

The Musculo-Elastic Theory of anorectal function and dysfunction

PETER PETROS (*) - MICHAEL SWASH (**)

(*) Royal Perth Hospital, University of Western Australia

(**) Dept of Neurology, The Royal London Hospital, London, UK

Abstract: The Musculoelastic Theory of anorectal function and dysfunction states “Anorectal dysfunction in the female is mainly caused by lax suspensory ligaments inactivating anorectal muscle forces”. *Anorectal closure.* The rectovaginal fascia inserts into perineal body, levator plate (LP) and the uterosacral ligaments. Contraction of levator plate stiffens rectovaginal fascia and both walls of rectum. Contraction of longitudinal muscle of the anus (LMA) against the uterosacral ligaments stretches the rectum around a contracted puborectalis muscle, to create the anorectal angle and closure. *Defaecation.* Puborectalis relaxes. Posteriorly acting LMA/LP vectors open out the anorectal angle; forward contraction of the pubococcygeus vector stiffens the perineal body, and anterior wall of anus; the rectum empties. *Pathogenesis.* Is similar to that described by the Integral Theory 1 for urinary incontinence: damaged ligaments decrease the force of opening and closure vectors. *Surgery according to this theory.* Reinforcement of damaged ligaments with precisely implanted polypropylene tapes restores structure and function.

Key words: Fecal incontinence; Constipation; Ligament laxity; Connective tissue; Anatomy; Integral Theory.

EXISTING CONCEPTS FOR NORMAL ANORECTAL FUNCTION.

The mechanics of defecation and fecal continence are poorly understood. Valvular theories for continence² rely on raised intra-abdominal pressure to force the anterior wall of the rectum downwards to close off the anorectal junction. Such theories are not consistent with EMG and radiological data which suggest a striated muscle sphincteric mechanism.³ It has been demonstrated that puborectalis and external anal sphincter muscles contract during effort, indicating a role for both in fecal continence.⁴ The internal anal sphincter is also said to be important for feces continence.⁵ The mechanism of defecation is even more poorly understood. According to one description,³ feces enter the anal canal, stimulate stretch receptors and produce the urge to defecate. The internal and external anal sphincters relax, decreasing the pressure within the anus. The rectum contracts and with the assistance of raised intraabdominal pressure (Valsalva) expels the feces. Shafik⁶ suggested an active muscular mechanism for anorectal opening and closure. In his proposal, during straining, the puborectalis, acting as the upper part of a triple loop system, contracts to close off the anal canal. No role is assigned by Shafik for levator plate contraction during anorectal closure. According to Shafik, during defecation, the puborectalis muscle relaxes, the levator plate contracts, and the “suspensory sling” lifts upwards to open out the anorectal canal prior to evacuation by rectal detrusor contraction.

MAJOR EXISTING HYPOTHESES FOR CAUSATION OF FECAL INCONTINENCE (FI)

Major hypotheses advanced for fecal incontinence causation include damage to the external anal sphincter (EAS), stretch-related pudendal nerve damage⁷ and internal anal sphincter (IAS) injury.⁵ These causes are by no means exhaustive. Bharucha,⁸ in a recent review article quoted the following causes: *Anal sphincter weakness Injury:* obstetric trauma; *Related to surgical procedures* (e.g., hemorrhoidectomy internal sphincterotomy, fistulotomy, anorectal infection); *Nontraumatic:* scleroderma, internal sphincter thinning of unknown cause; *Neuropathy stretch injury:* obstetric trauma, diabetes mellitus; *Anatomical disturbances of pelvic floor:* fistula, rectal prolapse, descending perineum syndrome; *Inflammatory conditions:* Crohn’s disease, ulcerative colitis, radiation proctitis; *Central nervous system disease:* dementia, stroke, brain tumors, spinal cord lesions,

multiple system atrophy (Shy Drager’s syndrome), multiple sclerosis; *Diarrhea:* Irritable Bowel Syndrome, postcholecystectomy diarrhea.

The majority of the above “causes” would appear to implicate either the external or internal anal sphincter in causation, or a disturbance of the stretch receptors or their cortical control paths. The Musculo-Elastic Theory concerns mainly the “idiopathic group”.

Like the nerve damage theory,⁷ the Musculo-Elastic Theory attributes bowel and bladder dysfunction to muscle insufficiency. Unlike the nerve damage theory,⁷ such muscle insufficiency is deemed mainly consequent upon laxity of the suspensory ligaments (*), the effective anchoring points of the closure muscles of urethra and anorectum.

