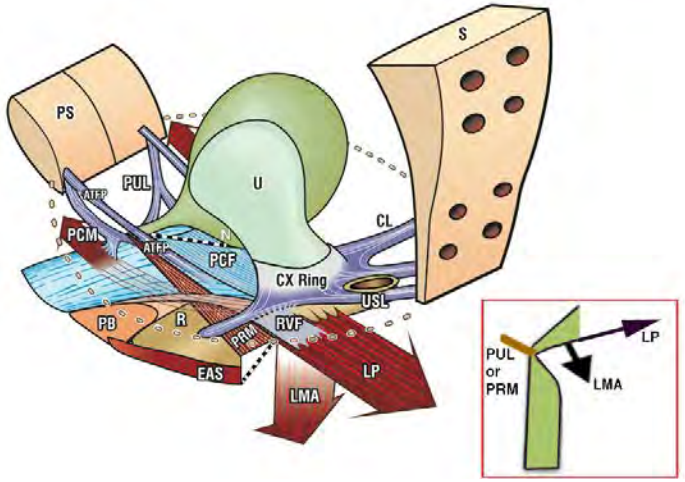
Structural and functional anatomy

The pelvic organs themselves contain smooth muscle fibres. These have characteristics which predispose to tonic contraction during evacuation, including unstable all-or-none action potentials, low-resistance pathways between smooth muscle cells and modulation by excitatory or inhibitory nerves (10). The organs are suspended superiorly by 5 paired ligaments attached to the pelvic bones (figures 1 and 2) and inferiorly by 4 paired striated muscles. Three directional muscle forces (figure 1; arrows), the pubococcygeus (PCM), levator plate (LP) and conjoint longitudinal muscle of the anus (LMA) contract against paired suspensory ligaments, i. e., the pubourethral ligaments (PUL) anteriorly and the uterosacral ligaments (USL) posteriorly (7,11). Resting tone and contraction of these muscles is activated by a feedback sensorimotor control system involving spinal cord, brainstem and frontal cortex (7,12). A fourth pair of striated pelvic muscle, the puborectalis (PRM), surround the rectum from its origin and insertion on the pubic symphysis; this muscle, like the external anal sphincter and external urethral sphincter is in a state of continuous low-level contraction during the maintenance of faecal continence (6,13). It relaxes just before defecation as part of the defecation response. It also has an important voluntary function, «squeezing» in an upward direction to interrupt urination or defecation (11). Micturition and defecation are induced by sensory input from organ filling, from surface sensory receptors and from stretch receptors in the

organs (figure 3). Evacuation of urine or feces occurs following activation of the micturition and defecation reflexes (14). Both continence and evacuation of organ contents are under conscious control through cerebral connections of spinal and brainstem afferents. In the normal state, these control processes can be voluntarily suppressed or facilitated (13,14). The term «reflex» (as summarized in figure 3) is conventionally used as a shorthand description of this much more complex integrated brain system program which is under conscious control. It is the complexity of this sensorimotor program that allows seamless integration of smooth, involuntary muscle and striated voluntary muscle activity in these functions.

In women, the pelvic floor ligaments and other connective tissues are the most vulnerable part of this system, due to damage sustained in childbirth, and the only components that can easily be repaired.

Figure 1. Five main ligaments suspend the organs from the skeleton to open or close the urethra and anus (view from above)



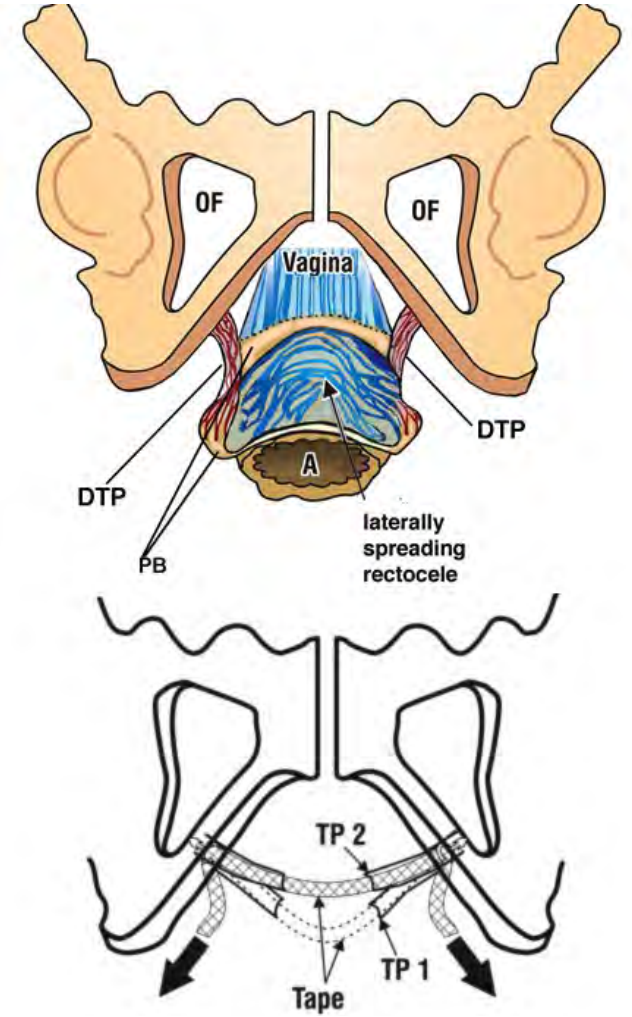
Pubourethral (PUL); Arcus Tendineus Fascia Pelvis (ATFP); Cardinal (CL); Uterosacral (USL); Perineal body (PB). urethra & anorectum show closed.

