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A critical analysis of integral theory statements concerning pathogenesis of LUTS and chronic pelvic pain

Jörgen QUAGHEBEUR[1,2], Peter PETROS[3], Jean-Jacques WYNDAELE[2], Stefan de WACHTER[1,2]

[1]Department of Urology, University of Antwerp, Edegem, Belgium
[2]Faculty of Medicine and Health Sciences, University of Antwerp, Wilrijk, Belgium
[3]Retired Reconstructive Pelvic Surgeon, Sydney, Australia

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ABSTRACT

Lower urinary tract symptoms (LUTS) and pelvic pain are highly prevalent in women and often poorly understood. The aim was to critically analyse the integral theory paradigm's (ITP) statements concerning anatomical pathogenesis of LUTS, in particular, chronic primary pelvic pain syndrome (CPPPS) and LUTS as defined by the 2002 ICS report. The ITP explains pelvic floor function is determined by three directional muscle forces: forward, backwards and downward-acting muscle vector forces supported by ligaments which result in an anterior and posterior resultant keeping the balance needed for optimal sphincter closing and voiding. Muscular weakness caused by loose ligaments may result in LUTS and pain in some patients. Loose uterosacral and pubourethral ligaments provoke dysfunctional bladder filling and evacuation. Weakened uterosacral ligaments (USL), often accompanied by pelvic organ prolapse, weaken the posterior resultant, as seen in the posterior fornix syndrome and may explain chronic pelvic pain, LUTS, and dysfunctional defecation in some. Further studies are needed to investigate the importance of the ITP when treating CPPPSs and LUTS, in particular, possible non-surgical options to support USLs, considering the possible complications of reconstructive surgery.

Keywords: Biomechanics; chronic pelvic pain; interstitial cystitis; neuroanatomy; painful bladder syndrome; posterior fornix syndrome; urinary stress incontinence

INTRODUCTION

Lower urinary tract symptoms (LUTS) and pelvic pain are highly prevalent in women of all ages and often poorly understood.[1] In women, 59.2% have storage symptoms, 19.5% voiding symptoms, and 14.2% reported post-micturition symptoms.[1,2] Lower urinary tract pain, appears to be more common in women than men.[1]

The integral theory (IT) suggests a comprehensive approach to pelvic floor sphincter function and dysfunction, possibly explaining the malfunction of the pelvic organs in some.[3]

Address for Correspondence: Jörgen Quaghebeur, Department of Urology, University of Antwerp; Faculty of Medicine and Health Sciences, University of Antwerp, Edegem, Wilrijk, Belgium
E-mail: jorgen.quaghebeur@telenet.be **ORCID ID:** orcid.org/0000-0002-3776-5969
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The IT postulates three directional muscle forces forming an anterior and posterior resultant to be responsible for normal bladder function. The pubococcygeus muscle (PCM) forms a vector in forward direction, the levator plate (LP) in backwards direction, and the conjoint longitudinal muscle of the anus (LMA) contracts in a downward direction. The back and downwards directed vector forces form a backward resultant. LUTS occur when the balance between these resultants is disturbed by loose ligaments and/or muscular weakness.[4] Tension in the pelvic floor fascia and bladder neck triggers proprioceptive afferents that may disrupt the micturition reflex perceived by patients as urgency.[4] The IT suggests tension in the vaginal wall and disturbed proprioceptive stimuli in the bladder base resulting from weakened muscular vector forces to be responsible for LUTS in some (e.g., OAB, urine retention, weak flow, urgency, nocturia) and pelvic pain.[4-6]

Testing with a haemostat gently supporting the pubourethral ligament (PUL) or speculum blade supporting the posterior fornix can show a possible indication for reconstructive surgery in some.[4,7,8] The IT suggests the use of an anterior, middle or posterior mesh to suspend regional weakness when treating incontinence, bladder or anorectal dysfunction and pain.[7-10] The IT in pelvic pain (e.g., vulvodynia) may show that uterosacral ligament (USL) weakness exists by injecting Xylocaine into the USL at the posterior fornix of the vagina (Bornstein test).[11,12]

