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Understanding the mechanics of closure is key to optimal midurethral sling technique

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Abstract

The animal experiments and prototype midurethral sling operations demonstrated that the sling provided new collagen to reinforce weak pubourethral ligaments (PUL). The now strengthened PULs were able to restore the contractile power of the 3 oppositely-acting directional closure forces. By contraction, these three forces exponentially altered the intraurethral resistance to flow when they closed the urethra to sustain continence. Relaxation of the forward force allowed the two posterior forces to uninhibitedly open the posterior urethral wall just prior to detrusor contraction, to facilitate evacuation of urine. The aim of this work is to examine the mechanics of the component anatomical structures which contribute to these functions, to analyse how subtle details impact on the actual surgical technique of the midurethral sling operations to optimize success, contribute to complications and how to prevent and fix them.

Keywords Midurethral sling · Urethral resistance · Obstructive micturition · Posterior fornix syndrome · Tethered vagina syndrome

Introduction

There are compelling reasons for understanding the anatomy of bladder closure and evacuation. The aim of performing the midurethral sling (MUS) is to repair the closure mechanisms of the bladder so the urethra can be closed on effort instead of opening; the mechanism of evacuation also needs to be known, as this impacts on the main complication of MUS, urinary retention; both mechanisms impact on how the sling itself restores continence and when it does not.

Despite the MUS being the most studied operation in the history of surgery with animal studies and prototypes extending from 1986 to 1996 [1–8], more than 1000 MUS surgical papers, 10,000,000 operations, it has been my experience that very few surgeons who perform the MUS have ever studied the anatomical mechanisms on which the MUS was based. Yet these very mechanisms, closure and evacuation, their component muscles, ligaments, tissues, constitute the very core of normal function, pathogenesis, the surgery itself and

complications of MUS surgery. The aim of this work is to revisit these mechanisms, how they explain pathogenesis, how the MUS works, why immediate post-op retention occurs, how to prevent and manage it; how surgery can unbalance these mechanisms to cause de novo symptoms such as pain, urge, nocturia, urinary retention, often months or years later and how to fix them.

The development of the MUS began in 1986 with an observation: mechanical support of the pubourethral ligament (PUL) at its origin immediately behind the pubic symphysis, controlled urine loss, but not always entirely; where there was some prolapse of the distal vagina, a fold of vagina was required to fully restore continence <https://youtu.be/0UZuJtajCQU>. From these studies [1] came a hypothesis: collagen weakness in the PUL caused stress urinary incontinence (SUI) [1]. Working on experimental animals with UWA Professor of Pathology, John Papadimitriou, we developed a new surgical principle, using tapes implanted in the precise anatomical position of the ligaments to create collagenous neoligaments [2] Fig. 1. The basis of this method was to use the collagenous wound reaction of an implanted foreign material (a tape) in a positive way, to reinforce collagen-deficient ligaments. This “neoligament” technology was successfully applied to the prototype MUS [3, 4] which showed that the MUS was safe, effective and proved the

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Fig. 1 Formation of artificial collagenous neoligaments. Specimen of vagina (V), vulva bladder (B) dissected from a dog 2 weeks after the implanted tape had been removed. Note the significant artificial collagenous neoligament created by tissue reaction against the tape (white arrows)

hypothesis, that the ultimate cause of SUI was collagen deficiency in the ligaments. These concepts are the basis of all midurethral (and other) sling surgery.

A firm pubourethral ligament is required to anchor the 3 oppositely acting directional urethral forces which close the urethra distally and at bladder neck, Fig. 2. <https://www.youtube.com/watch?v=3vJx2OvUYe0>.

The detailed anatomy of urethral closure Figure 3 is exactly as was described in 1990 [1]. It details how closure occurs at bladder neck when the muscle forces pull on PUL, which is sited at midurethra. This diagram also outlines why a retropubic sling is anatomically different from a TOT (“T”). In addition to providing a new PUL fulcrum point, the sling also reinforces the pubovesical ligament “PVL”(broken yellow lines).