(*) and so it is potentially reversible with ligament repair.

THE COMMON CAUSATIVE RELATIONSHIP BETWEEN ANORECTAL DYSFUNCTION AND OTHER PELVIC SYMPTOMS

In a study published in 1999,⁹ simultaneous cure of urinary stress and idiopathic fecal incontinence was observed in a group of 25 patients following a midurethral sling procedure performed for cure of stress incontinence. This operation created a collagenous pubourethral neoligament; only connective tissue was repaired. Normal internal anal sphincters were found in 72% of these patients, and normal external anal sphincters in 100%.⁹ Three patients from this group subsequently reported recurrence of their fecal incontinence simultaneously with occurrence of vaginal vault prolapse and posterior zone bladder symptoms (Fig. 1). Repair of the prolapse with a posterior sling in the position of the uterosacral ligaments (USL) cured the prolapse, the urinary and the feces incontinence.⁹ Three hypotheses followed naturally from that study.

1. Both pubourethral and uterosacral ligaments were essential for anorectal closure.

2. The same muscles acting against these ligaments for bladder/urethral closure 1 were most likely also involved in anorectal closure.

3. Damaged connective tissue was most likely a major cause of both urinary and idiopathic fecal incontinence, as only connective tissue was repaired.

In the section which follows, we have tried to describe the function and dysfunction of the anorectum in a way which can be tested directly for truth or falsity.¹⁰

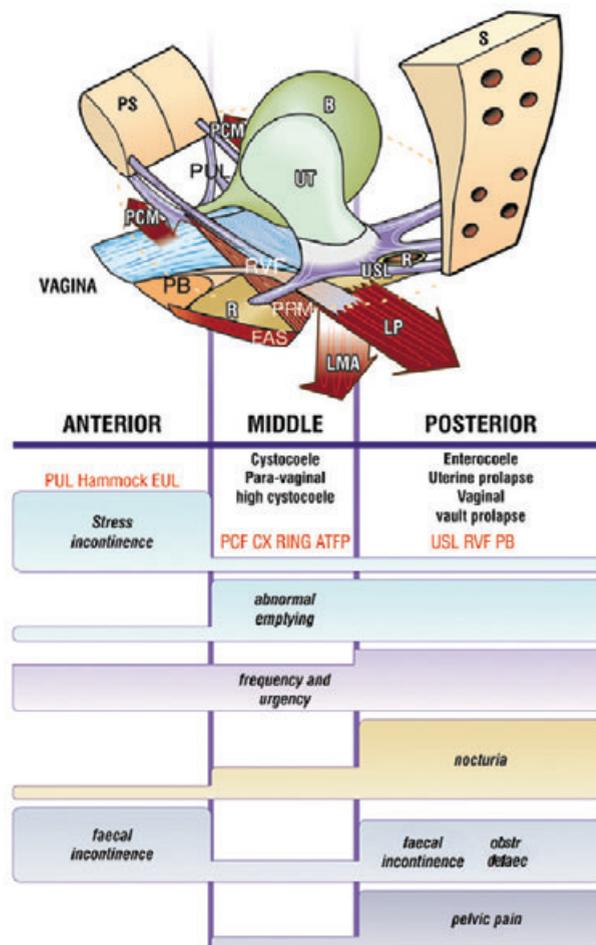


Fig. 1. – The Pictorial Diagnostic Algorithm summarizes the relationships between structural damage in the three zones and urinary and fecal symptoms. The size of the bar gives an approximate indication of the prevalence (probability) of the symptom. The same connective tissue structures in each zone (red lettering) may cause prolapse and abnormal symptoms. *Anterior zone*: External urethral meatus to bladder neck; *Middle zone*: bladder neck to cervix; *Posterior zone*: vaginal apex, posterior vaginal wall and perineal body. R = rectum; RVF = rectovaginal fascia; PB = perineal body; PRM = m. puborectalis; LP = m. levator plate; LMA = m. longitudinal muscle of the anus; PCM = m. pubococcygeus; PUL = pubourethral ligament; USL = uterosacral ligament.