Four muscles control function (4) Three involuntary directional muscles (arrows), pubococcygeus muscle (PCM); levator plate (LP); conjoint longitudinal muscle of the anus (LMA) contract against suspensory ligaments PUL and USL to open and close urethral and anal tubes; the 4th muscle, puborectalis muscle (PRM) is both voluntary and involuntary and contracts only against pubic symphysis (PS); EAS = external anal sphincter;U = urethra; V = vagina; R=rectum.

Broken lines indicate position of bladder and anorectum in the evacuation phase.

INSET simplified 'kinking' closure mechanism of urethra/anus effected by LP/LMA. PUL or PRM stabilize urethra/anus. LP/LMA rotate bladder or rectum around PUL or PRM to close urethra at bladder neck, or anorectal angle, much like kinking a garden hose.

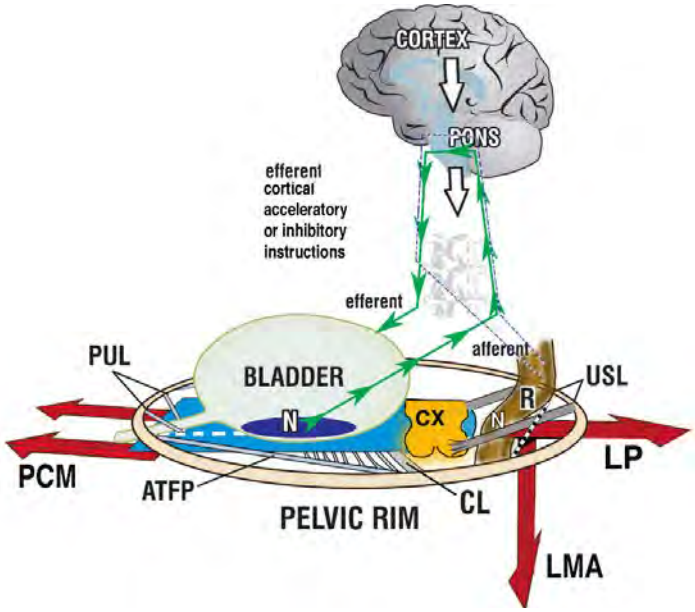
Figure 2. Perineal body anatomy



Upper figure: deep transversus perinei ligaments (4) attach perineal body 'PB' behind the descending ramus between its upper 2/3 and lower 1/3. The ligaments are elongated. PBs have been separated into two parts during childbirth, by stretching of their central part, allowing a rectocele to develop and spread laterally to cover the ligaments.

Lower figure: shortening and reinforcing elongated deep transversus perinei ligaments by TFS mini slings inserted through a single incision elevates and approximates PBs to cure descending perineal syndrome to close perineocele. TP1=original position of transversus perinei ligaments; TP2=position after shortening and elevation.

Figure 3. Control of micturition and defecation reflexes-the trampoline analogy



Normal control Afferent impulses from stretch receptors «N» in the bladder and bowel transmit (small green arrows) to the cortex, interpreted as «fullness». If not convenient to evacuate, the evacuation responses are cortically suppressed (white arrows); efferents activate the peripheral muscles PCM, LP, LMA (3 large arrows) which stretch the organs in opposite directions to support the stretch receptors «N» from below. If convenient, the evacuation responses are activated. Central suppression (white arrows) ceases. For micturition, PCM (pubococcygeus muscle) relaxes; LP/LMA pull open the posterior urethral wall (broken lines below bladder and behind rectum). For defecation, PRM relaxes (not seen here); LP/LMA pull open the posterior rectal wall (broken lines). Opposite stretching of the organs supports «N» (stretch receptors) prevent activation of the evacuation reflexes, micturition and defecation. LP=levator plate; LMA=conjoint longitudinal muscle of the anus; .PUL=pubourethral ligament; USL=uterosacral ligament; CL=cardinal ligament; ATFP= arcus tendineus fascia pelvis.

Dysfunction Because the 3 muscles pull against PUL and USL, any weakness in the ligaments will also weaken muscles forces. The muscles cannot properly close the urethral/anal tubes to cause incontinence; cannot open them (evacuation difficulties and retention of urine and feces); cannot stretch the organs sufficiently to support the stretch receptors «N» which give now give off excess afferents to activate the micturition and defecation reflexes, perceived by the cortex as urge.

