

An anatomical pathogenesis of lower urinary tract definitions from the 2002 ICS report symptoms, conditions, syndromes, urodynamics

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Abstract

Aim: To present an anatomical pathogenesis parallel with the 2002 International Continence Society Lower Urinary Tract (LUTS) definitions standardization Report 2002.

Methods: Each LUTS section is discussed using the same numbers as the Report.

Results: *Normal function* Bladder control is binary, with two reflexes alternating, either closure (dominant) or open (micturition), with the same cortical and peripheral components: three directional muscle forces contracting against pubourethral (PUL) and uterosacral (USL) ligaments for closure, two against uterosacral ligaments for micturition. *Dysfunction* OAB symptoms reflect a prematurely activated micturition; PUL/USL weakness prevents muscle forces from controlling afferent urothelial emptying signals. Stress urinary incontinence is a consequence of weak PULs allowing posterior muscle forces to open the urethra during effort. Lax USLs weaken contractile force of the posterior urethral opening vectors, so detrusor has to contract against an unopened urethra. This is experienced as “obstructive micturition.”

Conclusions: Anatomical analysis indicates the ICS definitions are fundamentally sound, except for “OAB” which implies detrusor causation. Minor changes, OAB to “overactivated” bladder allow causation outside of bladder. This construct supports OAB and its component symptoms as a syndrome, as intuited by the Committee, (albeit as a prematurely activated micturition), retains the acronym, explains OAB cure by ligament repair, and incontinence pathogenesis from two post-2002 syndromes

which need an addition to the definitions, Posterior Fornix Syndrome (of which OAB is a component) and Tethered Vagina Syndrome, which is the basis for skin-grafting cure of the 30%–50% of women who continue leaking urine massively after successful obstetric fistula closure.

KEYWORDS

binary control, ICS definitions, ligaments, OAB, prematurely activated micturition

1 | INTRODUCTION

The 2002 Standardization Report from the International Continence Society (ICS)¹ was a milestone paper. It defined as comprehensively as possible, known lower urinary tract symptoms (LUTS), conditions, syndromes, and urodynamic manifestations thereof. As such, it remains an important reference work for LUTS. Its aim was not to discuss causation, but to “enable effective communication by investigators who use urodynamic methods,” and “to restate that symptoms, signs, and conditions are separate categories.” The aim of this study is, where possible, to present anatomical pathogenesis for each 2002 ICS LUTS definition.¹

1.1 | Normal function

As regards the normal state, it is universally accepted that the bladder is a storage container which empties by voiding through the urethra; during voiding, the micturition reflex is activated; bladder funnels to evacuate. We can deduce from this that the bladder control is binary. It has only two modes EITHER closed OR open. The normal individual senses when the bladder is full, but, if it is inconvenient, can delay evacuation, often for some hours. A woman out shopping with some urge problems knows she can cross her legs, squeeze upwards and gain some valuable time while she seeks a toilet. We can deduce from the former that the brain automatically controls the afferent signals of fullness from the bladder and from the latter, that the pelvic muscles have a role in controlling afferent signals of fullness, at least temporarily. The video ultrasound shows how straining (or coughing) reflexly activates three opposite muscle forces, forwards, backward, downwards, as in the model, Figures 1–3. Video 1 <https://www.youtube.com/watch?v=3vJx2OvUYe0>.

Observe in the ultrasound, that when the woman strains, the forward movements reflexly close the distal urethra and simultaneously, the backward/downward movements reflexly close the bladder neck.

Because the muscle forces act in opposite directions, we can deduce that they stretch the vagina as shown in Figure 1.

Figure 1, is based on video X-ray levator myogram and EMG studies^{2,5,6} whose essential elements are summarized in Figure 2: Three opposite pelvic muscle forces (arrows) act reflexly against the pubourethral ligament (PUL) anteriorly and uterosacral ligaments (USL) posteriorly during urethral closure; two posterior muscles (arrows) act against USL to stretch open the posterior urethral wall to funnel it immediately before micturition, Figure 2. Video 2 <https://www.youtube.com/watch?v=eiF4G1mk6EA%26feature=youtu.be>.

1.2 | Dysfunction

With reference to Figure 1, any anatomical damage in any part of the nervous system, brain, spinal cord, (e.g., tumor) afferent or efferent nerves (multiple sclerosis) the muscles, ligaments, or the urothelial stretch receptors “N,” whether cancer or external pressure (e.g., fibroid) or scarring (iatrogenic or obstetric fistula) may interfere with either binary function, closure or opening, to create urinary retention or incontinence.

2 | METHODS

Where possible, LUTS symptoms, conditions, syndromes from the 2002 Report will be examined with reference to Figures 1–8. Actual numbers from Abrams et al.¹ are utilized.

2.1 | Storage symptoms

2.1.1 | OAB (urgency, frequency, and nocturia) and nocturnal enuresis

All three OAB symptoms are different, but related manifestations of an uncontrolled micturition, Figure 1: inability of the peripheral mechanism to stretch the vagina

