

Late-occurring pain/other dysfunctions in midurethral sling class actions are likely caused by uterosacral ligament weakness, not implant or surgeon

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Abstract

Background: Large sums of money have been awarded against manufacturers of midurethral slings (MUS) because of complaints of pain and other complications, even though pelvic pain is rarely seen at the 6–12 weeks review.

Hypothesis and Aims: Pain/other posterior fornix symptoms (urge, frequency, nocturia, and abnormal emptying) may appear weeks or months after MUS surgery due to dislocation of already weakened uterosacral ligaments (USL), a consequence of diversion of pelvic forces from pubourethral ligaments strengthened by the sling.

Methods: Review for prevalence, pathogenic pathway from damaged USLs to pain, OAB, emptying, and other late complications by reference to data from functional anatomy imaging, mechanical support of USLs (speculum test), and post-USL surgical repair.

Results: Pelvic pain and other pelvic symptoms frequently co-exist pre-operatively with SUI, but are not volunteered because patients complain of one main pelvic symptom, others being “under the surface” (Pescatori Iceberg). Late de novo occurrence of symptom complications beyond perioperative MUS surgery may occur: pain (5.7%), retention (5.4%), UTI (9.3%), and OAB (10.2%).

Xray/ultrasound evidence of pelvic forces acting on USLs support the hypothesis of diversion of forces. Improvement of pain and urgency by the “speculum test” indicates USL causation, as do cure of pain and other pelvic symptoms by USL slings.

Conclusions: Late-occurring PFS symptoms are the fault of neither implant, nor surgeon, but more likely consequences of pelvic forces acting on USLs already weakened by childbirth/age. Bladder/bowel/pain symptoms need to be sought out preoperatively and discussed before MUS surgery.

Brief Summary: Late MUS complications, OAB, pain, retention subject to class actions, may be caused by uterosacral dislocation from pre-existing structural weakness, not surgeon or device.

KEYWORDS

class action, implant, midurethral sling, pelvic pain, posterior fornix syndrome, uterosacral ligament

1 | INTRODUCTION

In a space of 20 years, the midurethral sling (MUS), has become the most utilized incontinence operation in the history of pelvic surgery, with an estimated 10 000 000 operations to 2018.¹ However, the MUS is not complication free. Complications such as immediate postoperative urinary retention and tape surfacings/erosions are known and well described. Overall, in over 100 000 MUS procedures, the rate of any complication was 9.8% after a tape for incontinence,¹ 20% of complications refer to tape extrusion. These predominantly present “early,” and as such represent a primary failure of healing of the incision. Fifty percent of all complications had voiding difficulties which normally resolved in a few weeks.¹ Ten percent of complications of tapes (1%–2% of total) relate to the development of urgency, with up to 2/3 a result from a worsening of pre-existing urgency rather than a new symptom.¹

Of the remaining 20% of the 9.8% (1.9% of the total of all MUS complications) “with significant pain which seems more specific to the tape procedures,”¹ the authors state: “Where it is a new symptom it may reflect a failure to observe or diagnose the pre-existing condition”¹; and, “The exact nature and cause of pain in this group is unclear, but appears to be quite diverse. Those with pre-existing pain disorders for example, fibromyalgia may be more susceptible to developing pain.”¹ This comment is supported by Brevik et al.’s findings, that chronic pain of moderate to severe intensity occurs in 19% of adult Europeans, seriously affecting the quality of their social and working lives.²

1.1 | In summary

Pain and de novo urge after MUS surgery constitute at most 3.9% of reported late complications, and even there, a very significant percentage were pre-existing symptoms.

The catalyst for this special presentation was the awarding of large damages in class actions against device companies and surgeons because of patient complaints of chronic pelvic pain (mainly) and other complications such as chronic retention, OAB after MUS surgery. Acute retention due to an overtight tape aside, such complications

are rarely seen at the 6–12 weeks postoperative visits by surgeons.¹ The question is, are these complaints, particularly pain, fake or real? Were such symptoms pre-existing but not sought out? Were they a direct consequence of the implant or surgeon error? The aim of this study is to critically examine the last two options as regards anatomical pathogenesis.

