Original article

Study No. 10: Fecal incontinence cure by surgical reinforcement of the pelvic ligaments suggests a connective tissue aetiology

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Abstract: AIM. To test the hypothesis that the pubourethral and uterosacral suspensory ligaments are an important cause of idiopathic fecal incontinence. METHODS. Thirty patients with urinary and fecal incontinence were tested with pre-operative endoanal ultrasound, pre- and post-operative anorectal manometry and pudendal nerve conduction times. Only 10 had stress incontinence. Polypropylene mesh tapes were inserted in the position of the pubourethral ligament (n = 3), uterosacral ligament (n = 9) and both ligaments (n = 18). Surgery was guided by the same diagnostic algorithm used to manage urinary incontinence (see Fig. 1, Theory paper). RESULTS. All patients were discharged within 24 hours of surgery. Post-operative assessment included anal manometry and pudendal nerve conduction time (PNCT) assessments. Twenty-five patients (83%) reported >85% improvement in their fecal incontinence symptoms. Low mean anal pressure, low squeeze pressure, prolonged pudendal nerve conduction times (n = 15) and thinned internal anal sphincters (n = 13) were not predictors of surgical success or failure, nor did these parameters change significantly with surgery. Only the functional anal length demonstrated a mildly significant improvement post-operatively (p = 0.049). Cure rates varying between 76% and 100% were reported for the various urinary symptoms. CONCLUSION. The results indicate that damaged pelvic ligaments may be an important cause of idiopathic fecal incontinence, possibly by inactivating the anorectal closure muscles.

Key words: Fecal incontinence; PNCT; Urinary incontinence; Connective tissue; Integral Theory.

INTRODUCTION

The cause of idiopathic fecal incontinence (FI) is at present unknown. In 1985 Swash et al. published a unifying theory of urinary and fecal incontinence based on striated muscle damage. This work was inspired by evidence of pudendal nerve damage in many patients with double incontinence (urinary and fecal).[1]

In 1993, based on obstetric ultrasound studies, Sultan et al hypothesized a link between damaged internal anal sphincters and fecal incontinence.[2] Neither of these theories [1, 2] assigns a role for damaged connective tissue in the causation of fecal incontinence. In 1999, it was observed that in 25 patients with double incontinence (stress urinary and idiopathic fecal incontinence) both types of incontinence were cured simultaneously following a midurethral sling operation.[3] These findings were replicated by Dr Ian Hocking (paper No. 9, this issue). Three patients from that cohort [3] subsequently reported recurrence of their FI coincident with development of uterovaginal prolapse. Both conditions, prolapse and FI, were cured with a posterior vaginal sling (infracoccygeal sacropexy).

Two hypotheses followed naturally from these observations

1. Both anterior (pubourethral) and posterior (uterosacral) suspensory ligaments may play a role in anorectal closure.

2. Damaged connective tissue was most likely a major cause of idiopathic fecal incontinence, as this was the only structure repaired.

The primary aim of this study was to prospectively test these hypotheses, by surgically implanting polypropylene tapes to reinforce damaged pelvic ligaments in patients with idiopathic fecal incontinence.

Royal Perth Ethics approval was obtained for the surgical procedures.

METHODS

Inclusion criteria were a history of solid or liquid fecal incontinence which, during episodes, occurred at least once a day. The only exclusion criterion was a torn external anal sphincter. The same pictorial diagnostic algorithm used to guide urinary incontinence surgery was applied to guide the site of surgery (cf Fig. 1, Theory, Part 1).

Polypropylene tapes were inserted in the position of the pubourethral ligaments (PUL) (n = 3), uterosacral ligaments (USL) (n = 9), or both (n = 18) (Fig. 1), using the IVS tunneller (Tyco, Norwalk CT, USA). The perineal body and rectovaginal fascia were repaired simultaneously in all patients who had the posterior sling. Pre and post-operative anal manometry and nerve conduction studies objectively monitored the clinical outcomes.

