Commentary

Invited comment: A new theory of anorectal function

(P. Petros, M. Swash et al. - issue 3, 2008)

General Comments

Thanks for asking us to comment on this manuscript by Petros and Swash, on various studies and case reports focusing on the central hypothesis that augmenting ligamentous support improves muscular loading which leads to improved muscular contractility. The studies on the continence mechanism also hypothesize that directional forces of muscular and ligamentous opposition are important.

Many studies have an running theme of a mid urethral sling procedure for urinary incontinence producing "cure" of concomitant "idiopathic" faecal incontinence. While we don't dispute the observational findings, these studies provide little objective evidence to support the hypotheses of the aetiological role of ligament and muscle loading abnormalities. There are several methodological limitations which we have commented on separately in turn.

Padmore in 1918 initially suggested the ship in a dry dock theory for uterine support, and later Delancey described the 3 layers of fascial support in the pelvic floor based on objective histological evidence. It would seem a plausible hypothesis that reinforcing fascial support should augment pelvic floor dynamics and function, however some of the claims of reconstituting anatomy are poorly founded with little evidence to support claims given in the discussions.

The term "idiopathic" faecal incontinence is used throughout without fully explaining how this group of patients is defined and on what specific clinical and investigational criteria. The aetiopathogenesis of urinary and faecal incontinence is rarely a singular process. Insults to the pelvic floor are usually multiple (including chronic straining, increased intra abdominal pressure effects, parturition and the menopause). Thus, it is difficult to make generalizations from case reports, retrospective analyses and uncontrolled studies, reflecting some of the evidence base presented here.

There is no doubt that different collagen types (such as those in patients with EDS III or benign joint hypermobility syndrome) are more prone to pelvic organ prolapse and ligamentous laxity [Alwari et al, Grahame R et al]. However surgery on this type of collagen has a higher risk of recurrence and it is unclear from this work how this type of surgery will benefit those with congenital collagen weakness.

Pelvic floor weakness is age and parity dependent. Collagen types change with age, from a more supple type I collagen to a more brittle type III collagen which is more prone to breakage. Addressing ligamentous laxity is an important one, however one should keep in mind that those with long term symptoms are likely to have multiple weakness throughout the pelvic floor or even global ligamentous laxity. With more type III collagen in an ageing pelvic floor, once ligamentous tensions exceed their modulus of elasticity for that tissue for stretch and recoil, it is likely that laxity begets further laxity.

Experimental Study No 1:
Directional muscle forces activate anorectal continence and defecation in the female

Previous attempts at correction of the puborectal angle do not result in improved continence and this is no longer attempted.[1, 2] It is unclear if indeed the outer longitudinal muscle of the rectum merges with the uterosacral ligaments proximally, as the authors contend; rather, it seems to enter the posterior rectovaginal facsia, and continues to the anal skin as the corrugator cutis ani.[3, 4]

The 25 patients with urinary and faecal incontinence are not described in a standardised or systematic fashion. Additionally, with only 4 control patients it is not possible to make meaningful comparisons (ideally they would be parity and age matched). The methodology is opaque – it is not clear how these muscular forces of opposition / contraction were measured or quantified. It is also unclear how "X" and "Y" were accurately and reproducibly placed without unwitting bias - what anatomical landmarks were used? What measurements were taken at rest, squeeze and strain to support the hypothesis? "T" pinching of the anterior rectal wall, is too high anatomically for the transverse perineii as shown. The authors quote all these dynamic movements, "consistent with anchoring of various muscle groups" with no objective measurement. They acknowledge that there are no differences in imaging between continent and incontinent groups after a procedure meant to alter anatomy, but then go on to advocate ligamentous reinforcement for faecal incontinence.

1. Matsuoka H, Mavrantonis C, Wexner SD, Oliveira L, Gilliland R, Pikarsky A. Postanal repair for fecal incontinence – is it worthwhile? Dis Colon Rectum 2000; 43: 1561-7.
2. van TetsWF, Kuijpers JH. Pelvic floor procedures produce no consistent changes in anatomy or physiology. Dis Colon Rectum 1998; 41: 365-369.
3. Lunniss PJ, Phillips RK. Anatomy and function of the anal longitudinal muscle. Br J Surg 1992; 79: 882-4.
4. Aigner F, Zbar AP, Ludwikowski B, Kreczy A, Kovacs P, Fritsch H. The rectogenital septum: morphology, function, and clinical relevance.Dis Colon Rectum 2004; 47: 131-40.