Urethral closure

The three reflexly controlled striated muscle forces are the dominant force vectors controlling bladder and bowel function**.** In urinary continence the forward-acting PCM pulls the suburethral vagina forwards against PUL to close the distal urethra. The backward/downward muscles, LP/LMA, contract down against the resistance of the USLs. This action rotates the bladder base around PUL to «kink» the urethra at the bladder neck, ensuring continence (inset, figure 1) (15,16). This is shown in the video at https://www.youtube.com/watch?v=3vJx2OvUYe0.

Evacuation of urine

The micturition reflex is activated when it is «convenient to go». PCM relaxes and LP/LMA pull the posterior urethral wall backwards to open out the outflow tract (broken lines, figure 1). This action exponentially lowers the intracavity resistance to flow according to Poiseuille's Law (15). The bladder contracts to drive out the urine through the now opened urethra. Micturition is shown at https://www.youtube.com/watch?v=eiF4G1mk6EA&feature=youtu.be.

Impaired urethral closure:

Stress incontinence develops if PUL is lax. This compromises the pubococcygeus muscle (PCM) force vector so that it cannot maintain sufficient contraction force to close the urethra from behind. Consequently, the posterior muscle vectors LP/LMA pull down the vagina and posterior wall of the urethra into the «open mode» (broken lines, figure 1), urethral resistance to flow falls exponentially (16) and urine is lost on effort (stress urinary incontinence).

Urinary retention (obstructed micturition) can develop if USL is lax, so that the downward vector (LMA) is weakened. The posterior wall of the urethra cannot then be adequately opened and the bladder detrusor muscle can only contract against a partially opened urethra. Internal resistance to urine flow increases exponentially (15). The patient senses this as «obstructed micturition». The bladder emptying time is increased, and micturition stops and starts, with incomplete emptying, or even urinary retention.

Anorectal closure

This is the default anorectal mode that maintains anorectal continence. The PRMs are in low-level tonic contraction, applying a forward force vector against the posterior wall of the rectum. The backward and downward contracting muscles, LP/LMA, contract against USLs to bend the rectum around the contracted PRMs closing the

anorectal angle (inset, figure 1). This is the main anorectal sphincteric mechanism for fecal continence (6,7,8).

Evacuation of feces-defecation

When it is «convenient to go», and there is sufficient afferent signalling of the need to evacuate feces, cortical reinforcement of the anorectal closure responses ceases and the defecation response is activated. The PRMs (figure 1) relax and LP/LMAs pull the posterior anorectal wall posteriorly to open out the anorectal outflow tract (16,17) (broken lines, figure 1). The external anal sphincter relaxes and the smooth muscle of the rectum contracts with associated voluntary levator ani muscle contraction to drive out feces through the anal canal. Defecation is shown in https://youtube/MS82AZoWn7U.

Impaired anorectal closure

Faecal leakage may occur if USLs are stretched and lax, so that the downward vector from LMA contraction cannot «kink» the rectum adequately against the contracted PRMs. Anorectal closure is then inadequate. Depending on the internal resistance achieved by this closure mechanism (16,17), symptomatic fecal incontinence may be experienced as difficulty controlling anal flatus, liquid bowel content or solid feces.

Impaired fecal evacuation

Difficulty in defaecation, e. g., «constipation» and «obstructed defecation», are subjective symptoms that develop if USLs are lax, since the downward vector (LMA) is then weakened. The posterior wall of the rectum cannot be opened adequately so that the rectum contracts against a partly unopened anal canal causing difficulty in defecation. This should be distinguished from idiopathic constipation due to slowed intestinal transit.

Perineal body anatomy

The perineal body is a 4 cm fibrous body located between the vagina and rectum. It is maintained in position by the deep transverse perinei ligaments which insert behind the descending rami at the junction of the upper 2/3 and lower 1/3 (figure 2). Stretching of these ligaments caused by childbirth trauma causes the descending perineum syndrome (6). This ligamentous damage is associated with damage to the innervation of the pelvic floor musculature (4,5,8), and sometimes by

direct trauma to these muscles, especially the external anal sphincter (5,8), further weakening these muscle vectors and leading to stress incontinence of feces and urine and also pelvic pain. Restoring pelvic floor muscle function by shortening and strengthening the deep transverse perineal ligaments using the tissue fixation system (TFS) applied to the perineal body (18), can be effective in alleviating these dysfunctional symptoms.