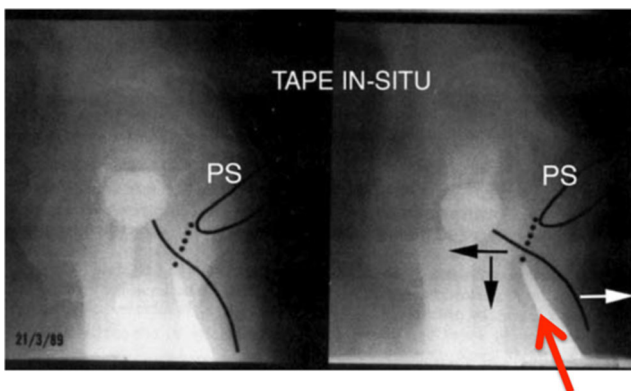


Fig. 2 Discovery that three directional forces act around the pubourethral ligament (broken lines, large arrow) to close urethra distally and at bladder neck. On application of a hemostat onto the intravaginal tape* (large red arrow) in the position of the pubourethral ligament, three directional forces became evident; forward to close distal urethra (white arrow); backward/downward (black arrows) rotate bladder around the pubourethral ligament to close urethra at bladder neck. *The tape of the 1st prototype was in the vaginal cavity

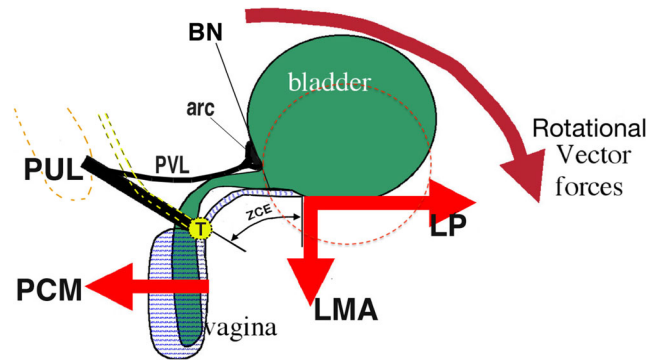


Fig. 3 The detailed anatomy of urethral closure as described in 1990 [1]. Pubourethral ligament (PUL) inserts into the midurethra and vagina. Pubovesical ligament (PVL) has the same origin as PUL; it inserts into a thickening of the anterior bladder wall called the pre-cervical arc of Gilvernet (“arc”). On effort, PCM (pubococcygeus muscle) contracts forwards against PUL to stiffen the posterior wall of distal vagina; LP (levator plate) pulls back against PUL to stiffen PVL and proximal urethra; the conjoint longitudinal muscle of the anus (LMA) pulls down against the uterosacral ligament (not shown here) to rotate bladder around the arc (broken lines) to close (kink) urethra at bladder neck. The vagina between PUL and bladder base “ZCE” (zone of critical elasticity) is elastic so it can stretch bilaterally to allow the distal (PCM) and proximal (LP/LMA) closure mechanisms to operate separately. The TOT MUS is inserted transversely at midurethra “T”. The retropubic MUS (broken yellow lines) crosses midurethra, follows PUL, crosses to reinforce PVL and proceeds retropubically