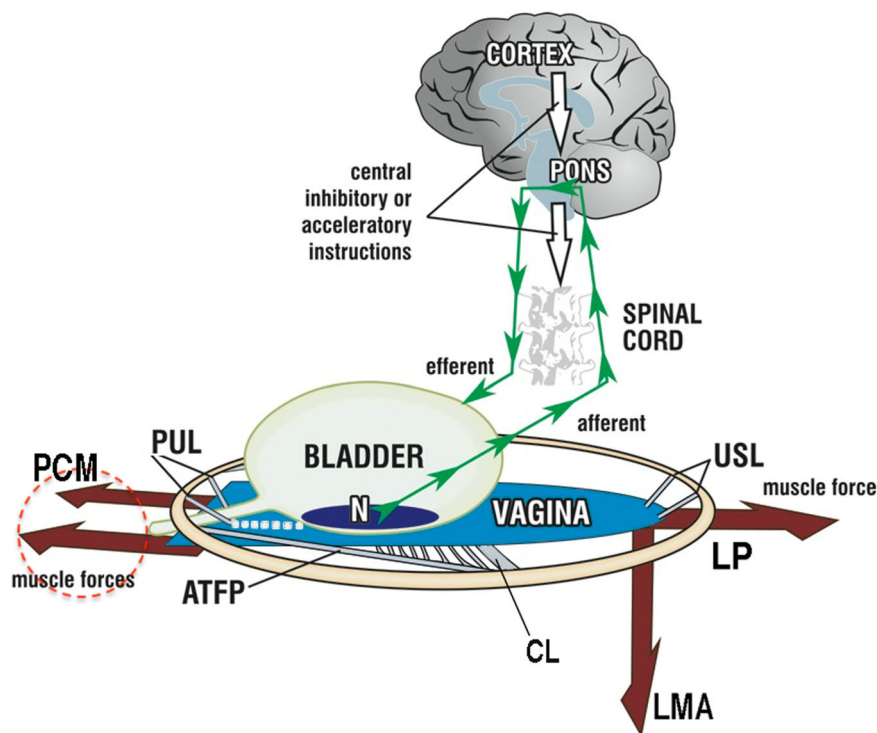


FIGURE 1 Binary model summarizes the anatomical basis of the hypothesis. System in normal closed mode. *Cortical control:* Afferent impulses from stretch receptors “N” are reflexly suppressed cortically (white arrows). When required, the cortex activates the micturition reflex. *Peripheral control* is by a musculo-elastic mechanism which responds to cortical efferents (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 urine column, preventing activation of the stretch receptors “N,” thereby decreasing afferent impulses to the cortex. *Micturition* Central control (white arrows) relaxes, as does PCM (broken circle); this allows the posterior muscles LP and LMA to unrestrictedly open out the posterior wall of urethra (white broken lines) just before bladder evacuation by global detrusor muscle contraction. ATFP, arcus tendineus fascia pelvis; CL, cardinal ligament; CX, cervix. *Dysfunction* weakness in the muscles PCM, LP, LMA, and/or the ligaments they contract against, PUL, USL, will affect the ability of the peripheral control mechanism to mechanically close urethra (incontinence), open it (obstructed micturition), or to suppress uncontrolled micturition by bilateral stretching of vagina by the three opposite muscle forces to support “N” (urge incontinence)

sufficiently to support the urothelial stretch receptors “N,” so excessive afferent impulses (small green arrows) are produced and these proceed to the cortex where they are interpreted as “urge”; “N” fire off at a low bladder volume, so the woman goes to the toilet more frequently (“frequency”). If it happens at night, the urge symptoms wake her to go to the toilet (“nocturia”). If the closure reflex cannot sufficiently control the afferent impulses from “N,” the micturition reflex, Figure 2, is activated, and the woman wets (urinary incontinence); if this happens to a child at night who does not wake up, it is called “nocturnal enuresis.”

2.1.2 | Stress incontinence

With reference to Figure 1, PUL is holding up the anterior vagina and urethra much like a wire strut on a suspension bridge; if PUL is weak, the posterior muscles

LP/LMA will pull open the posterior urethral wall like a trapdoor, (Figure 1, broken white lines). This is better seen in Figure 3. A hemostat at “L” immediately behind the pubic symphysis (PS), Figure 3, prevents elongation of a weak PUL to “L” and SUI is controlled.⁹ Video 3 <https://youtu.be/0UZuJtajCQU>.

2.1.3 | Mixed urinary incontinence

With reference to Figure 1, the vagina requires stretching from both ends to stretch it sufficiently to support “N” from below. If PUL is weak, so too, is the PCM force which contracts against it. At a critical point, “N” may fire off uncontrolled afferent impulses which the cortex interprets as urge. The same hemostat test for SUI, Video 3, will diminish urge in 50% of cases. If so, both SUI and urge may be cured.^{4,9}

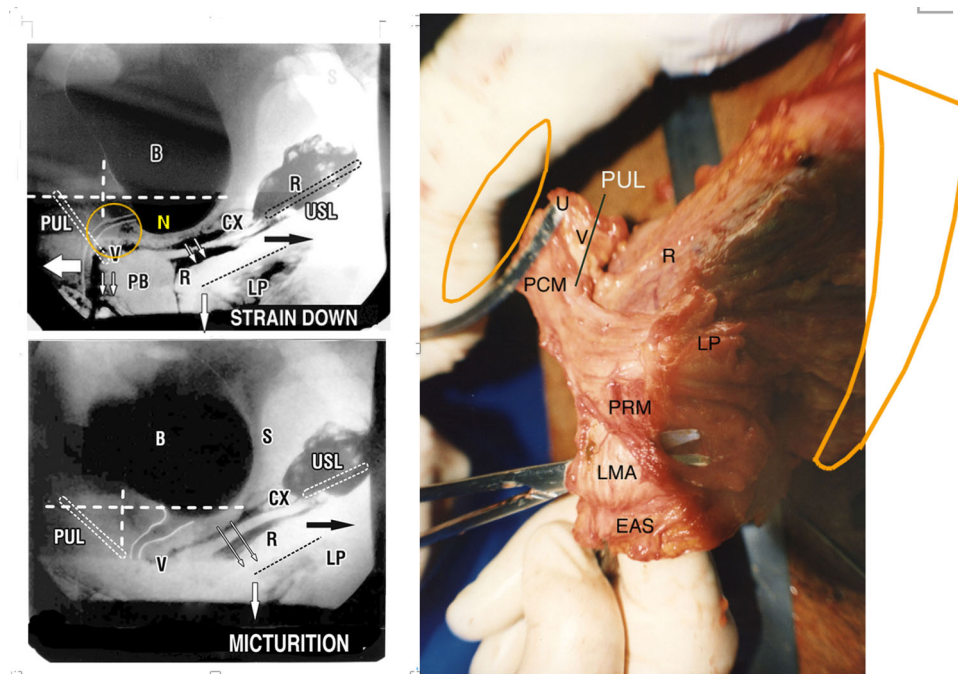


FIGURE 2 Anatomy and mechanics of the urethral closure and micturition reflexes validate. Figure 1. **Right figure:** Anatomical specimen from a female cadaver, cut away from its bony insertions. Bladder and vagina had been excised at the level of bladder neck. U = urethra; V = vagina. PCM = pubococcygeus muscle sweeping behind rectum (R) to merge with the contralateral side to form part of levator plate (LP) which inserts into the posterior wall of rectum. PRM = puborectalis muscle which surrounds rectum; LMA = conjoint longitudinal muscle of the anus which inserts into LP, PCM, and EAS; PUL = insertion of pubourethral ligament into medial side of PCM; EAS = external anal sphincter. **Left upper figure-closure reflex:** Vertical and horizontal broken lines are drawn from bony co-ordinates. NOTE stretching of vagina in opposite directions² by the oppositely-acting muscle vectors (arrows). The now stretched vagina supports stretch receptors “N” from below. *Straining down* Fast twitch directional vector forces stretch the vagina in opposite directions² (arrows), forwards (PCM), backwards (LP), against pubourethral ligament (PUL), and downwards (LMA) against uterosacral ligament (USL). Adequate elasticity is required in the bladder neck area of vagina “Zone of Critical Elasticity” (circle)² so as to allow the opposite muscle forces to operate independently of each other. CX = cervix; U = urethra; LP = levator plate muscle. PCM = pubococcygeus; LMA = conjoint longitudinal muscle of the anus.; v = vagina; R = rectum; PB = perineal body. **Left lower figure- micturition reflex:** Forward muscle (PCM) relaxes.² This allows backward LP muscle forces to stretch the vagina and distal urethra backwards behind vertical line; the downward muscle force (LMA) angulates the tip of LP downwards to open out the posterior urethral wall.² Evident connective tissue attachments between rectum, vagina, and bladder base in the cadaver specimen explain the funneling in the micturition X-ray. This external opening vastly expands the urethral channel to exponentially lower the urethral resistance to bladder evacuation

2.1.4 | Bladder sensations (all)

With reference to Figure 1, we can assume that the sensitivity of the stretch receptors “N” for any individual as regards the production of afferent impulses follows a normal distribution curve, with excess sensitivity at one extreme and little to none at the other. However, the balance of muscle forces stretching vagina to support “N” from below is also important. A recent study examining urinary retention,⁸ found that high preoperative mean bladder volumes (598 ml) in women with UAB symptoms and retention reduced to mean 301 ml following USL repair which cured/improved the emptying symptoms and residual urine.⁸ The anatomical explanation proposed was that the forward (PCM) and

backward (LP/LMA) forces, Figure 1, are balanced. Weak USLs also weaken LP/LMA which contract against them. Under autonomic instructions from the brain, PCM stretches the vagina further forwards, so a higher hydrostatic pressure, Figure 1, is required to activate the urothelial stretch receptors for evacuation to occur. See also Figure 7.