Feedback control of bladder and bowel

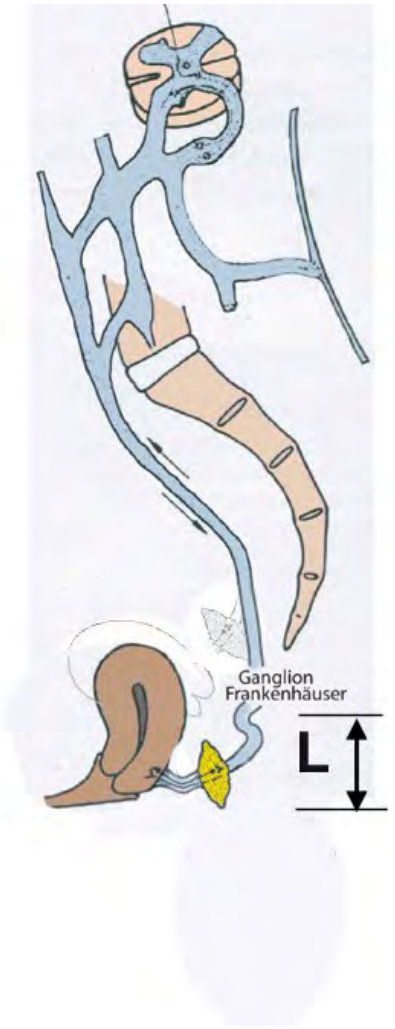
Continence of bladder and bowel is maintained by reflexly supported, low-level continuous activity of the pelvic floor striated and smooth musculature modulated by the central nervous system (see above) (figure 3). The resting state of this system is anorectal and urethral closure, thus maintaining continence. When the afferent impulse stream from organ filling (small green arrows, figure 3) reaches a self-determined critical level, the brain senses a need to evacuate feces or urine. If not convenient to evacuate the urge response is suppressed (figure 3). Alternatively, the control system switches to open mode and the evacuation programs in the central nervous system for micturition or defecation are activated.

Dysfunction of micturition and defecation (figure 3)

The high cure rates for bladder/bowel dysfunction achieved by ligament repair (19-41) cause us to consider pelvic ligament damage in the peripheral control mechanism as a critical point of susceptibility in the system. If PULs or USLs are stretched and lax, the muscles contracting against them are in effect lengthened, and their contractile force reduced. This is particularly problematic when these muscles are already weakened by neurogenic damage or by previous direct trauma, as in childbirth injury. The vaginal membrane cannot then be tensioned sufficiently to support the stretch receptors («N», figure 3) and afferent input cannot therefore be sensitively modulated. There is then a risk that micturition or defecation reflexes are activated inappropriately causing an overwhelming feeling of impending evacuation of urine or feces; i. e., «urge incontinence». A useful test consists in applying an intravaginal hemostat immediately behind the symphysis to support PULs, thus temporarily restoring the anatomy. This will immediately relieve stress continence, whether urinary or fecal (42,43). In addition, any neurological lesion along the afferent or efferent pathways (figure 3), caused by disease or, especially, childbirth damage (6,14), may interfere with both the closure and the evacuation responses. Nocturia can be relieved by a gauze roll inserted in the posterior vaginal fornix (29) as can bladder emptying difficulties.

Anatomical pathways in chronic pelvic pain (figure 4)

Figure 4. Pathogenesis of chronic pelvic pain



The Ganglions of the Frankenhauser T11-L2 and the Sacral Plexuses S 2-4 are supported by uterosacral ligaments (USL) at their uterine end. 'L' indicates ligament laxity. 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 impulses and be perceived as pain by the cortex.

The critical role of USLs in chronic pelvic pain was first described by Heinrich Martius in the German literature in 1938 (44) and in the English literature in1996 (45). Chronic pelvic pain is characteristically perceived in a wide distribution that is not consistent with involvement of specific peripheral nerves or nerve roots. Its distribution and description suggests referred pain consistent with involvement of sacral nerve roots (figure 4); i. e., in lower abdomen, groin, and lower sacrum (35); in paraurethral or introitus distributions (46) or as interstitial cystitis (47), or deep dyspareunia. Martius pointed out that inability of the uterosacral ligaments to support the pelvic sacral nerve plexuses may cause them to be stimulated inappropriately leading to diffuse, chronic pelvic pain (figure 4). This suggestion is easily verified by inserting a vaginal speculum to mechanically support the USLs («simulated operation» as in figure 5), in women with pain and /or urge and observing the immediate change in symptoms (48). The paired ganglia of Frankenhauser, located adjacent to the lateral sides of the lower uterus and cervix, are probably the source of the painful afferent nervous activity generated in response to mechanical stimulation in the context of a weak, stretched pelvic floor, especially in the seated position. These paired ganglia are derived from the inferior hypogastric plexus. They are unique in that they contain both parasympathetic and sympathetic neurons and nerve fibres, forming the uterovaginal plexus. Small diameter afferents subserving deep pain project to the spinal cord and thence to the brain from these ganglia.

Wu *et al.* (46) reported relief of pelvic pain and suburethral tenderness by insertion of the lower part of a bivalve speculum to support the posterior fornix (42) (figure 5). Bornstein relieved vulvodynia by local anesthetic injection into the USLs close to the uterine cervix (49) as did Petros in 3 patients with interstitial cystitis, abdominal pain and suburethral tenderness (47). Another test, used to confirm the role of lax USLs in nocturia, is to place a cylindrical pessary, or large tampon, in the posterior vaginal fornix overnight (48). Gunnemann reversed anterior rectal wall intussusception by pushing a cylindrical vaginal pessary 3x6cm into the apex of the vagina under 2D ultrasound control. This manoeuvre effectively tightened USL support (48).