A systematic review of MUSs at minimum 36 months after surgery confirms that de novo symptoms occur beyond the 6–12 weeks perioperative period. Similar % symptom complication rates for retropubic and transobturator MUS, (transobturator in brackets) occurred: pain, 1.8% (5.7%); retention, 5.4% (4%); urinary tract infection (UTI) 9.3% (3%); de novo urgency, 10.0% (10.2%); infective, 2.7% (3.8%); tape erosions, 2.1% (2.7%).³

These symptoms³ are almost identical to those reported for the posterior fornix syndrome (PFS),⁴ and are summarized in the rectangle, Figure 1: predictably co-occurring symptoms of frequency, urgency, nocturia, chronic pelvic pain, abnormal emptying caused by loose or damaged uterosacral ligaments (USL), cured/improved by repair thereof.⁴ Figure 1 summarizes the relationship between specific prolapses, symptoms, and ligament laxity.

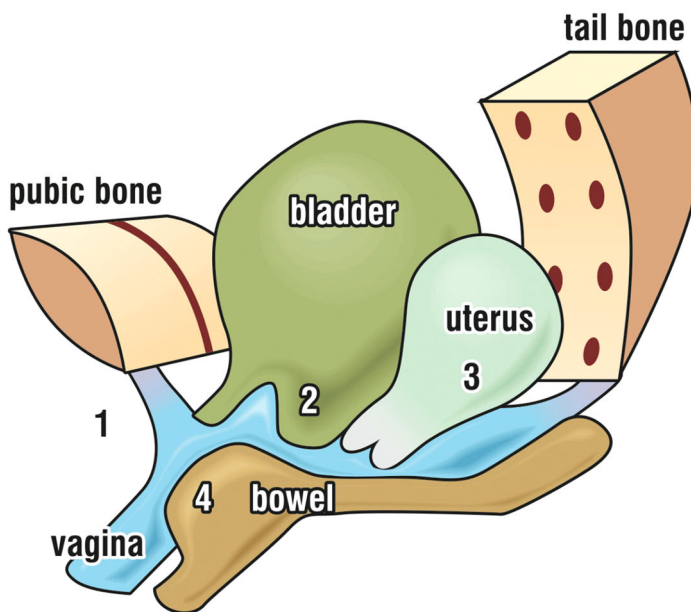
In a 35 year career spanning some thousands of MUSs, I have seen many delayed PFS complications occurring weeks, months, or years after the initial MUS in the proportions reported.³

An important clue to delayed appearance of posterior zone symptoms and prolapses, Figure 1 was the report by Shull, of greater than 30% “blowout” of prolapses in other parts of the vagina, some significant time after site-specific repair.⁵

The hypothesis for the pathogenesis of such delayed symptoms is that reinforcing the pubourethral ligament “1,” Figure 1, with a sling reflexly diverts intra-abdominally generated pressure posteriorly, thus adding sufficient extra stress to dislocate cardinal (CL) “2,” uterosacral (USL) “3,” perineal body “4” ligaments (Figure 1), ligaments which may be already compromised by birth and age, to cause cystocele, uterine prolapse or perineocele (Figure 1).

The aim of this study is to examine the causes of late symptom appearance post-MUS surgery. Is it the device, the surgeon, or is it from already compromised USL dislocation?

FIGURE 1 The pictorial diagnostic algorithm² relates symptoms to prolapse and ligament weakness. Symptoms in each of three zones indicate which ligaments have been damaged. The height of the bar indicates the probability of association of a symptom and a prolapse in a particular zone. The connective tissue structures causing prolapse and pelvic symptoms fall naturally into three zones. Anterior zone: external meatus to bladder neck. Middle zone: bladder neck to anterior cervical ring. Posterior zone: posterior cervical ring to perineal body. The rectangle indicates the symptoms associated with uterosacral ligaments laxity (USL). Chronic pelvic pain and nocturia are uniquely caused by USL laxity. ATFP, arcus tendineus fascia pélvis; CL, cardinal ligament; LMA, conjoint longitudinal muscle of the anus; LP, levator plate; PCF, pubocervical fascia; PCM, pubococcygeus muscle; PUL, pubourethral; ligament; hammock, suburethral vagina