THE DYNAMIC ANATOMY OF ANORECTAL FUNCTION AND DYSFUNCTION ACCORDING TO THE MUSCULO-ELASTIC THEORY

According to this theory, co-ordinated muscle forces acting against competent suspensory ligaments selectively open or close the anorectal tube. It is damage to these ligaments and their fascial attachments which cause continence and evacuation problems.

THE KEY LIGAMENTS OF THE PELVIC FLOOR (Figs. 2, 3)

The Pubourethral Ligament (PUL)

The pubourethral ligament (PUL) originates from the lower end of the posterior surface of the pubic symphysis, and descends like a fan to insert medially into the midurethra and laterally into the pubococcygeus muscle.^{11, 12} PUL is the effective anchoring point for m. pubococcygeus and levator plate.

The Arcus Tendineus Fascia Pelvis (ATFP)

The ATFP arises just superior to the pubourethral ligament (PUL), and inserts into the ischial spine. The vagina is suspended across the ATFPs. The muscle forces (arrows, Fig. 3), tension the ATFP and vagina.

The Uterosacral (USL) and Cardinal Ligaments

The uterosacral ligaments (USL) suspend the cervix and apex of the vagina. USL is the effective anchoring point for the longitudinal muscle of the anus 'LMA'^{13, 14} and through its attachments to the rectovaginal fascia, levator plate 'LP' (Fig. 3). The cardinal ligaments "CL", insert into the anterior portion of cervical ring, and they attach the cervix laterally to the pelvic wall.

The *perineal body (PB)* is approximately 4 cm long, and it occupies half the posterior vaginal wall. Its components are similar to ligaments, collagen, elastin, smooth muscle blood vessels nerves, but it also has striated muscle, being attached to all the superficial and deep perineal muscles.

THE MUSCLES OF THE PELVIC FLOOR (Figs 2,3)

There are 3 layers of muscles (Fig. 2).

Upper layer. The anterior part of pubococcygeus muscle (PCM) inserts into the lateral part of the distal vagina, and contracts forwards against PUL and the perineal body (PB).¹ The levator plate complex (LP) inserts into the posterior wall of the rectum, and it stretches the organs backwards. It acts against PUL, USL, and PB (Fig. 3).

Middle layer. The longitudinal muscle of the anus (LMA) connects levator plate, pubococcygeus and puborectalis to the external anal sphincter.^{13, 14} It creates a downward vector, which acts against USL, (Fig 3).¹ The puborectalis muscle (PRM) originates just medially to PCM. It is closely applied to the lateral walls of the rectum and inserts into its posterior wall. As well as its pivotal role in anorectal closure, the puborectalis muscle is the voluntary muscle activated during 'squeezing' when it elevates the whole levator plate (LP) and also, rectum, vagina and bladder (see X rays, study No 1, in this issue).

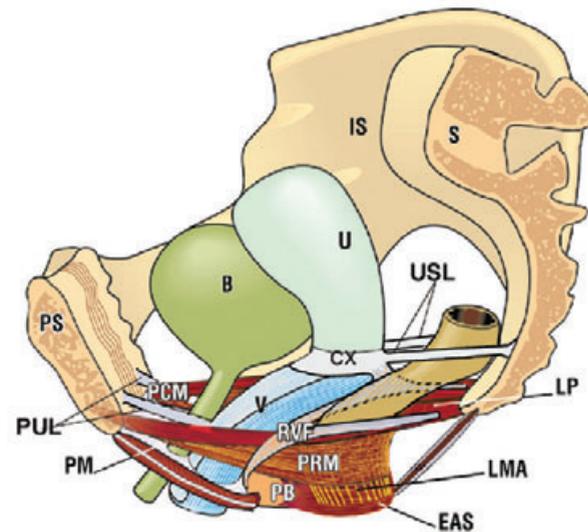


Fig. 2. – Muscles and ligaments of the pelvic floor 3D schematic sagittal view, ano-rectum closed. *Organs*: B = bladder; R = rectum; V = vagina; U = uterus; *Ligaments and fascia*: pubourethral ligament (PUL); uterosacral ligament (USL); PB = perineal body; RVF = rectovaginal fascia; Cardinal ligament (not shown) attached to anterior part of cervical ring (CX). *Muscles upper layer*: PCM = anterior portion of m. pubococcygeus; LP = levator plate; *middle layer*: LMA = longitudinal muscle of the anus; PRM = m. puborectalis; *lower (anchoring) layer*: EAS = external anal sphincter; PM = muscles of the perineal membrane.

