Foreword

A Musculo-Elastic Theory of anorectal function and dysfunction in the female

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

FOREWORD

We present a series of 13 works in 2 parts. Part 1 states the Theory, and presents 7 works which test core aspects of the Theory, in particular, the role of the suspensory ligaments and muscle forces. Part 2 directly challenges the Theory with 5 different surgical works which track the fate of anorectal and other pelvic symptoms following repair of specific suspensory ligaments and their related fascia.

In this series of papers, we bring together two apparently divergent concepts for the causation of fecal incontinence in the female, the muscle damage theory, and the ligament/connective tissue damage theory. Data from a blinded muscle biopsy/midurethral sling study provided the key to our theory. It indicated that where there are lax ligaments, there were also histological pattern of muscle damage due to several different mechanisms. The implications of these findings are that (1) Lax ligaments will cause muscle dysfunction by unloading the relevant muscles, as in tenotomy, and (2) This muscle dysfunction is potentially reversible by surgical reinforcement of the damaged ligament, even when there are major changes histologically in the pelvic floor muscles.

HISTORICAL BACKGROUND

In 1990 the Integral Theory of urinary incontinence suggested that stress and urgency both derived from laxity in the vagina or its supporting ligaments.[1] The initial focus was on correcting stress urinary incontinence with a midurethral sling. In 1993 the theory was expanded to include abnormal bladder emptying, nocturia, and pelvic pain,[2] a consequence of uterovaginal prolapse. These functional problems, including prolapse, were cured or substantially improved after a posterior sling procedure.[3] Though the focus at the time was on urinary dysfunction and pelvic pain, repeated voluntary anecdotal statements by patients that their fecal incontinence had also been cured, as well as their stress urinary incontinence by the slingplasty procedure created the impetus for a prospective systematic study. In 1997, simultaneous cure of urinary stress (88%) and idiopathic fecal incontinence (100%) was reported at 6 month review in a group of 25 patients with a midurethral sling.[4] Three of these patients were nulliparous. Endoanal ultrasound examination by a radiologist skilled in the procedure found that the external anal sphincter appeared of normal thickness and consistency in all these patients, and the internal anal sphincter appeared normal in 72%. Subsequently 6 patients from this group developed uterine prolapse, with recurrence of FI in 3 of those patients. In those 3 patients, the prolapse, and fecal incontinence were cured with a posterior sling. We concluded that the pathogenesis of FI was similar to that suggested for stress urinary incontinence:[1, 2] that is, ligamentous laxity in the suspensory ligaments of the vagina, disabled the anorectal closure mechanism by impairing the three directional muscle forces , the anterior portion of pubococcygeus (PCM), levator plate (LP), longitudinal muscle of the anus (LMA), that function in concert with the puborectalis muscle (PRM) to close the anorectal angle and so to maintain continence (Fig 1). It had been previously demonstrated,[1, 2, 5] that pubococcygeus (PCM) and levator plate (LP) contraction tensed the the pubourethral ligament (PUL) (Fig. 1), and LP and LMA contraction tensed the uterosacral (USL) suspensory ligaments of the vagina (Fig. 1). It was concluded therefore that the sling procedure worked by creating an artificial collagenous neoligament [6] in the position of PUL and USL (Fig. 1). This restored the three muscle vectors (arrows, Fig. 1) acting against these ligaments, and therefore, facilitated the anorectal closure mechanism and continence.

Though these findings emphasized the role of connective tissue (in particular, collagen) they did not invalidate a role for pelvic muscle damage in the causation of fecal incontinence (FI), as these muscles form an essential and integral part of the anorectal closure mechanism. All striated muscles in the body act against ligaments and the pelvic floor muscles are not exceptional in this regard. The elastic liga-

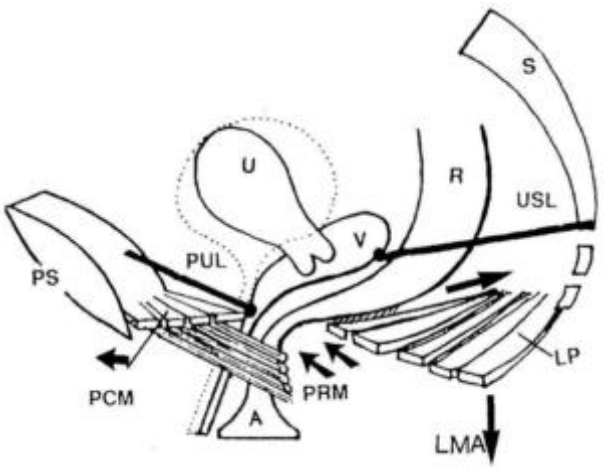

Fig. 1. – Bladder neck and anorectal closure. The same directional muscle forces contract against the suspensory ligaments PUL (pubourethral ligament) and USL(uterosacral) to create bladder neck and anorectal closure. Schematic 3D representation with the anorectal angle in the closed position. The middle part of pubococcygeus muscle has been cut away to reveal the underlying puborectalis muscle (PRM). Arrows=directional muscle forces. PCM= anterior portion of pubococcygeus muscle; LP=levator plate; LMA=longitudinal muscle of the anus; PS=pubic symphysis; S-sacrum; U=uterus; A=anus; V=vagina; R=rectum. The bladder is represented by dotted lines. (Petros,[4] by permission, Int J Urogynecol).