	Front ligaments (PUL)	Middle ligaments (ATFP & CL)	Back ligaments (USL & PB)
		2 cystocele	4 rectocele
			3 uterine/apical prolapse
1 stress incontinence			
		abnormal emptying	abnormal emptying
	frequency and urgency	frequency and urgency	frequency and urgency
			nocturia
faecal incontinence			faecal incontinence obstructed defecation
			pelvic pain
		tethered vagina	

2 | METHODS

Qualitative imaging methods were used to validate the hypothesis: demonstration of intraabdominal forces acting on pubourethral (PUL) and USL ligaments during closure and micturition. The diagnostic algorithm, a foundation stone of the integral theory system, Figure 1, was used as a diagnostic guide for the surgical studies.⁶⁻⁸

The algorithm, Figure 1, shows how the relationships between symptoms and loose ligaments, rectangle Figure 1, may cause pelvic dysfunctions. Finally showing how the hypothesis of USL causation of pain/bladder/bowel dysfunctions, can be partly tested by speculum or pessary tests, and more fully by referral to published works which validate cure such symptoms following repair of USLs.⁶⁻⁸

3 | RESULTS

Straining and micturition, Figure 2, show reflex directional forces acting on both PUL and USL as evidenced by the significant movement of bladder, vagina, and rectum forwards, backward, downwards around these ligaments which act much like fixed structures. See ultrasound closure VIDEO.

<https://www.youtube.com/watch?v=3vJx2OvUYe0>

For micturition, posterior muscle forces actively open out posterior urethral wall. See micturition xray VIDEO <https://www.youtube.com/watch?v=eiF4G1mk6EA&feature=youtu.be>

Typical pressures recorded during straining were 0–72 cm H₂O and for micturition 20–110 cm H₂O.

3.1 | Pathogenesis of retention

Bladder with reference to Figure 3, weak USLs cause the LP/LMA muscles which contract against them to lose contractile strength. The posterior urethral wall cannot be adequately opened out. Because the resistance to urine flows is exponentially determined, Figure 4, the detrusor cannot easily expel the urine. Urodynamically, a higher detrusor pressure is recorded; emptying time is prolonged; flow is slow and may “stop and start” with (usually) raised residual urine.¹² In the longer term, detrusor muscle is likely to hypertrophy.

3.2 | Why even minor USL laxity may cause “obstructive” symptoms

The forward and backward muscle forces, Figure 3, balance at bladder neck. 0-0 signifies the neutral point of the forces. If USLs are weak, the muscles, LP/LMA which contract against USL weaken; PCM is relatively stronger. The neutral point moves to 1-1. The section of vagina and urethra between 0-0 and 1-1 cannot be opened out (funneled) so the detrusor must contract against a relatively unopened urethra. The exponential nature of urine expulsion affects clinical symptoms. With reference to Figure 4, even a minor narrowing of the urethral diameter from 4 to 3.5 mm increases the head of pressure required by the detrusor to expel urine from the bladder from 100 cm water to 172 cm. Conversely, expanding from 4 to 6 mm (yellow lines), much further reduces the head of detrusor expulsion pressure required to 20 cm water.⁹ It follows that even minor prolapse (minimal weakness) may cause major symptoms. Surgical cure of “obstructive” urinary symptoms and post-void residual after USL repair were recorded.^{7,10}

3.3 | Urodynamic testing

Attempts to *consistently* relate micturition pressures to urine flow, residual urine, emptying time and postvoid residual urine were not successful.⁹ One hundred patients were investigated with micturition pressure,

