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Interstitial cystitis symptoms as defined are indistinguishable from posterior fornix syndrome symptoms cured by uterosacral ligament repair

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ABSTRACT

Objective: To test our hypothesis that female interstitial cystitis (IC) and Posterior Fornix Syndrome (PFS) are one and the same.

Materials and Methods: A retrospective study. We extracted raw CPP data from patients who had TFS surgery for cure of uterine/apical prolapse, along with bladder, pad and urodynamic data. We critically compared known IC phenotypes with PFS symptoms to check our hypothesis for truth or falsity. We used a validated Integral Theory System Questionnaire (ITSQ), "simulated operations" testing with the speculum test (which reduces urge and pain if USLs are weak), transperineal ultrasound, urodynamics, surgery and post-operative urodynamics.

Results: Forty-six patients with CPP had 157 urinary symptoms. The cure rate was CPP 76%, urge incontinence 74%, abnormal emptying/retention 80%, nocturia 75%, frequency 50%.

Conclusion: PFS data accord exactly with the ICS definitions for IC, except that PFS patients were cured or improved by USL repair, IC patients, not. Further testing of our hypothesis will require wide-ranging testing with the ITSQ (which diagnoses both PFS and IC), and the simulated operations "speculum tests" to confirm that USL weakness is indeed the cause.

Keywords: Interstital cystitis; posterior fornix syndrome; chronic pelvic pain; uterosacral ligaments; urge incontinence; nocturia; urinary retention

INTRODUCTION

Recently, the first surgical cure Interstitial Cystitis (IC) of histologically validated Hunner's ulcer by Tissue Fixation System (TFS) minisling repair of cardinal/uterosacral ligaments was reported,[1] except that cure occurred by using the diagnostic and surgical protocols of another paradigm, the Posterior Fornix Syndrome,[2] itself part of the Integral Theory of Female Urinary Incontinence.[3] This discovery is potentially transformative, because, if further evidence can be provided that the bladder and pain symptoms which comprise IC according to it definitions, IC, a condition which afflicts 8%–10% of women, may become generally curable by uterosacral ligament (USL) repair, a fairly minimal procedure.[1,4]

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Though only a singular case,[1] the IC cure was extremely well validated. Symptoms consistent with IC were recorded from a validated questionnaire.[2] The diagnosis of Hunner's ulcer was histologically confirmed, with findings of large numbers of mast cells within the muscular layer. Glomerulations were noted on bladder filling. Pre- and post-operative urodynamics showed increased urodynamic bladder capacity, from 300 ml to 700 ml. Repeat cystoscopy following cure showed no evidence of Hunner's ulcer.

PFS was first described in 1993.[2] It comprises symptoms of chronic pelvic pain (CPP), frequency, nocturia, urgency, abnormal emptying/retention, caused by USL laxity, and cured or improved by USL repair, either native USL plication,[2] or posterior sling.[1,4]

The authors,[1] did not set out to cure IC. They followed the standard protocol for diagnosis and management of the PFS. They concluded that IC may be a manifestation (or phenotype) of PFS.[1]

IC, renamed "Bladder Pain Syndrome" (BPS) by ESSIC, the International Society for the Study of BPS, is a debilitating condition, said to occur in up to 8%–10% of women of all ages.

The current International Continence Society (ICS) definition for IC/BPS[5] is *"persistent or recurrent CPP, pressure, or discomfort perceived to be related to the urinary bladder, accompanied by at least one other urinary symptom such as an urgent need to void or urinary frequency diagnosed in the absence of any identifiable pathology which could explain these symptoms."* Hunner's lesion, and glomerulations, previously hallmarks of IC, are no longer considered essential for diagnosis.[5]

Even at ESSIC, there is considerable debate about the very concept of IC. In 2018, Jane Meijink,[6] president of the Interstitial Cystitis society ESSIC stated *"With all patients being bundled together as one 'syndrome,' with no subtyping, study data have been unreliable and even meaningless. This is further exacerbated by the fact that there is still no international consensus on nomenclature, terminology, and definitions nor on diagnostic criteria. This is damaging for research, data sharing, and comparing and ultimately for the patient and treatment since the evidence from drug studies is erratic and inconsistent."*

Butrick,[7] in his classical paper of IC manifestations in accord with ICS definitions, sounded a note of caution as regards the broadening of the definition of IC, as it may have led to reports of higher IC prevalence. Butrick commented,[7] *"Some authors reported the prevalence to be as high as 26%. It is clear that BPS/IC represents a spectrum of bladder and pelvic pain disorders that are quite heterogeneous, yet many clinicians consider this higher symptom-based prevalence to be excessive and worry that the new diagnosis is overly inclusive. Patients who are ultimately diagnosed to have PBS/IC may present with various chief complaints such as recurrent bladder infections, dyspareunia, urinary frequency, or CPP. A bladder component of pain is found in 38%–85% of women with CPP. Patients with BPS/IC often have more than one pain generator and can present with chief complaints not related to the bladder at all. They often report multiple pain syndromes that involve not only the pelvis but also other parts"*

