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The mechanics of urethral closure, incontinence and midurethral sling repair. Part 1 original experimental studies. (1990)

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Aims: To summarize the mechanics of urethral closure, incontinence, and midurethral sling repair, a work in 3 parts Part 1. Original scientific studies (1990). Part 2. Experimental validation of reliance of the closure mechanisms on a competent PUL (1993–2003). Part 3. Surgery (1990–2016).

Methods: Part1. Two unrelated observations in the mid 1980s led to the discovery of the MUS: a hemostat applied on one side of the midurethral area of the vagina, controlled urine loss on coughing *without bladder neck elevation;* an implanted Teflon tape cause a collagenous reaction. It was hypothesized that urinary stress incontinence (USI) was caused by collagen loss in the pubourethral ligament (PUL) and a tape implanted in the exact position of PUL would reinforce it and cure USI. A tape removable at 6 weeks was configured as an inverted "U" in the vagina and lowered sequentially.

Results: At a certain point, the patient was continent on coughing but was able to pass urine freely. This proved the mechanism for continence was not obstructive. Post-op xrays showed no elevation of bladder neck. This invalidated Enhorning's Theory. Ultrasound showed closure of distal urethra from behind and descent of vaginal fornix on straining. This indicated there were two closure mechanisms, distal urethral, and bladder neck. Three months following sling removal, there was a 50% failure rate.

Conclusions: The 1990 results indicated a permanent sling was required for the MUS. Further proofs were required for the proposed musculoelastic mechanisms.

KEYWORDS
arc of Gilvernet, midurethral sling, neoligament, pubourethral ligament, pubovesical ligament, stress incontinence

1 | INTRODUCTION

The original experimental studies leading to the MUS were begun in the mid 1980s by the 1st author at The Royal Perth Hospital. The gold standard operation at the time was the Burch Colposuspension,[2] whose theoretical basis was Enhorning's pressure transmission theory.[3] The Burch was performed via a low transverse abdominal incision. The vagina in the area of bladder neck was sutured to the pectineal ligament. This was a traumatic operation which had high rates of post-operative pelvic pain and notably, urinary retention, often up to 14 days, occasionally for months.

Roger Dmochowski led the peer-review process as the Associate Editor responsible for the paper.

Two unrelated observations in the mid 1980s led to the discovery of the MUS. It was observed that if a hemostat was applied on one side of the midurethral area of the vagina, VIDEO 1, urine loss on coughing was controlled *without bladder neck elevation.* The second observation came from operations on patients who presented with a sterile abscess in reaction to the Teflon tapes used in the Cato Operation. The Cato was a type of colposuspension performed only in Western Australia.[4] It used 2 mm Teflon tapes to suspend the bladder neck area of the vagina across the rectus sheath. At operation, it was observed that the implanted Teflon tape created a dense collagenous cylinder around it. The tape was removed simply by pulling it out of the collagenous cylinder.

It was hypothesized that the cause of urinary stress incontinence (USI) was a loose pubourethral ligament (PUL), a consequence of collagen deficiency in the ligament, and that a tape implanted in the exact position of PUL would create new collagen to reinforce it.

Based on experiments on a hemi pelvis in the Dept of Anatomy, University of Western Australia, an instrument was invented which could implant the tape by sliding it around the back of the pubic symphysis.

The next step was to test the methodology. A Mersilene tape was implanted in 13 canines. This resulted in the discovery of a new surgical principle which used the positive aspects of tissue reaction to implanted materials to create of an artificial collagenous neoligament,[5] Figure 1. A prototype MUS was performed in 30 women between 1988 and 1989[6] under local anaethesia/sedation. The position of the bladder neck relative to the symphysis was determined preoperatively with radio-opaque dye in the balloon of a Foley catheter at rest and straining. The Mersilene tape was placed at midurethra and was configured as an inverted "U" on rectus sheath with its arms exiting into the vagina. Holes 0.5 cm apart were placed in the ends of the tapes to secure sutures, so that the tape could be lowered in stages by cutting each suture in turn if the patient could not pass urine. At a certain point, all 30 patients could pass urine freely and were continent on coughing. This proved that the mechanism for the MUS was not obstructive. A 2nd standing xray was taken in this position. At 6 weeks, all tapes were removed. Immediately following removal, all patients reported cure. However, within 3 months, 50% of these prototype operations had failed.