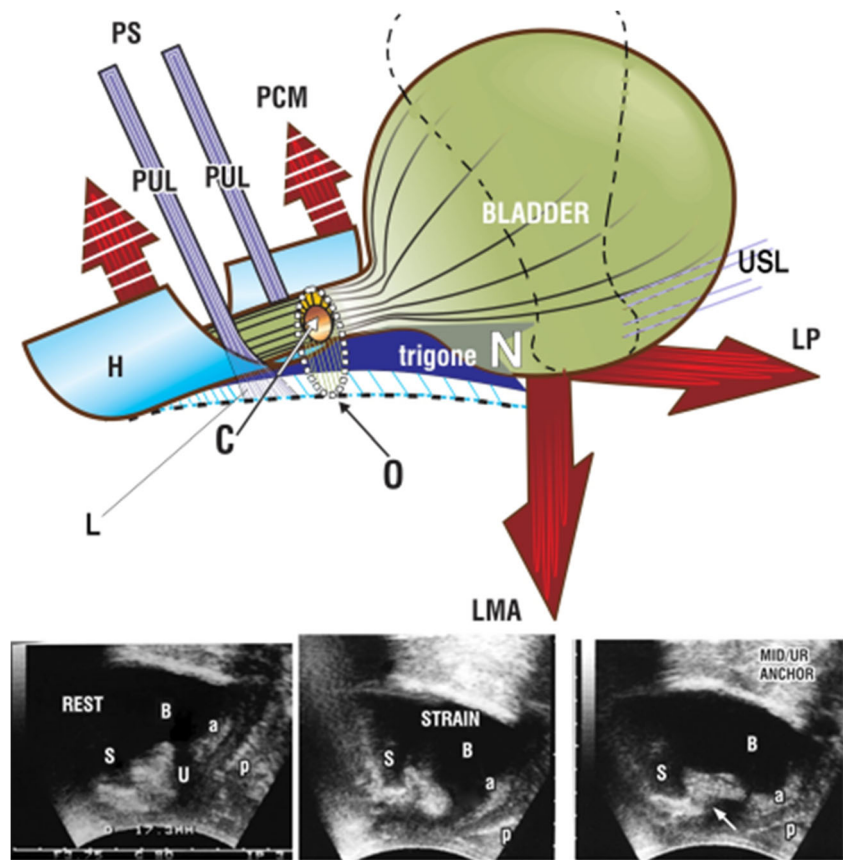
Pathogenesis of SUI, Fig. 4

The pubourethral ligament inserts 1.5 cm above and behind the lower end of the pubic symphysis [9]. If PUL is weak, it lengthens on effort “L”, Fig. 4. Because PUL is loose, it cannot support the posterior urethral wall and vagina which are pulled down to open the urethra from “C” closed, to “O” open; because PUL is lax, the forward muscle vector, pubococcygeus (PCM) is weakened, and cannot close the distal urethra¹; the posterior vector (levator plate “LP”) also weakens, as does the rotation closure mechanism at bladder neck; urine is lost, SUI. Supporting a weak PUL with a hemostat, white arrow, lower ultrasound diagram, Fig. 4, prevents PUL stretching (“L”). The vector forces (large arrows) can now contract optimally: with reference to the right ultrasound figure, the distal urethra closes, the funnelling disappears and urethra closes at bladder neck.

Testing for a weak PUL The only way to test PUL’s biomechanical competence is to insert a finger or hemostat on one side of the urethra immediately behind the lower end of the symphysis and press gently upwards. <https://youtu.be/0UZuJtajCQU>.

¹ A striated muscle requires a firm insertion point to contract efficiently [10]; a weak PUL insertion point Fig. 4, weakens both the distal and bladder neck closure mechanisms.

Fig. 4 Pathogenesis of Stress Urinary Incontinence. *Upper figure* On effort, a weak PUL is stretched down by LP/LMA muscle forces to lengthen, “L”. PUL + L cannot support the posterior trigone and posterior vaginal wall (broken lines) which are pulled down. Urethral cavity is forcibly opened out from C (closed) to O (open). Both closure mechanisms fail. Urine is lost - SUI. *Lower figure Transperineal ultrasound* reflects upper figure. At rest urethra is closed. On strain, note how the anterior “a” and posterior “p” vaginal walls are stretched back and down; urethra U is forcibly opened out (funnels) at bladder neck and distally. On placement of hemostat at midurethra, (white arrow) bladder neck and distal urethral closure are immediately restored. B = bladder; S = symphysis



How the test works A hemostat placed at the base of pubourethral ligament “PUL” (white arrow, Fig. 4), prevents a weak PUL elongating to “L”, Fig. 4, which invalidates both closure mechanisms, as pubococcygeus muscle “PCM” and LP/LMA (levator plate/ conjoint longitudinal muscle of the anus) forces require a firm insertion point to contract efficiently [10]. One-sided support means it is impossible to compress the urethra. This simple method immediately invalidates the many hypotheses which state that the MUS works by compression of the urethra. What these studies are documenting with their ultrasound observations, is evidence of faulty surgical technique, indentation of urethra at the time of surgery. The aim of the tape is to prevent elongation of a weakened pubourethral ligament, thus restoring the two urethral closure mechanisms, not indent the urethra.

Knowledge of urethral opening helps to better understand post-op urinary retention

Discussed in the 1990 prototype operation [3, 4], was the exponential effect of tape tightness on urine flow (Poiseuille’s Law) and therefore, urinary retention. In the normal woman, prior to bladder emptying by detrusor contraction, PCM, Fig. 4, relaxes (broken large arrow) allowing the

trigone and posterior urethral wall to be actively opened out by the posterior vectors LP/LMA [11]. See VIDEO micturition <https://www.youtube.com/watch?v=eiF4G1mk6EA&feature=youtu.be>

This active opening by external muscle forces exponentially decreases the resistance to the flow of urine through the urethra which is determined by a complex formula [12, 13]

$$\Delta P = P_{ves} - P_0 = \frac{8\rho Q^2 L f}{\pi^2 d^5} + \frac{1}{2} \rho V^2 - \rho g \Delta h$$

More simply stated, the pressure required to drive a head of urine through the urethra is inversely proportional to the 4th power of the radius (see pressure/flow graph Fig. 5) [12, 13].