Butrick[7] described 408 patients based on ICS definitions,[6] who presented to a referral urogynecologic pain centre with varied pelvic floor disorders, Table 1, who, after an initial evaluation, were diagnosed as BPS/IC based on ICS symptom-based definitions and diagnostic criteria.[5]

Table 1. Interstitial cystitis (n=408) specific complaints

Bladder installations: improvement; no cure
Bladder pain/interstitial cystitis (n=157)
Chronic pelvic pain (n=98)
Vulvodynia dysfunction (n=70)
Dyspareunia (n=54)
SUI (n=24)
POP (n=21)
Hunner's ulcer (n=18)

SUI: stress urinary incontinence; POP: pelvic organ prolapse; n: Number

Following publication of the IC cure,[1] we extracted raw data, patients who had CPP, from a previous surgical study which used the TFS tensioned mini-sling to cure pelvic organ prolapse. Our aim was to answer the research question, are PFS and IC similar conditions? If they are, patients with IC *who have a positive speculum test* are at least theoretically, potentially curable by USL repair?'

MATERIALS AND METHODS

A retrospective study. We examined data from 46 women who had CPP and USL repair using the TFS for prolapse repair. Surgery was performed by both surgeons PP and PR either at Royal Perth Hospital or Bentley Hospital Perth, WA between 2004 and 2009.

Patient characteristics: the median age of the women was 66 years (range: 35–87) Median parity was 3 (range: 0–8), and median weight was 70 kg (range: 38–117 kg). Of the 46 patients, 18 had only 1st degree prolapse, and 28 had 2nd degree or greater prolapse. The mean number of previous incontinence or vaginal repair operations was 1.6 per patient (range: 0–6 operations). Only 12 patients had not had prior incontinence or vaginal surgery; 25 patients (50.4%) had undergone prior hysterectomy.

Pre-operative assessment

All patients completed a 24-hour urinary diary, and a self-administered Integral Theory System Questionnaire (ITSQ),[8] which was completed at home in the patient's own time. Pre-operatively, a speculum test, Figure 1, and 24-hour pad test were also administered. Symptoms derived from the questionnaire were ticked off in a diagnostic algorithm, Figure 2. The algorithm visually condenses the diagnosis from the ITSQ as regards ligament and fascial damage to three zones of the vagina, anterior, middle or posterior. In this classification. anterior zone extends from external urethral meatus to bladder neck, middle zone from bladder neck to cervix or hysterectomy scar, posterior zone from apex to perineal body.

Inclusion/exclusion criteria

Patients who had stress urinary incontinence (SUI) were excluded from the study.

Inclusion criterion for surgery were patients with typical PFS symptoms as per the red rectangle, Figure 2, symptoms of urgency, frequency, nocturia, abnormal emptying, urinary retention, pelvic pain, and who, like Scheffler's study, had a positive speculum test for urge or pain.

Objective assessment

Using the symptom-based algorithm as a guide, the three zones of the vagina were examined for evidence of ligament or fascial damage; anterior zone: SUI; middle zone, evidence of high cystocele (cardinal ligament damage) central cystocele, perineal body, posterior vaginal wall (rectovaginal fascia), and for the degree of prolapse of the vaginal vault or uterus. (USL damage). Transperineal ultrasound was performed with a Toshiba 3.5 Mhz curvilinear probe to check organ position at rest and straining and to confirm clinical diagnosis of prolapse. Urodynamic testing was performed with Gaeltec microtransducers to assess for maximal urethral closure pressure, flow, residual urine, and "overactive bladder". The patients were monitored at 6 weeks, and at 3 monthly intervals thereafter using a 24-hour urinary diary, structured self-administered questionnaire, cough stress test, 24-hour pad test, transperineal ultrasound, and urodynamic investigation. Residual urine was assessed by catheter after the patient had voided.

Statistical analysis

We applied the McNemar x^2-tests to test for significance changes in the symptoms' incidence-frequency from baseline (preoperative) to the postoperative phase. For each symptom the null hypothesis H_0: P(baseline) = P(12 months after surgery) versus H_1: P(baseline) =P(12months after surgery) was tested, with P indicating prevalence or incidence rate. An $\alpha = 0.05$ was accepted as the nominal level of significance. Because of multiple testing the p-values of the tests were compared to a Bonferroni corrected α (say α^*) for keeping the type I error less or equal to 0.05.

Ethics

This is a retrospective study of previous surgical data. There are no Ethics Committee issues.

