

Summary

The Musculo-Elastic Theory for anorectal function and dysfunction in the female

Part 2: Summary. Surgical proofs of the Theory

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The Theory states: “Anorectal dysfunction in the female is mainly caused by lax suspensory ligaments inactivating anorectal muscle forces”.

In this section, the theory is directly tested by comparing pre and post-operative symptoms, and objective tests such as anorectal manometry, pudendal nerve conduction times, and defecating proctography, following surgical placement of polypropylene tapes in the position of lax suspensory ligaments. The tapes work by creating a linear deposition of collagen to reinforce the damaged ligament. According to the theory, a competent ligament is required to restore the muscle’s ability to open or close the anorectum more efficiently. Failure to do so would severely compromise the theory.

Experimental study No. 8 by Petros, Swash and Kakulas. In this histological/surgical study (n = 47), evidence of muscle damage, consistent with that seen in patients with tenotomy, was found in 20/21 multiparous patients with stress urinary incontinence (SUI) and also in 4 older nulliparas. Biopsies were normal in 4 younger nulliparas and one multipara. Fibrous tissue only was found in 17 biopsies. 88% of all patients were cured of their SUI with a midurethral sling. This study indicates that muscle damage may be secondary to ligamentous laxity, and both may be improved by a midurethral sling.

Experimental study No. 9 by Hocking, directly challenges the role of a lax pubourethral ligament in the causation of ‘double incontinence’, stress urinary incontinence and fecal incontinence (FI). It is a prospective clinical study encompassing 62 patients who had ‘double incontinence’, all of whom had intact external anal sphincters. Hocking reported a 92% cure rate for this group of patients, supporting the role of the pubourethral ligaments in maintaining both urinary stress and fecal continence in the female.

Experimental study No. 10 by Petros and Richardson describes 30 patients with idiopathic fecal incontinence who also had a range of bladder and other symptoms attributed to lax uterosacral ligaments. Only 10 patients had SUI. This study also challenged the use of the Pictorial Algorithm as a diagnostic tool for patients with idiopathic FI. Post-operative assessment included anal manometry and pudendal nerve conduction time (PNCT) assessments. Twenty-five patients (83%) reported >85% improvement in their fecal incontinence and other pelvic symptoms. Low mean anal pressure, low squeeze pressure, prolonged pudendal nerve conduction times and thinned internal anal sphincters were not predictors of surgical success or failure, and these parameters did not change significantly after surgery. Only the functional anal length demonstrated a mildly significant improvement post-operatively (p = 0.049).

Experimental study No. 11 by Abendstein, Petros and Richardson, involves a different group of 81 patients with multiple anatomical defects, pubourethral ligament (n = 43), cystocele (n = 39) and uterosacral ligaments (n = 72). These defects were repaired simultaneously using a new ‘minisling’ technique, the TFS (Tissue Fixation System). In these patients the ATRP and cardinal ligaments were repaired in patients with cystocele. The cure rates for FI and bladder symptoms were similar to studies 9 & 10, indicating the important principle was not the actual surgical technique, but repair of the damaged ligaments. It was further concluded that damage to the ATRP and cardinal ligaments was unlikely to be a factor in FI causation.

Experimental study No. 12 by Abendstein et al., prospectively assessed 48 patients who had symptoms of fecal incontinence, obstructed defecation syndrome, uterovaginal prolapse and radiologically diagnosed anterior rectal wall intussusception. Only the uterosacral ligaments, and associated posterior zone structures were repaired. A high cure rate was achieved for ‘obstructed defaecation’, anorectal mucosal and fecal incontinence. Anatomic restoration was proven by post-operative defecography. It was concluded that the competent uterosacral ligaments were a key element in preventing both FI and anterior rectal wall intussusception.

Conclusions. The studies as presented lend support to many of the theory’s predictions, sufficient to encourage a new integrated and holistic approach to research into the causation of FI. However, more studies involving selective ligament anchoring (“simulated operations”) during anal manometry, nerve conduction studies and, if possible, during ultrasound or fluoroscopy, may shed more light on the importance of the pelvic ligaments in pelvic floor disorder.

Key words: Integral Theory; Posterior IVS; Fecal incontinence; TFS; Obstructed defecation; Rectal intussusception.