2.2 | Voiding symptoms

The posterior muscle forces, Figure 1, stretch open the posterior urethral wall: micturition Video 2 <https://www.youtube.com/watch?v=eiF4G1mk6EA%26feature=youtu.be>.

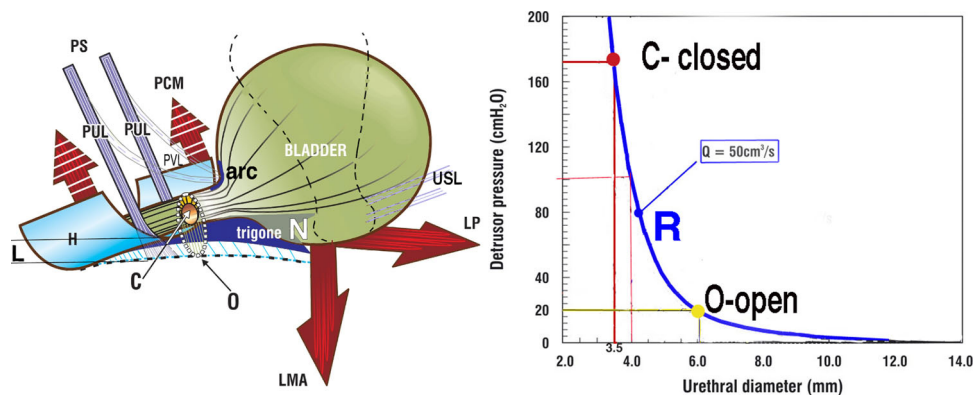


FIGURE 3 Stress urinary continence (SUI) mechanism in the female.² Schematic 3D model. *Left figure:* Three directional forces (VIDEO 1) <https://www.youtube.com/watch?v=3vJx2OvUYe0> close distal urethra and bladder neck: m. pubococcygeus (PCM) stretches the suburethral vaginal hammock “H” against a competent pubourethral ligament (PUL) to close the distal urethra from behind. Levator plate (LP) pulls backward against PUL to stretch the vagina, proximal urethra, trigone, and bladder base backward, to render the tissues semi-rigid.² LP also tensions the pubovesical ligament (PVL) which inserts into the arc of Gilvernet, a smooth thickening on the anterior wall of the bladder.² The role of PVL and arc is to hold steady the anterior bladder wall while the downward vector, conjoint longitudinal muscle of the anus (LMA) contracts against the uterosacral ligaments (USL) to rotate the now semirigid bladder around the arc to close the urethra at bladder neck.² *Right figure* Graphically shows the effect of narrowing the urethra on the resistance “R” to urine flow. This is exponentially determined inversely to the fourth power of the radius.³ For example, if the urethra can be pulled open by LP/LMA from “C” to “O,” say from 3.5 to 8 mm, urine will leak with a generated pressure of 5 cm H₂O. Conversely if this cannot happen because of weak USLs, then at 3.5 mm diameter, 180 cm H₂O is required to empty at 50 ml/sec flow rate. **Incontinence.** On effort, a weak PUL stretches down to “L” so it can no longer support the distal vagina “H” into which it inserts.⁴ LP/LMA instead of closing bladder neck (upper X-ray, Figure 2), actually pull down vagina and trigone. This actively opens out the posterior urethral wall from “C” (closed) to “O” (open exactly) as happens during micturition, (incontinence). **Voiding, obstructed micturition, and urinary retention.** *Left figure* The geometry of SUI is identical to that for micturition, except that during micturition, PCM relaxes (broken lines) and LP/LMA open out the urethra to decrease resistance to urine expulsion. If USL is weak, the opening force LMA also weakens. The posterior urethral wall cannot be opened adequately. The patient experiences symptoms of obstructed micturition. *Right figure* If USL is weak, the opening mechanism driven by LMA also weakens. Resistance “R” at 3.5 mm diameter is very high. A pressure of 180 cm is needed to drive urine through the urethra to empty it

Inability to open the urethra before micturition means that the detrusor must work harder to push urine through “C”, Figure 3, during micturition, resulting in a higher expulsive pressure. Even at a moderate diameter of 3.5 mm, a pressure of 180 cm is required to drive urine out at 50 cm/s velocity. The individual voiding symptoms¹ are clinical manifestations of this explanation.

2.2.1 | Slow stream

The urethra cannot be adequately opened from “C” to “O”, Figure 3, because a loose USL weakens the opening forces, LP/LMA; the detrusor has to push urine out through a narrower tube; the stream is slow.

2.2.2 | Splitting or spraying

Dislocation of one of the pubococcygeus muscles “PCM” Figure 1, unbalances the forces exerted against the symphysis and causes the opposite healthy muscle to pull the urethra towards it, to cause a divergent stream or spraying.³

2.2.3 | Intermittent stream

Whereas EMG during normal voiding subsides, it rises repeatedly in women with an intermittent stream indicating the muscles cannot “grip” sufficiently to open the urethra from “C” to “O”, Figure 3. **Hesitancy** This indicates initial difficulty in the pelvic muscles LP/LMA, Figure 3, externally opening the urethra.

2.2.4 | Straining

Once the urethra has been pulled open and urine is flowing, straining increases abdominal pressure, which helps the detrusor drive out the urine.

2.3 | Feeling of incomplete emptying

Striated muscles LP/LMA Figure 3, tire. That is why women who cannot empty adequately may have to return in 20 min to empty more urine.

