Review Article | Basic and Mechanism Sciences

A brief physiology and pathophysiology of the anorectum based on the Integral Theory paradigm

Darren M. Gold[1]^, Michael Swash[2]^, Ahmed Farag[3]^, Shuqing Ding[4]^, Giulio Santoro[5]^, Giuseppe Dodi[5]^

[1]Department of Surgery, St Vincent's Clinical School, University of NSW, Sydney, NSW, Australia; [2]Royal London Hospital, Barts and the London School of Medicine, QMUL, London, UK; [3]General and Colorectal Surgery Department, Cairo University, Giza, Egypt; [4]Northwestern Health Sciences University, Bloomington, MN, USA; [5]Department of Colorectal Surgery, University of Padova, Padova, Italy

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Correspondence to: Darren M. Gold, MSc, FRACS, FRCS(Gen), FRCS(Eng), MBBS. Senior Lecturer, Department of Surgery, St Vincent's Clinical School, University of NSW, 390 Victoria Street, Darllinghurst, Sydney, NSW 2010, Australia. Email: dandjgold@googlemail.com.

Abstract: The remit of this review is confined to the experimental scientific works and surgeries based on the Integral Theory paradigm. The video abstract summarizes the anorectal function, how ligaments cause dysfunction and cure of fecal incontinence and obstructed defecation by ligament repair. Anorectal function is reflex and binary, with cortical and peripheral components. The same three oppositely acting reflex muscle forces which open and close the bladder, contract against the pubourethral (PUL) and uterosacral (USL) ligaments: (I) to close the anorectum for continence when the puborectalis muscle (PRM) contracts forwards; (II) to open the anorectum prior to evacuation when the PRM relaxes; (III) to stretch the rectum in opposite directions to support the anorectal stretch receptors "N" to prevent premature activation of the defecation reflex, (fecal urgency). Weak or loose PULs or USLs may cause dysfunction of closure, of evacuation, and inability to control the defecation reflex (fecal urgency). Repair of the PUL and USL can improve or cure these dysfunctions. The perineal body (PB) acts as an anatomical support for the distal vagina, anorectum and external anal sphincter (EAS). It serves as an anchoring point for the forward action of the pubococcygeus muscle (PCM), which tensions the anterior rectal wall during closure and defecation. Bladder and bowel dysfunction have a similar pathogenesis, ligament laxity, mainly pubourethral and uterosacral, with added PB damage for anorectal dysfunction. PB damage can cause obstructive defecation and descending perineal syndrome (DPS). Repair of damaged PUL and USL can restore the closure and evacuation functions of both bladder an anorectum. DPS can be cured by repair of the PB's suspensory ligaments, deep transversus perinei.

Keywords: Rectum; anus; binary control; fecal incontinence (FI); obstructed defecation

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^ ORCID: Darren M. Gold, 0000-0003-4478-9747; Michael Swash, 0000-0002-8717-8914; Ahmed Farag, 0000-0002-1580-906X; Shuqing Ding, 0000-0002-5875-8629; Giulio Santoro, 0000-0002-0086-3522; Giuseppe Dodi, 0000-0002-8683-5468.

Introduction

The key points of the article are summarized in the video abstract (Video S1).

The remit of this review is confined to the experimental scientific works and surgeries of the Integral Theory paradigm (ITP). Anorectal function is reflex and binary, with cortical and peripheral components (1). The musculoelastic theory of anorectal function and dysfunction is part of the ITP (1). A full account of the original scientific work on which the colorectal part of the ITP is based, can be found in the 12 original experimental studies (see https://www.researchgate.net/publication/267778578_The_Musculo-Elastic_Theory_of_anorectal_function_and_dysfunction) (1).

Functional anorectal anatomy

There are four main reflex pelvic muscles, pubococcygeus muscle (PCM), levator plate (LP), conjoint longitudinal muscle of the anus (LMA), and puborectalis muscle (PRM). The PCM and LP contract against pubourethral ligaments (PULs); the LMA and LP contract against the uterosacral ligaments (USLs); the PRM contracts only against the pubic symphysis. These four muscles contribute to the binary control of anorectal functions (*Figure 1*).

Four reflex striated pelvic muscles open and close the anorectum and control fecal urgency

With reference to *Figure 1*, the same three reflex forces, PCM, LP, LMA, which control bladder continence and evacuation, perform the same functions for the bowel, and contract against the PUL and USL:

(I) To close the anorectum for continence when the PRM contracts forwards (*Figure 1*, Video S2).

(II) To open the anorectum prior to evacuation when the PRM relaxes (*Figure 1*, broken lines; Videos S3,S4).

(III) To stretch the rectum in opposite directions to support the anorectal stretch receptors "N" to prevent premature activation of the defecation reflex (fecal urgency) (*Figure 1*).

Role of the perineal body (PB)

The PB attaches to the distal vagina, anorectum and external anal sphincter (EAS) and acts as an anatomical support for the distal vagina, rectovaginal fascia and anus (*Figure 1*). The PB is suspended from the descending pubic rami by the deep transversus perinei ligaments (DTPs) (*Figure 2*). DTPs are about 4 cm long, 7 mm in diameter, and attach behind the rami exactly between the upper 2/3 and lower 1/3. The PCM contracts forwards against the PB to tension the anterior rectal wall and anus during closure and defecation. The PB is an insertion point for the EAS.

