Original article

How the midurethral sling works

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Abstract: Though the midurethral sling (MUS), the gold standard operation for cure of urinary stress incontinence has been in use for almost 30 years, few surgeons understand the anatomical basis for how it works. The original explanation given in 1990 was that the pubourethral ligament (PUL) weakened due to birth related collagen damage. The MUS tape shortened and reinforced the PUL by creation of a collagenous neoligament. This neoligament restored the distal and proximal urethral closure mechanisms. The closure mechanisms were further validated by simultaneous measurement of intra and extra urethral and bladder pressures, video ultrasound imaging after placing a haemostat on one side of the vagina to support the damaged PUL and in 2014 by a mathematical model which showed that any explanation which relied on the pressure equalisation theory would need a pressure The midurethral sling (MUS) is the most studied operation in the history of Urogynecology, validated by more than 2000 studies. Few know its history, fewer how it works. Even today, what some consider as the peak body for incontinence science, the International Consultation for Incontinence, attributes urethral closure to pressure transmission theories, none of which can explain why a tape placed at midurethra can restore continence. Absent from ICI is any mention of the Integral Theory (1) on which the first commercial MUS, the TVT was based (2). The aim of this comment is to present the original scientific work which led to the MUS and further experiments which sought to validate the original mechanisms proposed.

Keywords: Midurethral sling; Integral theory; Pressure transmission; Pubourethral ligament.

INTRODUCTION

1986-1990

The discovery of the MUS began with two unrelated observations in the mid 1980s. A hemostat applied at midurethra, fig. 1, (so it could not obstruct) controlled urine loss on coughing *without bladder neck elevation*. The second observation was an implanted Teflon tape created a dense collagenous tissue reaction around it. It was hypothesized that the cause of urinary stress incontinence (USI) was collagen deficiency in the pubourethral ligament (PUL) and that a tape implanted in the exact position of PUL would create new collagen to reinforce it. Experimental implantation of the tape in 13 canines validated this hypothesis and created a new surgical principle, creation of an artificial collagenous neoligament[3]. Between 1988 and 1989, prototype MUS were performed in 30 women. 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 midurethrally placed Mersilene tape was configured as an inverted 'U' on rectus sheath with its arms descending into the vagina, so the tape could be lowered if the patient could not pass urine. At a certain point, all 30 patients could pass urine freely and were continent on coughing. A 2nd standing xray was taken[1].

Xray findings All 30 patients were initially cured with no bladder neck elevation, even those with pre-op bladder neck below the lower border of symphysis. These findings invalidated the concept of a 'pressure equalization zone'. Other xray findings were: 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. Only muscle forces could explain these observations; forwards: anterior part of pubococcygeus muscle (PCM); backwards: levator plate (LP); downwards: conjoint longitudinal muscle of the anus.

Conclusions from the 1990 experiments

A muscle-based closure mechanism was acting against a PUL of optimum length to close both distal urethra and bladder neck.

* a rise in intraabdominal pressure closes the urethra

Question Why a PUL of optimum length? A striated muscle requires a firm anchoring point to contract efficiently. If the insertion point, in this case PUL is loose, the closure muscles effectively lengthen, their contractile forces weaken and urethra cannot be closed. This explanation can be easily tested by gently supporting the PUL with a haemostat placed on one side immediately behind the symphysis. This controls urine loss on coughing.

A further clinical test (late 1980s). A small group of patients with USI were asked to arrive with a full bladder. These were given extra fluid until they felt an urge to empty. Application of the one-sided hemostat test controlled USI. With the haemostat in place, they were asked to relax and pass urine. Those who succeeded were asked to strain down. This accelerated the stream. This test demonstrated 1. The mechanism of the MUS is not obstructive. 2. If the pressure transmission theory were valid, the urine stream should have diminished or stopped.