We explain the possible anatomical pathogenesis of disturbed bladder and sphincter function using the 2002 standardization report from the ICS on LUTS[13] and discuss CPPPS and pelvic organ dysfunction from a critical point of view. Chronic primary pain is used unless another diagnosis could better explain the pain and symptoms present.[14] Such other diagnoses are referred to as “chronic secondary pain”, where pain may initially be assigned as a symptom secondary to an underlying disease.[14]

OBJECTIVE

The aim was to critically analyse the Integral Theory Paradigm’s (ITP) statements concerning anatomical pathogenesis of lower urinary tract symptoms, in particular, chronic primary pelvic pain syndrome (CPPPS) and LUTS as defined by the 2002 ICS report.[4,13]

METHODS

Explain normal bladder function and LUTS as postulated by the Integral Theory, according to the 2002 ICS report.[4,13]

1. Function and Dysfunction of the Lower Urinary Tract

Normal bladder function

Afferent and efferent stimuli controlled by higher centres determine the normal function of the lower urinary tract.[15] The control system regulates the continence and emptying of the bladder and is determined by cortical and peripheral factors. Sensory stimuli from stretch receptors in the bladder wall initiate the sensation of a full bladder and the need to void. Muscular and ligament proprioceptive afferent stimuli from the small pelvis also trigger the higher centres and periaqueductal grey matter, the pelvic organ stimulation centre and the pelvic floor stimulation centre in the brainstem.[15] There is a balance between the muscular open and closing forces and tension in ligaments, that control the proprioception around the bladder in a normal situation. Three oppositely acting directional muscles determine the tension in the vaginal wall supporting the bladder. Figure 1 shows the ligaments and muscular vector forces of the pelvic floor. The PCM contraction pulls on the PULs in a forward direction, the LP on the PUL in a backwards direction, and the conjoint LMA contracts in a downward direction on the USL. The back and downwards directed vector forces form a backward resultant (Figure 1).[4]

Dysfunction

Any anatomical damage in the afferent or efferent system or higher centres may cause bladder continence or voiding

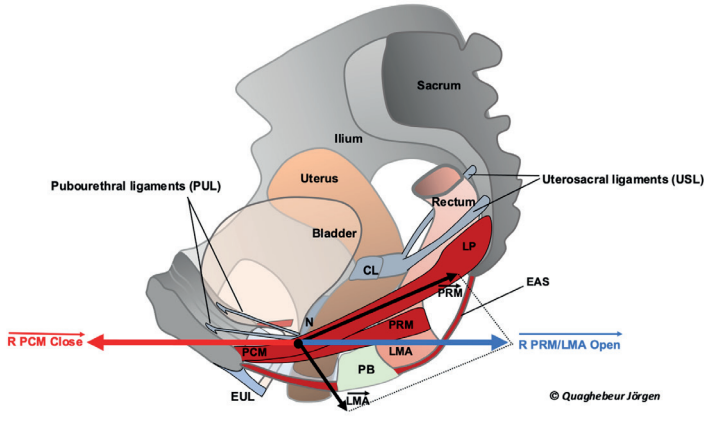

Figure 1. The three directional muscle forces of the pelvic floor and the anterior and posterior resultant

CL: cardinal ligament; EAS: external anal sphincter; EUL: external urethral ligament; LMA: longitudinal muscle anus; LP: levator plate; N: bladder stretch receptors; PB: perineal body; PUL: pubourethral ligaments; USL: uterosacral ligaments

The figure shows zero resultant force at the level of the bladder neck. Weak PUL decrease forward muscle forces, so the stronger backwards-pulling muscles move the zero force backwards. Weak USLs decrease the backward vector forces, so the stronger forward muscles pull the zero-force forwards

dysfunction and disturb the control of the micturition reflex, as seen in overactive bladders. Damage in the central nervous system can be in the brain, spinal cord (e.g., tumor), peripheral nerves (multiple sclerosis), and disturb the micturition reflex. Tension or damage of the pelvic muscles, ligaments, and urothelial stretch receptors "N" (e.g., cancer or external pressure by fibroid, scarring by iatrogenic or obstetric fistula), can disturb the peripheral control. Disturbance of the binary control of the bladder (continence, evacuation) results in bladder dysfunction, such as deficiencies in closure (incontinence), evacuation (urinary retention) or premature activation of the micturition reflex (OAB).[4] Figure 2: The afferent and efferent nervous system innervating the bladder wall, ligaments and muscular pelvic floor.