Measuring pelvic floor dysfunction determines management

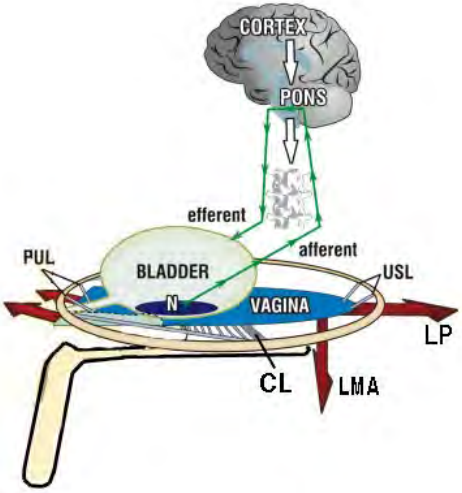
Collagen and elastic tissue are the key structural component of ligaments. Ligamentous laxity can be congenital, but the most frequent cause is stretching due to softening of ligaments in pregnancy and, especially, stretch-induced damage during childbirth (figure 6). In older women this is exacerbated by age-related collagen breakdown leading to organ and pelvic floor prolapse and progressive pelvic sphincteric symptoms (figures 7 and 8). The initial injury to the pelvic floor ligaments, muscles and their innervation, occurs during childbirth, when the fetal

head may stretch or tear muscles, nerves, ligaments and other connective tissue as it descends through the pelvis. At the pelvic inlet, injury to USLs («1», figure 6) may stretch and elongate them, so that they lose their normal innate elasticity, leading to vulnerability to uterine prolapse and enterocele. The USLs are attached to the lateral rectal wall by multiple fine ligaments that may become stretched leading to anterior rectal wall intussusception. Injury to the cardinal ligament as it attaches to the anterior cervical ring («2», figure 6), will also damage the pubocervical fascial attachment of the vagina to cause a transverse defect («high cystocele»). Damage to the perineal body during childbirth (figure 5), will also stretch its attachments to the descending ramus by the deep transverse perineal ligaments (see figure 2) causing perineal descent and a low rectocele (perineocele) on straining. Pressure by the fetal head during delivery («1», figure 6) immediately behind the symphysis pubis can injure PULs and stretch the collagenous insertion of the pubococcygeus to the pubic symphysis causing a central cystocele. Stretch-induced damage to somatic pelvic nerves has been demonstrated post-delivery and has been shown to be related to bladder and bowel incontinence by causing muscular weakness (14). During recurrent perineal descent these nerves may be further damaged by stretch injury, leading to the progressive dysfunction that is so often observed in clinical practice.

Diagnosis

The pattern of symptoms is an accurate indicator of specific weakness and ligament damage (2,3,7,11). The pelvic floor ligaments can be considered as acting in 3 zones and the relationship between lax ligaments and specific symptoms is summarized in figure 7. This algorithm can be used to understand all the symptoms with which a patient may present. Pelvic symptoms occur in multiple groupings (figure 7) but, almost invariably, one symptom is dominant. However, this is but the «tip of the pelvic symptom iceberg» (figure 8). Applying this algorithm to 198 patients presenting with chronic pelvic pain, Goeschen and Gold noted (23) many other initially occult symptoms (figure 8), often elicited only by direct questioning based on the algorithm. Our experience is consistent with common clinical experience, that major symptoms may occur with minimal or no apparent prolapse. Prolapse does, however, become evident during examination under anesthesia.

Figure 5. Speculum test



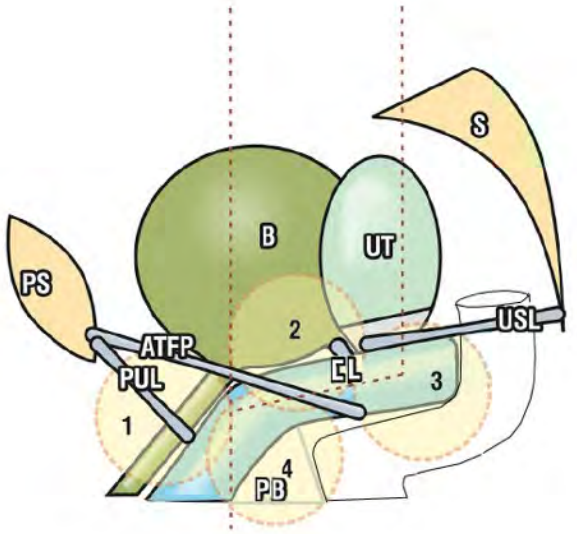
3D view of the bladder located on the anterior vaginal wall, which is suspended from the pelvic brim by pubourethral (PUL), cardinal (CL), uterosacral (USL) ligaments. A speculum inserted into the posterior fornix mechanically supports lax USLs and with it, the nerve plexuses S2-4, T11-L2. Firm USLs restore the contractile strength of the posterior muscle forces (arrows LP, LMA), which contract against them. These now tension the vagina like a trampoline to support the stretch receptors «N», decreasing the afferent impulses which on passing a critical mass, are interpreted as urgency.