Anorectal and bladder control

Anorectal and bladder control are very similar (1) (*Figure 3*). Both are binary controlled, and are regulated by two cortically controlled reflexes, either closed (closure reflex) or open (micturition, defecation reflexes). These reflexes comprise cortical and peripheral components (striated pelvic muscle/ligaments) and are mutually exclusive. The binary control system works like an electric switch which controls two different circuits, one at a time, either closed or open. Though control is autonomic, the cortex exerts voluntary control over both closure and evacuation reflexes (2,3).

With reference to *Figure 3*, the binary cortico/peripheral control of bladder and bowel is virtually identical. Afferent fibres from stretch receptors "N" in the bladder and bowel transmit afferent nerve emptying impulses to the cortex which interprets them as "fullness". By reflexly stretching the bladder and bowel bidirectionally (large arrows), the muscles tension the underlying supports of the stretch receptors "N" of each organ to prevent them from firing off emptying impulses prematurely, thereby controlling inappropriate activation of the micturition and defecation

Highlight box

Key findings

- Four reflex muscles forces open and close the anorectum. Dysfunction is mainly caused by pubourethral ligament, uterosacral ligament (USL) and perineal body (PB) laxity and can be cured or improved by repair thereof.

What is known and what is new?

- Pathogenesis of fecal incontinence (FI) and obstructive defecation syndrome (ODS) is said to be multifactorial.
- Binary cortico/peripheral control of bladder and bowel is virtually identical: pelvic muscles contract against pelvic ligaments to open and close the anorectum. Damaged ligaments are the main cause of anorectal dysfunctions, FI and ODS.

What is the implication, and what should change now?

- The pathogenesis of FI and ODS is almost identical and both can be cured or improved at the same time by USL and/or PB repair.

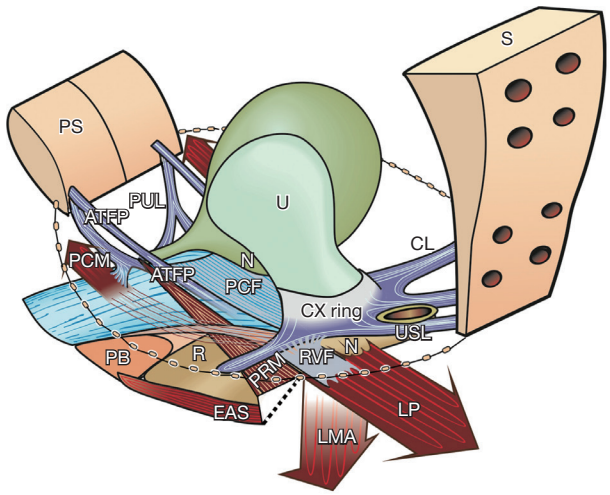

Figure 1 Integrated structural anatomy of the pelvic organs, ligaments and muscles. Bone: PS, S. Suspensory ligaments: PUL, ATFP, USL, CL. Anorectal stretch receptors N, which respond to pressure of rectal contents to activate the defecation reflex (felt as urge to defecate); "N" at bladder base, urothelial stretch receptors which activate the micturition reflex (urinary urge) and rectum. Muscles: PCM, LP, LMA, PRM. Supporting fascia: PCF, RVF. Perineal anchoring structures: PB, EAS. Reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd edition. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright. U, uterus; R, rectum; CX ring, cervical ring; PS, pubic symphysis; S, sacrum; PUL, pubourethral ligament; ATFP, arcus tendineus fascia pelvis; USL, uterosacral ligament; CL, cardinal ligament; PCM, pubococcygeus muscle; LP, levator plate; LMA, conjoint longitudinal muscle of the anus; PRM, puborectalis muscle; PCF, pubocervical fascia; RVF, rectovaginal fascia; PB, perineal body; EAS, external anal sphincter.

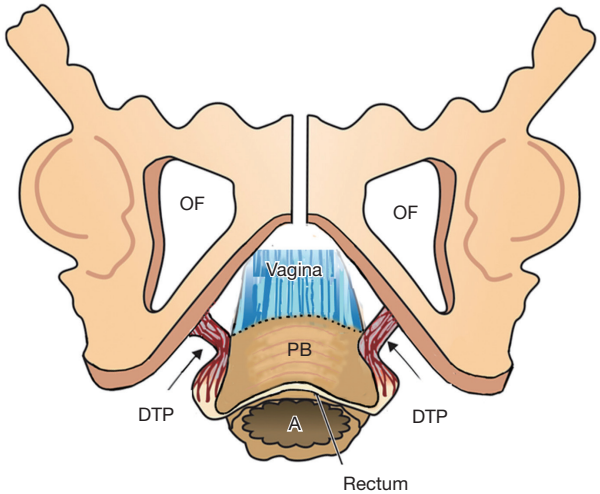

Figure 2 PB. The lower part of the vagina has been cut away. The "PB" is suspended from the pubic rami by the transverse perineal ligaments "DTP". These are 4 cm long. They are inserted behind the descending rami, exactly between the upper 2/3 and lower 1/3. Reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd edition. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright. OF, obturator fossa; PB, perineal body; DTP, deep transversus perinei; A, anus.

reflexes, which is sensed by the cortex as urgency. If convenient to empty, the closure reflex shuts down, and the emptying reflexes (micturition and defecation) are activated. The posterior walls of the urethra and anorectum are actively pulled open (broken lines) by the LP/LMA immediately prior to evacuation. This external opening exponentially decreases resistance to urine or fecal flow, thereby facilitating evacuation (4).