At the 1st visit, all patients had a structured assessment [4] including a self-administered semi-quantitative questionnaire, vaginal and rectal examinations, incontinence diary, transperineal ultrasound, urodynamics, and 2 pad tests, 10

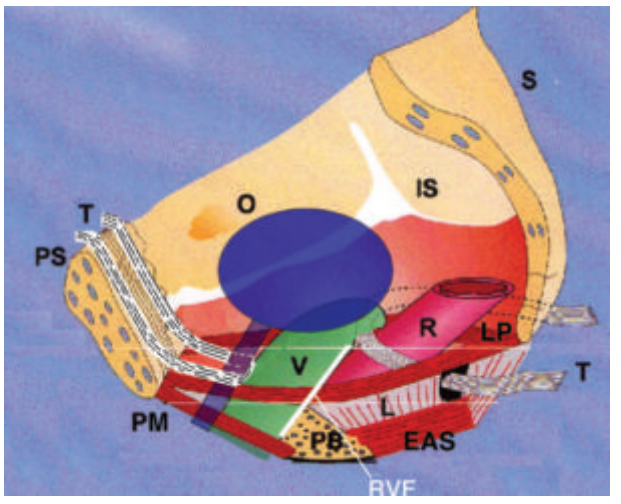

Fig. 1. – Surgery schematic 3D sagittal section. Implanted "tension-free" tapes 'T' create collagenous pubourethral neoligaments anteriorly and uterosacral ligaments posteriorly. V = vagina; LP = levator plate; L = longitudinal muscle of the anus; PB = perineal body; EAS = external anal sphincter; LP = levator plate; IS = ischial spine; O = obturator membrane; RVF = rectovaginal fascia. LMA = m. longitudinal muscle of the anus; PCM = m. pubococcygeus; PUL = pubourethral ligament; USL = uterosacral ligament.

coughs to measure urine loss for stress incontinence, and a 24 hour pad test to assess 24 hour urine loss. These were performed by the gynecological team. On a second pre-operative occasion, a radiologist skilled in the procedure performed endoanal ultrasound to detect damage to the internal and external sphincters. On a 3rd pre-operative occasion the patients were independently assessed by the Dept of Colorectal surgery which also tested anal pressures (manometry) and pudendal nerve conduction times (PNCTs). The decision as to which ligaments to repair was guided by the symptoms and examination findings as summarized by the pictorial diagnostic algorithm.[4] Post-operative anal manometry and PNCTs were performed by colleagues from the Dept of Colorectal surgery, who also reviewed the patients with regard to the fate of their fecal incontinence. The gynecological team objectively assessed the fate of the urinary incontinence with post-operative pad tests, ultrasound, and urodynamics. The patient self-assessed the percentage rate (0-100 scale) of improvement in her fecal incontinence, and urinary urgency. Frequency (>10/day) and nocturia (>2 night) were assessed with a validated semiquantitative questionnaire.[4]

RESULTS

Thirty patients were studied, all with "idiopathic" fecal incontinence, two of whom had thinned, but not torn, external anal sphincters. Only 10 patients had stress incontinence. The others had other bladder symptoms such as urgency, frequency and nocturia. Their mean age was 57 years (range 32-74 years), mean parity 2.7 (range 0-5). The main criterion for fecal incontinence cure, and also, frequency, urgency and nocturia cure, was a symptomatic improvement of 85% or more as assessed by the patient using a 0-100 scale. Of the 25/30 patients (83%) who were cured of fecal incontinence, 20/25 attended for post-operative manometry and PNCTs. No post-operative testing was performed in the 5 patients with failed surgery. Only one patient was nulliparous, and she was cured of fecal incontinence with a midurethral sling operation. In addition to fecal incontinence, 10 patients had stress urinary incontinence (SI), and all were cured of their SI. Twenty-five patients had urinary frequency, nocturia and urgency (FNU), and 19 (76%) were cured of their FNU. All patients were discharged within 24 hours of surgery, and were reviewed at 6 weeks and at 6 monthly intervals thereafter. The mean post-operative assessment time was 16 months (range 6-24 months).

Mean anal pressure (MAP), squeeze pressure, pudendal nerve conduction times (PNCT) and thinned internal anal sphincters were not predictors of surgical success or failure. Only the functional anal length demonstrated any significant change post-operatively, and then only minimally ($p = 0.049$).