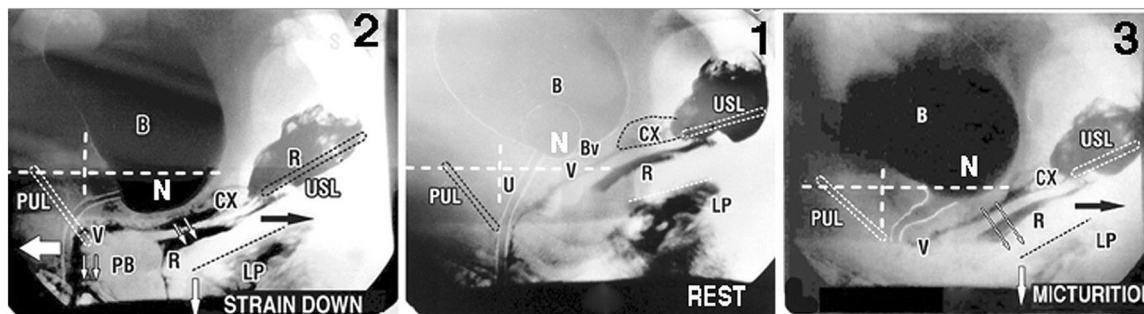


FIGURE 2 External directional muscle vector forces open and close the urethra, White lines represent vertical and horizontal bony coordinates. 1. Middle figure. At rest, asymptomatic patient. Slow twitch muscle forces and inherent elasticity combine to close urethra and prevent activation of the micturition reflex. N, stretch receptors for micturition reflex; LP, levator plate; U, urethra; B, bladder; U, urethra; CX, cervix; Bv, attachment of bladder base to anterior vagina; PUL, pubourethral ligament; USL, uterosacral ligament; R, rectum. 2. Left figure. Same patient. Straining—urethral closure. The PUL, USL do not stretch but are angulated downwards. With reference to the vertical bony co-ordinates, fast twitch muscles forces stretch the distal vagina forwards against PUL (arrow) to close distal urethra; backward/downward vectors (arrows) contract against USL to stretch and rotate the proximal urethra, proximal vagina, and rectum downwards around PUL, to effect bladder neck closure. **Typical pressure rises due to straining** were (mean 29.75 cm water, range 0–72 cm) PB, perineal body. 3. Right figure. Same patient. Micturition. The ligaments do not stretch. USL is angulated downwards. There is absence of a forward vector which has been relaxed by the micturition reflex. The distal vagina and urethra can now be unrestrictedly stretched backwards behind the vertical bony coordinate. With reference to the horizontal co-ordinate, proximal vagina and rectum are stretched backwards/downwards, against USL to open out the posterior wall of urethra