Surgical technique

The posterior TFS sling consists of two polypropylene soft tissue anchors through which is inserted an adjustable polypropylene tape. A transverse full thickness vaginal incision was made 1 cm below the hysterectomy scar, or cervical ring. The uterosacral ligament was identified by Allis forceps. A 4-5 cm channel was dissected immediately lateral to the uterosacral ligaments. Using a special applicator, the TFS anchors were inserted into the uterosacral ligaments. The tape was tightened via a one-way system at the anchor base, and this reduced the apical/ uterine prolapse to a normal anatomical position.

The criteria for symptomatic improvement: nocturia: change in patients having >2 episodes per night; abnormal emptying: self-assessed improvement >80%, using a 0–100 Visual Analogue Scale (VAS) expressed as a percentage; urge incontinence: change in the number of times a patient wet per day; frequency: change in patients having >8 episodes per day; pelvic pain and fecal incontinence: self-assessed improvement >80%, also a 0–100 VAS scale; otherwise objective measures such as pad weights and residual urine measured by catheter were used.

RESULTS

Forty-six patients with CPP had 157 urinary symptoms, urge incontinence, frequency, nocturia, abnormal emptying/ retention, Table 2 This equates to three urinary symptoms per pain symptom, fulfilling the ICS diagnosis for IC in all 46 patients in this study. Mean hospital stay was 1.5 days (1–3 days). One patient was lost to follow-up.

Mean post-operative review time was 12 months. The operation results are summarized in Table 2. There was one recurrence of prolapse which needed re-operation.

CPP: of 46 women with CPP, 35 (76%) reported >80% improvement of their pain and 3 (7%), >50% improvement.

Nocturia: there was a reduction from 168 episodes to 61 episodes. Based on ICS definition, less than 2 episodes/night, cure rate was 27/36 (75%).

Table 2. Symptom outcome (n=46)						
		Symptom change with surgery			% cure in brackets	
	Fecal incontinence (n=12)	Frequency >8/day (n=45)	Nocturia >2/night (n=40)	Urge incontinence >2/day (n=33)	Abnormal emptying (n=39)	Pelvic pain (n=46)
%	100	50	75	74	80	76
p	<0.005	0.05	0.005	0.005	0.005	0.005
n: number						

Urge incontinence: the number of women who had urge incontinence episodes reduced from 33 to 9. Cure rate was 74%. The total number of episodes (getting up at night counting even once per night) within these women reduced from 86 to 17.

Frequency: based on ICS definitions, frequency being >8/night, reduced from 27 women to 14 (50%). The total reduction in episodes in the 27 women was from 385 to 125/day.

Fecal incontinence (FI): a total of 12 women had FI. Nine were 100% cured and 3>80% cured.

Severe urge incontinence (UI): was present in 17 women, mean loss 215gm/24-hour pad test (range: 20 gm–644 gm). At 6 weeks review, 10/17 patients said they were >95% cured. Their mean 24-hour urine loss was 6 gm (range: 0–12gm). The results were statistically significant (p=<0.05) (Student's 2 tailed t-test). In the other 7/17 patients from this group with OAB who were not considered cured of their symptoms, their mean 24-hour loss reduced from 320 gm (range: 25–388 gm) to 223 gm (range: 35–720 gm). Of these seven patients, four were cured of nocturia, three of daytime frequency, and six of other symptoms such as fecal incontinence, pelvic pain and abnormal emptying.

Abnormal emptying and residual urine: symptomatically, 23/39 patients reported more than 80% improvement, with another 11 reporting more than 50% improvement. A residual urine >50 ml (50–600 ml) was seen in 29 patients. In this group, the mean reduction of residual urine was from 271 ml preoperatively to 53 ml post-operatively (p=0.005). One patient who self-catheterized four times daily pre-operatively, was restored to normal emptying. Mean emptying time for this group (n=29) decreased from mean 41 seconds (12–130 secs) to 31 seconds (7–130 secs) (p=0.005). There was no significant change in peak flow, (41 ml/sec pre-operatively to 37 ml/sec post-operatively).

Urodynamics: forty-six patients underwent urodynamic testing preoperatively, and 43 postoperatively. Two glomerulations were noted, but no Hunner's ulcers. There were eight cases of "overactive bladder" (OAB) as defined by the International Continence Society associated with significant urine loss. Five of these eight patients considered themselves more than 95% cured, and a sixth more than 80% cured (mean change from 288 gm–17 gm). Two reported no improvement at all.

Prolapse repair: mean post-operative review time was 12 months. The operation results are summarized in Table 2. There was one recurrence of prolapse which required reoperation.