Taking 40 mm Hg as normal, mean anal pressure (MAP) was low in 14 patients, and only 2 of these were not cured of fecal incontinence by the surgery. Post-operatively, MAP increased in 8 patients, decreased in 7 and remained similar in 5. Of the 7 patients cured of fecal incontinence but whose post-operative MAPs remained well below 40mm Hg, 5 improved their MAP only marginally, while 2 recorded a definite fall in MAP.

Taking 100 mmHg as a normal squeeze pressure, 11 patients were normal pre-operatively. Post-operatively, 11 patients showed a demonstrable increase, and 9 a decrease in their pre-operative squeeze pressures.

Pre-operatively, taking 2.2 seconds as a normal conduction time, 15/30 patients had abnormal pudendal nerve conduction times (PNCT) times on the right side, and 10/30 patients on the left side.

Of the 20 post operative PNCTs performed, allowing 0.2 seconds as a margin for measurement error, 5 patients increased their conduction times, and 2 decreased their conduction times in either the left or right side. The other PNCTs were unaltered.

Functional anal length (FAL) increased in 10 patients, decreased in 2, and was unaltered in the other 8. This was the only significant result ($p = 0.049$).

The internal anal sphincter was thinned in 13 patients (43%) and 2 of these were not cured of fecal incontinence.

Of the 5 patients not cured of fecal incontinence, 3 had normal PNCTs at least on one side, 3 normal MAPs, and one had normal squeeze pressures. Two of the 5 patients were cured entirely of their bladder problems, but not their FI problems. Of the other 3, two were cured for a short period of 6 weeks of both fecal incontinence and urinary incontinence, and both recurred after 6 weeks, first the urinary then the fecal.

One of the 2 patients with a thinned (but not ruptured) external anal sphincter (EAS) was cured of passive liquid soiling but continued to have fecal incontinence with "intestinal hurry". She was cured of the latter with a subsequent EAS and post-anal repair. For the purposes of this study, she has been classified as an operative failure. The other patient with thinned EAS was cured of her fecal incontinence and reported return of the ability to discriminate between wind and liquid feces following repair of both pubourethral and uterosacral ligaments. One patient reported 50% cure of her FI after repair of her anterior ligaments, and 100% cure after her posterior ligaments were repaired at a later date. Another patient reported 70% cure after the anterior tape repair, then 100% cure after the posterior tape repair. Two patients initially failed to respond to the posterior ligament reconstruction and remained incontinent of both urine and feces. Both were obese and it was reasoned the tapes had slipped. On repeating the posterior sling procedure, both were restored to continence of urine and feces. Both were classified as cured. No significant complications such as organ perforation, tape erosion, haemorrhage or infection occurred.

DISCUSSION

This study differs considerably from Hocking's study (No. 9, this issue), and a previous study.[3] Only 10/30 patients from this group had associated urinary stress incontinence symptoms (pubourethral ligament defect (Fig. 1). Twenty patients had no SI whatsoever. Their associated symptoms were FNU (frequency, nocturia, urgency), consistent with a uterosacral ligament defect. The results indicate that posterior ligament laxity may also be an important cause of idiopathic FI, and this has been validated in studies No. 11&12.

It was radiologically demonstrated (Part 1), that the pelvic floor muscles effectively contract against the pubourethral and uterosacral ligaments. The theory predicts that: laxity in these anchoring ligaments may not allow the closure muscles to work properly, so that leakage of both urine and feces may occur; reinforcing these ligaments with polypropylene tapes will restore the weakened muscle forces, and therefore, continence. This was largely validated by the surgical study, but not entirely. A significant number of patients (17%) were not cured of their FI symptoms. Others achieved a good, but suboptimal improvement (85%), suggesting either incomplete anatomical restoration, or other causation. The inherent inaccuracy of the diagnostic algorithm, and other as yet unknown causes may explain the former. The muscle damage1 hypothesis provides a rational explanation for the latter, given the fundamental tenet of the Musculo-Elastic Theory as described in Part 1, that an adequate muscle force is required to effect anorectal closure (continence).