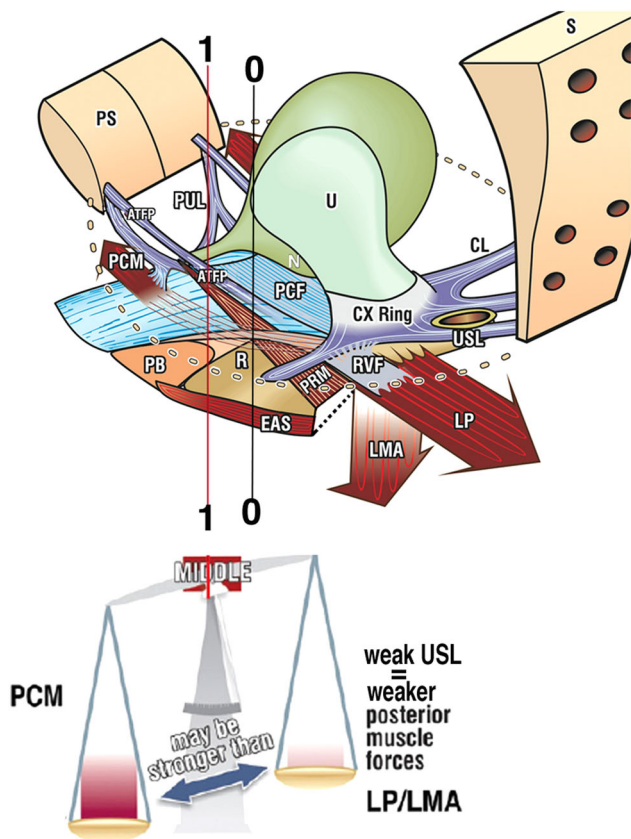


FIGURE 3 In the normal patient, the opposite directional vector forces are always in balance at bladder neck (black vertical line 0-0). The posterior vectors LP/LMA require a firm uterosacral ligament (USL) insertion point. If USL is loose, the posterior vectors weaken and the system loses balance, so the pressure equilibration point moves to the left to the red vertical line 1-1. The weakened LP/LMA cannot open the posterior wall of the urethra (obstructed micturition) or posterior anorectal wall (obstructed defecation). PCM compensates by contracting forwards. LMA, conjoint longitudinal muscle of the anus; LP, levator plate; N, bladder base stretch receptors; PCM, pubococcygeus muscle; PUL+, pubourethral ligament

studies.⁹ Frictional and dynamic losses were estimated at various flow rates using a mathematical model.⁹ Approximately 25% of all patients recorded micturition pressures below 11 cmH₂O at widely varying peak flows (mean 23 cmH₂O, range 0–91). Large inter- and inpatient variations in micturition pressures were recorded on retesting.⁹ The low pressures were explained by an external opening mechanism. The low pressures observed⁹ were explained by an external opening mechanism, Figure 2: backward stretching of the vagina during micturition by the muscles of the pelvic floor. This, most likely, was the cause of the large variability in observed micturition pressures and urine flow on retesting,⁹ as even minor changes in urethral radius are magnified by the fourth power (Poiseuille's Law),

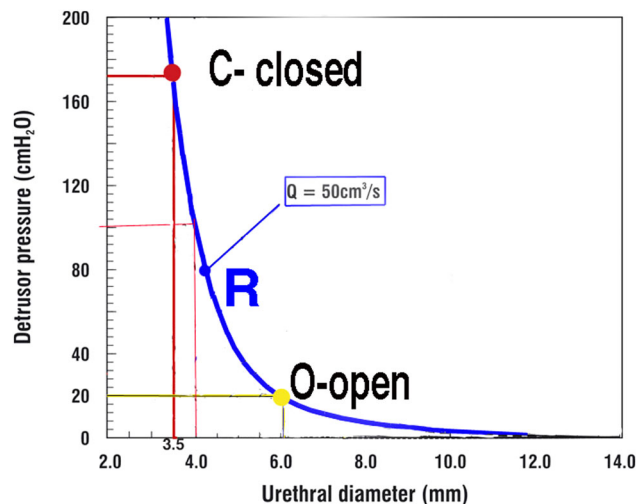


FIGURE 4 Exponential nature of urine flow is related to urethral diameter, based on a flow rate of 50 ml/s (thick blue line) which represents the total internal urethral resistance to flow “R.” Inability of the external muscle forces to open the posterior urethral wall before micturition, increase the detrusor pressure required for expulsion from 20 cm at 6.0 mm to 100 cm at 4.0 mm to 170 cm at 3.5 mm diameter

Figure 4. It was concluded that micturition itself, and the components for pressure generation, are complex nonlinear entities which appear to be greatly modified by the external striated pelvic floor opening mechanism.^{9,12}

3.4 | Anorectum

The biomechanics of ODS are very similar to those for the bladder as regards internal resistance to fecal expulsion (Figure 4). For closure, Figure 3, puborectalis muscle, PRM, contracts; LP/LMA rotate rectum around PRM to close the anorectal angle and “kink the rectum like a hose to close it.” For defecation (broken lines, Figure 3), PRM relaxes; LP/LMA open out anorectal angle (broken lines). External opening exponentially decreases resistance to fecal expulsion, thus assisting evacuation. Similarly to the bladder, if USLs are weak, LP/LMA forces weaken; the anorectal angle cannot be opened, the patient reports “constipation” or ODS (<https://www.youtube.com/watch?v=eiF4G1mk6EA&feature=youtu.be>)

4 | PATHOGENESIS OF OAB AND PAIN- SPECULUM TEST

The speculum test relieves/improves urge and pain symptoms in 70%–80% of women with PFS symptoms (PP unpublished data). OAB and chronic pelvic pain