Experimental Study No 2:
A direct test for the role of the pubourethral ligament in anorectal closure

This single case presentation produced both faecal and urinary continence from a mid urethral sling procedure. It is unclear what the mechanism of incontinence here was, with no mention of structural or functional assessment. Urethral tapes are a common procedure for urinary stress incontinence – with the common co-prevalence of urinary and faecal incontinence, it would be expected that more faecally incontinent patients would benefit. This group requires a closer look to determine the structural anorectal changes with a sling that may produce continence. Unfortunately the authors do not advance an explanation of the mechanism of action.

Study No. 3:
Reflex contraction of the levator plate increases intra-anal pressure, validating its role in continence

It is known that both vaginal and anal distention produce rises in voluntary squeezes of the external anal sphincter. Two fingers in the vagina producing an unknown force produces a higher rise in the control rather than the incontinent groups in this study. It is feasible to explain this through a sensory mechanism rather than a mechanical one - the digits would produce sensory biofeedback to enhance reflex contraction.

Experimental Study No. 4
Abdominal pressure increase during anorectal closure is secondary to striated pelvic muscle contraction

The authors report no significant differences in increases intra abdominal pressure on straining and squeezing. They used a solid state catheter, which depends on direct compression to demonstrate a pressure rise. For measurement of pressure inside a hollow viscus this is optimally measured

directly (via needle technique) or indirectly (via measurement of intravesical intraabdominal pressure and transduction of this pressure through a column of water).

Experimental Study No. 5:

A prospective endoanal ultrasound study suggests that internal anal sphincter damage is unlikely to be a major cause of fecal incontinence.

The author uses the term "idiopathic" loosely, using it in a previous study to describe faecal incontinence in study 1 as patients with intact sphincters. Most authorities would disagree with the contention that the internal sphincter does not contribute to incontinence; the internal anal sphincter contributes 80% of the resting sphincter pressure. One of the frequent causes of faecal incontinence in the elderly is internal sphincter atrophy. The internal anal sphincter thickness is also age dependent and to arbitrarily say < 2 mm is abnormal is over-simplistic.

The gold standard for endoanal imaging, is using a dedicated endoanal probe with a 360 degree field of view, at a frequency of 10 -15 MHz , the probe used in this study is a rectal probe with linear array sector scanning at 7 MHz.

We agree that not all patients with an internal sphincter injury will be incontinent, but again incontinence is multifactorial and all aspects of the continence mechanism including the internal anal sphincter structure and function should be assessed with the correct instruments.

Experimental Study No. 6:

Correction of abnormal geometry and dysfunction by suspensory ligament reconstruction gives insights into mechanisms for anorectal angle formation

This is a case study of a patient with some functional symptoms of pain, urinary stress leakage and rectal evacuation difficulties helped by perineal digitation. Imaging is suggestive of a non-relaxing puborectalis which fits her "functional" type of symptomatology. The patient applies perineal pressure, but with an attenuated perineal body and the passage of only a small amount of contrast one wonders if the patient is anally digitating.

It is unclear how this posterior sling is inserted and how it augments and supports the uterosacral ligaments. The post operative images still show a non-relaxing puborectalis as the anorectal angle increases with straining. It would be surprising if this patients evacuation actually improved in light of the images shown. Postoperative proctography would have better illustrated the anorectal angle relaxation during evacuation rather than straining films which are less physiological method of illustrating puborectalis movement.