With reference to Figures 1 and 2, control of the bladder is binary, with only two modes, EITHER "closed" (closure reflex) OR "open" (micturition reflex).[13,16] "Closed" is the dominant mode. "Open" is activated when the afferent signals of fullness from the bladder base stretch receptors "N" signal a need to empty.[16] Anatomical damage in some part of the control system, Figure 2, in particular collagen-based weakness in the suspensory ligaments, Figure 1, can disturb the closed/open binary balance, to cause premature activation of an otherwise normal micturition reflex,

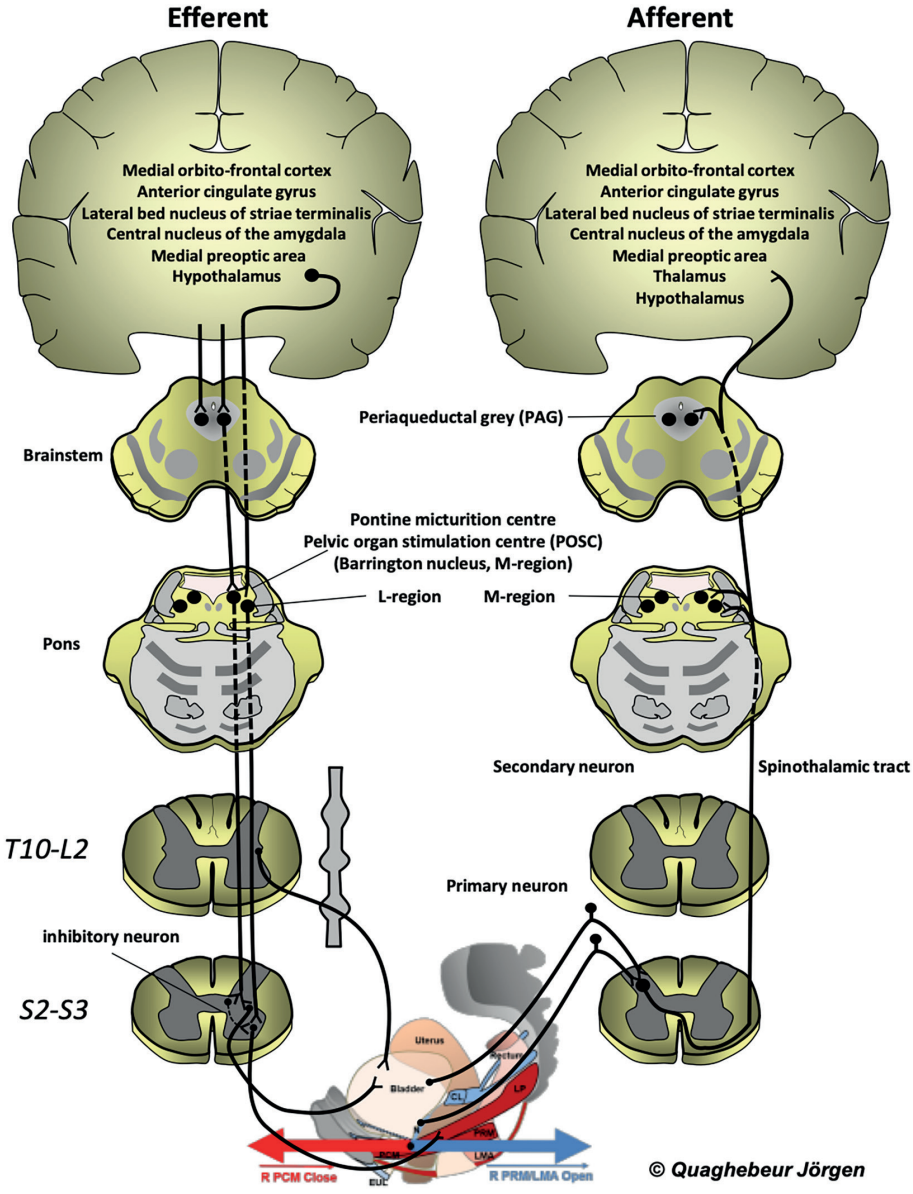

Figure 2. The afferent and efferent nervous system innervating the ligament and muscular pelvic floor
CL: cardinal ligament; EAS: external anal sphincter; EUL: external urethral ligament; LMA: longitudinal muscle anus; LP: levator plate; N: bladder stretch receptors Figure shows the higher centres, afferent and efferent innervation of the bladder and pelvic floor sphincter

which has been shown to be equivalent to urodynamic "detrusor instability".[17]