(CPP) are related to bladder/bowel evacuation difficulties by virtue of the same anatomical defect, loose or weak USLs.¹² USL causation of urge and pain are directly testable by insertion of a speculum, Figure 5. As regards CPP, it has been known since 1938 in the German literature,¹¹ and 1996 in the English literature,⁸ that a major cause of “idiopathic” CPP has been the inability of loose USLs to support the T11-L2 (sympathetic) and S2–4 (parasympathetic) visceral plexuses, “VP,” (Figure 5). The hypothesis is that stimulated either by gravity “G,” or muscle movement in the upright position, unsupported VPs send afferent impulses to the brain. These are (falsely) interpreted as coming from the end organ itself, so the patient feels pain at the appropriately innervated endorgan, either T11-L2, S2–4, or both, depending on which afferent axons are involved. As regards urge incontinence, lax USL ligaments “L,” weaken the muscles LP/LMA which contract against them (wavy arrows). Muscle weakness affects the ability of the 3 opposite muscle forces to tension vagina to support the stretch receptors “N” from below and prevent the afferent impulses from stimulating the micturition reflex to empty. Beyond a threshold level, these impulses are interpreted by the cortex as urge symptoms.

4.1 | Speculum test

The speculum mechanically supports the USLs; this prevents the action of gravity “G” on the visceral plexuses “VP.” If the speculum test is positive, the pain is immediately relieved or improved. USL support also restores the LP/LMA contractile forces; the vagina is tensioned to support “N”; afferent impulses to the micturition center decrease; the urge symptoms improve or disappear.

4.2 | Direct proof of the hypothesis by USL repair by posterior sling

Table 1 is from a multicentre study of 611 patients where USLs were repaired by the TFS mini sling.⁶ Assessment was at 12 months. Prolapse was predominantly 2nd & 3rd degree. Almost all women were discharged the same day or after an overnight stay. Table 2 is from a single surgeon in 198 women who presented with SUI, chronic pelvic pain and prolapse, also 2nd & 3rd degree.⁷ Surgery was midurethral and uterosacral ligament repair using the IVS tunneler. Hospital stay was 3–4 days.

*Cure of CPP of unknown origin by native USL repair.*⁸ In 1996, the author reported, for the first time in the