The mechanics of normal anorectal closure and opening

With reference to *Figure 1* and Video S2, for anorectal closure, the PRM contracts forward against the symphysis; the LP contracts backward against the PUL to tension the rectum; the LMA contracts downward against the USL to rotate the rectum around a contracted PRM to close the anorectal angle.

For defecation, in *Figure 1*, (broken lines behind rectum) and Video S3, the PRM relaxes; the LP/LMA pull the posterior rectal wall backward/downward to open out the anorectal angle; the PCM contracts forward to stiffen the anterior rectal wall; the lateral walls of the rectum are pulled outwards, most likely by contraction of the iliococcygeus muscle via its insertion into the lateral wall of PCM (Video S4). The rectum opens radially and appears to shorten by downward shrinking of the walls, as demonstrated in both sagittal and anterior-posterior views (Video S4). These opening actions exponentially decrease the internal anal resistance to the passage of feces to facilitate fecal evacuation (4,5).

Dysfunction

Damage to any part of the binary system may interfere with

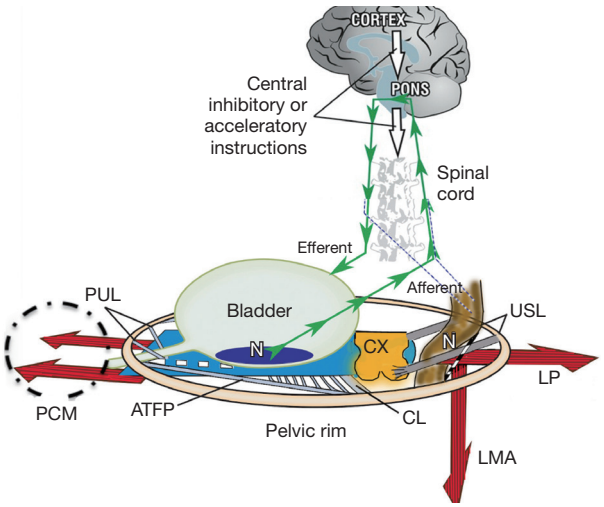

Figure 3 Binary cortico/peripheral control of bladder and bowel. Binary control of bladder and bowel is virtually identical. "N" are the stretch receptors in the bladder and anorectum which send afferent impulses of fullness to the brain to compute activation by the micturition or defecation reflexes. Broken lines below the urethra and behind the rectum indicate "open" (evacuation position). Anatomical damage to any part of the system may interfere with the binary control of all bladder and anorectal closure and opening functions. Reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd edition. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright. LP, levator plate; LMA, conjoint longitudinal muscle of the anus; USL, uterosacral ligament; CX, cervix; CL, cardinal ligament; ATFP, arcus tendineus fascia pelvis; PUL, pubourethral ligament; PCM, pubococcygeus muscle.

the binary control of all its functions to cause anorectal (and bladder) dysfunctions (*Figure 3*). For example, these include damage to the facilitatory or inhibitory centres of the brain or spinal cord; to the afferent or efferent nerves (for example by multiple sclerosis); to any part of the peripheral control system, be it muscle or ligament or the organ itself; by infection or local pressure by cancer to the stretch receptors "N" or by external pressure on "N" by cervical fibroids for the bladder, or rectal mucosal prolapse for the anorectum.

Fecal incontinence (FI)

In an individual woman, FI may have many causes. The scope of this review is limited to the role of loose ligaments in FI, as indicated in the diagnostic algorithm (*Figure 4*). Note how FI can be caused by both PUL and USL weakness. The most common cause of FI is USL weakness or laxity. This type of FI is associated with uterine/apical prolapse (often minimal) and other posterior zone symptoms (pain and bladder symptoms) (*Figure 4*, 3rd column). Other co-occurring manifestations of USL laxity are obstructive defecation, and sometimes, anterior rectal wall intussusception (*Figure 5*). A not so well recognized cause of FI is PUL weakness, which co-occurs with stress urinary incontinence (SUI), which is known as "double incontinence". The pathogenesis of "double incontinence" is explained by reference to *Figure 6*: a weak or loose PUL anchoring point may weaken the backward contraction of the LP and its ability to stretch the rectum into the semirigid structure required for it to be pulled down and "kinked" like a garden hose around a contracted PRM. Any looseness in the PUL can result in FI as well as SUI. A hemostat applied at the midurethra on one side as in *Figure 6*, will control both urinary and FI seen on coughing (7). Both the SUI and FI of "double incontinence" may be cured by a midurethral sling (6).

Obstructive defecation syndrome (ODS)

ODS is a form of constipation. With reference to *Figure 1*, a loose USL anchoring point may weaken pelvic muscle ability to open the anorectal angle (broken lines), so that the rectum contracts against an unopened anus. This is perceived as ODS. Both FI and ODS can co-occur with anterior rectal wall intussusception (*Figure 6*), and both can be cured by a posterior sling inserted to reinforce the weakened USLs (8).

