Conclusions and future directions

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

The aim of Part 2 was to directly challenge the Theory by repairing specific ligaments, pubourethral only (Study No. 9), pubourethral and uterosacral (Study No. 10), pubourethral, uterosacral, ATFP, cardinal, and perineal body (Study No. 11), and uterosacral and perineal body only (Study No. 12). We used the pictorial algorithm (Fig. 1) to guide which zone to repair, anterior, middle or posterior. Clinical results from Studies 9-12 appeared to validate these assumptions.

Accurate diagnosis of structural damage

The first step in understanding causation is the appreciation that all structures work together inextricably as a system. Though our results indicated that laxity in the pubourethral and/or uterosacral ligaments appeared to be the main cause of the idiopathic fecal incontinence, these cannot be the only causes. It is theoretically possible for any of the structures in Fig. 1, red lettering, to contribute to the genesis of fecal incontinence. All these structures work synergistically, and each structure may contribute a different weight to the system. The varying size of each bar (Fig. 1), expresses this variation pictorially.

Though external anal sphincter damage was specifically excluded from this study, it needs to be diagnosed and repaired where present. The EAS has a key role in our theory. With reference to Fig. 1, EAS is a tensor of the perineal body, and the inferior insertion point of the downward rotating muscle force 'LMA' (arrow). Inability to tension the perineal body may invalidate backward stretching of the posterior vaginal wall by LP (backward arrow), with similar consequences to those reported in Study No. 12.

One consequence of the theory is that there has to be a critical mass below which a severely damaged muscle will not have sufficient contractile force to effect organ closure. Even tenotomy muscles can atrophy to a point of no return. Severe muscle damage may explain the lower cure rate in Study No. 10, which we consider was the worse affected group. At present we have no method for diagnosing severe muscle damage.

Surgery

One biomechanical consequence of our theory, which has surgical implications, is that the ligaments and fascia require a critical length and tension for optimal muscle contraction. The advent of the TFS provides a tool which can, for the 1st time, restore tension as part of the ligament/fascia reconstruction. The results of Study No11 are encouraging, but far more data will be required to fully assess the effects of surgical tensioning on restoration of function.

Finally there is the consequence of operating on damaged tissues. Study No 10 reports instances where repair of one ligament may divert the pelvic forces to stretch another (subclinically damaged) ligament to cause de novo symptoms. For this reason, it may be prudent to consider routinely repairing both the pubourethral and uterosacral ligaments and in patients with idiopathic fecal incontinence, and the uterosacral ligaments, perineal body and rectovaginal fascia in patients with 'obstructed defecation'.

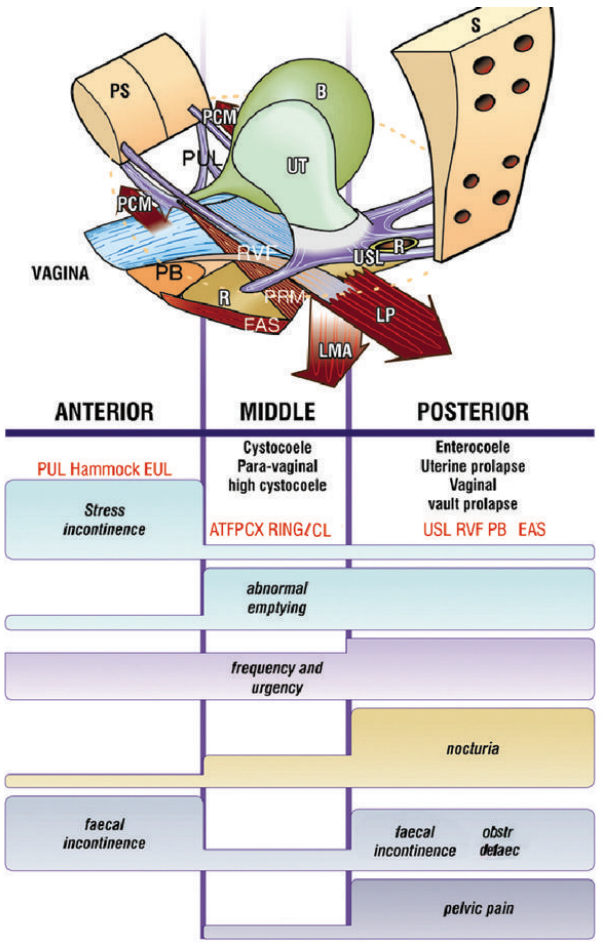

Fig. 1. – Pictorial diagnostic algorithm. The algorithm related prolapse and symptoms to 3 zones of damage. The structures (red lettering) denote the connective tissue structures in each zone which are liable to be damaged. PUL: pubourethral ligament; EUL:external urethral ligament; ATFP: arcus tendineus fascia pelvis; cx ring; cervical ring; CL: cardinal ligament; USL: uteosacral ligament; RVF: rectovaginal fascia; PB: perineal body; EAS: external anal sphincter.