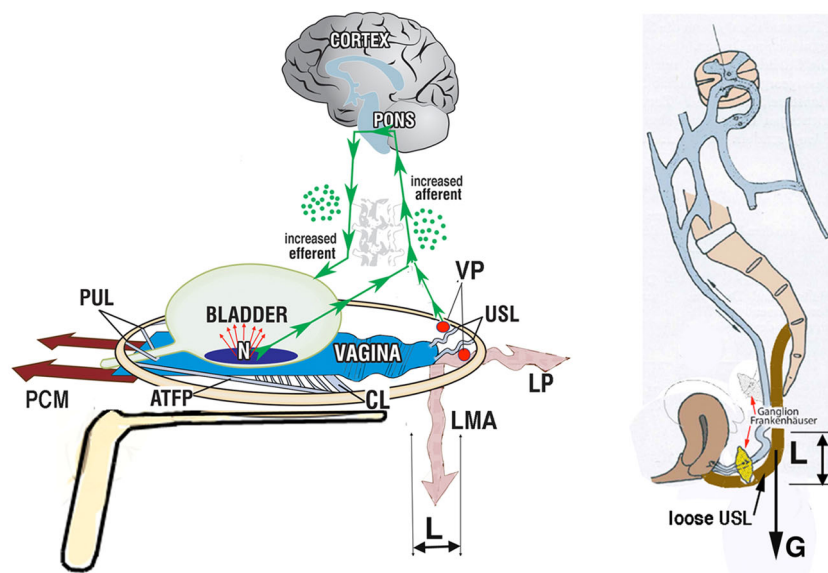


FIGURE 5 Speculum test diagnoses USL (uterosacral ligament) causation of chronic pelvic pain and urgency. *Left:* Cortical/peripheral feedback binary control system, with only two modes, closed (continence) or open (micturition). LMA, conjoint longitudinal muscle of the anus; LP, levator plate; N, stretch receptors; PCM, pubococcygeus muscle; PUL, pubourethral ligament; USL, uterosacral ligament; VP, visceral plexus. *OAB* Lax ligaments “L” weaken the muscles LP/LMA forces which contract against them (wavy arrows). Vagina cannot be stretched to support “N”. “N” fires of excess afferent impulses to activate micturition at a low bladder volume (“urge incontinence”). *Pain control:* The visceral plexuses (VP) are supported by USLs. *Speculum* mechanically supports USLs to prevent VPs firing off. Also, to create a firm anchoring point for LP/LMA to restore their contractile strength to tension vagina to support “N” and relieve urge. *Right:* Pain circuit via sympathetic (Frankenhauser) plexus activated by gravity “G” acting on VPs unsupported by lax USLs

TABLE 1 Cure rate of symptoms after TFS uterosacral ligament repair from Liedl et al.⁶

Symptom	Patients with symptoms/total patients		
	pre-TFS	post-TFS	CURE (%)
Pelvic pain	194/611	42/611	77
Nocturia	254/611	77/611	68
Urge/urge incontinence	317/611	51/611	85
Frequency	310/611	55/611	83
Fecal incontinence	93/532	34/532	65
Apical prolapse	611/611	63/611	90

literature, 70% cure of “idiopathic” CPP in 28 women at 12 months after native USL repair. Laparoscopy showed no obvious cause in any of the women. Six women had been admitted as emergencies; two of these were nulliparous. VAS scores were used but were difficult to monitor precisely. The ultimate criterion used was zero sum: presence of pain or not.

Role of USL laxity in causation of urinary retention, post-void residual urine and delayed emptying time. Of 36 patients with symptoms of “obstructed micturition,” 34 who had USL reconstruction with a TFS sling were reviewed at 12 months. Emptying symptoms were cured or improved beyond a VAS self-assessed >80% level in 76% of patients ($p > 0.001$); mean emptying time improved from 52 to 26 s ($p > 0.001$); mean residual volume from 201 to 39 ml ($p > 0.001$).

5 | DISCUSSION

Late de novo symptom complications such as pain, OAB, urinary retention, which occur in up to 10% of women having MUS,² are consistent with the hypothesis of USL dislocation by diverted pelvic forces. Based on data from 100 000 cases, pain and de novo urge after MUS surgery constitute at most 3.9% of reported late complications, **and even there, a very significant percentage were pre-existing symptoms.**

How does all this translate to a surgeon or device company who are the subjects of legal action? Shull's observation⁵ is very important: 30% of site-specific pelvic operations have a “blow out” prolapse in another part of the vagina. A tape at PUL creates new ligament composed of collagen¹³ which is immensely strong, breakdown strength being 18 000 lbs/sq inch.¹⁴ Pelvic pressures impacting on such a powerfully reconstructed PUL are diverted to other structures often compromised by age or childbirth, to weaken the ligaments and cause

TABLE 2 Symptom cure rate after posterior IVS sling from Goeschen et al.⁷

Symptom	pre-PIVS	post-PIVS	CURE (%)
Pelvic pain	198/198	52/198	74
Nocturia	63/198	13/63	79
Urge/urge incontinence	55/198	11/55	80
Frequency	127/198	26/127	80
ODS	59/198	12/59	80
Residual urine > 50 ml	44/198	20/44	55

further prolapse, schematically represented by CL“2’, USL”3”, PB”4’ (Figure 1), different prolapses, as reported by Schull.⁵ Next, understanding that posterior fornix syndrome (PFS) symptoms co-occur in predictable groupings, rectangle Figure 1, helps to diagnose USL causation for such pain. Two PFS symptoms, pain and nocturia, Figure 1, belong uniquely to USL laxity.

The speculum test, Figure 5, allows confirmation of urge and pain originating from loose USLs. For urinary retention, co-occurrence of other grouped symptoms as in Figure 1, give a clue. Tampons in the posterior fornix anecdotally improve emptying problems (if they can be retained during micturition).