The International Continence Society (ICS) standardization committee classified the terminology of LUTS as storage symptoms and voiding dysfunction (e.g., obstructed micturition, urinary retention).[13] This ICS report includes the description of LUTS, urodynamic observations, symptoms associated with sexual intercourse, pelvic organ prolapse, and chronic pelvic pain syndromes.[13]

We summarized how the IT may play a role in some patients the anatomopathological explanation of LUTS, based on the IT in Table 1.

Storage symptoms include overactive bladder symptoms (OAB), nocturnal enuresis, stress and mixed urinary incontinence, and disturbed bladder sensation. Deficient contractility of the bladder is a storage problem accompanied with urine residue.

The IT explains OAB symptoms (e.g., frequency, urgency, and nocturia) as manifestations of damage in some part of the

Table 1. Integral theory and LUTS
Storage symptoms
Overactive bladder symptoms (OAB) and nocturnal enuresis
The three directional forces fail to stretch the vagina sufficiently, continuously triggering the proprioceptors at the bladder base (N) with urgency and frequency at a low bladder volume. When the closure reflex cannot control the micturition reflex, urge incontinence can occur. Nocturia occurs when the urgency wakes the patient to go to the toilet at night. Nocturnal enuresis occurs when a child with an immature bladder does not wake up to void.[1]
Stress urinary incontinence (SUI)
When a weak pubourethral ligament (PUL) cannot structurally support the middle part of the urethra and the LP/LMA muscles pull down the distal vagina, trigone and posterior urethral wall in an open position, SUI occurs. A haemostat placed immediately behind the pubic symphysis (PS) prevents the elongation of a weak PUL and decreases the incontinence confirming the SUI mechanism.[1-3]
Mixed urinary incontinence
Loose PUL weaken the PCM force triggering the stretch receptors (N) at the bladder base, which sends uncontrolled afferent impulses to higher centres. This is perceived as urgency. A haemostat positioning behind the pubis diminishes urge in 50% of cases and indicates further treatment.[1,3,4]
Bladder sensations
The balance of forward (PCM) and backwards (LP/LMA) muscle forces, stretching the vagina to support "N" from below, is disturbed when loose uterosacral ligaments (USLs) weaken LP/LMA posterior resultant. Higher hydrostatic pressure is required to activate the urothelial stretch receptors for evacuation because autonomic stress responses from higher centres let the PCM stretch the vagina further forwards. Urinary retention occurs as seen in under active bladder.[5]
Voiding, obstructed micturition, and urinary retention
The voiding mechanism is similar to SUI, except during micturition, the PCM relaxes, and LP/LMA opens out the urethra to decrease resistance to urine expulsion. Weak USLs reduce the opening force of the LMA, and the posterior urethral wall opens inadequately. Symptoms of obstructed micturition occur.[5,6]
Slow stream
Loose USLs weaken the LP/LMA posteriorly directed opening forces, and the urethra cannot open adequately. Therefore, the detrusor must push urine out through a narrower tube with a slow stream.[1]
Splitting of spraying
Dislocation of one of the pubococcygeus muscles causes "PCM" to unbalance the forces against the symphysis. The opposite healthy muscle pulls the urethra towards it, causing a divergent stream or spraying.[1,7]
Intermittent stream
The EMG must decrease during normal voiding, although in women with an intermittent stream, it repeatedly rises, indicating that the muscles cannot sufficiently open the urethra.[1]
Hesitance
The LP/LMA muscles externally open the urethra insufficiently due to weak USLs causing hesitance.[1]
Straining
Straining increases the abdominal pressure and helps the detrusor to evacuate once the urethra has been pulled open during the flow.[1]