DISCUSSION

Forty-six patients with CPP had 157 urinary symptoms, urge incontinence, frequency, nocturia, abnormal emptying/retention, all PFS symptoms, Table 2. This result equates to three bladder symptoms per pain symptom and therefore, consistent with the ICS definition for IC.[5] Our pain data comprises many different phenotypes of CPP, including vulvodynia, paraurethral pain, and those specified in the 1996 paper attributing CPP to originating from visceral plexuses,[9] lower abdominal pain, contact dyspareunia, coccygeal pain, paraurethral pain. Whereas it is possible to attribute symptom cure in Table 2 as a direct result of ligament reinforcement by a TFS USL sling, the pathway for symptom occurrence in Tables 1 and 2, needs to be elucidated more scientifically, in order to prove that IC as defined,[5] and PFS[2] are one and the same condition.

Normal bladder function for retention, evacuation and urge control[3,10-19]According to Petros and Ulmsten[3] and Petros and Bush[10-19], the key to understanding dysfunctions of closure (stress incontinence), urge control for OAB and emptying dysfunction known as "retention", "underactive bladder" (UAB), is the interaction of three the reflex opposite directional forces and the ligaments pubourethral (PUL) and uterosacral (USL) which they contract against. Note the opposite muscle force action of the reflex muscles in Figure 3 and Video 1: US three directional forces "https://www.youtube.com/watch?v=3vJx2OvUYe0"

During micturition, m. pubococcygeus (PCM) relaxes (broken circle, Figure 3). Levator plate (LP) and conjoint longitudinal muscle of the anus (LMA) contract against USL to actively open out the urethra (broken white lines).[10-19] This action exponentially reduces the urethral resistance to flow inversely by the 4th power of the radius (Poiseuille's Law), Figures 4 and 5.

Video 2 micturition shows active opening of the posterior

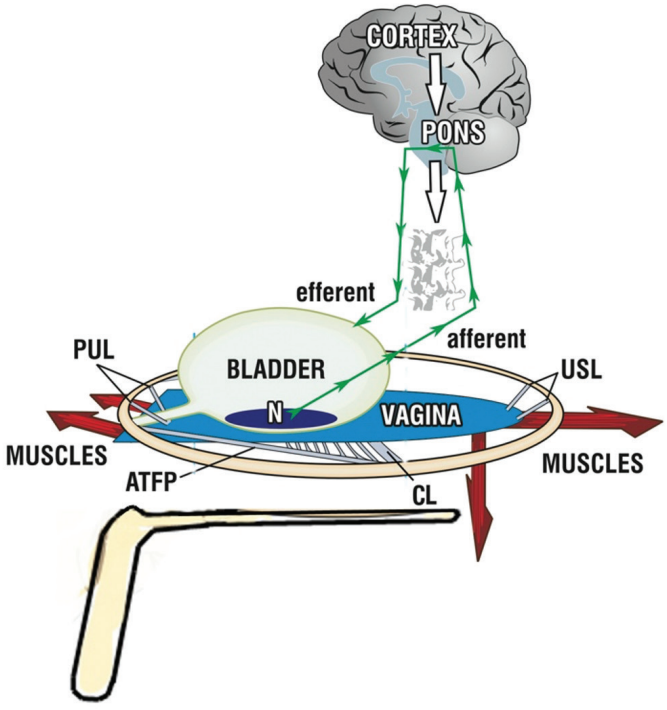

Figure 1. Speculum test. This is a 3D view of the bladder sitting on the anterior vaginal wall. The vagina is suspended from the pelvic brim by ligaments, pubourethral (PUL), cardinal (CL) and uterosacral USL. A speculum inserted into the posterior fornix mechanically supports lax USLs and the nerve plexuses S2-4, T11-L2 supported by USLs. Firm USLs restore the contractile strength of the posterior muscle forces (arrows) which contract against them. These now tension the vagina like a trampoline to support the stretch receptors "N", decreasing the afferent impulses which on passing a critical mass, are interpreted as urgency.
USL: uterosacral

urethral wall by the posterior muscle forces. Note how the anterior lip of LP is pulled down to open the posterior urethral wall. Micturition Video 2 "

https://www.youtube.com/watch?v=eiF4G1mk6EA&feature=youtu.be"

If USLs are weak, LP/LMA contractile force weakens, the posterior urethral wall is not optimally opened out, and the detrusor has to contract against a partially unopened urethra. The detrusor may not empty adequately (retention). The patient experiences this as "obstructed micturition", inability to evacuate, "stopping and starting" etc.

In this next section, specific data from Butrick's[7] classical IC study are compared to data from this work. The pathogenesis detailed is based on original scientific studies.