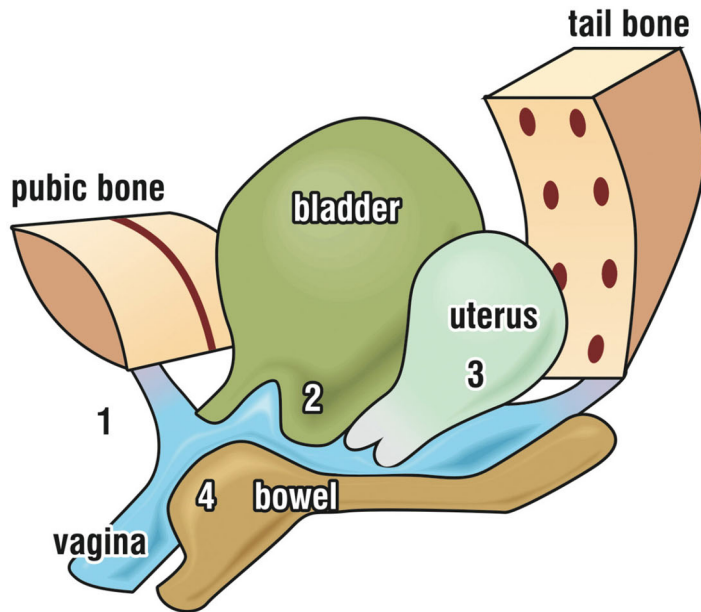


FIGURE 4 Diagnostic algorithm: Symptom prevalence in the posterior zone (rectangle), % cure after posterior sling in brackets.² Symptoms occur in predictable groupings, are associated with uterine/apical prolapse and/or enterocele and have been shown to be cured by uterosacral ligaments (USL) repair Data from Goeschen et al.⁷

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 68 (54)
frequency and urgency	frequency and urgency	frequency urge incont 127 (80) 55 (80)
		nocturia 63 (79)
faecal incontinence		faecal incontinence obstructed 59 defecation (80)
		pelvic pain 198 (74)
	tethered vagina	

2.3.1 | Postmicturition dribble

Striated muscles LP/LMA Figure 3, tire; the urethra begins to close under influence of its elastic tissues, exponentially raising resistance to detrusor emptying; the detrusor can only expel urine at a very slow flow.

2.4 | Incontinence during sexual intercourse

In a live anatomical study, the lateral part of PUL was noted to insert into the lateral part of PCM and directly into the vagina.⁹ With reference to Figure 3, a penis thrusting backward from the introitus may stretch the vagina sufficiently to

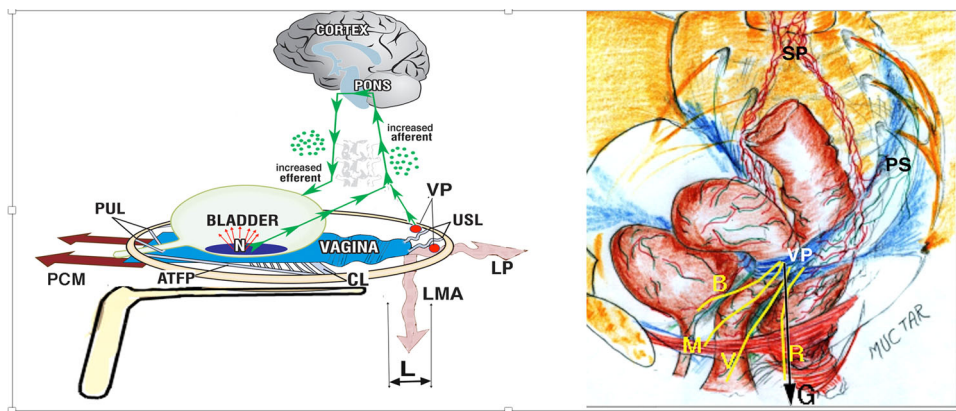


FIGURE 5 Lax USLs may cause chronic pain and urgency. *Left figure Speculum test.* This is a 3D view of the bladder sitting on the anterior vaginal wall. The vagina is suspended from the pelvic brim by ligaments, pubourethral (PUL), cardinal (CL), and uterosacral USL. “L” signifies ligament laxity. The wavy form of the arrows signifies weakened muscle force of the muscles LP (levator plate) and LMA (conjoint longitudinal muscle of the anus) which contract against USL. A speculum inserted into the posterior fornix mechanically supports lax USLs, the nerve plexuses S2–4, T11–L2 and also, the stretch receptors “N” to decrease afferent impulses to the brain. *Right 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 by permission, Muctar. S

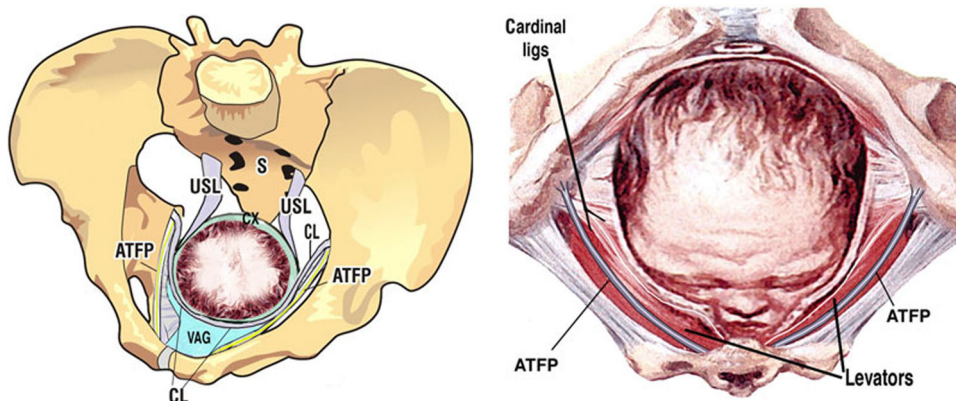


FIGURE 6 Birth damage to ligaments. *Left side* 10 cm dilatation, entry to the birth canal; there is intense pressure on already stretched cardinal (CL) and uterosacral (USL) ligaments which may overstretch the collagen beyond its ability to return to normal length postnatally, thereby causing laxity, prolapse, and bladder/bowel/pain symptoms. *Right side* Pressure of the head on exit from the birth canal may damage the insertion of pubourethral ligaments, levators and arcus tendineus fascia pelvis (ATFP)

pull open the urethra if PUL cannot sustain the backward stretching from the penis, which elongates PUL to “L”; this allows the posterior forces (large arrows, Figure 3) to stretch open the urethra from “C” to “O”. Such cases respond to midurethral sling surgery.

2.5 | Symptoms associated with pelvic organ prolapse

“Backache”, “dragging” is consequential on the uterus being pushed into the vagina by intraabdominal pressure and gravity pulling on USLs which contain myelinated

and unmyelinated nerves.¹⁰ Digitally supporting the perineum prevents diversion of feces into the rectocele during defecation.