Table 1. Continued
Feeling of incomplete emptying
Incomplete emptying
The striated LP/LMA muscles tire with repeated efforts to evacuate. The EMG is disturbed, incomplete evacuation follows, and further emptying is needed in 20 min.[1]
Postmicturition dribble
Repeated efforts in the EMG occur when the LP/LMA striated muscles tire. The urethra closes under the influence of its elastic tissues, exponentially raising resistance to detrusor during emptying. The detrusor can only expel urine at a prolonged flow.[1]
Incontinence during sexual intercourse
The lateral part of the PUL inserts into the lateral part of PCM and directly into the vagina.[1,4] A penis thrusting backwards from the introitus may stretch the vagina sufficiently to pull open the urethra. If the PUL cannot sustain the backward stretching from the penis, the PUL elongates. The elongation allows the posterior forces to stretch open the urethra. Such cases respond to mid-urethral sling surgery.[1]
Symptoms associated with pelvic organ prolapse
In uterus prolapse, the uterus descends into the vagina by intraabdominal pressure and gravity pulling on USLs, which contain nerves causing backache and dragging.[1] Digitally supporting the perineum prevents the diversion of faeces into the rectocele during defecation.

control system, disturbing reflex control of the micturition reflex.[4] Disturbed tension of the vaginal wall caused by a weak sphincter mechanism triggers the proprioceptive stretch receptors "N" which the cortex perceives as urgency and lower bladder volume develop as a consequence. Urge symptoms at night wake the patient to void "nocturia".[4] Urge urinary incontinence occurs when the closure reflex cannot sufficiently control the afferent impulses from "N", and the micturition reflex is continuously activated. In children sleeping, nocturnal enuresis manifests when afferent impulses activate the micturition centre without waking up.[4]

The IT explains stress urinary incontinence as a result of a weak PUL that cannot structurally support the middle part of the urethra. The anterior resultant decreases, and the LP/LMA muscles pull down the distal vagina, trigone and posterior urethral wall to open the urethra.

Three directional forces close the distal urethra and bladder neck. The PCM stretches the suburethral vaginal hammock against a competent PUL to close the distal urethra from behind.[4,10] The LP pulls backwards against PUL to stretch the vagina, proximal urethra, trigone, and bladder base backwards to render the tissues semi-rigid.[4,10] The LP also tensions the pubovesical ligament (PVL), which inserts into a smooth thickening on the anterior wall of the bladder[4,10] (arc of Gil-Vernet).[18] Normally, the pubovesical ligament (PVL) and the arc hold the anterior bladder wall steady, while the conjoint LMA (downward vector) contracts against the USL rotating the now semi-rigid bladder around the arc to close the urethra at the bladder neck.[4,10] A weak PUL stretches down on effort and does not support the distal vagina into which it inserts.[19] A haemostat or finger gently placed behind the pubic symphysis (PS) and above the urogenital diaphragm supports the PUL preventing the elongation of a weak PUL and decreasing the loss of urine, showing the need for anterior pelvic floor support.

The vagina requires stretching from both ends and supports the bladder neck from below. Mixed urinary incontinence occurs if loose PUL weaken the PCM force and accompanies uncontrolled afferent impulses interpreted by the cortex as urgency. A haemostat test gives support just behind the pubis, diminishes urge in 50% of cases, and indicates SUI and urge cure.[4,19,20]

The balance of the forward and backward resultant muscle forces stretches the vagina to support "N" from below and is essential for optimal bladder sensation. LP/LMA vector forces weaken when the USLs are loose. Autonomic impulses from higher centres in the brain activate the PCM and stretch the vagina further forwards. This requires a higher hydrostatic pressure to activate the urothelial stretch receptors for evacuation. High preoperative mean bladder volumes (598 mL) in women with under active bladder symptoms and retention decreased to a mean of 301 mL following USL repair, which cured/improved the emptying symptoms and residual urine.[21]