How uterine prolapse may cause anterior rectal wall intussusception

The USLs are loosely attached to the lateral walls of the rectum with a number of fine small narrow ligaments (9). With reference to *Figure 5* (bottom figure), childbirth may stretch and elongate the USLs, dragging the anterior rectal wall forwards, loosening it, so it prolapses inwards as an intussusception. *Figure 5* (bottom figure) shows how the attachment of USLs to the lateral wall of the rectum drags the more distensile lateral wall with them; as the USLs lengthen, they splay laterally. The anterior rectal wall, therefore, also elongates laterally, weakening it structurally. Its collagen concentration lessens and the anterior rectal

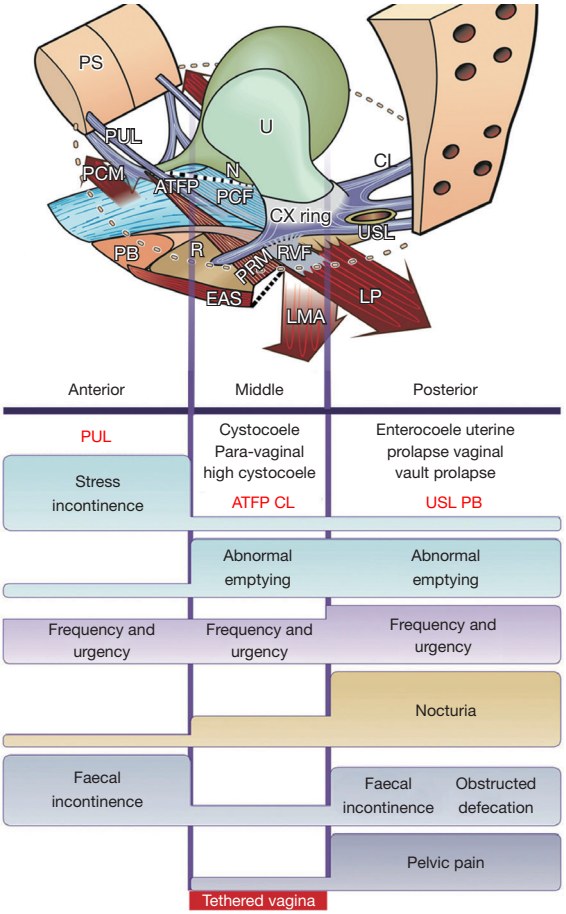

Figure 4 Symptom-based diagnostic algorithm. The ligaments fall naturally into the 3 zones of causation: PUL, CL, ATFP, USL, PB. Anterior zone: urethral meatus to bladder neck. Middle zone: bladder neck to cervix. Posterior zone: cervix to perineal body. Boxes are ticked with questionnaire answers, even if a symptom occurs only "sometimes". The prolapses in the algorithm correlate with ligament damage in the columns. Symptom groupings in the columns help deduce which ligaments cause which symptoms. The broken lines indicate the open positions of the bladder and bowel. Bladder and bowel symptoms which co-occur, as in the posterior zone are all potentially curable by USL repair, with or without perineal body repair for rectocele. Reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd edition. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright. U, uterus; R, rectum; N, bladder base stretch receptors; CX ring, cervical ring; PS, pubic symphysis; S, sacrum; PUL, pubourethral ligament; ATFP, arcus tendineus fascia pelvis; USL, uterosacral ligament; CL, cardinal ligament; PCM, pubococcygeus muscle; LP, levator plate; LMA, conjoint longitudinal muscle of the anus; PRM, puborectalis muscle; PCF, pubocervical fascia; RVF, rectovaginal fascia; PB, perineal body; EAS, external anal sphincter.

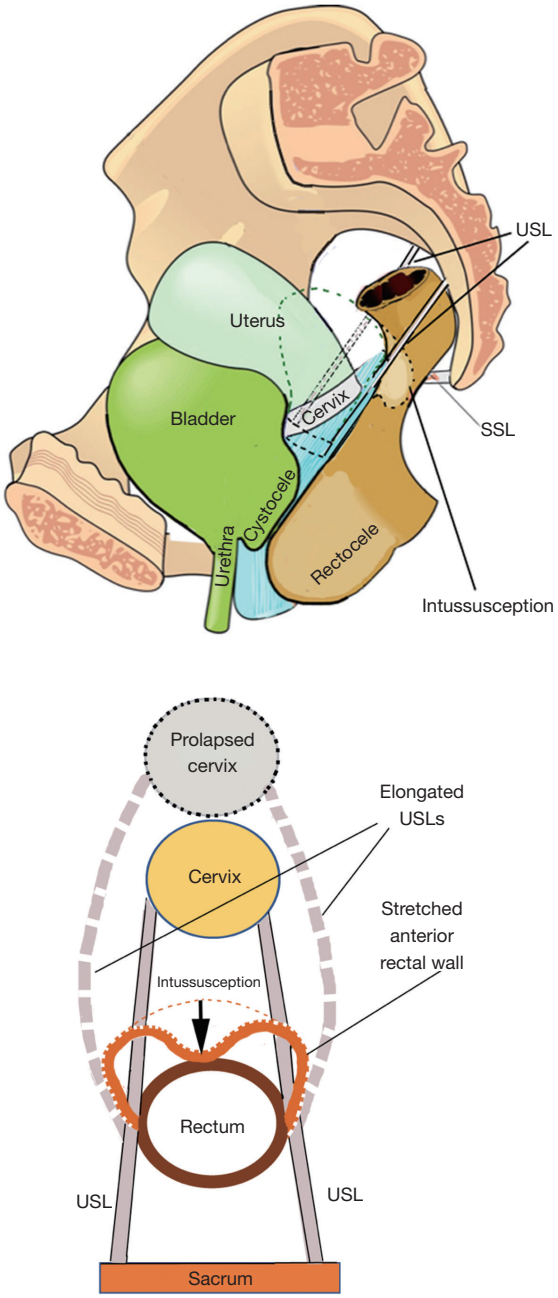

Figure 5 Anterior rectal wall intussusception caused by uterine prolapse. Top figure: sagittal view. Elongation of USL. Bottom figure: view from above. USLs splay laterally as they lengthen with the prolapse, as does the anterior rectal wall which invaginates to cause intussusception. Adapted and reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd edition. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright. USL, uterosacral ligament; SSL, sacrospinous ligament.