2.6 | Genital and lower urinary tract pain

Pain was not defined in the ICS definitions.¹ Neuroscience online¹¹ defines pain as “an unpleasant sensation and emotional experience that links to tissue damage. It allows the body to react and prevent further tissue damage*. People feel pain when a signal

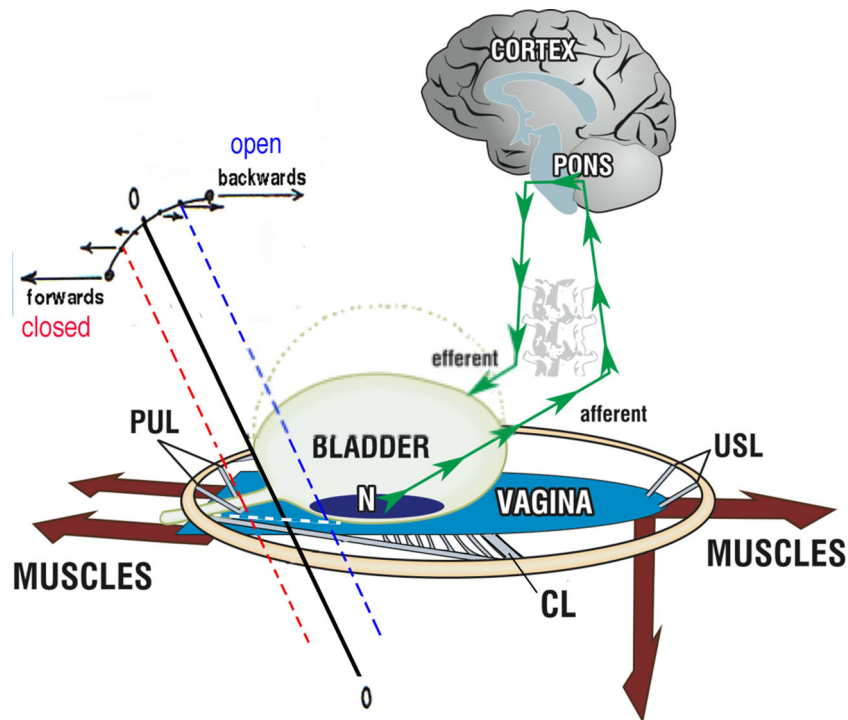


FIGURE 7 3D view of bladder showing opposite muscle forces (large arrows) balanced at rest with zero resultant force 0-0 at bladder neck. Weak pubourethral ligaments (PUL) weaken forward muscle forces, so the stronger backward muscles pull the equilibrium point 0-0 backwards (blue broken lines) to open urethra and cause stress urinary incontinence. Weak uterosacral ligaments (USL) weaken the backward vector forces, so the stronger forward muscles pull the vagina and equilibrium point 0-0 forwards (red broken lines). This may overtension the distal vagina and close the distal urethra more tightly as reported in Fowler's Syndrome⁸ and may create a much firmer support below the urothelial stretch receptors "N". A greater hydrostatic pressure (higher bladder volume) is needed to activate the afferent impulses required to activate the micturition reflex. This explains the greater resting bladder volumes and retention reported in "underactive bladder" and Fowler's Syndrome⁸

travels through nerve fibers to the brain for interpretation."

*This definition explains the inflammatory reaction, mast cells, leukocytes, even neuroproliferation, seen at visceral nerve receptor sites such as, the urothelium (Hunner's lesion) and vaginal pain syndrome (see also Figure 5).

2.7 | Genito-urinary pain syndromes and symptom syndromes suggestive of LUTD

"Syndromes describe constellations or varying combination of symptoms but cannot be used for precise diagnosis"¹***.

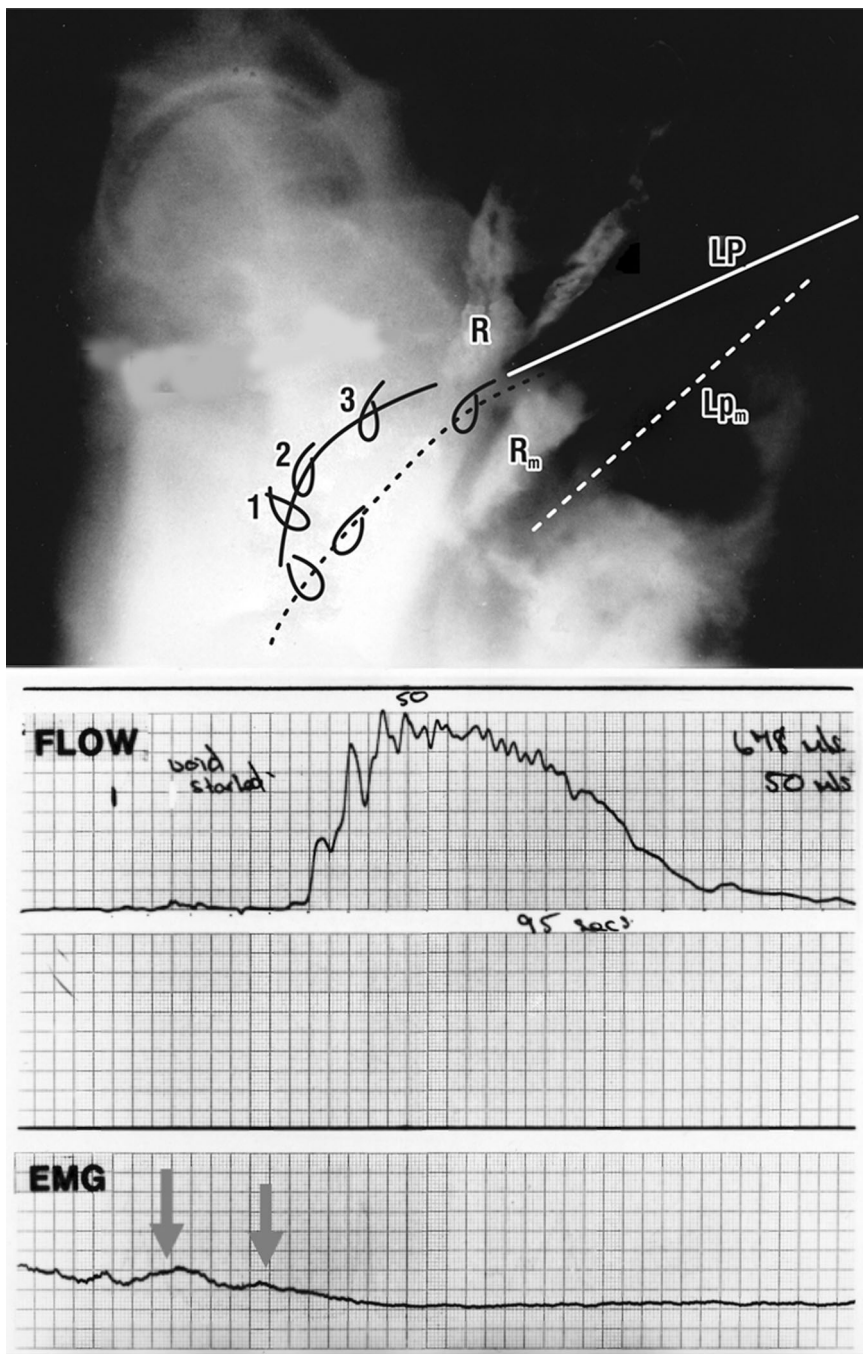
**Data from Wagenlehner,¹² Goeschen⁷ (and many others) who apply the Posterior Fornix Syndrome (PFS), rectangle, Figure 4, contradicts this underlined statement and will be discussed below.