Voiding, obstructed micturition, and urinary retention can also be caused by a disturbed peripheral muscular and ligament mechanism. The SUI and micturition mechanisms are similar, except that during micturition, the PCM relaxes, and LP/LMA opens out the urethra to decrease resistance to urine expulsion. The LMA opening force weakens when the USLs are loose. Obstructed micturition is experienced when the posterior urethral wall opens inadequately.[22] Lax PUL weaken forward

muscle forces, and the posteriorly directed resultant pulls the equilibrium point backwards, broadening the urethra and causing SUI.[21,22] Lax USLs weaken the backward vector forces, and the stronger forward muscles pull the vagina and equilibrium point forward.[21,22] Imbalanced muscle forces may over-tension the distal vagina and close the distal urethra more tightly. Imbalanced muscle forces may create much firmer support below the urothelial stretch receptors "N".[21,22] To activate the micturition reflex, a greater bladder volume is necessary to activate the afferent impulses required and explains the more significant resting bladder volumes and retention as reported in "underactive bladder" and Fowler's syndrome.[21,22]

Symptoms such as slow stream and hesitance can be caused by a weak posterior resultant (LP/LMA opening forces) due to loose USL. The urethra cannot adequately open, and the detrusor pushes the urine out against resistance with a slow stream.[4]

Splitting or spraying can be caused by the dislocation of one of the PCMs provoking unbalance of the "PCM". In divergent streams or spraying, the opposite healthy muscle pulls the urethra towards it.[4,23] During normal voiding, the EMG repeatedly rises in women with an intermittent stream, although it must decrease. The muscles cannot "grip" sufficiently to open the urethra.[4] An increased abdominal pressure by straining helps the detrusor to empty, and once the urethra has been pulled open and urine is flowing.[4]

The lateral part of PUL inserts into the lateral part of PCM and directly into the vagina.[4,20] Coital incontinence may occur when a penis thrusting backwards from the introitus stretches the vagina sufficiently to pull open the urethra. If the PUL cannot sustain the backward stretching from the penis, the PUL elongates. The elongation allows the posterior forces to stretch open the urethra provoking urinary leakage during sexual intercourse. Such cases respond to mid-urethral sling surgery.[4]

In uterus prolapse, the uterus descends into the vagina pulling on USLs, which contain myelinated and unmyelinated nerves and causing backache and dragging.[4] Digitally supporting the perineum prevents the diversion of faeces into the rectocele during defecation and repair may improve the prolapse and symptoms.[24,25]

Chronic pelvic pain and the posterior fornix syndrome

Damage to the posterior suspension system in the pelvis (e.g., USLs) is mainly responsible for prolapse-induced visceral plexus, chronic lumbosacral, and pelvic pain often accompanied by LUTS and anorectal dysfunction (e.g., urgency, frequency, nocturia, and obstructive miction and defecation).[26,27] The USLs connect the sacral vertebrae (S2-S4) with the fornix and contain autonomic nerve fibres and nociceptive afferents.[26,28] The posterior fornix syndrome (PFS) is caused by laxity or injury of the USLs and causes LUTS, chronic pain, and obstructive defecation that can be relieved by the suspension.[25,29] Gentle support using a lower blade of a bivalve speculum generally relieves the pain, and excessive caudal traction will exacerbate it.[4,11,29,30] Some cases of vulvar pain syndrome also will determine the pain's origin by the posterior fornix's support.[12,24,31,32] Permanent traction on the hypogastric or sacral plexus and irritation of the Frankenhäuser's ganglion caused by USLs laxity and POP may also be the reason for chronic neuropathic pain mechanisms and gynaecological low back pain.[26]

DISCUSSION

Although the IT suggests an all-encompassing explanation for bladder dysfunction and bowel evacuation problems, CPPPS and LUTS comprise a more complex pathophysiology.

The CPPPS are determined by exclusion of infection, inflammation, or obvious pathology and defined as "primary pelvic pain syndrome" if there is adequate evidence for its use.[33] When the pain can be localised to an organ, the subclassification must be attributed to a more specific term adding "primary" in front of the organ attained.[14] When chronic pelvic pain or LUTS are accompanied with USL-related POP, as suggested in IT, the pain and symptoms are secondary to prolapse and must not be classified as a "primary" pain syndrome. However, USL-caused chronic pelvic pains usually co-occur in multiple sites. These disparately-sited pains are generally relieved simultaneously either by the speculum test, by local anaesthetic injection into the lower end of the USL (Bornstein test)[11] or by USL surgery.[22] These data would seem to require revision of the "regional pain syndrome" classification of multiple organ involvement. It is considered a regional pain syndrome if multiple organs are involved.[33] Determining a specific end organ phenotype also involves the evaluation of significant emotional stress, functional disability, sexual and behavioural problems.[14,33]