Study No 7:

Role of puborectalis muscle in anal continence - comments on original 4D pelvic ultrasound data from Chantarasorn & Dietz

Recent publications have detailed the importance of puborectalis and the remaining pubovisceral sling in the continence mechanism. Levator trauma in obstetric trauma produces urinary stress incontinence, particularly following forceps-assisted delivery. It is true that few of these patients have faecal incontinence, however in the long term it is unclear if these injuries produce the delayed incontinence often reported 20 or more years later. Puborectalis weakness and atrophy are genuine entities that contribute to faecal incontinence.[1,2] 4D ultrasound was used in this retrospective study, however MRI is the gold standard for pelvic floor imaging of levator injuries, better yet would be the use of an MR endocoil.[3,4]

1. Bharucha AE, Fletcher JG, Harper CM, Hough D, Daube JR, Stevens C et al. Relationship between symptoms and disordered continence mechanisms in women with idiopathic faecal incontinence. Gut 2005; 54: 546-555.
2. Azpiroz F, Fernandez-Fraga X, Merletti R, Enck P. The puborectalis muscle. Neurogastroenterol Motil 2005; 17 Suppl 1: 68-72.
3. DeLancey JO, Kearney R, Chou Q, Speights S, Binno S. The appearance of levator ani muscle abnormalities in magnetic resonance images after vaginal delivery. Obstet Gynecol 2003; 101: 46-53.
4. Terra MP, Beets-Tan RG, Vervoorn I, Deutekom M, Wasser MN, Witkamp TD, Dobben AC, Baeten CG, Bossuyt PM, Stoker J. Pelvic floor muscle lesions at endoanal MR imaging in female patients with faecal incontinence. Eur Radiol 2008; 18: 1892-901. Epub 2008 Apr 4.

Study No. 8:

Stress urinary incontinence results from muscle weakness and ligamentous laxity in the pelvic floor

This study assesses the histology of pubocccygeus biopsies during a mid urethral sling procedure. Importantly no comparisons were made with control tissue which we realize will be difficult to recruit for but the stated findings are meaningless otherwise. This sling procedure corrects the anatomical abnormality caused by ligamentous laxity and or injury, but this study does not show how this procedure improves muscle contraction which is the contention of the discussion. At least some post operative histology would be needed in the long term, before the word "cure" can be used.

Study No. 9:

Double incontinence, urinary and fecal, cured by surgical reinforcement of the pubourethral ligaments

This study appears to be prospectively collected data on the mid urethral sling procedure, retrospectively analysed for patients with faecal incontinence. It is not made clear what type of faecal incontinence these patients had (passive leakage, urge incontinence or post-defaecation soiling). In the era of endoanal ultrasound to use an examining finger to assess sphincter integrity is insufficient, and makes this an even more heterogenous group to rationalize and promote the use of a midurethral tape for.

Study No. 10:

Fecal incontinence cure by surgical reinforcement of the pelvic ligaments suggests a connective tissue aetiology

It is not mentioned what criteria were used to determine positioning of this synthetic mesh, whether anteriorly in 3, posterioly in 9 or both in 18 patients. The "pictoral diagnostic" algorithm offers little to the reader in explanation of which technique is applied for this heterogenous group of patients. What was the basis of the prevalence data? What was the reference for this algorithm. What are the grounds for assuming that the prevalence (frequency) and probability (likelihood) of symptoms are equivalent? In pelvic floor parlance the terms anterior, middle and posterior often refer to the various compartments, the author has cystocele under the middle compartment and uterine and vault prolapse under the posterior compartment. Why is nocturia under the posterior and faecal incontinence under anterior, is the idea to explain pathogenesis of ligamentous laxity? How does symptom frequency relate to treatment in this algorithm. This illustration attempts to stratify pathogenesis, symptoms and therapy but it fails to show this.

Importantly the endopelvic fascia and ligaments all work in concert through all compartments in the pelvic floor, and surgery on one compartment often affects the others.

The results are presented in a rather irregular way. The timings of the follow up visits are not mentioned. It would have strengthened the argument to have undertaken testing on the patients who did not respond to surgery, as it would have been the ideal control compared to those with symptomatic improvement. It is not stated how mean anal pressure was calculated. Pudendal nerve terminal latencies are a poor choice of physiological measure – they are notoriously poorly reproducible and reflect only the fastest conducting fibers in the pudendal nerve. How was functional anal canal length measured?

It would have strengthened the data enormously to have used one of the validated scoring systems or questionnaires of quality of life. In the discussion there is no objective evidence from the data presented that ligamentous support improves muscular force and continence.