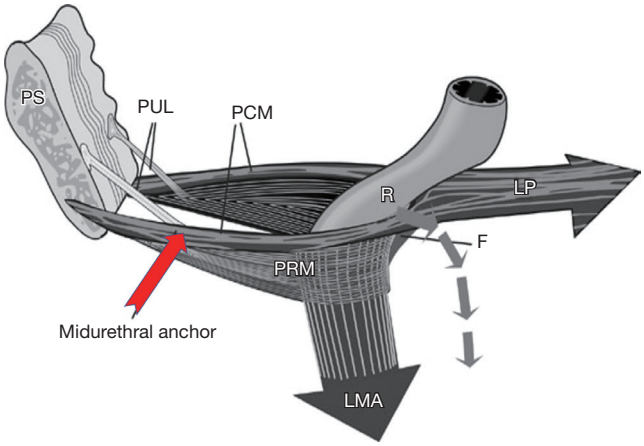

Figure 6 Mechanism of double incontinence (stress urinary and stress fecal incontinence). A hemostat (red arrow) is placed in the midurethral position in the vagina. It acts as an anchor for the PUL ligaments against which the "PCM" and "LP" contract to control urine and fecal loss on effort. "F" (small arrows), represents the "LP/LMA" rotational vector forces which stretch the rectum around a contracted "PRM" to close the anorectum. Reused with permission from *Pelviperineology* (6). PS, pubic symphysis; PUL, pubourethral ligaments; PCM, pubococcygeus muscle; R, rectum; PRM, puborectalis muscle; LMA, conjoint longitudinal muscle of the anus; LP, levator plate.

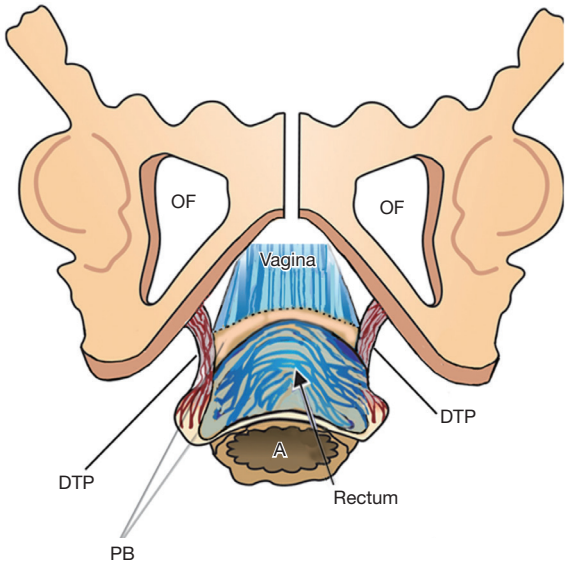

Figure 7 DPS. The perineal body "PB" has been stretched, narrowed and flattened; the rectal serosa has been broken and rectal mucosa (rectum) has spread laterally to adhere to the vagina, PB, and the DTPs (10). Reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd edition. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright. OF, obturator fossa; DTP, deep transversus perinei ligament; PB, perineal body; A, anus; DPS, descending perineal syndrome.

wall invaginates to cause intussusception.

Pathogenesis of perineocele and descending perineal syndrome (DPS)

The pressure of the fetal head exiting the vagina stretches the PB laterally and downwards. The DTPs are stretched and elongated (*Figure 7*). Pressure on the anterior rectal wall stretches it laterally and often splits its serosal and muscular layers, leaving the thin anal mucosa exposed and adherent to the posterior vaginal wall which now bulges forward to cause perineocele (low rectocele) (10). If the PB and DTP are sufficiently stretched, the perineum may descend as "DPS".

Anorectal muscle vectors require firm PUL and USL for balanced closure and defecation

The four opposite striated muscle forces (*Figure 8*, large arrows), are always in balance. The forward vectors (PCM, PRM) and backward vectors (LP/LMA) act externally on the anorectum to facilitate normal closure and opening of the anorectal angle "A" (*Figure 8*). The PRM surrounds the rectum, but it is not attached to it. During closure, the PRM, which lies below the LP, contracts forward to pull the posterior wall of the rectum upwards and forwards. The backward muscle forces (LP/LMA) contract against competent USLs, to stretch the rectum down and around the PRM to close it in a "kinking" action, which also narrows the anorectal angle "A" (*Figure 8*). The PCM contracts forwards against the PUL during anorectal closure to steady the anterior rectal wall. During defecation, the PRM relaxes, the LP/LMA open out the posterior rectal wall enlarging the anorectal angle "A" (*Figure 8*). The PCM continues to contract during defecation; its role is to stretch the PB and anterior wall of the anus forwards to enlarge the diameter of the anal tube (11).