Urinary retention

Butrick[7] Urinary retention and/or voiding dysfunction was reported by 70% of his cohort, and urodynamics demonstrated

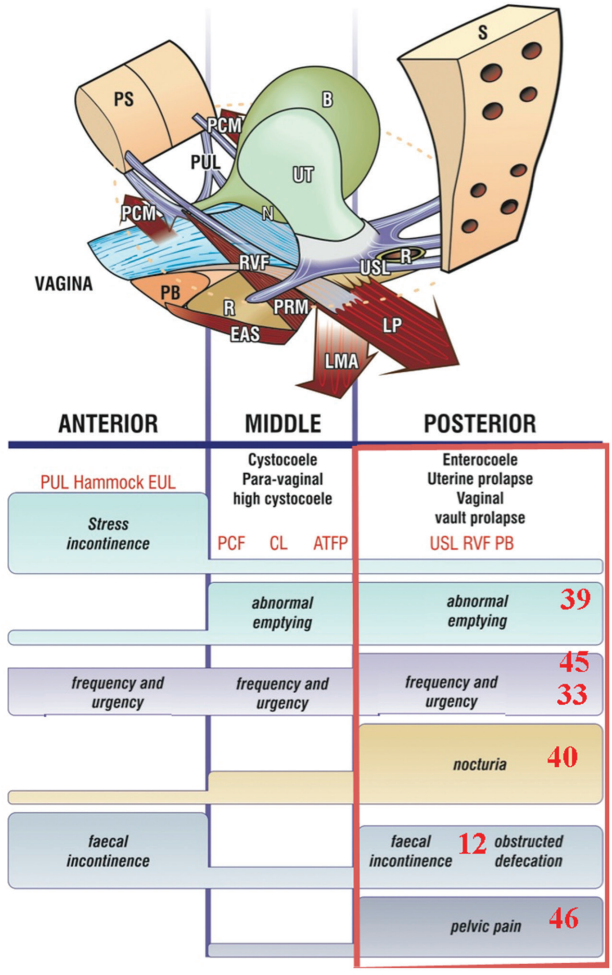

Figure 2. The pictorial diagnostic algorithm. Symptoms indicate which ligaments are damaged
The numbers in red indicate the number of symptoms co-occurring with the 46 symptoms of chronic pelvic pain to confirm the diagnosis of USL damage. The three reflex muscle forces (arrows) tension the organs and the vagina to support urothelial stretch receptors "N".
The height of the bar indicates probability of association of a symptom with a particular zone. The connective tissue structures causing prolapse and pelvic symptoms fall naturally into three zones.
Anterior zone: external meatus to bladder neck PUL: pubourethral ligament; hammock: suburethral vagina; EUL: external urethral ligament
Middle zone: bladder neck to anterior cervical ring. CL: cardinal ligament; PCF: pubocervical fascia; ATFP: arcus tendinous fascia pelvis.
Posterior zone posterior cervical ring to perineal body (PB): USL uterosacral ligaments; RVF: rectovaginal fascia; PB. The rectangle indicates the symptoms associated with USL laxity and the posterior fornix syndrome.
Chronic pelvic pain and nocturia are uniquely caused by uterosacral (USL) ligament laxity.

voiding dysfunction in 80%. Butrick[7] also reported women with recurrent urinary tract infections, a known consequence of inability to empty.

This study, Table 1 Following USL repair, in women with raised residual urine (>50 ml), there was a mean reduction from 271 ml to 53 ml (p=0.005). One patient who self-catheterized 4 times

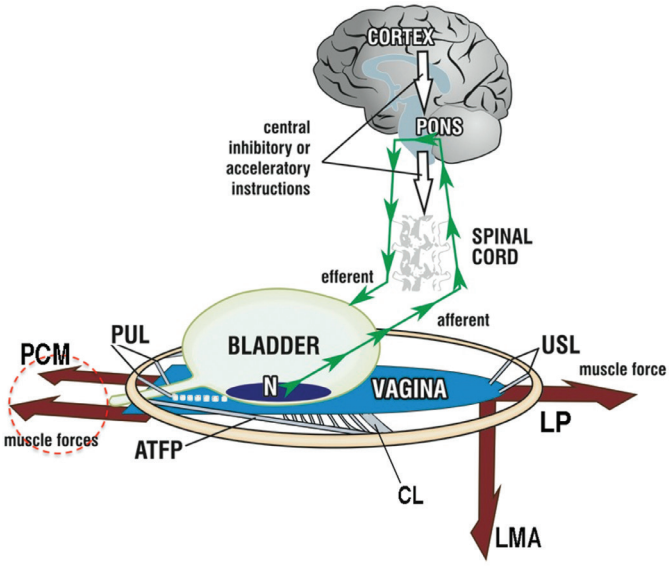

Figure 3. Control of bladder is binary. Schematic 3D sagittal view. System in normal closed mode.

Cortical control: In a woman with no bladder dysfunction, the afferent impulses from the stimulated stretch receptors 'N' are reflexly suppressed cortically (white arrows). When the bladder requires to evacuate, the cortex activates the micturition reflex.

Peripheral control is by a cortically controlled musculo-elastic mechanism which responds to cortical efferents output (small arrows).