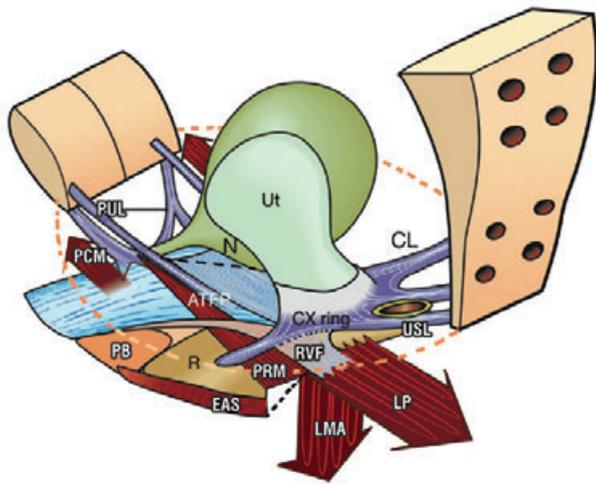


Fig. 3. – The related dynamics of anorectal and bladder closure and opening, pathogenesis and surgery. Arrows denote directional muscle forces. ATRP = arcus tendineus fascia pelvis; CL = cardinal ligament; circular broken lines = pelvic brim; Anorectal closure. The rectovaginal fascia (RVF) inserts into perineal body (PB), levator plate (LP) and the uterosacral ligaments (USL). Contraction of LP stiffens RVF and both walls of rectum. Contraction of LMA (longitudinal muscle of the anus) against USL stretches the rectum around puborectalis muscle (PRM), to create the anorectal angle and closure. Defaecation. PRM relaxes. LMA/LP vectors open out the anorectal angle (broken lines); pubococcygeus (PCM) vector stiffens PB and anterior wall of anus; rectum (R) empties. Urethral/bladder neck closure. The forward vector (PCM) closes urethra from behind; LP/LMA vectors rotate bladder around pubourethral ligaments (PUL) to close off bladder neck. Micturition. Forward vector (PCM) relaxes. Posterior vectors (LP/LMA) stretch open posterior urethral wall (broken lines). Bladder contracts. Pathogenesis. Damaged ligaments decrease the force of opening and closure vectors for urethra and anus. Surgery according to this theory. Reinforcement of damaged ligaments with implanted polypropylene tapes, Fig. 5.

Lower (anchoring) layer. The perineal body is the key anchoring point for contraction of bulbocavernosus and the external anal sphincter (EAS). The deep transverse perinei stabilizes the perineal body laterally. As well as being a sphincter in its own right, the EAS is the tensor of the perineal body and principal insertion point of the longitudinal muscle of the anus. The bulbocavernosus stretches and anchors the perineal body. Between EAS and the coccyx is the postanal plate, a tendinous structure which also contains striated muscle. It is proposed that the internal anal sphincter's role is limited to creating water and air-tight closure.

A new dynamic musculoelastic sphincter for anorectal closure (Fig. 3). The backward/downward muscle forces ('LP/LMA' arrows) stretch the rectum and rectovaginal fascia (RVF) around a contracted puborectalis muscle (PRM) to create the anorectal angle, and anorectal closure. Contraction of the forward force ('PCM' arrow) and perineal membrane muscles, tension and stabilize the perineal body (PB) and anterior wall of anus. Contraction of the external anal sphincter (EAS) creates a firm anchoring point for LMA, constricts the lower end of the anal canal, and tensions the perineal body. A similar mechanism closes urethra and bladder neck (Fig. 3).

Opening (defecation). With reference to Fig. 3, PRM relaxes. Anorectal stretch and volume receptors initiate a defecation reflex. Levator plate (LP) contraction stretches the posterior rectal wall backwards to full extension against the perineal body, and uterosacral ligament (USL). LMA (downward vector) contracts against the uterosacral ligament 'USL', to open out the tensioned posterior wall and

anorectal angle (broken lines, Fig. 3). Forward contraction by pubococcygeus 'PCM' 'splints' the perineal body and anterior anal wall to convert the anus to a semi-rigid tube. This "semi-rigidity" allows the posterior anal wall to be firmly pulled open, broken lines. This action reduces the internal resistance of the tube, facilitating the expulsion of feces during rectal contraction. A similar mechanism opens out the posterior wall of the urethra during micturition. During defecation, we hypothesize that at least initially, EAS must contract for LMA contraction to open out the anorectal angle (broken lines, Fig. 3). EAS then relaxes once the fecal bolus reaches the lower part of the anus, whereupon LP contraction would stretch the rectum up over the bolus, facilitating evacuation.