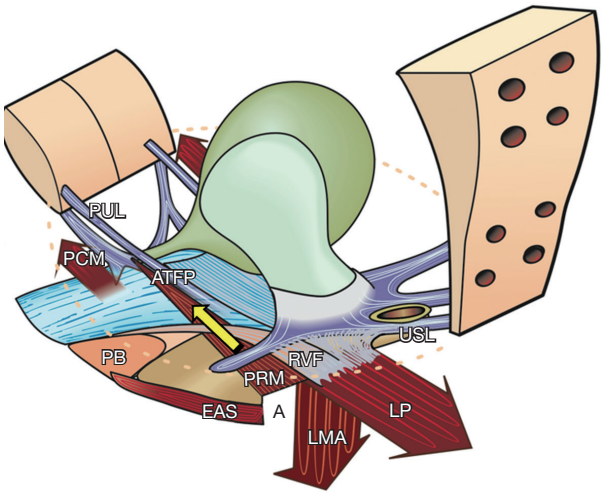

Figure 8 Anorectal angle formation. Anorectal angle. If "PUL" and "USL" are competent, the opposite muscle forces, "PCM", "LP/LMA" are in balance, and the anorectal angle "A" is normal. The small yellow arrow represents unbalanced forward action of the "PRM" when the "LP/LMA" weaken because of a weak "USL". Reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd edition. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright. PUL, pubourethral ligament; PCM, pubococcygeus muscle; ATFP, arcus tendineus fascia pelvis; PB, perineal body; RVF, rectovaginal fascia; PRM, puborectalis muscle; EAS, external anal sphincter; USL, uterosacral ligament; LP, levator plate; LMA, conjoint longitudinal muscle of the anus.

Imbalance of forward and backward forces may cause anorectal dysfunction

The anorectal angle "A" (*Figure 8*) is formed by the balanced backward/downward contraction of the LP/LMA against forward contraction of (mainly) the PRM. USL laxity will weaken LP/LMA forces which contract against USL. In relative terms, the PRM contracts more strongly than the LP/LMA; consequently, the directional muscle system (*Figure 8*, 3 large arrows) becomes unbalanced, and the PRM indents the posterior rectal wall, giving the impression of "paradoxical contraction" or "spasm" of the PRM as seen with defecating proctograms and ultrasound examinations (*Figure 9*). *Figure 10* demonstrates how repair of the PUL and USL restored the anatomy both at rest and on straining, and also restored the function (see case report below).

Ultrasound monitored loss of pelvic muscle equilibrium cured by PUL & USL repair

Experimental study No. 6: reused from (11). Copyright 2008, with permission from *Pelviperineology*.

The patient, 49 years old, parity 3, presented with 2nd degree prolapse, difficulty with defecation, FI, SUI, nocturia and chronic pelvic pain (1). Preoperative defecating proctogram demonstrated an acute anorectal angle similar to that of the preoperative ultrasound (*Figure 9*). There was no rectocele or rectal intussusception. PUL and USL

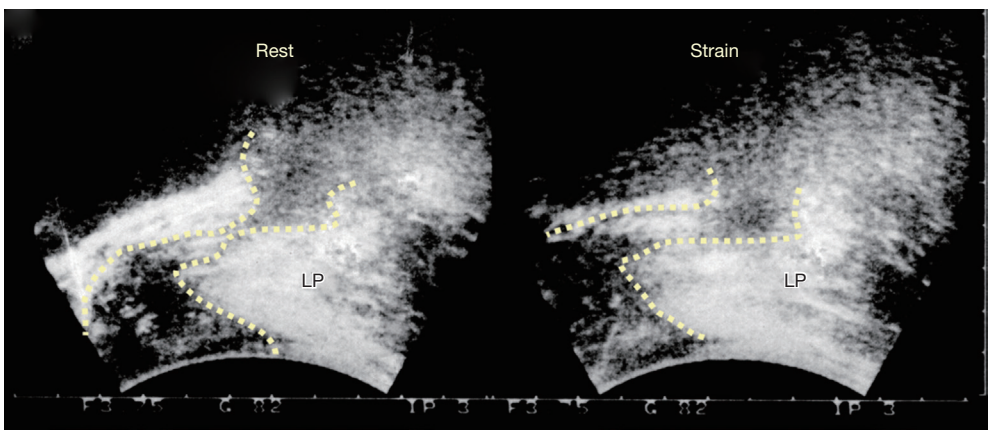

Figure 9 Transperineal ultrasound prior to PUL and USL reconstruction. Acute anorectal angle at rest and straining. Note excessive indentation of the posterior rectal wall at rest by LP. On straining (non-evacuatory), there is descent of the posterior rectal wall, but no significant change in the anorectal angle. Dotted lines demonstrate rectal wall. Reused with permission from *Pelviperineology* (11). LP, levator plate; PUL, pubourethral ligament; USL, uterosacral ligament.