The three directional muscles (large arrows), forward, pubococcygeus muscle "PCM ", backward, levator plate" LP", and downward, conjoint longitudinal muscle of the anus "LMA" contract against the supporting ligaments, PUL (pubourethral) and USL (uterosacral), to stretch vagina tightly, much like the membrane of a drum. The stretched vagina supports the pressure of the urine column. This prevents activation of the stretch receptors "N", thereby decreasing afferent impulses to the cortex.

Micturition: When convenient to empty, central control (white arrows) relaxes, as does the forward contractile force of PCM (broken circle); this relaxation allows the posterior muscles LP and LMA to unrestrictedly open out the posterior wall of urethra (white broken lines) immediately prior to bladder evacuation by global detrusor muscle contraction (actually spasm- see micturition video). CX: cervix; CL: cardinal ligament; ATFP: arcus tendineus fascia pelvis.

Dysfunction If there is weakness in the reflex muscles PCM, LP, LMA and/or the ligaments they contract against, PUL, USL, the peripheral control mechanism cannot adequately close the urethra (incontinence), open it to empty (obstructed micturition) or control micturition by bilateral stretching of vagina by the three opposite muscle forces to support "N" (urge incontinence).

daily pre-operatively, was restored to normal emptying. Mean emptying time (n=29) decreased from mean 41 seconds (12–130 secs) to 31 seconds (7-130 secs) (p=0.005).

Cure of retention: The TFS (or other) tape irritates the tissues to create new collagen to repair the damaged USL [20–30], Table 2, restore the contractile force of LP/LMA, to open the urethra and restore urine flow.

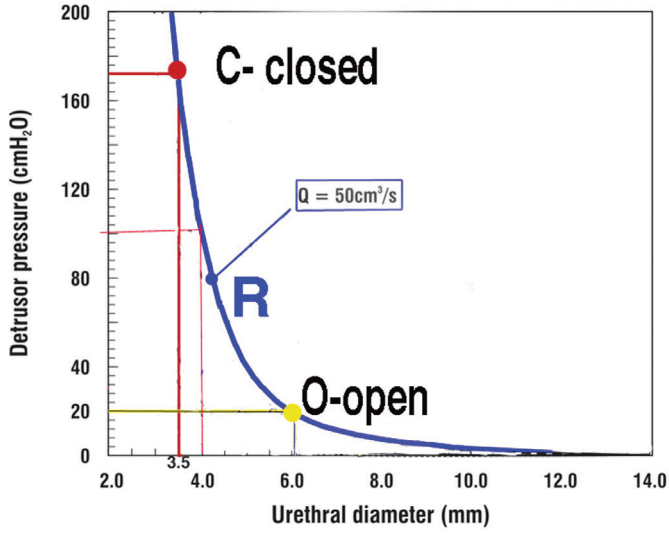

Figure 4. Urine flow is inversely and exponentially related to urethral radius. For a flow rate of 50 ml/sec (thick blue line), opening the urethral diameter from 3.5 mm to 4 mm reduces the head of pressure required by the detrusor to expel urine from the bladder from 172 to 100 cm water. Expanding to 6 mm (yellow lines), reduces the head of pressure to 20 cm water. The blue line represents the total urethral resistance to flow, which is composed of dynamic and frictional flow components.

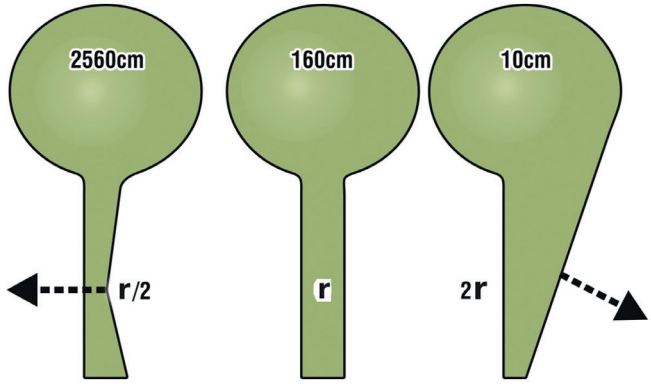

Figure 5. Effect of the external striated muscle mechanism (arrows) on urethral diameter during closure (r/2 left) and opening (2r right) exponentially alters urethral resistance and therefore head of pressure required for the detrusor to drive the urine through the changed diameter. Pressures indicated are based on a nominal pressure of 160 cm for radius 'r'; calculated pressure apply the 4th power law of Poiseuille. Halving the radius (r/2) increases the expulsion force required by a factor of 16, to 2,560 cm H_2O. Doubling the radius (2r), decreases that force by a factor of 16 to 10 cm H_2O.

Retention If we take the front arrow to represent overcompensation by the distal urethral closure mechanism in IC or PFS patients who have loose USLs, very little extra activity is required to close the urethra sufficiently to make it impassable to urine flow (retention). After Petros PE, The Female Pelvic Floor, 3rd Ed Springer, 2010.

