

Underactive bladder may be caused by uterosacral ligament laxity – a critical review of two paradigms

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Introduction The aim of this study was to compare the posterior fornix syndrome (PFS), (abnormal bladder emptying, urge, frequency, nocturia, chronic pelvic pain) cured/improved by uterosacral ligament (USL) ligation, with ‘underactive bladder’ (UAB) [2], whose cause and cure of UAB are said to be unknown [2].

Material and methods A limited literature search was carried out for the words posterior fornix syndrome; obstructed micturition; post-void residual.

Results We found the diagnostic criteria used for UAB to be identical with PFS. Also, individual symptoms could be improved in the short term with squatting-based pelvic floor exercises, native tissue cardinal/uterosacral ligament repair, but requiring posterior ligament slings for a long term cure.

Conclusions Because the similarity in symptoms may not be sufficient in the first instance to recommend surgery for UAB, we advise the use of a roll gauze or large tampon placed in the posterior fornix to support USLs ('simulated operation'), always with a full bladder, then observe any changes in PFS symptoms such as urge, pain, urine flow and post-void residual as a screening test before proceeding to surgery.

Key Words: DUA ↔ nocturia ↔ obstructed micturition ↔ post-void residual
↔ posterior fornix syndrome ↔ underactive bladder

INTRODUCTION

Our aim is to compare the posterior fornix syndrome (PFS) [1], with the description of underactive bladder” (UAB) [2]. PFS was first described in 1993 [1] as a syndrome comprising of abnormal bladder emptying, urge, frequency, nocturia and chronic pelvic pain. It was cured/improved by uterosacral ligament ligation. Whereas all 4 PFS symptoms are all potentially curable by USL repair, there is no cause or cure deemed possible for UAB [2].

‘Underactive bladder’ and its urodynamic correlate ‘detrusor underactivity’ (DU) are definitions introduced by the International Continence Society (ICS) [2].

Urodynamically diagnosed DU is “a contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or a failure to achieve

complete bladder emptying in a normal time span” [2]. UAB is characterized by slow urinary stream, hesitancy and straining to void, with or without a feeling of incomplete bladder emptying and dribbling, often with storage symptoms, without evidence of any outlet obstruction [2].

Uren et al. [3] demonstrated storage lower urinary tract symptoms (LUTS) (nocturia, increased daytime frequency, urgency, and incontinence), and voiding symptoms (slow stream, hesitancy, and straining) in over half the patients with DU. Also, high post-void residual, history of urinary tract infections (UTIs) and a history of retention episodes [3]. All the above are classic PFS diagnostic criteria.

A systematic review by Osman et al. using the search words ‘underactive bladder’, ‘detrusor underactivity’, ‘contractile bladder’, ‘detrusor failure’, ‘detrusor areflexia’, ‘atonic bladder’, ‘chronic retention’,

and ‘impaired bladder contractility’ concluded there had been little progress in the etiopathogenesis, diagnosis, and treatment of the problem [4].

The UAB definitions were questioned recently by Uren and Drake [5]. “Because the terminology does not make the contrasting storage and voiding functions clear, patients can often be confused by the possibility that they could have both an overactive bladder (OAB) and an UAB, and the health-care professional then has to explain the relationship between these terms and the micturition cycle. No consensus has yet been achieved on how to overcome this limitation.”

The posterior fornix syndrome, ‘PFS’ (abnormal bladder emptying, urge, frequency, nocturia, chronic pelvic pain) was published in 1993 [1].

Not all patients had every PFS symptom. At 12 months, the cure rate following native tissue uterosacral ligament (USL) approximation was >50% for all symptoms [1]. The subsequent cure rate deterioration was attributed to insufficient collagen in the USLs. A posterior USL sling using the same ‘artificial neoligament principle’ used in the tension-free vaginal tape TVT* was introduced with much higher cure rates at a 20 month review [6].

- In effect, a ‘reverse TVT’. The tape was applied along the length of the damaged USLs.

The first urodynamically controlled pre and post-op report regarding the cure of PFS in the female was in 1997 [6] in patients who had midurethral and posterior slings to repair damaged pubourethral ligaments (PULs) and USLs. A total of 50% of patients had 1st degree apical prolapse and 50% 2nd or more. At (mean) 21-month follow-up, cure rates were: stress incontinence 88% (n = 85), frequency 85% (n = 42), nocturia 80% (n = 30), urge incontinence 86% (n = 74), emptying symptoms 50% (n = 65). Mean objective urine loss (cough stress test) was reduced from 8.9 g preoperatively to 0.3 g postoperatively, mean residual urine decreased from 110 ml to 63 ml, $p \leq 0.02$. Urodynamically diagnosed detrusor instability (DI) was present in 36/85 patients preoperatively (42%) and in 13/61 postoperatively (21%). Of these 13 patients, 12 had no incontinence symptoms. Of the 5 operative failures who were tested postoperatively, 4 had a stable detrusor.