How this translates to MUS surgery

It is evident from Fig. 4 and from the hemostat video, that the principal anatomical structure which needs to be repaired is the PUL. This is done by a midurethral sling, Fig. 5.

A sling placed too tightly below the urethra will not allow the posterior wall to be opened out fully (“funnelled”) during micturition. It means that even a very small amount of excess tensioning by the tape can require a much higher head of pressure to drive the urine through the urethral tube. For

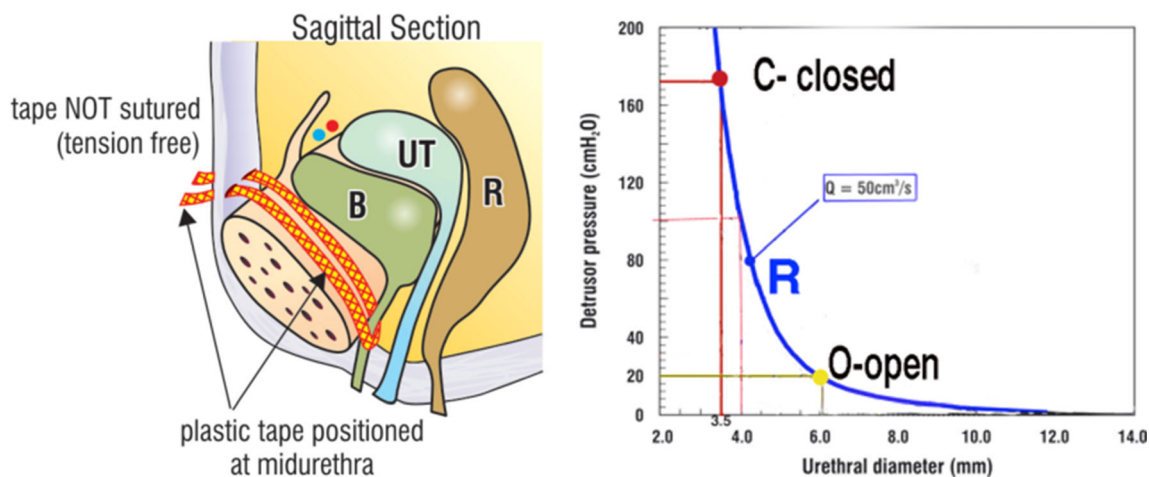


Fig. 5 Why even a minor tape constriction may cause post-operative urinary retention. For a urine flow of 50 ml/s, the urethral resistance to flow “R” increases exponentially as the diameter of the urethra narrows.

example, in Fig. 5 (right), a 2.5 mm difference in urethral diameter (6 mm reduced to 3.5 mm) requires 170 mm H₂O of detrusor pressure to expel the urine instead of 20 mm H₂O.

Correlation of closure/opening anatomy with MUS surgical technique

Repair of the proximal closure mechanism (midurethral sling)

I emphasize what I have written is based on my own surgical experience. It is meant only as a general guide.

1. It is very important to always bear in mind during the surgery, the exponential relationship between urethral diameter and resistance to urine flow, Fig. 5.
2. As a safety measure, it is recommended that an 18G catheter stay in the urethra during tensioning of the tape. Even minor excess tensioning may cause relative obstruction, Fig. 5. (A silicon catheter is less compressible than a rubber catheter).
3. Measure the length of urethra. Some urethras are only 2 cm long. It is very easy to place a tape at bladder neck to cause obstruction or even to perforate the bladder. Some urethras are 6 cm long; a tape 2 cm from meatus may leave the tape too distal. This will unbalance the two closure mechanisms for a suboptimal result.
4. A non-stretch tape gives built-in safety. Touch but do not indent the urethra. An elastic tape is necessarily stretched when it is pulled up; elastic retraction post-surgery may compress the posterior urethral wall to narrow it, with consequences for flow, Fig. 5, retention, or even urethral perforation as reported in the early TVTs.