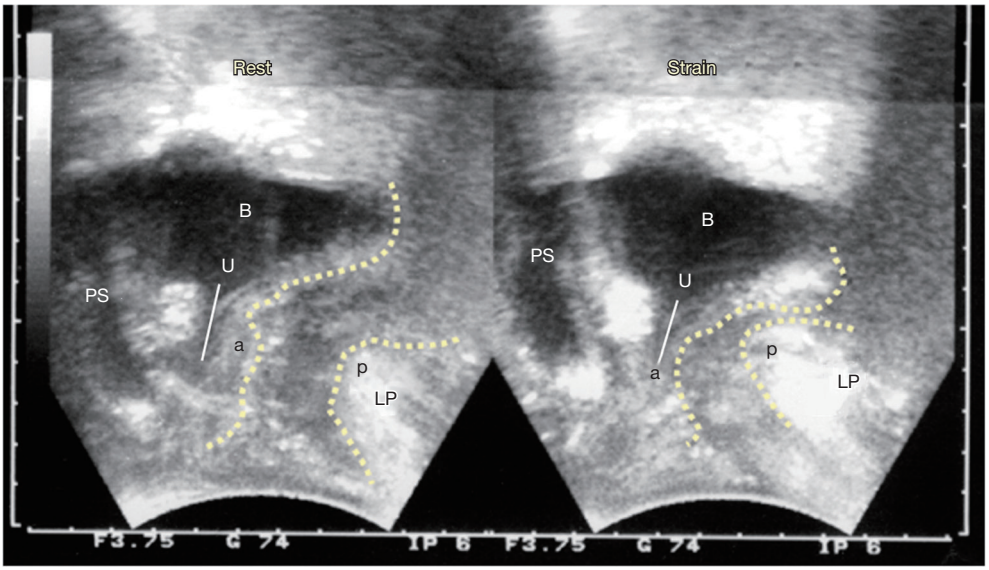

Figure 10 Transperineal ultrasound after PUL and USL reconstruction. Postoperative perineal ultrasound, at rest and straining. Note restoration of normal anatomy "at rest", and also, a more acute A/R angle on straining following repair with PUL and USL slings. During straining (non-evacuatory), an upward/forward force seems to have been exerted on to "p" (posterior rectal wall). This force at "p", can only be from the PRM. It lifts the rectum forwards and upwards towards the upper part of the "U" and bladder neck; meanwhile, the anorectal angle has become more acute by the rectum being pulled downward and backwards around "p" (PRM). This sequence of events supports those detailed for anorectal closure in *Figure 1*. The vertical white line "U" indicates urethra; a&p indicate the anterior and posterior walls of the rectum. Dotted lines demonstrate rectal wall. Reused with permission from *Pelviperineology* (11). PS, pubic symphysis; B, bladder; LP, levator plate; PUL, pubourethral ligament; USL, uterosacral ligament; A/R, anorectum; PRM, puborectalis muscle.

sling repairs cured or substantially improved both bladder and bowel symptoms; also restored was the appearance of normal anatomy on straining in the transperineal ultrasound (*Figure 10*) which was attributed to restoration of the balance of the four directional forces from *Figure 8*. With reference to *Figure 9*, the indentation of the posterior wall of the rectum, both at rest and on straining, is consistent with the inability of the LP/LMA to balance the forward contraction of the PRM in *Figure 8*. On comparing *Figure 9* to *Figure 10*, following USL repair (again with reference to *Figure 8*), the LP/LMA could now stretch the rectum backwards and downwards against USLs, in a rotating motion, to close ("kink") the anorectal angle. At the same time, the PRM contracts forwards to provide a firm point for rotation of the rectum, and the PCM contracts forward to stabilize the anterior wall of the anus. A firm USL is required for the LP/LMA to exert maximum contractile force, and a firm PUL for PCM. The PRM contracts directly against the symphysis, so its contractile force is not affected by any ligament damage.

Surgical validation of USL causation of bladder/bowel dysfunctions

In addition to cure of several bladder dysfunctions (urge, frequency, nocturia, emptying) application of USL slings achieved cure rates ranging between 65% and 85% for dysfunctions of anorectal closure ("FI") and evacuation ("ODS") (12-19). If women with either FI or ODS have co-occurring bladder or pain symptoms (*Figure 4*, posterior zone), there is a high probability of surgical cure of both FI and ODS with USL repair, as demonstrated (12-19).

Prima facie, these results go some way towards validating ligament pathogenesis for these conditions. According to the Theory (1), however, it is the pelvic muscles which close and evacuate the anorectum (albeit by contracting against ligaments). Therefore, it is possible that failure to cure FI and ODS may have been due to pelvic muscle damage (20), which raised the question, which is the main pathogenic factor, damaged ligaments, or damaged muscles? This question was answered indirectly by a blinded biopsy muscle