Two further urodynamically controlled studies reported cure of PFS symptoms and post-void residuals Goeschen [7]: USL intravaginal slingplasty (IVS) (mean age 62 years) (n = 198): 80% cure for frequency (127/198), 80% for nocturia (63/198), and improvement of 44 patients with residual urine >50 ml to 26 <10 ml (65%).

Petros et al. [8]: cardinal ligament (CL)/USL TFS sling (mean age 65 years) (n = 69), pre and post-op

urodynamically controlled study: emptying symptoms (36/69): 76% cured; mean emptying time improved from 52 to 26 seconds, mean residual volume from 201 ml to 39 ml. Two patients who were self-catheterizing pre-operatively were restored to normal micturition, with postoperative residuals of 50 ml and 32 ml. Nocturia: 149 episodes pre-op; 53 episodes post-op. Frequency: 11.8 /day pre-op to 8.4 post-op; urge incontinence: 68 wet episodes/day to 16/day.

Two large studies, n = 611 [9] and n = 1420 [10] with 4 and 5 year longer-term data [11, 12] and other studies with 1–2 year data [13–17] reported similarly high surgical cure rates for PFS symptoms based on USL reconstruction.

Abendstein et al. extended PFS diagnostic criteria to the anorectum with a pre- and post-op study showing cure of obstructed defecation and rectal intussusception cure following a USL posterior sling [17].

The improvement in PFS symptoms is not confined to surgery. Patricia Skilling [18] demonstrated >50% improvement in PFS symptoms in younger women at 3 months following squatting-based pelvic floor exercises which strengthen the 3 directional muscle forces and the ligaments against which they contract, PUL and USL, (Figure 1A).

How the Integral Theory explains overactive bladder (OAB) and detrusor underactivity (DU) (Figures 1A to 1C)

The Integral Theory emphasizes the primacy of pelvic ligaments in bladder function. Simplistically, 3 oppositely-acting pelvic muscle forces contract against the pubourethral (PUL) and uterosacral (USL) ligaments, to tension the vagina bilaterally to support the bladder base stretch receptors ‘N’, from below, Figure 1A. This support prevents premature activation of the micturition reflex which is interpreted as urge by the cortex. On relaxation of the forward force PCMA (arrow, Figure 1A), the two posterior forces (arrows) pull against the uterosacral/cardinal ligaments (USL/CL) to stretch open the posterior urethral wall just prior to micturition (broken lines Figure 1A), radiograph, Figure 1B. This action further stimulates ‘N’ to accelerate emptying. If USLs are loose, the activation of the micturition reflex by ‘N’ is understimulated; funnelling, Figure 1B, is suboptimal creating greater resistance to flow **; the striated muscle opening forces LP/LMA cannot ‘grip’ and they contract repeatedly to open the outflow tract, Figure 1C; all these factors contribute to prolonged micturition, the key symptom of ‘UAB’; the altered mechanical and peripheral neurological process explains the described characteristics of UAB and DU: stopping and starting, slow

stream, hesitancy, straining, inability to empty, post-void residuals and the characteristic flow pattern, Figure 1C.

** The resistance to urine flow is very finely balanced, being inversely proportional to the 4th power of urethral radius (Poiseuille's Law), so even minor changes in the dynamic muscle/ligament anatomy may cause UAB.