Simulated corrective procedures validate diagnoses predicted by the algorithm

One extra step is required before a surgical management can be planned. A «simulated operation» involves mechanically supporting a specific pelvic ligament in an outpatient setting and observing the effect on symptoms. For example, to plan cure of urge and pain, a speculum is gently inserted into the vagina to support USLs (figure 5). For stress urinary incontinence (SUI), inserting a haemostat immediately behind the symphysis on one side at the insertion point of PUL will stop urine loss on coughing (see https://youtu.be/0UZuJtajCQU). This will also control loss of feces on coughing (42) and subjective urgency in patients with mixed incontinence. Done under ultrasound control for SUI, this test will simulate restoration of the bladder neck and associated distal urethral closure (43). Nocturia can be tested by placing a large tampon or a roll gauze in the vagina overnight (29). Gunnemann demonstrated that an anterior rectal wall intussusception can be reversed by gently pushing a cylindrical pessary up into the fornix of the vagina under ultrasound control (48).

Supporting specific ligaments in this manner can normalize both urodynamic urethral pressure measurements (49) and anorectal manometry measurements (50).

Figure 6. Muscle/ligament injury by descending fetal head



The fetal head may injure specific connective tissue structures or muscles as it descends through the pelvis. «3»: USL (uterosacral ligament)uterine prolapse; «2»: CL (cardinal ligament) transverse defect cystocele; «4»: PB (perineal body) rectocele, descending perineal syndrome; «1» PUL (pubourethral ligament) stress urinary incontinence. «1» injury to the insertion of the pubococcygeus muscle to pubic symphysis (PS) hiatal ballooning, low (central) cystocele. Arcus tendineus fascia pelvis (ATFP) can be injured at its insertion point at the ischial spine or at PS. S=sacrum; B=bladder; UT=uterus.

Non-surgical treatment is theoretically dependent on specifically exercising the three directional pelvic muscle forces and the ligaments they contract against (figure 1), using squatting-based exercises (https://www.youtube.com/watch?v=3vJx2O-vUYe0). In premenopausal women improvement may occur in anterior and posterior zone symptoms (51) (see Tables 1 & 2) but, unsurprisingly, given the background of complex pelvic floor injury the duration of this beneficial effect is limited. These exercises may also be helpful in children aged 6-11 with day/night enuresis (52).

Surgical treatment according to this ligament-based system of assessment is based on tightening and reinforcing the 5 main ligaments, and re-attachment of the vaginal fascia (anterior «PCF» and posterior «RVF», figure 1). Plication of USLs in women with minimal prolapse gave good initial cure rates for the «posterior fornix syndrome» (PFS) (19) (rectangle, figure 7) a disorder caused by lax USLs, characterized

by urge, frequency, nocturia, abnormal bladder emptying and chronic pelvic pain. However, because of rapid deterioration of symptoms in the subsequent two years, a posterior TFS sling was subsequently added to reinforce USLs, in essence a reverse TVT sling. This posterior sling reinforcement procedure gave high cure rates at 12 months for urge, frequency, nocturia and urinary retention (24,25,30,32).

Conventional reconstructive surgery is often unsatisfactory in older women, as shown by Shkarupa *et al.* (29) who compared results after native tissue cardinal/uterosacral ligament plication in pre and post-menopausal women with overactive bladder (OAB) and prolapse. Initial cure rates were similar at 3 months, but rapidly deteriorated in the post-menopausal group at 18 months. There was 80 % cure of prolapse in the premenopausal group as against 17 % in the post-menopausal group, and for OAB, 68 % cure against 17 %. Unlike Shkarupa's data, a 70 year-old cohort from the Kamakura clinic in Japan showed only minimal change in the cure rate for prolapse, from 90 % at 12 months to 79 % at 5 years. When all 5 ligaments were repaired by a single incision TFS mini-sling technique (30); symptom cure at 5 years for SUI (82 %) and for OAB (91 %) were similarly maintained. Similar cure rates using posterior slings for prolapse, bladder/bowel dysfunction, and pain by USL slings have been reported in other studies, notably Hocking's results for double incontinence with a midurethral sling (36) and the xray validated report of Abendstein *et al.* (22) who relieved fecal incontinence, obstructive defecation and anterior rectal wall intussusception using the sling technique.

Practical implications

The «engine» for this musculo-elastic ligament-based paradigm is the predictive algorithm shown in figure 7. The inter-relationships of functional disorders tending to present specifically to Urologists, Gynecologists and Colorectal surgeons are listed in Table 1. The algorithm describes disorders in three pelvic compartments. High cure rates by specific PUL and USL ligament repair of conditions in the anterior and posterior pelvic compartments, shown in figure 5, validate the relevance of the algorithm. With reference to gynaecological disorders (Table 1), urine loss associated with intercourse is curable with a midurethral sling. All the different manifestations of chronic pelvic pain can be alleviated by USL repair, using native tissue ligament repair for premenopausal and TFS slings for postmenopausal women. Alleviation of cystocele requires cardinal ligament repair if the cause is a transverse defect, but treating a central cystocele is more complex as it is usually caused by dislocation of the pubococcygeus muscle (PCM) from the symphysis pubis. A welcome surprise for the authors has concerned the management of colorectal disorders. Every symptom, including fecal incontinence, perineal descent with anorectal pain or incontinence, ODS, anterior rectal wall intussusception, even hemorrhoids, has

been cured or improved with repair of the uterosacral ligaments, deep transverse perineal ligaments, or both.