1995

Local anesthesia methodology for MUS allowed an experiment which tested both pressure transmission theory and the proposed musculoelastic mechanism[1] for truth or falsity. Pressure transducers were placed in equivalent positions inside and outside the bladder and urethra[4]. Pressure readings were made during coughing. As expected, the pressure inside the bladder was lower, between 2 and 5 cm H_2O less than outside the bladder. However, the pressure inside the urethra was in every case between 5 and 21 cm H_2O *greater* than the pressure outside. These findings confirmed the action of a reflex muscular mechanism and at the same time invalidated the pressure transmission theory. The pressure readings were carried out with vaginal incision open and repeated when the vagina was sutured. With open flaps, all patients lost large streams of urine on coughing, even when there were major rises in intraurethral pressure during coughing: in the 4 patients tested, midurethral pressure rise was 78, 94, 112, and 170% greater than resting. On tightening the flaps (without elevation) full continence was restored in all patients.

1999

Under video ultrasound control[5], a haemostat placed exactly in the position of PUL, on one side of the urethra,

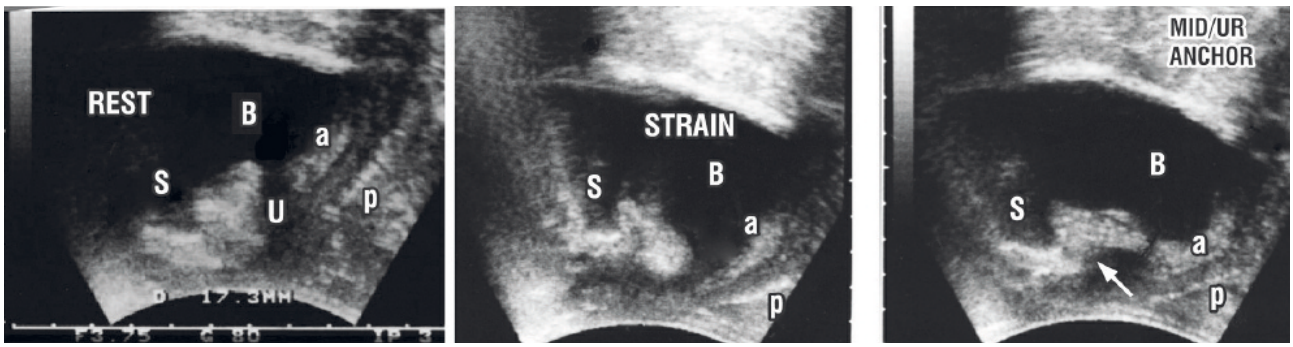

Figure 1. – A firm pubourethral ligament 'PUL' is required for urethral and bladder neck closure.
Left figure, 'REST' S=symphysis; U=urethra; B=bladder; a=anterior vaginal wall; b=posterior vaginal wall.
Middle figure 'STRAIN' A lax PUL cannot support the posterior urethral wall. This allows the posterior pelvic muscles to stretch the vaginal walls 'a' and 'b' backwards; this pulls open the posterior urethral wall. The urethra opens out (funnels) both proximally and distally.
Right figure 'MID/UR ANCHOR' Pressing upwards with a hemostat (arrow) restores PUL length and the strength of the muscle forces which contract against PUL; 'a'&'b' visibly tension; distal urethral and bladder neck closure.

controlled most of the urine loss on coughing, fig. 1. In those patients where there was some residual leakage on coughing, plicating the suburethral vagina with a 2nd curved haemostat restored continence. Both closure mechanisms as hypothesized[1] were validated . Note closure at bladder neck and distal urethra in fig1. It is evident on examination of fig. 1, that a PUL of specific length is required to restore continence and urethrovesical geometry. Pressure transmission theories cannot explain fig. 1. Only oppositely acting vector forces acting against a competent PUL can explain distal urethral and bladder neck closure.

2014

A mathematical model based on known biomechanical tissue characteristics estimated that a bladder pressure 2 orders of magnitude (100 times) was needed to open out the urethra from the inside for normal micturition[6]. The same pressure requirement would apply for closure from the outside in, the pressure transmission theory.

CONCLUSION

The MUS works by restoring the anchoring point for the opposite closure muscles PCM and LP.

CONFLICTS OF INTEREST

Burghard Abendstein has no conflicts of interest. Peter Petros is the co-inventor of the midurethral sling.

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