Pathogenesis of urge and nocturia ("OAB")[9-30] with reference to Figure 3, Weakness in the muscles PCM, LP, LMA and/

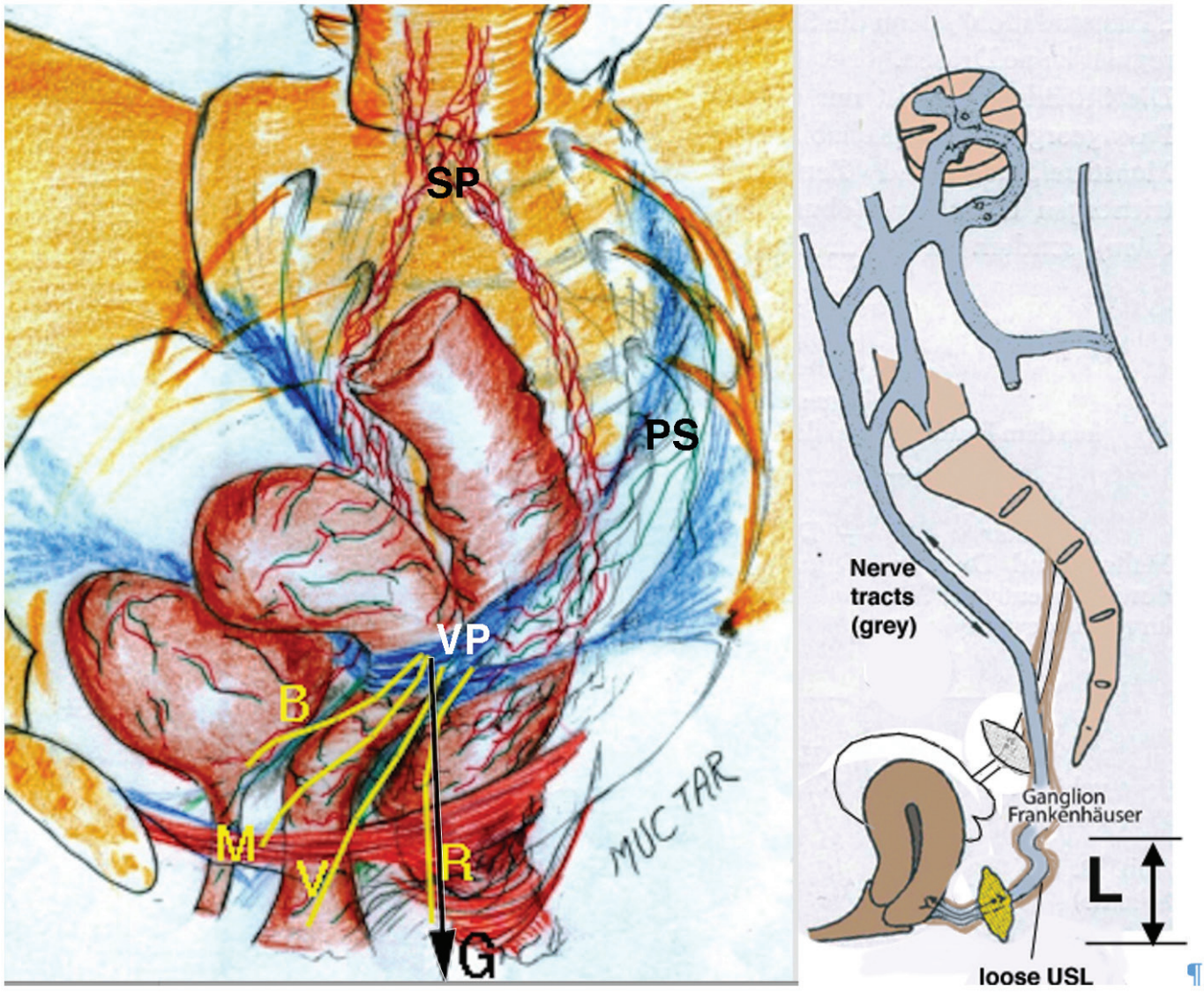

Figure 6. Pathogenesis of chronic pelvic pain from unsupported visceral plexuses
Left figure: Visceral plexus (VP) with components SP (sympathetic T11-L2) and parasympathetic (S2-4). Endorgan afferent nerves M (muscles), B (bladder), V (vagina), R (rectum) travel to VP which serves as a type of relay junction. G signifies forces of gravity acting on these nerves in the upright position.
Right figure: Ganglion Frankenhauser (sympathetic) supported by USLs (uterosacral ligaments). Laxity 'L' in USLs means they no longer can support VPs, so they can be stimulated by 'G'. Arrows upwards: afferent impulses from VP to brain. Right figure by permission, Muctar. S. Left figure after Martius H.
Video 1. https://www.youtube.com/watch?v=3vJx2OvUYe0
Video 2. https://www.youtube.com/watch?v=eiF4G1mk6EA&feature=youtu.be