Damage to the 5 main ligaments and the 4 main muscles may cause dysfunction of bladder/bowel closure (incontinence) or evacuation difficulties (figure 3), inability to control the micturition and defecation reflexes (urge incontinence), and chronic pelvic pain (figure 4). Figure 5 illustrates how the descending fetal head may damage supporting ligaments and attached fascias during childbirth to cause the various prolapses. However, it will also cause muscle damage as demonstrated histologically by PCM biopsy (39), and nerve damage associated with bladder/bowel incontinence developing after delivery (4,5) and progressing subsequently in association with straining at stool, i. e., an evacuation difficulty (6,8). The remarkable short-term cure rate (86 %) with squatting-based exercises in day/night enuretic children (52) has been attributed to creation of new collagen (53) although without objective supportive evidence. However, Shkarupa's high cure rate of prolapse and urgency by cardinal ligament and USL repair in premenopausal women is probably due to new collagen laid down around the repaired ligament. The poor results from «native tissue» surgery after the menopause, may be explained by inability to lay down new collagen since at this time in life collagen is slowly being broken down and excreted as hydroxyproline (54). New collagen is required for success after surgical repair of damaged pelvic ligaments. This occurs (55) following insertion of a polypropylene sling in the exact position of damaged ligaments, the only proven method for cure of prolapse-related symptoms in the longer term. Slings induce local deposition of new collagen as a wound reaction to create a collagenous neoligament. This is the principle underlying the midurethral sling procedure.

What role does neurogenic or direct traumatic muscle damage play in pathogenesis? This will vary from case to case but weakened muscles will perform much better when their damaged, lax ligamentous insertions are tightened. In a blinded muscle biopsy study of 50 women with SUI, almost all had evidence of major muscle damage but 89 % reported cure of their SUI the day following insertion of a midurethral sling (39). Swash *et al.* (6) demonstrated nerve damage that was often progressive due to perineal descent syndrome, associated with later development of bladder or bowel incontinence after childbirth. Nerve damage which fails to recover will diminish contractile muscle power. This alone could account for the varying failure rates reported (56).

A new concept needs to be explanatory and predictive

The fundamental test for any theory is its predictive and explanatory value. We have therefore tested the concepts presented here against 5 recently published papers.

Alketbi *et al.* (57) reported high rates of cure for fecal incontinence and dyschezia (constipation) occurring after repair of ruptured puborectalis muscle (PRM). PRM is especially important for anorectal continence as it anchors the anorectum from behind while the two posterior vectors rotate the rectum around PRM to close the anorectal angle. The high cure rate for fecal incontinence achieved validates our concepts for anorectal closure (figures 3 and 4B). We attribute cure/improvement in anorectal symptoms to re-attachment of the pubococcygeus (PCM) and puboure-thral ligament (PUL). PCM is inextricably bound to PRM at the insertion point to the symphysis and PUL inserts into PCM.

Re-attachment of PCM and PUL reconstitute the insertion point of the levator pla-te re-activating the backward stretching of the rectal wall required for defecation (16,17).

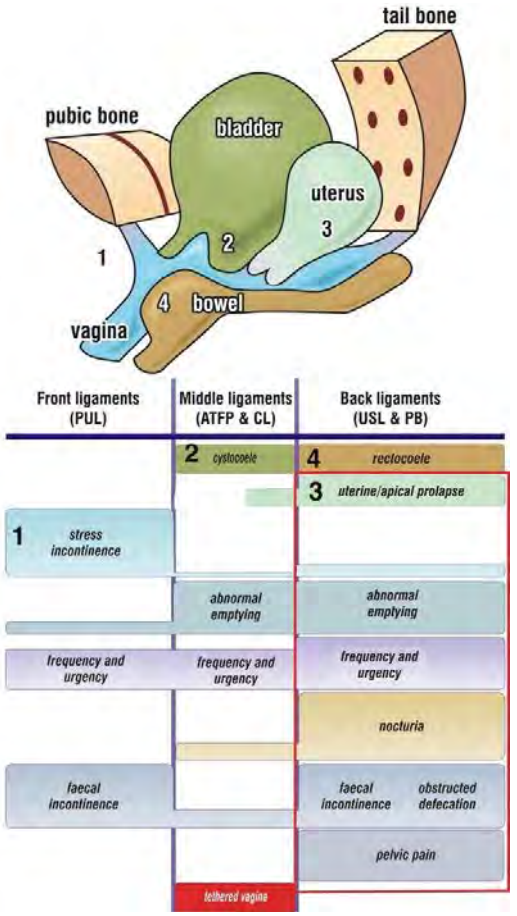
Torrijo *et al.* (58) showed a statistically significant worsening of nocturia at 6 months ($p = 0.002$) and 1 year after rectal cancer surgery ($p = 0.037$) in women. The utero-sacral ligaments (USL) are attached to the lateral walls of the rectum and are the anchoring points for downward rotation of the rectum around a contracted PRM. If the excision of the carcinoma also excised USLs, the downward vector would be affected and the anorectal angle could not be closed.