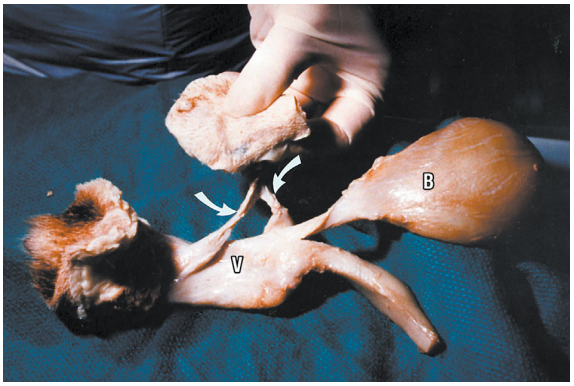

FIGURE 1 Formation of artificial collagenous neoligaments. Tapes were inserted in the position of the pubourethral ligaments (white arrows). Specimen of vagina (V), vulva, bladder (B) dissected from a dog 2 weeks after the tape which had been implanted for 12 weeks was removed. Note the significant artificial collagenous neoligament created by tissue reaction against the tape (white arrows)

Xray findings, Figure 2. All 30 patients were initially cured. There was no bladder neck elevation, even in those patients where the bladder neck was below the lower border of symphysis preoperatively, Figure 2. These findings invalidated the concept of a "pressure equalization zone." Other xray findings were the discovery of three directional forces which seemed to activate proximal and distal urethral closure with two separate mechanisms (see middle xray, Figure 2): a forward force pulling distal urethra forwards against the tape: a backward force pulling the Foley balloon backwards against the tape; a 3rd downward force pulling the Foley balloon downward, Figure 1.[2] The only muscles which could create these forces were: forwards: anterior part of pubococcygeus muscle (PCM); backwards: levator plate (LP); downwards: conjoint longitudinal muscle of the anus.

1.1 | Hypothesized urethral closure mechanism (1990)

The role of PUL and subsidiary structures in normal urethral closure were examined and a hypothesis for their role in urethral closure was proposed[1] (Figure 3). With reference to Figure 3, the pubourethral ligament (PUL) and pubovesical ligaments (PVL) have a common origin behind symphysis pubis, 1–1.5 cm above its lower border (Figure 3). PUL inserts into the lateral walls of midurethra and distal part of pubococcygeus muscle (PCM). PVL inserts into a fibrous thickening on the anterior surface of the bladder, the precervical arc of Gilvernet. In Figure 3, ZCE is the "zone of critical elasticity" (ZCE) is, the elastic part of anterior vaginal wall which allows separate function of the distal and proximal urethral closure mechanisms.

The distal closure mechanism, Figure 3. PCM stretches the vagina forwards against PUL to close the distal urethra. Its main function is to immobilize and seal the distal urethra. The classical symptoms of EUL defect are small leakages on walking, moving quickly accompanied by a feeling like "a bubble of air escaping."

The proximal closure mechanism, Figure 3, is the principal closure mechanism. The levator plate (LP) contracts backwards against PUL and the immobilized distal urethra.