or the ligaments they contract against, PUL, USL, will affect the ability of the three opposite muscle forces to support the stretch receptors "N" from below. These send increased afferent impulses to the micturition centre. If the impulses cannot be suppressed cortically, the micturition reflex may be activated. The cortex perceives this as urge incontinence. If this occurs at night, the patient may be woken with a need to empty her bladder. Another characteristic of "OAB" is a smaller bladder capacity. After USL repair, Scheffler increased bladder capacity from 300 ml to 700 ml.

Cure of OAB/nocturia: The TFS (or other) tape irritates the tissues to create new collagen to repair the damaged USL,[18-30] Table 2, restore the contractile force of LP/LMA, restore stretching of the vagina to support stretch receptors "N" from below, to decrease afferent impulses to the micturition centre.

Pathogenesis of chronic pelvic pain, Figure 6 The hypothesis of lax USLs causing CPP was first described in the German literature in 1938 by Heinrich Martius[31] and in the English literature by Peter Petros[9] in 1996.

With reference to Figure 6 (left) the visceral plexus (VP) comprises component nerves SP (sympathetic T11-L2) and parasympathetic

(S2-4). VPs are mechanically supported by USLs at their lower end. The yellow lines represent the afferent and efferent visceral nerves from the endogans, muscles, bladder, vagina, rectum. The afferent nerves proceed to the visceral plexus "VP", fig6, which is best regarded as a type of nerve junction. If USLs are loose, the force of gravity of muscle movements may stimulate afferent axons from specific end organs, often more than one at a time. (This explains co-occurrence of several sites of CPP). The afferents pass up to the brain which (falsely) interprets them as an injury (pain) coming from the particular end organ(s). As well as pain, in both vulvodynia and IC, actual inflammatory cells may be seen at the end organs themselves, mast cells, T cells etc. This can be explained by the brain sending efferent signals via the visceral nerves to the end organ, to activate resident "sleeper cells" to produce mast and other inflammatory cells, even the neuroproliferations sometimes seen on biopsy in vulvodynia and in Hunner's ulcer. One could postulate that an ulcerated Hunner's ulcer could be an extreme manifestation of this inflammatory reaction perhaps aided by ischaemia from cutting off low pressure venous drainage by a full bladder descending as a cyctocele. The latter was the explanation accorded for glomerulations.[1] Scheffler cured both Hunner's ulcer, and glomerulations with TFS sling ligament repair.[1]

Cure CPP: With reference to Figure 6, the tape creates mechanical support which prevents stimulation of the nerves within "VP", Table 2. The speculum test works by mechanically supporting USLs. This support temporarily restores the structural integrity of USL to decrease "pain" afferents and likewise "urge" afferents by supporting the urothelial receptors "N", Figure 3.

Muscle spasm and/or pain

With reference to Figure 3, the forward and backward muscle forces are balanced and equal. If USLs are weak, the striated muscles which contract against them also weaken. The system becomes unbalanced. The forward muscles forces (m. pubococcygeus) may over-react to close the urethra excessively. This explains the high urethral pressure described by Butrick[7] More importantly, the excessive contraction may cause pain and spasm to explain the "myofascial pain and hypertonic pelvic floor dysfunction with BPS/IC, well, over 70%." described by Butrick[7]. However, Wu et al.[32] reported immediate improvement in paraurethral pain with the speculum test and the first author (P.P.) after successful posterior sling surgery. At this stage such spasm and pain relief remains a hypothesis to be further tested.

CONCLUSION

We were to show that, at least as regards the ICS definition, IC and PFS are substantially one and the same condition. The descriptions of Butrick regarding the different manifestations of pain and bladder dysfunctions, bear a striking similarity to the PFS as originally described[2] is part of the 1993 Integral Theory.[3] Direct proof of CPP, OAB, nocturia, retention, fecal incontinence cure by a posterior sling to reinforce USL as described, Table 2, must be accepted as proof of USL causation. We were largely able to explain the pathogenesis of these conditions by reference to several of the basic science discoveries of the Theory, [3,33] and the basis for ligament surgery, creation of an artificial collagenous ligament to explain dysfunction and return of function.[34]

Contributions

Surgery and data: P.P., P.R., Concept: P.P., P.R., Analysis of data: P.P., Writing: P.P., P.R.

Ethics

Ethics Committee Approval: This is a retrospective study of previous surgical data. There are no ethics committee issues.

Informed Consent: Written consent was obtained from all patients.

Peer-review: Externally peer-reviewed.

DISCLOSURES

Conflict of Interest: No conflict of interest was declared by the authors.

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