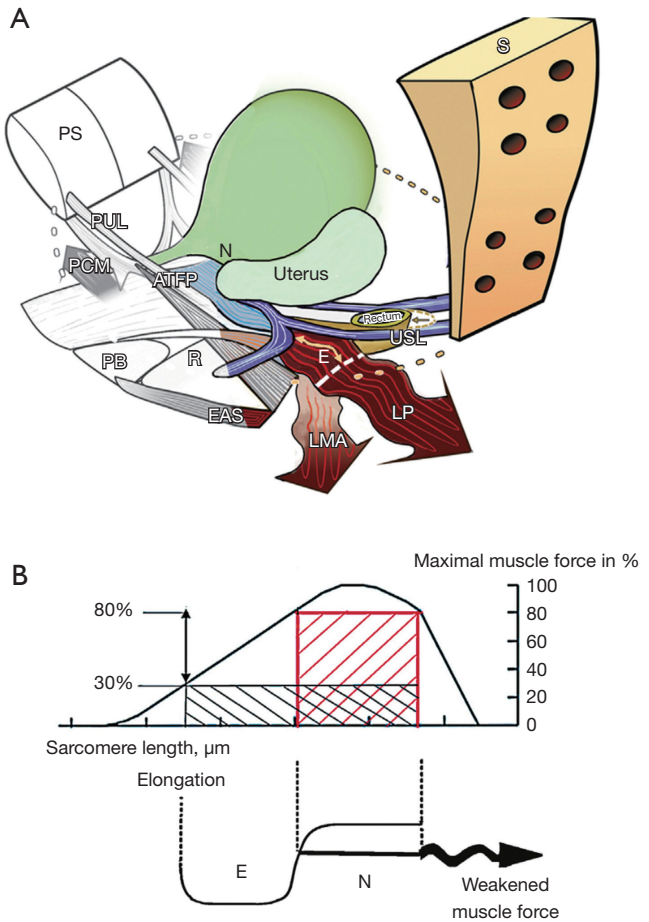

Figure 11 Striated muscles require firm insertion points for optimum contractility. (A) Three-dimensional view from above. The uterus has prolapsed to first degree. The "USLs" have elongated by "E". The "LP" and "LMA" have also effectively lengthened because they contract against the "USLs". The rectum also has descended, by virtue of its attachments laterally to the elongated "USL". The wavy shape of the "LP" and "LMA" indicate diminution of contractile strength. (B) Gordon's law (22): a striated muscle contracts optimally over a short normal length only ("N", red square). Elongation of the muscle results in rapid loss of contractile force ("E", black rectangle), from 80% to 30%. Adapted and reused from Petros P. The female pelvic floor function, dysfunction and management according to the Integral Theory. 3rd edition. Heidelberg: Springer Berlin; 2010. With permission from Peter Petros; retains ownership of the copyright. PS, pubic symphysis; PUL, pubourethral ligament; PCM, pubococcygeus muscle; ATFP, arcus tendineus fascia pelvis; N, bladder base stretch receptors; R, rectum; PB perineal body; EAS, external anal sphincter; USL, uterosacral ligament; LP, levator plate; LMA, conjoint longitudinal muscle of the anus; S, sacrum.

study of 47 women having a midurethral sling for SUI. The histology demonstrated that the vast majority had muscle injury, yet 90% were cured of their SUI the next day (21). Further evidence can be deduced from the anatomical pathway hypothesized by the anorectal theory for FI and ODS (1). This is graphically represented in *Figure 11*. The USLs, the insertion point of the LP/LMA, are loose and the uterus has prolapsed. The LP and LMA also effectively lengthen by "E", "A" (*Figure 11*). Consequently, their contractility diminishes ("B", *Figure 11*), as the striated muscle sarcomere requires a firm insertion point for optimum contractility (22).

Conclusions

Within the context of the ITP, bladder and bowel dysfunctions co-occur mainly because of similar ligamentous pathogenesis. Both can be cured or improved at the same time by appropriate ligament repair. Though high cure rates have been achieved with ligament repair for either dysfunction, consideration must be given to the possible effects of nerve and muscle damage in an individual patient, which cannot be diagnosed and, therefore, cannot be surgically repaired.

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Footnote

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All clinical procedures described in this study were performed in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patients for the publication of this article, accompanying images and videos. Human participation in the videos was by patients permission on the basis it was deidentified.

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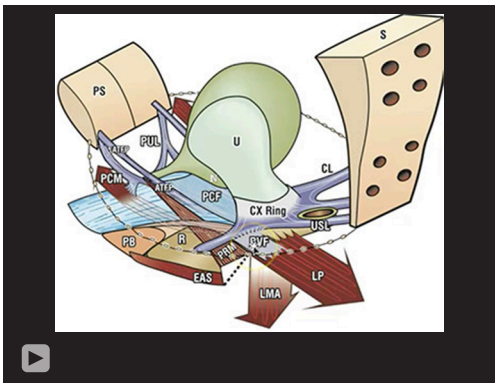
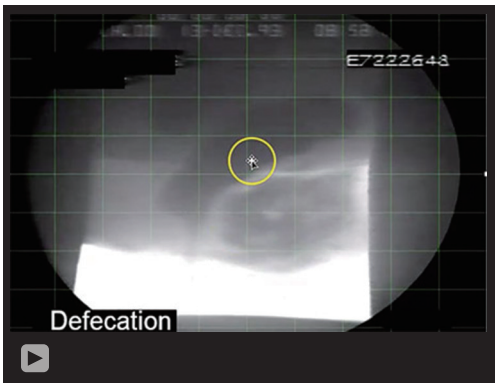
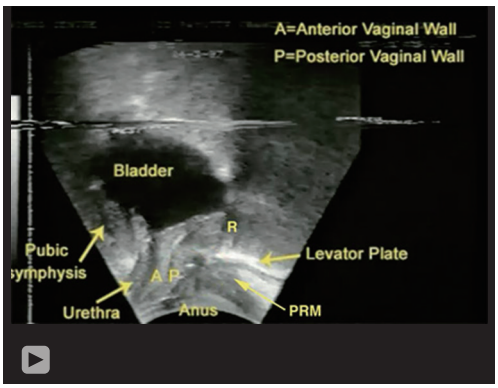
Supplementary



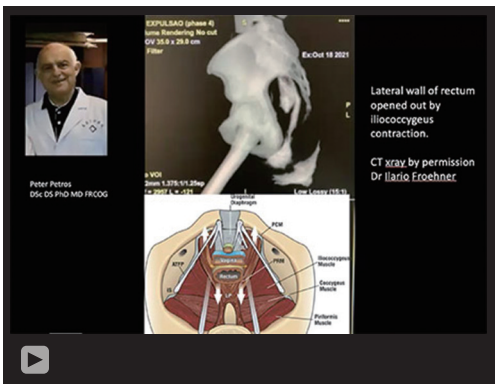
Video S1 Video abstract.



Video S3 Defecation sagittal view. X-ray defecography.



Video S2 Anorectal closure. Ultrasound.



Video S4 Lateral distension of rectal walls during defecation. CT scan. By permission Dr. Ilario Froehner Jr.

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