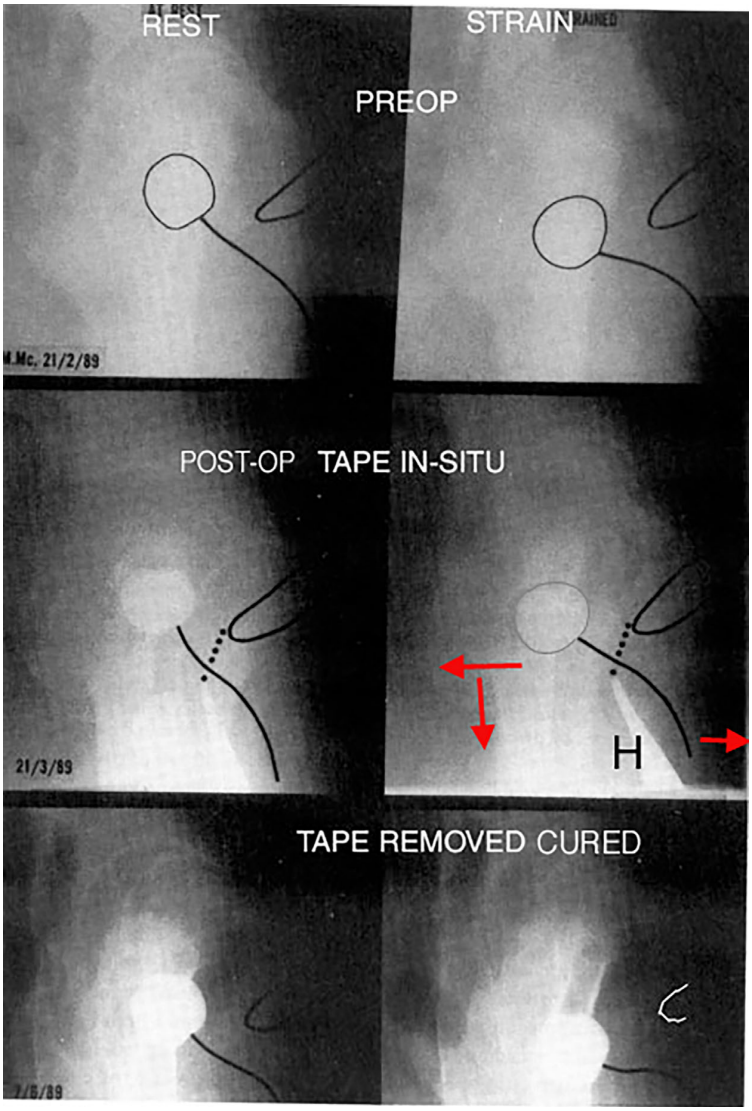

FIGURE 2 Standing sagittal xrays from 1990 prototype MUS, Same patient. Left column: at rest. Right column:straining. Middle xray right, post-op. Intravaginal tape grasped by a haemostat "H" reveals three directional forces acting in the position of PUL. Bottom Xray, post-op. Bladder neck below symphysis at rest and during straining. Patient cured. It was concluded that there were two closure mechanisms acting against a PUL of optimum length, one at distal urethra and the 2nd at bladder neck, activated by oppositely acting muscle forces, Figure 2

The PVL tensions the arc of Gilvernet to anchor the anterior wall of bladder neck while the posterior vectors LP/LMA rotate the bladder downwards around the arc to close off the bladder neck, like kinking a hose.[1]

1.2 | Initial ultrasound proofs in 1990

Forward muscle forces were demonstrated. These were seen to stretch the distal vagina from behind to close the urethra (1, p22-23).

A downward muscles force which stretched the proximal vagina downwards was demonstrated by ultrasound (1,p24).

1.3 | Causation of urinary stress incontinence

Fundamental to the Integral Theory's concept of dysfunction is the pathogenesis of ligament laxity: its elongation and weakening mainly from childbirth, age, and menopause.[7] Collagen depolymerizes to lose 95% of its strength prior to labor,[8] allowing the cervical ring, ligaments and vagina to stretch considerably during birthing. If the collagen fibrils do not return to their pre-stretched state, they remain elongated and may weaken the contractile force of the vectors acting on them to cause USI, Figure 4.

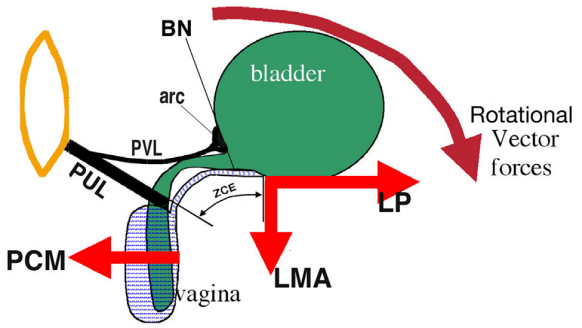

FIGURE 3 Normal urethral closure This is the schematic urethral closure mechanism as originally described in 1990.[1] The anterior portion of pubococcygeus muscle PCM (forward arrow), pulls against the pubourethral ligament (PUL) to close the distal urethra; levator plate (LP) (backward arrow) pulls backwards against PUL to stiffen the pubovesical ligament (PVL), bladder and vagina; The conjoint longitudinal muscle of the anus (LMA) (downward arrow), pulls against the uterosacral ligament to rotate the now stiffened vagina and bladder base around the precervical arc of Gilvernet (arc) to kink the bladder neck. Elasticity is required in the bladder neck area of the vagina (ZCE=zone of critical elasticity) to allow their separate actions. The directional arrows indicate the vector closure forces

The prototype operations proved that a PUL of specific length was required to restore continence. Figure 4 graphically shows how if the ligament (PUL) against which the PCM and LP directional closure forces lengthens, the muscles effectively lengthen; the contractile force of the (PCM) and backward (LP) vectors weakens,[9] Figure 5, to