mentous structure, e.g., in tendons, modulates muscle force and acts in some muscle systems as a mechanical energy store for sudden or slow release.

Linkage between urinary and fecal incontinence had been previously established by Swash and colleagues [7, 8] who presented extensive data showing that both fecal and urinary incontinence are associated with weakness of the pelvic floor sphincter musculature, and the pelvic floor diaphragm itself.[1] They also noted the importance of laxity of the pelvic floor, presenting as pelvic floor descent on straining, and pointed out the importance of direct sphincter damage, e.g., tears of the external anal sphincter, in the causation of feces incontinence.[7, 8] They found histological and electrophysiological evidence of damage to the nerve supply of these muscles, especially of the external sphincters and of the puborectalis sling, and showed that this occurred during childbirth, correlating with adverse events such as prolonged childbirth, the need for forceps assistance in delivery, and also with anal sphincter tears.[2] The latter, in particular, was an indication of the likelihood of the development of fecal incontinence later in life, and subsequent work has shown that sphincter tearing is, perhaps not surprisingly, an independent predictive factor in the causation of later-developing incontinence.[9]

However, it has always been evident that direct damage to pelvic floor sphincters, or damage to their innervation, could not be the sole factors leading to the delayed development of fecal incontinence, often many years after childbirth. Studies of hormonal factors, associated with the menopause, did not reveal plausible mechanisms that might have been in part causative of pelvic floor weakness. An association with intractable constipation and straining patterns of defecation, seemed to be an associated factor in some women but, again, this was not a universal feature of the syndrome of fecal and urinary incontinence. Progressive damage to the innervation of pelvic floor muscles due to stretch injury associated with perineal descent during straining was demonstrated, but it could not shown that this was the sole cause of the functional deficit leading to fecal and urinary incontinence.

Data from a blinded muscle biopsy/midurethral sling study provided the key to our theory. It indicated that where there are lax ligaments, there was histological evidence of muscle damage of varied causation. Weakened muscles, whether caused by partial denervation, due to stretch injury to perineal nerves in childbirth, often itself associated with direct injury as shown by sphincter tearing, are further prevented from developing normal tension by these weakened, stretched and eventually non-elastic ligaments. A muscle cannot develop normal tension if its ligamentous attachment is lengthened or if, as in the case of the pelvic floor, the elastic ligamentous structure of the pelvic floor is deranged by loss of normal ligamentous elasticity. The immediate restoration of continence following the sling procedure demonstrates the importance of this integrated functional mechanism, even when there is continuing uncorrected muscular weakness.

We do not discount the role of internal (IAS) and external (EAS) anal sphincters in their contribution to anorectal continence. Rupture of the latter is an easily recognizable and correctible cause of FI. However, even here, we consider the role of EAS is more complex than a stand-alone sphincter, given that EAS is both the tensor of the perineal body, and the insertion point of the longitudinal muscle of the anus. We believe that the role of IAS is limited to assisting air and watertight closure of the anal canal, which may be important in anal sensory function, as suggested by Bartolo.[10]

METHODOLOGY AND LIMITS OF OUR WORK

We present a series of papers in part 1 which test the anatomical basis of the Musculo-Elastic theory of fecal incontinence. In part 2 a series of surgical observations tests the theory by examining the effects of repair of the suspensory ligaments and their related fascia, directed by the same pictorial algorithm used to guide surgery for urinary dysfunction. In attempting to test our theory we have followed the guidelines of Karl Popper,[11] who considered that scientific theories are universal statements. Therefore, a scientific theory should suggest a causal explanation, and predict singular events deriving from the theory. Ideally a theory should be simply expressed, internally consistent, and expressed in such a way that it can be falsified. One major contradiction is sufficient to invalidate a particular theory.

Popper proposed two rules of methodology in assessing scientific theories:

1. Scientific statements can never be finally verified.

2. An existing hypothesis stands unless it can be directly falsified by a valid observation, or replaced by another hypothesis which better explains the data.

In accordance with Popper's approach, the papers presented here set out to test our Musculo-Elastic Theory and also other anorectal theories for truth or falsity.

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