HYPOTHESIZED ROLE OF THE PUBORECTALIS IN CONTROL OF THE DEFECATION REFLEX

Voluntary puborectalis muscle 'PRM' contraction stretches the anus and rectum like a trampoline, to support the feces bolus, relieving its pressure on the stretch receptors. This decreases the afferent stimuli to the cortex, and so prevents the activation of the defecation reflex.

PATHOGENESIS

The role of collagen in anorectal dysfunction. The pelvic muscles contract against pelvic ligaments to open or close the anal canal. The main structural element of the pelvic ligaments is collagen. Collagen may stretch or weaken with age or childbirth (circles, Fig. 4), or it may be congenitally weak.¹⁵ A muscle requires a firm insertion point to contract efficiently. As the suspensory ligaments are the effective insertion points of all three directional forces (arrows, Fig. 3), both closure (continence) and opening (defecation) may be compromised. This explains how FI and "constipation" may co-exist in the same patient. Childbirth may cause dislocation of the collagenous "glue" connecting the mucosa to the anal serosa. Mucosal prolapse or anterior wall intussusception may cause a "motor" type of fecal incontinence

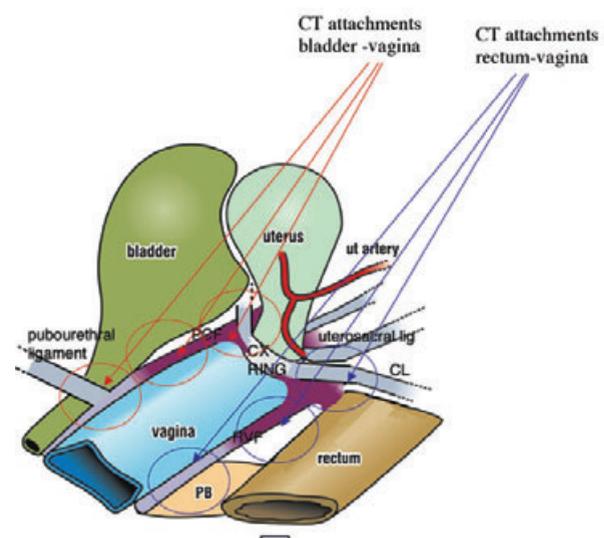


Fig. 4. – Pathogenesis-role of vaginal connective tissue in bladder and anorectal dysfunction. The circles indicate the intimate relationship between the fascia and ligaments ensheathing bladder, vagina and anorectum; also, overdistension of ligaments and fascia by the fetal head. Red circles: attachment points of ligaments and fascia to vagina and bladder; blue circles, to vagina and rectum. The circles pictorially explain the relationship between birth, urinary and feces dysfunction. CL = cardinal ligament; PB = perineal body.

by causing a constant stimulation of nerve receptors in the anal mucosa.

The effect of age and pregnancy on connective tissue

Age-related degenerations of connective tissue superimposed on childbirth damage explain the late onset of feces incontinence (FI) in most patients. Connective tissue in the pelvis is hormonally sensitive¹⁶ and loses elasticity with age¹⁷ and childbirth. This may explain the incidence of FI during pregnancy (hormonal depolymerization of collagen), after childbirth (connective tissue damage), and in old age (connective tissue degeneration). Cure of nulliparous females by reinforcing the pubourethral ligament with a midurethral polypropylene sling⁹ further supports a connective tissue hypothesis, in this instance, genetic ligamentous laxity. A relationship between genetic connective tissue dysfunction and stress incontinence has been previously described.¹⁵

Fecal incontinence

With reference to Fig. 3, the pubourethral (PUL) and uterosacral (USL) ligaments are the effective anchoring points for backward muscle force, levator plate (LP), and USL for the downward muscle force, longitudinal muscle of the anus (LMA). Laxity in either ligament may invalidate anorectal closure by the 2 directional muscle forces, LP and LMA (arrows, Fig. 3).

Minor degrees of inadequate closure may lead to leakage of wind; greater degrees to liquid or solid feces incontinence. Clearly a severely damaged external anal sphincter muscle may also cause FI. This is a different type of FI to that proposed by this theory, and will not be cured by using implanted tapes to repair damaged suspensory ligaments, Fig. 5. In addition to weakening its direct sphincter effect, a damaged EAS would invalidate the rotation of rectum around anus required to create the anorectal angle and watertight closure.