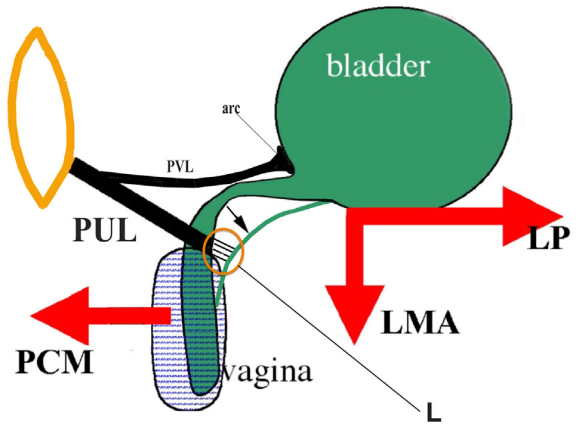

FIGURE 4 How a loose PUL causes stress incontinence The extension of PUL to "L" (circle) indicates PUL loosening; PCM and LP directional closure forces effectively lengthen and so their contractile force weakens (9); the anterior urethrovesical wall remains anchored by PVL and arc of Gilvernet. The downward action by LP/LMA opens out the posterior urethral wall. This is indicated by small diagonal arrow. The patient loses urine on effort. A tape implanted along PUL is elevated to remove looseness "L," thereby restoring the two closure mechansims and continence

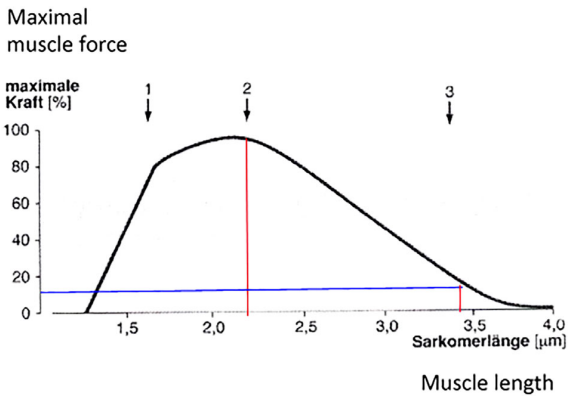

FIGURE 5 Gordon's Law A striated muscle contracts optimally over a short length only, between 1.7–2.5 µm on the sarcomere length. Lengthening the muscle from 2.5 to 3.5 µm, results in a rapid loss (85%) of contractile force

cause USI. Figure 4 explains why a tape placed along PUL as in the original TVT needs to be elevated to remove looseness "L" so that it may restore the anatomy, geometry, and continence.[10] This explanation is further validated experimentally in Part 2.

2 | CONCLUSIONS

The following were demonstrated by the prototype operations. A permanent sling was required for the MUS. Further proofs were required for the proposed musculoelastic mechanisms; this had to be proven by further experiments. An artificial collagenous pubourethral ligament of a specific length was needed to remove looseness "L," Figure 4, so as to restore anatomy and cure urinary stress incontinence. Xray and ultrasound demonstrated three directional forces acted during coughing and straining, suggesting they were involved in urethral closure. The experiments detailed in[1] led to the 1990 Integral Theory of Female Urinary Incontinence: *"Stress and urge, mainly occur, for different reasons, from laxity in the vagina, or its supporting ligaments, a result of altered collagen/elastin.*" The pathway to continence of a PUL sling was hypothesized to be restoration of the contractility of the three directional muscle forces, these being consequent on PUL repair. Part 2 summarizes some of the experiments which sought to validate the Theory and improve the MUS. Part 3 analyses the contributions of the findings of Parts 1&2 to the optimal surgical technique recommended by the authors.

CONFLICTS OF INTEREST

The 1st author PP is the co-inventor of the MUS, Integral Theory and TFS. BA has previously acted as a teacher for the MUS.

VIDEO 1 A haemostat applied on one side (so it cannot obstruct the urethra) directly behind the symphysis in the position of the pubourethral ligament so as to support it. This controls urine loss during coughing in more than 80% of patients with USI.

VIDEO 2 A haemostat applied on one side (so it cannot obstruct the urethra) directly behind the symphysis in the position of the pubourethral ligament so as to support it. This does not entirely control urine loss during coughing in patients with USI. Tightening the suburethral vagina is required for complete continence in about 15–20% of patients with USI.

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