2.7.1 | Genito-urinary Pain Syndromes

"These are all chronic in their nature. Pain is the major complaint, but concomitant complaints are of lower urinary tract, bowel, sexual or gynecological in nature."¹ This description of "pain syndrome" accurately fits the 1993 description of Posterior Fornix Syndrome "PFS"¹³: predictably grouped symptoms of urge, frequency, nocturia, chronic pelvic pain, and abnormal emptying, caused by uterosacral ligament laxity, Figure 4, cured or improved by repair thereof.^{7,10,13}

Cross sensitization¹⁴⁻¹⁷ and neuroplasticity^{18,19} might also explain the co-occurrence of genito-urinary pain syndromes with other symptoms related to chronic pain and dysfunction (e.g., pudendal pain syndrome, pelvic floor pain syndrome, and IBS),²⁰⁻²² and entrapment of peripheral nerves of the lumbosacral plexus.^{23,24}

FIGURE 8 Biomechanics of normal micturition. *Upper figure.* Superimposed X-rays, at rest and micturition. Vascular clips have been applied to 1. midurethra, 2. bladder neck, 3. bladder base. Unbroken lines at rest. Broken lines micturition. LP = levator plate at rest; LP_m at micturition. R = rectum at rest; R_m at micturition. Note backward/downward extension of all three vaginal clips during micturition, validating active muscle action apparently by downward angulation of LP and rectum, to which LP is attached. *Middle figure* FLOW chart (normal). EMG in vaginal fornix denotes pelvic muscle contraction before urine flow starting and ceases once flow is established. *Lower figure* FLOW chart (slow flow). Urethra cannot open. “stopping and starting” Muscle cannot “grip” to open out urethra



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2.7.2 | The specific pain syndromes

The specific pain syndromes, such as bladder, urethral, vulval, and vaginal perineal,¹ (to which can be added low abdominal pain, coccygeal pain, and contact dyspareunia¹⁰) co-occur in the female. With the obvious exception of “Scrotal pain syndrome”, these pain phenotypes have been relieved by the speculum test which mechanically supports USLs and visceral plexuses (VP), Figure 5, or by the Bornstein test²⁵ which injects local anesthetic (LA) into the VPs, 2 cm proximal to USL

insertion to the cervix. The LA relieves the pain for 20 min. Also relieved by the speculum test were para-urethral pain, low abdominal pain, sacral pain and these were 70% cured, at 12 months by native USL repair.¹⁰

The pain, bladder, bowel, and prolapse symptoms comprising the more evolved posterior fornix syndrome (PFS), rectangle, Figure 4, also almost entirely incorporate stated LUTS symptoms (2.7.1).¹ With reference to the listed pain phenotypes “2.7.1,”¹ a 70% cure of CPP at 12 months was achieved in 28 women in multiple sites of CPP following USL plication.¹⁰

It was hypothesized that the cause of the CPP was inability of lax USLs to support the visceral plexuses (VP) T11–L2, S2–4¹⁰: these were stimulated by gravity “G”, Figure 5, to send afferent impulses to the cortex which were interpreted as pain coming from an injury to the end organ.¹⁰ As gravity “G,” Figure 5, does not distinguish between the multiple nerves bunched together in the plexuses, multiple sites of pain were experienced,¹⁰ and simultaneously cured by USL plication.¹⁰

The hypothesis for CPP pathogenesis was proposed 60 years earlier in the German literature by Heinrich Martius.²⁶ Anatomical studies of VP support this hypothesis: in 1996 myelinated and unmyelinated nerves in USLs¹⁰; sympathetic, parasympathetic, nociceptive, and sensory-motor nerves were found in cardinal ligaments and USLs.²⁷ Sympathetic nerve ending density was dominant in the lateral part of uterosacral ligaments, which were considered “a major pathway for autonomic nerves to the pelvic organs.”²⁷

Causation of USL laxity or damage

Distension of USLs at childbirth, Figure 6, surgical cure of PFS symptoms by USL repair in primiparas¹⁰ and aged women^{7,12} give a pathogenesis rationale for congenital, birth and age, the last by collagen breakdown after the menopause.²

Testing the USL hypothesis for pain

The speculum test mechanically supports lax USLs to relieve pain and supports urothelial stretch receptors “N”, Figure 5, to relieve urge, both symptoms, in up to 70%–80% of women.

*Bornstein test*²⁵ Local anesthetic injected into VP relieves pain totally or partly in several sites simultaneously.²⁸

USL repair gives high cure rates for CPP by USL plication in younger women¹⁰ or posterior sling in older women.^{7,12}

OAB syndrome components, urgency, frequency, nocturia have been urodynamically demonstrated to be an uncontrolled, but otherwise normal micturition, Figure 1.²⁹ Grouping urgency, frequency, nocturia into a syndrome “OAB” fits with our concept of OAB as a prematurely activated uncontrolled micturition; this concept holds in all instances, male, female, children, diseases, degenerative, and infection, tumors in any part of the control system, Figure 1. In the female, cure of OAB along with other Posterior Fornix Syndrome symptoms, Figure 4, by surgical operations which repair USL,^{7,10,12,13,30} confirms OAB as an integral part of PFS.

2.8 | Measuring the frequency, severity, and impact of lower urinary tract symptoms

The recurring difficulty with objective measurement of any parameter is the enormous variation of frequency, severity and impact, often day to day. Such variation is a natural consequence of the nonlinear feedback and exponential effect of diameter on urethral resistance,^{31,32} Figure 3. Each phase in the feedback control system, Figure 1, retention, normal, unstable (OAB), can be mathematically expressed by a nonlinear Chaos Theory feedback equation $X_{NEXT} = cX(1 - X)^{33}$ where X = fraction of possible nerve impulses emanating from the stretch receptors “N” in the micturition circuit, Figure 1. Numeral “1” = maximum possible number of impulses in the circuit “c”, represents the closure reflex’s inhibition of micturition, central, and peripheral.³³

3 | URODYNAMIC OBSERVATIONS AND CONDITIONS¹

Comment: The original objective of urodynamics in the First ICS Report in 1976 was to objectively reproduce the conditions which cause the urge incontinence component of “OAB.” The criterion in 1976 was a 15 cm pressure rise during filling cystometry. This was abandoned in 1988, with a “wavy” pattern now sufficient for diagnosis. Pitted against objective testing for OAB were two problems. (1) There is *invariably* wide variation in all component OAB symptoms. (2) The brain accurately records, orders and averages urge symptoms over a long period, while urodynamic testing occurs in a fraction of that period, 30 min. This explains how, even with provocative measures, the yield is only 50% for urodynamic testing against a patient’s testimony.

3.1 | Ambulatory urodynamics

It measure normal filling pressure. The finding of 61% patterns of detrusor overactivity (“DO”) in women with no urge symptoms support earlier analysis in this study that “DO” is a urodynamic expression of an activated micturition reflex.²⁹

3.2 | Filling cystometry

Rapid bladder filling stimulates “N,” Figure 1; “N” sends afferent impulses which activate the closure (or

“guarding”) reflex cortically (white arrows) and peripherally. Action of rhabdosphincter and forward contraction of PCM may combine to increase urethral pressure.

3.2.1 | Bladder sensations during filling cystometry

Bladder sensations during filling cystometry are a consequence of cortical interpretation of afferent impulses from “N,” which are driven by hydrostatic pressure on “N,” Figure 1.