Bowel emptying disorders

With reference to Fig. 3, laxity in the suspensory ligaments, may weaken the forces stretching open the posterior anorectal wall (downward/backward arrows, 'LP/ LMA'), so the canal may not be fully stretched open during defecation. Laxity or rupture of the rectovaginal fascia (rectocele) or perineal body may also prevent the levator plate from stretching the rectal wall into a semi-rigid structure. Inability to open out the tube may vastly increase the intraluminal resistance to feces evacuation, given that the resistance within a tube varies inversely with the 4th power of the radius, Poiseuille's law. All this may lead to bowel emptying difficulties, which may lead to straining, which may further damage the connective tissue, if not the nerve supply of the organ. The patient interprets this evacuation disorder as "constipation". The feces may bulge into the vaginal cavity. In some instances the patient may need to "manually assist" evacuation by pressure on the perineal body, or the posterior vaginal wall. In this context, the role of the perineal body (PB) is very important. Not only does the PB occupy 50% of the posterior vaginal wall, it is the main anchoring point of the rectovaginal fascia, and therefore, levator plate, (Fig. 3), both critically important for the rectal stretching which precedes evacuation.

Mucosal prolapse and intussusception

With reference to Fig. 4, there is a close interconnection between the uterosacral ligaments and rectovaginal and pre-rectal fascia. The uterosacral ligaments suspend the fascial attachments of vagina and anterior rectal wall. Laxity in the uterosacral ligaments may cause sagging of the anterior

rectal walls. Inability to empty may cause straining, and this, in turn, may cause intussusception of the anterior rectal wall. Abendstein has reported a high rate of surgical cure of anterior rectal intussusception with a posterior polypropylene sling (n = 48) plus repair of rectovaginal fascia and perineal body, validated by post-operative evacuating proctography (Part 2, study No 12).

DIAGNOSIS OF DAMAGED CONNECTIVE TISSUE STRUCTURES

Diagnosis of the site of ligamentous damage is made using the pictorial algorithm (Fig. 1). As fecal incontinence may be caused by connective tissue damage in either the anterior or posterior zone, the presence of associated urinary and other symptoms (Fig. 1), helps designate the zone of damage. The site of damage may be confirmed by vaginal examination. Even minor degrees of damage may cause FI, or other pelvic floor symptoms.

TREATMENT OF DAMAGED CONNECTIVE TISSUE STRUCTURES

Non-surgical Treatment

New pelvic floor exercises adding squatting exercises¹⁸ and more recently, sitting on a rubber "fitball" instead of a chair to strengthen the slow-twitch muscles, have demonstrated an up to 78% improvement in patients with abnormal bowel symptoms such as constipation. The anatomical principle underlying these exercises is that strengthening a muscle also strengthens its ligamentous (or tendinous) insertion points. Clearly such techniques are contraindicated in patients with significant organ prolapse.

Surgical Treatment

Based on the Diagnostic Algorithm (Fig. 1) and clinical examination, polypropylene tapes are inserted to re-inforce damaged ligaments, Fig. 5. In addition to previous data⁹ greater than 80% improvement for patients with FI has been noted by Hocking (Study 9), Petros & Richardson (Studies No10 & 11), and Abendstein (Study No 12).

CONCLUSIONS

The theory predicts that lax ligamentous insertion points may cause suboptimal muscle contraction, and therefore

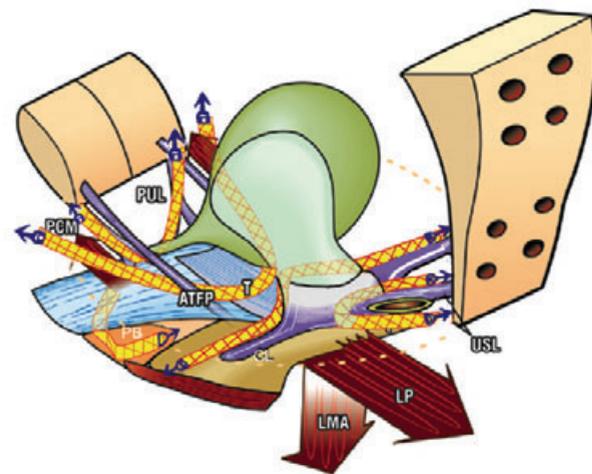


Fig. 5. – Surgical repair of damaged ligaments Polypropylene tapes "T" (in this case with attached soft tissue anchors) may be used to reinforce the main suspensory ligaments, pubourethral (PUL), uterosacral (USL) cardinal (CL) and arcus tendineus fascia pelvis (ATFP).