Mege *et al.* (59) described 40 patients who underwent dynamic graciloplasty; 31 patients [77 % women, median age 57 years (range 17-74 years)] were later stu-died, with a mean long-term follow-up of 11±6 years. Postoperative, surgical and major morbidity occurred in 20 (64 %), 17 (55 %) and 7 (23 %) patients, respecti-vely. At the end of follow-up, 18 patients still used their stimulation device (58 %). If the dynamic graciloplasty was placed in the anatomical position of the PRM, which is always below the insertion point of the levator plate, it would serve as a rotation point for closure exactly as normally does the PRM.

Ascanelli *et al.* (60) performed a prospective cohort pilot study on 30 patients with dyssynergic defecation. There was improvement in the biofeedback group ($p = 0.021$). Our perspective on dyssynergic defecation is based on loss of balance between the opposing muscle forces (figure 4). If USLs are weak, the posterior vec-tors LP/LMA weaken, impairing anorectal opening that precedes defecation. If PRM overcompensates involuntarily, dyssynergic defecation occurs. We would advise managing the problem by USL repair (61).

Muctar *et al.* (63) described successful surgical cure with a USL TFS sling in 3 women with intractable faecal incontinence who were booked for colostomy. They were treated according to the diagnostic algorithm (figure 7).

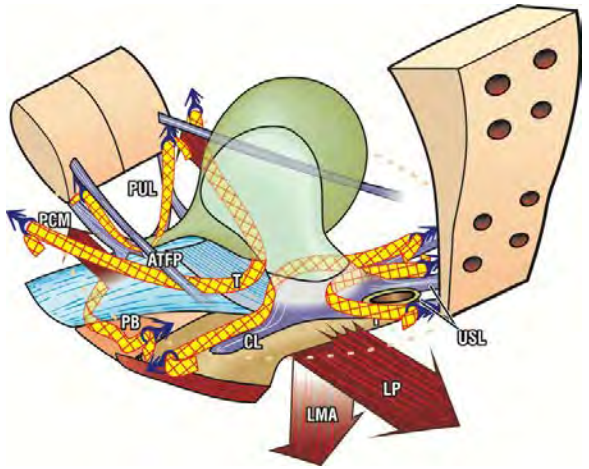
Figure 7. The pictorial diagnostic algorithm uses symptoms to locate damaged ligaments and as such, guides surgery. The connective tissue structures causing prolapse and pelvic symptoms fall naturally into 3 zones, front (meatus to bladder neck), middle (bladder neck to cervix), back (cervix to perineal body). The height of the bar indicates probability of association of a symptom with a particular zone.



How to use the algorithm Tick every symptom box. Chronic pelvic pain and nocturia are uniquely caused by USL laxity, stress urinary/stress fecal incontinence by PUL. Symptom grouping assists location of other ligaments.

PUL=pubourethral ligament; USL-uterosacral ligament; ATFP=arcus tendinous fascia pelvis; CL=cardinal ligament; PB=perineal body.

Figure 8. Tapes create new collagen to strengthen ligaments 3D view from above



This figure shows one way adjustable TFS (Tissue Fixation System) tapes. Thin 7mm wide polypropylene tapes inserted in the exact anatomical position of damaged ligaments create new collagen to restore anatomy and function of the 3 reflexly controlled muscles, PCM (pubococcygeus), LP (levator plate) conjoint longitudinal muscle of the anus (LMA). Tapes cut from a large mesh sheet placed tension-free after native ligament plication will similarly create new collagen to reinforce damaged ligaments[48]. Pubourethral (PUL); Arcus Tendineus Fascia Pelvis (ATFP); Cardinal (CL); Uterosacral (USL); Perineal body (PB).

Perspective and conclusions

We have described a holistic pelvic floor understanding dependent on a muscu-lo-ligamentous based paradigm to explain bladder/bowel function and dysfunction. How does all this translate to day to day management? Surgical repair by minimally invasive sling techniques focused on ligament and fascia repair can be very effective in relieving symptoms in older women. The pelvic floor algorithm is an important guide to detect occult symptoms, since it guides assessment for specific ligamentous and fascial defects in the three vaginal zones. Each of these defects has a specific phenotype seen during vaginal examination (53). Finally, «simulated operations» to confirm if a specific ligament is causing specific symptoms are valuable in directing successful corrective ligament repair. This unique approach to pelvic floor problems has major implications for practice in these three surgical specialties, extending from paediatric (63) to geriatric (30).

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