Conversely, “R” falls exponentially when the urethral diameter increases, as happens during micturition, when the posterior urethral wall is actively pulled open by the posterior vectors, or in SUI when PUL support fails

5. It is the posterior urethral wall which is vulnerable to compression. Tensioning a tape placed under a bare urethra and pulling down with a No8 Hegar as an after measure is a very coarse way to dilate a urethra given that resistance to flow is exponentially determined, Fig. 5. Better a Hegar dilator in urethra which allows precise tensioning of the tape, mm by mm. This technique, adjusting the tape more precisely, is especially indicated in women with Intrinsic Sphincter Defect (ISD). Nakamura et al. reported 90% cure of ISD at 12 months using precise mm by mm tensioning methodology [14].
6. Suggested technique for precise sling tensioning: do it after cystoscopy with 300 ml in the bladder to check for tape perforation. Wait 3-5 s after removal of cystoscope and check if there is any continued leakage. If there is large continued leakage, it means the tape is too loose. If too loose, insert a No8 Hegar dilator, and tighten tape very slightly over the Hegar, *never over a bare urethra*. Press on bladder and see if there is a leak. You may have to repeat this manoeuvre 2-3 times. A slight leak when you press is a good sign.
7. What to do with an elastic tape? Insert scissor tips between tape and urethra to account for elastic retraction, always with an 18G Foley in place.
8. Tape placed near bladder neck. Some surgeons wrongly teach that if a woman has funnelling as in Fig. 4 “STRAIN” on ultrasound, the sling should be moved closer to bladder neck. This will hinder the stretching open of the posterior urethral wall prior to micturition by LP/LMA and may very significantly increase the resistance “R”, Fig. 5, leading to symptoms of “obstructive micturition” if not retention. One more step during the ultrasound, placing a hemostat at midurethra as in Fig. 4 (white arrow), would have demonstrated why a tape

should be at midurethra. It would have restored anatomy and continence.

9. If there is total urinary retention by 48 h, my advice is to take the patient back to OR, insert 18G Foley catheter, undo sutures and loosen tape carefully, sufficient to have a very slight leak on pushing on the bladder suprapubically. Because the wound is still raw, all this can be done quickly and easily. Tape re-adjustment is much easier if the tape used is nonstretch.
10. Why it is unwise to wait beyond 48 h? The answer is in “R” Fig. 5. Given enough time, the bladder *may* ultimately learn to contract harder to empty, but even then, the stream is often reduced. Another consideration is the effect of ageing on the neo-collagenous ligament which is created, Fig. 1. Collagen contracts and shortens with age. A woman with a tape slightly tight may present with retention 20 or even 10 years later because the collagen of the neoligament contracts with time. I have seen several such cases attending with acute retention 20 years after a sling, requiring section of the tape.

Repair of the distal urethral closure mechanism

The distal urethral closure mechanism is an often ignored but an important part of the MUS. Repair of the distal closure mechanism is especially important when operating on women with SUI and ISD. The original MUS protocols always recommended routine repair of the distal closure anatomy, Fig. 6 [3, 4, 8]. According to the Integral System, the role of the distal closure mechanism is to seal the urethra. However, it was demonstrated in that in about 20% of women, the closure mechanism did contribute significantly to urethral closure [15].

The components of the distal closure mechanism are the anterior portion of the pubococcygeus muscle, Fig. 6, PUL, external urethral ligament (EUL), Laxity in the EUL is invariably related to laxity in the suburethral vaginal hammock “H”, Fig. 6.

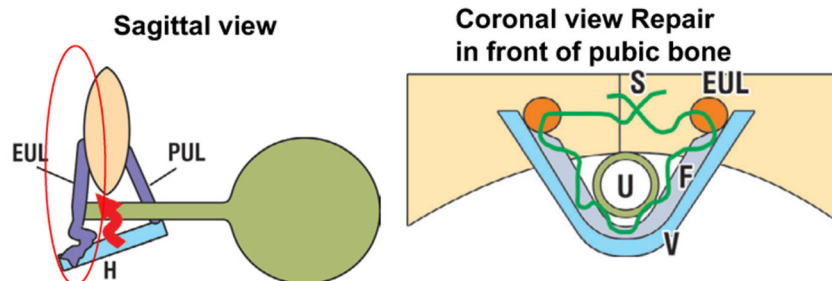


Fig. 6 repair of the distal closure mechanism <https://youtu.be/PIHTF5hh32A>. The aim of this continuous suture is to take the looseness out of the suburethral vagina. *Left* The red circle shows the distal closure mechanism anterior to the pubic bone with a normal PUL (pubourethral ligament), loose external urethral ligament (EUL) and loose

Technique for repair of the distal closure mechanism <https://youtu.be/PIHTF5hh32A>

The midurethral incision is extended to within 0.5 cm of external urethral meatus. With reference to Fig. 6 (right side) and with no 18 Foley catheter in situ, a continuous 2-0 Vicryl suture penetrates the ligamentous attachment of external meatus (EUL) on the right side; then the smooth muscle layer of vagina on both sides, finishing into the contralateral EUL. The suture is gently tightened over the catheter.