3.2.2 | Phasic detrusor overactivity

Phasic detrusor overactivity according to the binary control, Figure 1, indicates a struggle for control between the closure reflex (high-pressure point) and the opening reflex, micturition, low-pressure point) with the wave period a consequence of the time delay in switching from “closed” mode to “open.”

Terminal detrusor overactivity

During evacuation, the micromotions from bladder smooth muscles coalesce to “contract as one,”³⁴ evident on observing the micturition Video 2 <https://www.youtube.com/watch?v=eiF4G1mk6EA%26feature=youtu.be>.

Provocative maneuvers

Local (e.g., cold water) stimulate “N,” Figure 1, while a tap or handwashing interrupt the cortical/spinal autonomic suppression; in cases with weak peripheral control, the micturition reflex may temporarily dominate; this is perceived as “urgency,” or, if the closure mechanism cannot regain control, the micturition reflex goes on to detrusor contraction and the patient wets.

3.2.3 | Bladder compliance during filling cystometry

Bladder scarring from radiation damage would inhibit detrusor distension. However, most low compliance arises from an activated but controlled micturition reflex.³³ During bladder filling, afferent impulses from the stretch receptors “N,” Figure 1, pass to the cortex. *If they cannot be fully suppressed cortically*, the peripheral part of the closure reflex is activated; the urethra is closed; micromotion segments of detrusor smooth muscle fibers coalesce to “contract as one”³⁴ against a urethra which remains closed; not sufficiently to evacuate the bladder, but sufficiently for detrusor pressure rises in a low compliance pattern³³; if the closure reflex is

sufficiently strong to retain closure, and the efferent impulses from the brain sufficient to inhibit micromotion coalescence, the patient does not wet. Release of cortical inhibition by say handwashing,³³ completes the micturition cycle and urine is lost.³³

3.2.4 | Cystometric capacity

As the bladder fills, hydrostatic pressure on the urothelial stretch receptors “N,” Figure 1, activates afferent impulses. The capacity assessed will depend on how successfully the micturition reflex can be suppressed by the closure reflex.

Low cystometric capacity

Weak PUL or USL, Figure 1, weaken muscle forces (arrows) which stretch the vagina to support the urothelial stretch receptors “N”; urge to empty occurs at a low bladder volume.

High cystometric capacity

Neurological damage, for example, a transected spinal cord, may prevent activation of micturition; efferent nerve lesions (multiple sclerosis) may prevent the impulses reaching the detrusor fibers to activate them. Weak USLs, Figure 7, may weaken the posterior forces; the equilibrium point of the opposing forces moves forwards from 0 to 0 (bladder neck) to the red broken line, Figure 7. Excess forward stretching may overtighten vaginal support of the stretch receptors “N.” A higher bladder volume is required to activate the micturition reflex for evacuation, as noted in urinary retention, “underactive bladder” and Fowler’s Syndrome retention.⁸

3.2.5 | Incompetent urethral closure mechanism

A weak PUL, Figure 3, allows the posterior muscle forces to forcibly pull open the posterior wall of urethra from “C” (closure) to “O” (open) on effort, to cause stress urinary incontinence (SUI).

Urethral relaxation incontinence¹ (leakage in the absence of a detrusor contraction)

Tanagho³⁵ urodynamically demonstrated what he called “urethral relaxation”*** (fall in urethral pressure), followed by detrusor contraction and urine evacuation. However, EMGs, Figure 8, show pelvic contraction prior to micturition, while X-ray Video 2 <https://www.youtube.com/watch?v=eiF4G1mk6EA%26feature=youtu.be> shows pelvic muscles actively opening out the urethra, as per Figure 2.

Note the depression of the anterior lip of levator plate which immediately precedes urine flow. On the pressure/diameter graph, Figure 3, beyond 8 mm diameter, the intraurethral resistance to flow is reduced sufficiently so urine flows with very little, even no, detrusor pressure. This is in accord with Griffiths' studies which showed that at a certain diameter, all the detrusor force is converted to flow to expel the urine.³⁶

***The "relaxation"³⁵ is actually in the striated closure muscle, PCM, which allows the posterior muscles to open out urethra from "C" closed to "O" open, vastly increasing urethral area, Figure 3 with consequent fall in pressure "P", as "P" = force/area.

Urodynamic stress incontinence

This is explained in detail in Figure 3.

3.2.6 | Urethral pressure

This is generated by the striated periurethral (rhabdosphincter) and pelvic floor muscles, the smooth muscle of the urethral wall, the fibroelastic tissues and submucosal vascular plexus³⁷; rise in urethral pressure on stress, however, is mainly the province of the striated periurethral muscle.

Maximal urethral pressure

This is at midurethra, the site of the thickest part of the rhabdosphincter.

Abdominal leak point pressure mechanism

The abdominal and pelvic muscles arise from the same myotome embryologically.³⁸ Straining reflexly contracts the three directional muscles.² With reference to Figure 3, a weak PUL allows the two posterior forces (backward/downward arrows) to pull open the posterior wall of urethra during straining, enlarging urethral diameter, reducing the internal resistance to flow. Abdominal leak point pressure is the pressure in the graph relative to the diameter opened out by straining, Figure 3, that needed to overcome the resistance to flow.

Detrusor leak point pressure

This is the point, Figure 3, where the resistance to flow is insufficient to stop urine leakage.

3.3 | Pressure flow studies

Flow mechanics within the urethra, especially turbulent flow, are extremely complex, nonlinear, exponentially determined by the fourth or even fifth power of the urethral radius, complicated by external opening forces and

the condition of the ligaments against which they contract,^{1,2,5} Figures 2 and 3. The graph, Figure 3, is based on bench studies of fluid through a tube³⁷ and computer-based studies³² calculating the forces required to stretch the tissues so they can open out the posterior urethral wall, lower X-ray, Figure 2. It was found that these forces were two orders of magnitude greater (X100) than pressures recorded during micturition.³² The same experiments applied in reverse for compression of the urethra show it is impossible for abdominal pressure to close the urethral tube.

3.3.1 | Continuous urine flow

EMGs in the posterior fornix of vagina,^{5,6} show contraction just before micturition, then electrical silence, Figure 8; urine being incompressible, keeps the urethra open throughout evacuation as the detrusor/urethral muscle shrink down around the urine to expel it. See Video 2.

Intermittent urine flow

If EMG, Figure 8 showed continuous muscle action throughout micturition, it would indicate the external muscle forces are not sufficiently strong to open the urethra, so they work continually to "grip" and pull open the urethra. With such continual movement, striated muscles tire, and this the relationship between slow flow, prolonged emptying time and urinary retention.

Flow rate

Figure 3, is exponentially determined. "Normal" micturition relies on the ability of the external muscle forces to open out the urethra. As such, it can never be precisely repeatable, often resulting in markedly different measurements.³⁹

3.3.2 | Pressure measurement during urine flow

With reference to the pressure/diameter graph, Figure 3, the pressure measured depends on the diameter of the urethral tube through which the detrusor has to push the urine to evacuate. Because the detrusor "contracts as one," to empty, the variation in the spikes of the pressure graph reflect the constantly varying diameter and therefore, resistance, of the emptying tube. The external opening mechanism adds to the many complex factors affecting urethral diameter. These include the urethral smooth muscle and its complex neural innervation.³⁷

3.3.3 | Detrusor function during voiding

The ICS definitions¹ are adequately explained by Figures 2 and 3.