inability to open or close the anorectal tube. According to Popper's deductive criteria,¹⁰ an essential "next step" in the validation of this theory is the objective demonstration of the directional muscle vector forces, and their ligamentous anchoring points, during anorectal closure and opening (defecation) using X ray video or other dynamic imaging methods; then to directly test the hypothesis prospectively in patients with idiopathic fecal incontinence by surgically implanting polypropylene tapes to reinforce the anterior and posterior suspensory ligaments.

REFERENCES

1. Petros P and Ulmsten U. An Integral Theory and its Method for the Diagnosis and Management of Female Urinary Incontinence, *Scand J Urol Nephrol.*, Vol 27 Supplement No 153 - 1993; 6: 1-93.
2. Parks AG. Anorectal incontinence. *Proc Royal Soc Med* 1975; 68: 681-90.
3. Bartolo DCC, and Macdonald ADH. Fecal continence and defecation. In: *The Pelvic Floor, its functions and disorders.* Eds Pemberton J, Swash M, and Henry MM, WB Saunders, London, 2002; 77-83.
4. Freckner B, Von Euler C. Influence of pudendal block on the function of anal sphincters. *Gut* 1975; 16: 482-489.
5. Sultan AH, Kamm MA, Hudson CN, Thomas JM and Bartram CI. Anal-sphincter disruption during vaginal delivery. *N Eng J Med* 1993; 329: 1905-11.
6. Shafik A. A new concept of the anatomy of the anal sphincter mechanism and the physiology of defecation. *Colo-proctology* IV, 1982; 1: 49-54.
7. Swash M, Henry MM, Snooks SJ. Unifying concept of pelvic floor disorders and incontinence. *Journal of the Royal Society of Medicine* 1985; 78: 906-911.
8. Bharucha AE. Fecal Incontinence. *Gastroenterology* 2003; 124: 1672-1685.
9. Petros PE. Cure of urinary and fecal incontinence by pelvic ligament reconstruction suggests a connective tissue etiology for both. *International Journal of Urogynecology* 1999; 10: 356-360.
10. Popper KR. A Survey of some fundamental problems. Theories. Falsifiability. The problem of the empirical basis. Degrees of testibility. Simplicity. *The Logic of Scientific Discovery.* Unwin, Hyman, London, 1980; 27-146.
11. Zacharin RF. A suspensory mechanism of the female urethra. *Journal of Anatomy* 1963; 97: 423-427.
12. Petros PE. The pubourethral ligaments-an anatomical and histological study in the live patient. *Int J Urogynecology* 1998; 9: 154-157.
13. Courtney H. Anatomy of the pelvic diaphragm and ano-rectal musculature as related to sphincter preservation in ano-rectal surgery. *American Journal Surgery* 1950; 79: 155-173.
14. Macchi V, Porzionato A, Stecco C, Benettazzo F, Stecco A, Parenti A, Dodi G, De Caro R. Histo-topographic study of the longitudinal anal muscle. *Pelviperrineology* 2007; 26: 30-32.
15. Ulmsten U, Ekman G, Giertz G, Malmstrom A. Different biochemical composition of connective tissue in continent and stress incontinent women. *Acta Obstet & Gynecol Scand* 1987; 66: 455-457.
16. Smith P, Heimer G, Norgren A & Ulmsten U. Steroid receptors in pelvic muscles and ligaments in women. *Gynecologic & Obstet Investigations* 1990; 30: 27-30.
17. Yamada H. Aging rate for the strength of human organs and tissues. *Strength of Biological Materials*, Williams & Wilkins Co, Balt. (Ed) Evans FG. 1970; 272-280.
18. Petros PE and Skilling PM. Pelvic floor rehabilitation according to the Integral Theory of Female Urinary Incontinence. First report. *European Journal of Obstetrics & Gynecology and Reproductive Biology* 2001; 94: 264-269.

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Correspondence to:
PETER PETROS
E-mail: kvinno@highway1.com.au