Not well known but important complications of MUS surgery and how to address them

Delayed de novo pain, urge, nocturia, urinary retention after MUS These symptoms may occur months or years later. It is evident from Fig. 4, that if a strong PUL is created by collagen from a mesh tape, the intraabdominal forces may be diverted to impact on the uterosacral ligaments (USL). If the USLs have been weakened by childbirth, they can give way and loosen. Loose USLs may (variously) cause grouped symptoms known as the “posterior fornix syndrome [16]: chronic pelvic pain, urge, frequency, nocturia, abnormal emptying and raised residual urine. In younger women, native USL repair can reverse these symptoms. In older women, because ligaments may be collagen deficient, a tape is usually required to reinforce the weakened USLs by creating new collagen [17–27].

Tethered vagina syndrome “TVS” *Not all urine loss on effort is SUI.* In women who have a scarred fibrotic vagina (either from multiple vaginal operations or large mesh sheets) and massive urine loss, classically, on getting out of bed in the morning, an MUS will only worsen the symptoms, as it intensifies the scarring. Understanding pathogenesis of TVS requires knowledge of the two oppositely-acting closure mechanisms. A mesh placed behind vagina across the “Zone of Critical

suburethral vaginal hammock (H). *Right* EUL is sited anterior to the pubic bone immediately lateral to the external urethral meatus “U”; F = fascia behind vagina “V”; S = symphysis pubis. Crooked red arrow signifies a weakened anterior portion of the pubococcygeus closure muscle

Elasticity² (ZCE), Fig. 3, may create sufficient fibrosis to “tether” the two opposite directional forces; the stronger posterior vectors overcome the weaker anterior vectors; the urethra is forcibly opened and urine may be lost uncontrollably when a major prolonged strain is placed on the muscles, such as getting out of bed in the morning (the classical sign of TVS). Often there is no urine lost on coughing. Transperineal ultrasound shows minimal downward bladder neck movement on straining. The cure for such women is a skin graft³ across ZCE to restore elasticity to ZCE.

Recommended is Professor Goeschen’s teaching module on the “TVS”. <http://www.pelviperrineology.org/archives/archive-detail/article-preview/tethered-vagina-syndrome/38678>

Discussion

The ban on the MUS in the UK seemingly ignored the huge body of scientific evidence validating the MUS over the 35 years since its inception and the recommendations of every learned surgical body. Older women in the UK have been left with few, very inferior options. Neither prolapse nor incontinence can be adequately cured with native tissue repair surgery when collagen is grossly lacking in the ligaments, as we find in old women. Collagen can be damaged at childbirth and it breaks down after the menopause to cause osteoporosis and organ prolapse. Excretion of hydroxyproline, a key breakdown product of collagen, doubles after the menopause and continues to excrete at that rate afterwards [19]. This process causes irreversible changes to the ligament/connective tissue structures which lose collagen, elastin and glycoproteins [28]. It follows, that a ligament truly deficient in collagen can only be repaired by improving its collagen component. To date, this has been done with a tape implanted in the exact position of the damaged ligament, [2], Fig. 1. This is the core principle behind the MUS and other ligament repair operations [27].

Conclusions

Where to now? The only way forward now for surgeons who have faith in the MUS and related operations, is to continue to present incontrovertible scientific data and well considered arguments which cannot be countered by anecdotal comments

² “Zone of Critical Elasticity, Fig. 3 demonstrates two different urethral closure mechanisms, distal and proximal, activated by oppositely-acting muscle forces (arrows). These can only function separately if these is adequate elasticity at “ZCE”, Fig. 3.

³ A free skin graft is best harvested from an area of elasticity, preferably lower abdominal wall. Another option is a vascular graft, such as skin-on Martius graft, or a “Singapore Flap” a skin-on graft from the groin.

which now pass for evidence by uninformed doctors, paramedics, patients and others [29].

Compliance with ethical standards

Conflicts of interest None.

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