*Detrusor function during voiding*¹

With reference to Figure 1, anything preventing afferent impulses reaching the cortex to activate micturition may cause UAB: reactionary overtightening of vagina by PCM because of USL laxity, Figure 7, requires higher hydrostatic pressure to stimulate “N”; interference with nerve transmission of afferent impulses from “N”, e.g., by multiple sclerosis.

Acontractile detrusor

Demonstration of “contraction” relies on observations of a rise in pressure. However, with reference to Figure 3, if the external mechanism can open urethra to say 10 mm, urine may leak with a detrusor actually contracting, but with little or any detectable pressure. In such situations, all the detrusor’s contractile energy is converted to flow, as described by Griffiths.³⁶

Postvoid residual

Unless the striated external opening muscles can fully open the urethra, the detrusor has to contract against the high resistance of a urethra inadequately opened by LP/LMA, and may tire with the repeated striated muscle contractions required to open it.

3.3.4 | Urethral function during voiding

The statement¹ “A urethra that opens and is “continually relaxed”¹ during voiding, is only partly correct. X-rays and EMGs in Figures 2, 3, 8, and Video 2 demonstrate that the pelvic muscles actively open the urethra just before and during voiding.

*Normal and abnormal urethral function*¹

See Figures 1–3, 8.

*Bladder outlet obstruction*¹

May be mechanical (enlarged prostate, urethral scarring) or functional, for example, overcompensation of PCM to block the distal urethra, because of lax USLs, Figure 7, was attributed as the cause of retention in Fowler’s Syndrome.⁸

Dysfunctional voiding

“Intermittent contractions of the periurethral striated muscle (PUSM) during voiding”¹ is mechanically possible, but PUSM is a very weak muscle and unlikely to be able by itself to narrow urethral area “A” sufficiently to cause

pressure rise. Because of global detrusor contraction, the force “F” of urine flow is constant; more likely, the closure reflex activates PUSM and also, the forward vector PCM, to act in concert; as pressure “P” = F/A, P increase reflects PUSM and PCM striated muscle contraction.

Detrusor sphincter dyssynergia

Straining before LP/LMA have opened out the urethra may activate the closure reflex, so PCM closes the urethra during detrusor contraction (see above).

Non-relaxing urethral sphincter obstruction

Relaxation of PCM, Figure 2, is essential to allow LP/LMA to open out the posterior wall of the urethra. A neurological lesion may prevent PCM relaxing as would imbalance of forces, Figure 7, seen in Fowler’s Syndrome.⁸

4 | CONDITIONS 4

4.1 | Chronic retention of urine

As per postvoid residual (3.3.3).

4.2 | Benign prostatic obstruction

An enlarged prostate which compresses the urethral tube will narrow it, requiring a higher detrusor pressure to drive emptying, usually with slow flow.

5 | TREATMENT

Surgical cure/improvement in OAB and other PFS symptoms, Figure 4, by PUL or USL sling repair, mainly in post-menopausal women,^{3,4,7,8,10,12,13,30,40–49} validated with pre and sometimes postoperative urodynamics, is consistent with restoration of the ligament-based peripheral control mechanisms, Figure 1. However, a key paper by Shkarupa et al. shows that at least as regards OAB and prolapse, native cardinal/uterosacral ligament repair may give good cure rates at 18 months, but only for premenopausal women, (postmenopausal in brackets)⁴⁹: prolapse:79.6% (16.7); frequency 60% (14.6); urgency 67.3% (16.7); and nocturia 87.7% (18.8).

6 | DISCUSSION

The aim of this study was to provide parallel anatomical explanations for the ICS definitions, which the ICS avoided as being beyond the remit of a

definition.¹ However, definitions must be a condensation of what they define,⁵⁰ “A precise statement of the nature, properties, scope or essential qualities of a thing.”⁵⁰ Our anatomical analysis indicates the 2002 definitions mainly meet this criterion, except for DO and OAB which imply pathogenesis is in the bladder itself. Our analysis supports the committee’s intuition that urge, frequency, nocturia are different manifestations of the same condition which they called “OAB,”¹ manifestations which our analysis considers part of a prematurely-activated micturition.² Minor definition changes, OAB to “overactivated” bladder and UAB to “underactivated” bladder converts cause to effect, indicating causation may be outside the bladder. DO as it stands, fails Sackett’s criteria for a diagnostic test.⁵¹ However, ultrasound⁵² and urodynamic monitoring⁵³ of “simulated operations,” mechanical support applied to PUL and USL can markedly alter the urodynamic graph, much in the way that surgical repair can restore function.^{3,4,7,8,10,12,13,30} With reference to urodynamics, this new method could be called “interventional urodynamics,” which supports the original vision of the ICS founders,⁵⁴ that urodynamics can be an effective diagnostic and predictive test for bladder dysfunction, albeit differently applied.

Some examples of clinical tests which can be applied urodynamically: SUI by PUL support: Video 3 (<https://youtu.be/0UZuJtjCQU>); urge and pain relief by USL support, Figure 5; nocturia by an overnight gauze in the fornix,⁴⁹ chronic pelvic pain by a pessary.¹⁰

*Urodynamic applications*⁵³: after midurethral anchoring, maximal urethral pressure increased from a mean of 33.25 cm H₂O to a mean of 58.06 cm H₂O ($p < 0.0001$); conversion of a $< 100\%$ CTR to $> 100\%$ CTR in the proximal urethra was observed in 14 of 22 patients ($p < 0.005$), with no significant change noted in the distal urethra. Further conversion of CTR was noted in six of the remaining eight patients with unilateral plication of suburethral vagina (“pinch test”²). Digital support of bladder can control DO.³³ Unilateral midurethral support can control cough-activated DO; a cylindrical pessary can improve “obstructive” flow charts (Petros unpublished observations).

7 | CONCLUSIONS

Anatomical analysis indicates the 2002 ICS definitions are fundamentally sound, except “OAB” which implies causation from the detrusor. Minor definition changes, OAB to “overactivated” bladder, UAB to “underactivated” bladder, broaden causation to structures

outside the bladder. This construct supports OAB and its component symptoms as a syndrome, as intuited by the Committee, (albeit as a prematurely activated micturition) and retains the acronym. It explains OAB cure from surgical repair of ligaments, and incontinence from two new syndromes which need to be added to definitions, Posterior Fornix Syndrome (of which OAB is a component) and Tethered Vagina Syndrome, which is the basis for skin-grafting cure of the 30-50% of women who continue leaking urine massively after successful Obstetric Fistula closure.⁵⁵

AUTHOR CONTRIBUTIONS

Planning, writing, reviewing, checking: Peter Petros, Jörgen Quaghebeur, and Jean-Jacques Wyndaele. Illustrations, video abstract: Peter Petros.

CONFLICT OF INTERESTS

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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