The explanation for improved continence in patients with suspected pudendal neuropathies and failure in nulliparous women is based on conjecture. The incompletely rationalised application of the laws of Laplace and Poiseuille may further confuse the reader as their link is not clearly supportive of the authors' hypothesis.

Study No 11:

Ligamentous repair using the Tissue Fixation System confirms a causal link between damaged suspensory ligaments and urinary and fecal incontinence

This pictoral algorithm is commented on above. Once again in this study it does not inform the reader of what specific criteria were used to determine which approach was used. These patients seem to form a heterogenous group with combined prolapse of varying degrees and different types of incontinence. The results presented makes no note of such things as POP-Q scores or symptom scoring perioperatively. These would be standard in any operative study ooking at outcome for surgery for prolapse and or incontinence, the stated primary aim of this study.

It is unclear why the cystocoele repairs were performed: if the objective was to show ligamentous support improves function, why were standard cyctocele repairs undertaken and then disbanded because of poor results? How did this fit in with the algorithm of treatment.

The results presented in tables I and II are an amorphous mixture of prolapse scores, and a list of procedures without knowing their indications. It is not clear what structural abnormalities these 33 faecally incontinent patients had. There are no descriptive statistics to support why the authors believe TFS outcome in faecal incontinence is equivalent to the tension free system. The table of results show the TFS for the anterior, transverse, posterior and sling procedure ;if the suggestion is that changing tension in the ligaments and muscles in the pelvic floor improves faecal incontinence, it is not represented here which tension systems work. There are no sub group analyses for this faecally incontinent group. There is also no mention of the incidence of constipation symptoms which is always relevant in treating continence.

The conclusions drawn from this study are overstated, and cannot be made on the results shown. There are no comparative or randomized data to show that both approaches were equivalent. How was "cure" of urinary and faecal incontinence defined over this mean of 12 month follow up?

Study No 12:

Role of the uterosacral ligaments in the causation of rectal intussusception, abnormal bowel emptying, and fecal incontinence-a prospective study

This study is presented in a more cohesive way in comparison to the previous ones but with major methodological flaws. There are numerous procedures previously described to reduce and prevent this intussusception some used for rectal prolapse as in rectopexy procedures and EXPRESS (external rectal pelvic suspension) procedure. Rectal intsussusception on proctography is a common finding in asymptomatic patients and care must be taken before deciding on surgery, symptomatic patients tend to have more full thickness rather than mucosal prolapse.[1,2] The degree of intussusception is not quantified, whether mucosal only, anterior only, circumferential, intra anal etc. Other important proctographic features of evacuation are not mentioned, such as rectocoele size, emptying, pelvic floor descent, and the degree or absence of "anismus" – a poor prognostic factor for surgical outcome.

A large proportion of patients presenting with solely an evacuation disorder have underlying psychological contributors to their symptoms, these patients also have higher surgical failures and many studies have shown that these patients do well with a conservative therapy, biofeedback. [3,4]

The stated aim of the study was to address the effect of uterosacral ligament reinforcement on the various anatomical abnormalities and incontinence. However the approach involves, in addition to the tension free IVS as the new treatment, a posterior repair as well as a perineal body repair. A posterior repair is one of the conventional approaches to treating a symptomatic rectocoele and a perineal body repair is sometimes used for faecal incontinence where the sphincter is sometimes involved in this procedure. This makes it difficult to say which procedure has worked for which symptom. Twelve patients also underwent hysterectomy. If this was performed at the same time this would surely complicate interpretation of the findings.

Was the "focused questionnaire" a validated tool? Was it generic, disease specific or quality of life related? How was "compete normalization" of defaecation defined, there is no mention of symptoms such as bowel frequency, straining, laxative use, manual manoevures etc, critical for defining symptomatic improvement. Which "numeric rating scale" for faecal incontinence was used, and why was not one of the numerous validated questionnaires available used.

Complications of rectal perforation and erosion in "expert" hands occurred in this study with relatively small numbers, which raises concerns as to who should be undertaking this procedure. This procedure does not seem minimally invasive as is the suggestion, and these are serious complications that shouldn't be down played. Unfortunately, in the absence of objective assessment, we do not share the same enthusiasm as the authors for promoting the novel idea of tension free augmentation of the uterosacral ligaments.

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