We explain cure of FI in patients with pudendal nerve damage in our study (prolonged PNCT) as follows: the same pelvic floor muscles responsible for anorectal closure contract to contain the intra-abdominal organs. This indicates these muscles may possess reserves of strength far greater than required just for anorectal closure. Restoring the effective insertion points for these muscle forces, the suspensory ligaments, enables a damaged muscle to contract more efficiently, sufficient in many cases to cure fecal incontinence.

Partial cure in 2 patients with restoration of the anterior ligament followed by total cure after posterior ligament reconstruction indicates that the control of fecal continence is likely to be synergistic, similar in fact to the mechanism proposed for continence control in the bladder.[4]

Occurrence of fecal incontinence in nulliparas [3, 5] can only be explained by congenital collagen deficiency. Surgical cure by tape implantation in this and a previous study 3 is consistent with a connective tissue aetiology such patients.

Of 13 patients with damaged IAS, 11 were cured. Seventeen patients had FI and normal IAS. This accords with previous data.[3] The IAS damage hypothesis cannot explain the results of this work.

There was no statistical correlation between mean anal pressure (MAP) and squeeze pressure in patients who reported clinical cure, suggesting that low intra-anal pressures may not be a major factor per se in the causation of fecal incontinence. Pressure is not necessarily the same thing as closure. Pressure within a tube = Force/Area over which the force is exerted. Closure implies a water-tight resistance to leakage. Many other factors may play a role in closure, for example, stretching and narrowing of the tube (Pressure = Tension/radius, 'Laplace's law'), resistance within the tube (Resistance varies with tube length/ 4th power of the radius, 'Poiseuille's law'), and anal mucosal sealing, which we attribute to the internal anal sphincter. A restored musculoelastic mechanism would stretch the rectal tube backwards and downwards more efficiently to narrow the lumen. This creates an exponentially raised resistance within the lumen.[6] For example, halving the anorectal diameter will increase the intra-anal resistance by a factor of 16. The exponential increase in intra-anal resistance with even a small decrease in diameter explains how evacuation difficulties may occur without any detectable mechanical obstruction.

Cure of urinary but not fecal incontinence in some patients indicates the causative relationship between urinary and fecal incontinence is not absolute.

CONCLUSIONS

Connective tissue damage in the anterior and posterior suspensory ligaments appears to be a significant cause of idiopathic fecal in continence. Anal manometry and nerve conduction studies do not appear helpful in predicting surgical success or failure, and they are not at all useful in predicting which ligament has been damaged. The pictorial diagnostic algorithm is helpful in this regard, but only as a guide to surgery. It cannot directly assess the condition of each ligament. Given this limitation, one solution would be to routinely repair both anterior and posterior ligaments simultaneously in all patients with FI. However, such a solution would not help if there were other contributing causes, such as middle zone defect (damaged ATFP, cardinal ligaments), or severe muscle damage. Future pre and postoperative morphological studies using MRI, 3D and 4D ultrasound may help to further elucidate the role of specific muscles and ligaments. Study No. 11 attempts to assess the adjunctive role (if any) of damaged ATFP and cardinal ligaments in FI causation.

REFERENCE

1. Swash M, Henry MM, Snooks SJ. Unifying concept of pelvic floor disorders and incontinence. Journal of the Royal Society of Medicine 1985; 78: 906-911.
2. Sultan AH, Kamm MA, Hudson CN, Thomas JM and Bartram CI. Anal-sphincter disruption during vaginal delivery, N Eng J Med 1993; 329: 1905-11.
3. Petros PE. Cure of urinary and faecal incontinence by pelvic ligament reconstruction suggests a connective tissue aetiology for both. International Journal of Urogynecology 1999; 10: 356-360.
4. Petros PE and Ulmsten UU. An Integral Theory and its method for the diagnosis and management of female urinary incontinence. Scand J Urol Nephrol 1993; 26 (Suppl. 153): 1-93.
5. Parks AG, Swash M, Urich H. Sphincter denervation in anorectal incontinence and rectal prolapse. Gut 1977; 18: 656-665.
6. Bush MB, Petros PEP, Barrett-Lennard BR. On the flow through the human urethra. Biomechanics 1997 30: 967-969.

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