I have seen late presenting cases of urinary retention following MUS. The cause was attributed to “obstruction” by the tape. Tape removal did not improve emptying. Rather, the SUI returned. Careful history revealed that the patients were discharged micturating adequately after the MUS and there was a delay in appearance of the retention, often weeks, months, even years. Clearly the reason for the emptying difficulties could not have been an overtight MUS.

Symptoms not mentioned by the patient, but sought out, may give a clue as to possible problems in the future. Pescatori's iceberg study¹⁵ demonstrated that on consulting a doctor, patients complain of one main symptom (tip of the iceberg). However, many other symptoms lie “below the surface.”¹⁵ Using the ITSQ validated questionnaire¹⁶ and Figure 1 as an aide memoire, Goeschen⁷ uncovered a range of other “below the surface” conditions, Figure 6. These 198 patients presented mainly with CPP. Some degree of uterine/apical prolapse was found in all women on examination.

The importance of considering other pelvic symptoms preoperatively was emphasized in a study by Norton et al.¹⁷ who reported on 2098 women with urinary incontinence symptoms:49.8% pure stress incontinence, 34.3% mixed incontinence and 15.9% pure urge incontinence. Norton found preoperative voiding

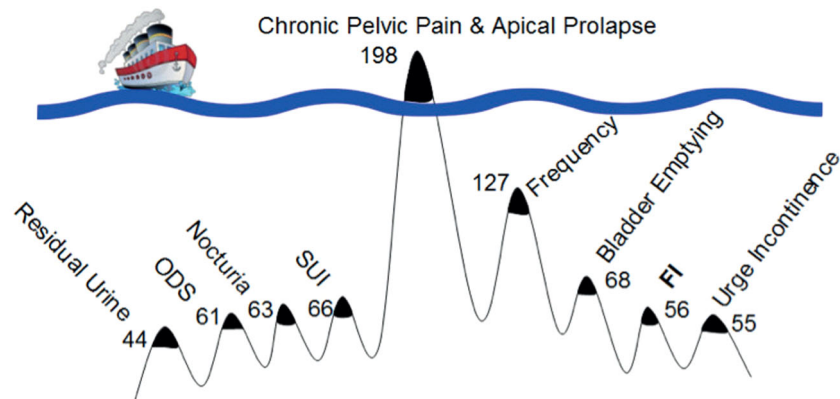


FIGURE 6 Pescatori Iceberg applied to chronic pelvic pain (CPP). The symptom prevalence is graphically indicated in the iceberg diagram, latent symptoms below the waterline. All symptoms are derived from the Integral Theory Symptom Questionnaire (ITSQ).¹¹ Numbers indicate combined total of the hysterectomy and nonhysterectomy cohorts

symptoms were a risk factor for incomplete bladder emptying at discharge after mid-urethral sling surgery. Urodynamic findings were not predictive of post-operative retention. In such cases,¹⁷ the algorithm, Figure 1, administered as a routine pre-operatively, is helpful in predicting those patients whose symptoms indicate there may already be a problem from damaged USLs. Urge and emptying problems together indicate a weakness in USLs and/or cardinals. If pain and nocturia were present pre-operatively, it is almost certain that USLs were already weak and vulnerable to dislocation after MUS surgery. In fact, chronic pain of moderate to severe intensity occurs in fully 19% of adult Europeans, seriously affecting the quality of their social and working lives.² To my knowledge, no account was taken of pre-existing CPP in the litigation cases. Instead, the device or the surgeon was blamed.

6 | CONCLUSION

Posterior fornix syndrome (PFS) symptoms may occur months or years after MUS, but may be present “below the surface” preoperatively, as shown by Goeschen⁷ and others.⁶⁻⁸ Late PFS symptoms are not necessarily the fault of the implant, or the surgeon. They are more likely a consequence of diverted pelvic forces onto USLs weakened from childbirth or aging. It is prudent for the surgeon operating for MUS to preoperatively to use the algorithm Figure 1, as a quick aide memoire and to meticulously search out, discuss and record coexisting symptoms. Furthermore, to be aware that such symptoms may occur months or years after MUS.

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CONFLICT OF INTEREST

The author declares no conflict of interest.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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