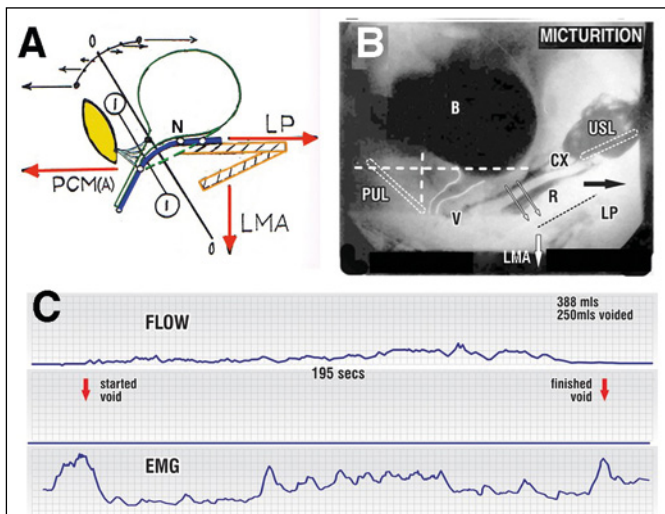


Figure 1. Mechanics of normal micturition and underactive bladder (UAB). **A.** Schematic sagittal view of bladder, vagina (thick blue line), levator plate (LP), conjoint longitudinal muscle of the anus (LMA) and anterior part of *m. pubococcygeus* [PCM(A)]. At rest: LP/LMA and PCM(A) vectors are in balance. On effort PCM(A) stretches the distal vagina forwards against the mid-urethral ligaments (PUL) to close distal urethra from behind; LP/LMA stretch bladder base backwards/downwards to 'kink' urethra at bladder neck (0-0). The stretched vagina (thick blue line) supports the stretch receptors 'N' from below. Micturition: PCM(A) relaxes, LP/LMA uninhibitedly stretch open the posterior urethral wall to funnel bladder/urethra (broken lines) forwarding bladder neck to 1-1. This 'funneling' exponentially lowers intra-urethral resistance to urine flow and stimulates N (stretch receptors) at bladder base, further activating the micturition reflex. Figure modified from 'An Integral Theory of Female Urinary Incontinence'. *Acta Obst. Gynecol. Scand. Supp.* 1990; 153: 21. **B.** Radiograph micturition video myogram, radiopaque dye in LP and organs: Normal patient sagittal view. Vertical and horizontal lines are bony co-ordinates. The posterior wall of urethra is pulled open by LP/LMA vectors acting against the insertion of uterosacral ligaments (USL) in the cervix (CX); R – rectum; PUL – pubourethral ligament **C.** Detrusor underactivity graph with prolonged flow indicating inability of muscles to open out posterior wall of urethra and enhance the micturition reflex. An EMG cylindrical electrode inserted into the posterior fornix of vagina records posterior muscle activity (LP/LMA). Note LP/LMA muscle activity preceding flow and repeated activity throughout flow.

How does uterosacral ligament (USL) repair reverse pathogenesis and symptoms?

A stronger USL creates stronger posterior vector forces to stretch the vaginal membrane to better support the stretch receptors 'N', Figure 1A. This prevents their premature activation during the day (urge, frequency) or at night (nocturia). The stronger backward force would better accelerate the micturition reflex and importantly better open out the out-flow tract, Figure 1B. The cure of chronic pelvic pain is explained by better support of the nerve plexuses T11-L2 and S2-4 contained within the USLs [19, 20].

DISCUSSION

Our data explains the dilemma articulated by Uren and Drake, how overactive bladder (OAB) and an underactive bladder (UAB) may co-exist [5]. At least in the female, the identical diagnostic criteria indicate UAB and PFS may be the same condition, raising the possibility that UAB is potentially curable by surgically reinforcing lax USLs, or in younger women, by strengthening the 3 directional pelvic muscle forces, Figure 1A, by squatting-based pelvic floor exercises [18].

Is a posterior sling necessary? The original USL ligations cured symptoms characteristic of both UAB and OAB in >50% of women [1]. In a native tissue cardinal/uterosacral ligament repair study (n = 88), Shkapura et al. (personal communication) demonstrated a marked difference in cure rates at 12 months between pre- and post-menopausal women, 80% and 20% respectively. Shkapura explained the poor cure rate in post-menopausal women as due to collagen leaching out of the cardinal ligament / uterosacral ligament (CL/USL) post-menopausally. In contrast, the tissue fixation system (TFS) posterior sling data of Inoue [11, 12] showed minimal deterioration at 4 and 5 years. We explain this by the creation of new collagen by the neoligament surgical principle on which the TVT and posterior slings are based.

How to prove that UAB and posterior fornix syndrome (PFS) are one and the same condition?

We suggest:

1. That interested physiotherapists or physicians use the validated ITSQ, Integral Theory System Questionnaire (published in *World Journal of Urology* in 2013), which will uniquely diagnose all PFS symptoms. Moreover, ITSQ will accurately indicate which ligaments are damaged (including the USL).
2. Perform a 'simulated operation': insert a roll gauze, cylindrical pessary, or 1–2 large menstrual

tampons to support USLs in the posterior fornix and observe the effect clinically and by flowmeter.

CONCLUSIONS

Squatting – based pelvic floor exercises [18], native tissue cardinal ligament / uterosacral ligament repair may improve posterior fornix syndrome

(PFS) (and underactive bladder) in younger premenopausal women, but not older post-menopausal women, who generally require tapes which create new collagen to reinforce the ligaments for long-term effect.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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