Functional urological (e.g., CPPPS, BPS/IC, OAB) and gastrointestinal disorders (IBS) are interrelated, and the stress response mediates the severity of symptoms via the sympathetic-adreno-medullar axis (SAM), the hypothalamus-pituitary-adrenal axis (HPA), and the immune system.[15,34-36] Any psychological or physical stimulus that disturbs the homeostasis of the human body causes an autonomic stress response.[37-40] This stress response is also determined by the phylogenetic development of the nervous system as described in the Polyvagal Theory.[15,41,42] Comorbid conditions often accompany chronic pelvic pain and LUTS, and cross-organ sensitization.[43-48] The pelvic organs share

the same innervation, making them susceptible to cross-organ sensitization.[36,46,49] Cross-sensitization at a neuronal level and the gut microbiome explain the co-occurrence of LUTS and bowel dysfunction.[50-52] Determining the origin of pelvic pain syndromes or LUTS is difficult because of the overlap of symptoms, emotional factors, comorbidity and associated conditions.

The IT postulates ligament weakness with pelvic floor dysfunction as an explanatory model for a variety of often poorly understood bladder dysfunctions, genitourinary pain (e.g., vulvodynia) and anorectal dysfunction. The IT model explains pelvic pain and LUTS in women, although chronic pelvic pain conditions and genitourinary pain exist in men as well, which suggests that loose ligaments and disturbed muscular vector forces in the pelvic floor seem not always the reason for pelvic organ dysfunction and unexplained pelvic pain. The symptoms in patients with CPPPS appear to be equal between gender.[53] The symptoms in men are challenging to explain with IT, even taking into account recent discoveries of male analogues for USL and PUL.[54,55]

Not all LUTS and pelvic pain syndromes can be explained by IT, and testing the support with a haemostat or speculum blade might indicate some cases with specific regional weakness. LUTS and pain accompany POP, but also minor prolapse must be seen as a pathological condition. In POP, symptoms such as urinary stress incontinence, urinary urge incontinence, abnormal bowel, bladder emptying and faecal incontinence may occur, with or without pain.[56] Loose USL may cause PFS, often accompanied by faecal urgency, pelvic pain, nycturia, and anorectal evacuation problems.[56] The IT suggests the Bornstein test to detect chronic pain due to loose ligaments; the Xylocaine injection works by its local analgesic (LA) effect on the visceral nerve plexuses at the lower end of the USL. A valid criticism of this LA test is it cannot *per se* directly diagnose USL weakness. The extra link to causation is the speculum test which mechanically supports USL and (when it works) immediately relieves several CPP sites,[29] and the results of surgical ligament strengthening by plication[25] or by slings.[26,27,32] It may indicate pain due to constant traction on nervous structures (visceral plexus). The IT suggests that pelvic organ dysfunction is provoked by disturbed muscular vector forces in the pelvic floor caused by ligament weakness and pelvic organ prolapse (POP) affecting the binary innervation and disturbing the pontine micturition reflex.[56]

LUTS and pelvic pain syndromes are often unexplained, but when in POP, they result from a disorder and are the consequence or "secondary to an underlying disorder".[14]

Neurodynamic testing showed significant mechanosensitivity in at least one lumbosacral plexus nerve in 88% of the patients with CPPPS, suggesting minor nerve injuries. Eighty-five per cent showed palpatory-provoked pudendal nerve mechanosensitivity. Neurodynamic testing also showed the involvement of multiple lumbosacral plexus nerves in 42%, suggesting abnormal impulse generation sites not caused by traction on the visceral plexus, as suggested by IT.[57] Compression and continuous traction are traumatic for nervous structures. Inside nerves, we find blood circulation, primo vascular channels, and lymphatic circulation in the perineurium. It remains unclear if a nerve injury provokes chronic pain and circulatory disturbances in the bladder wall and mucosa resulting in fibrosis and Hunner's lesions at a later stage.

Scheffler et al.[58] suggested a cure for BPS/IC by repairing the ligaments outside of the bladder (cardinal, uterosacral) according to the PFS protocols[24] of the 1993 Integral Theory.[59] Goeschen and Gold[8] revisited clinical data (n=198) of women who had presented with chronic pelvic pain (CPP) plus varying degrees of uterine/apical prolapse. Posterior intravaginal slingplasty repair of the USLs using a collagen-producing tape variously cured several bladder symptoms and pain.[8,60] The cure rate after USL sling repair was CPP 74%, urge incontinence 80%, frequency 79.6%, abnormal emptying 53%, nocturia 79%, and obstructive defecation 80%. Goeschen et al.[60] suggested that the PFS is similar to BPS/IC but still further rigorous scientific investigation, preferably by RCT, is required. It remains unclear whether lesser visceral plexus problems or only a part of the plexus provokes BPS/IC or causes different effects in the bladder wall. CPPPS co-occurs in multiple sites. It would be interesting when looking at the data from the studies, if it becomes possible to link the loss of support grade or side to plexus problems limited or global to the symptoms. It remains unclear whether plexus problems explain why the impact of bladder inflammation can be broad or restricted. Probably the pain response on visceral plexus traction is global because Wu et al.[29] reported relief of several sites with the speculum test, and a Bornstein test carried out in 3 women with BPS/IC relieved several pain sites simultaneously.[11] Research on the lack of pelvic floor support and BPS/IC is needed but can only be considered a start based on the limited data in the literature. The actual findings indeed show the need for testing the support system, excluding POP, when assessing CPPPS and LUTS during the patient's physical examination.

The IT in its formal statement states pelvic organ dysfunctions are mainly caused by collagen-induced ligament weakness; the IT predicts "strengthening the ligament will restore the function".[61] The strengthening can be non-surgical (as demonstrated by the speculum test for USL weakness), but to date, treatment for ligament weakness has been overwhelmingly surgical: Midurethral slings for SUI, and USL slings for LUTS and POP, with

high cure rates and low direct tape reaction.[26,27,32]

However, multiple publications describe mesh complications (e.g., pain, erosion, voiding dysfunction, infection, recurrent urinary tract infections, fistulae, organ perforation, bleeding vaginal scarring, neuromuscular alterations, LUTS, bowel complications, and immune disorders).[62-65] A study showed that 42% of women had at least one adverse event, and 12% had at least one serious adverse event.[66] A systematic review showed that using polypropylene pelvic mesh becomes highly controversial because of serious complications provoked by an essential mismatch between its viscoelastic properties and the structure of the surrounding tissue. In multiple countries, polypropylene mesh for treating POP has been banned, with no available alternative.[67] An important distinction was drawn between mesh usage for POP and tapes mesh sheets block descent of the prolapse, but do not cure it; tapes repair the prolapse by creating new collagen for its structural supports, the ligaments.[68]

CONCLUSION

As a theory, the ITP describes how ligament and muscular factors under peripheral and central nervous control determine how the bladder functions, how in specific cases, anatomical damage to pelvic muscles, ligaments or their nervous control can disturb the equilibrium between anterior and posterior resultant muscular forces and to explain LUTS and chronic pelvic pain dysfunctions, collagen being the weakest link. Also as a theory, the ITP is obliged to make firm statements that are falsifiable, which never cover every possible cause. Though surgical repairs such as the midurethral sling have dominated the ITP to date, equally important is intravaginal mechanical support of pubourethral and USL described, which has been shown to immediately reduce SUI, urge, pain and nocturia on application. This is a promising non-surgical future way forward for the ITP, as it avoids the complications inevitably encountered with reconstructive surgery. Looking ahead, the main task now, as we see it, are further studies, non-surgical, to assess the impact of ligament weakness on POP, LUTS and chronic pelvic pain.

ETHICS

Contributions

Surgical and Medical Practices: P.P., J-J.W., S.D.W.; Concept: J.Q., P.P.; Design: J.Q.; Data Collection or Processing: J.Q.; Analysis or Interpretation: J.Q., P.P., J-J.W., S.D.W.; Literature Search: J.Q., P.P.; Writing: J.Q.

DISCLOSURES

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