DOI: 10.34057/PPj.2020.39.04.002
Pelviperineology 2020;39(4):107-114

FOWLER'S SYNDROME: WHAT IT IS AND WHAT IT'S NOT

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ABSTRACT

We review the clinical problem of idiopathic non-obstructive urinary retention and voiding difficulty in young women, thought by Fowler and associates to be due to urinary sphincter spasm. We conclude that this voiding disorder in Fowler's syndrome (FS) is not due to sphincter spasm associated with a so-called characteristic electromyography pattern in the urinary rhabdosphincter, since the latter has been shown to occur in normal young women. We point out that non-obstructive urinary retention and voiding difficulty is also a feature of the posterior fornix syndrome (PFS) and we suggest that these two syndromes are one and the same. In idiopathic non-obstructive urinary retention, as described in PFS and in FS, the voiding disorder is usually associated with laxity of the uterosacral ligament (USL)with resultant loss of balance in the pelvic floor muscular vectors, causing impaired opening of the urethra for voiding. The various associated features of these two syndromes, especially enlarged bladder, urgency and frequency, chronic pelvic pain, and secondary psychological symptoms are all relieved when voiding difficulty is resolved by successful management strategies, including physiotherapy, tape-based reinforcement of the USL, and neuromodulatory management. We suggest that, given its simplicity and long-term effectiveness, surgical reinforcement of the USL, by plication in younger women or tape reinforcement after the menopause, is the optimal first line treatment for posterior fornix syndrome (PFS). Nonetheless, we caution that our conclusions described here do not exclude other, central neurological causations for this voiding disorder in some patients.

Keywords: Fowler's syndrome; posterior fornix syndrome; underactive bladder; urinary retention; chronic pelvic pain; OAB; nocturia

INTRODUCTION

The clinical problem

In 1988 Fowler et al.[1] suggested that idiopathic non-obstructive urinary retention in young women was a distinct syndrome often associated with certain characteristic features (Table 1). At that time urinary retention in young women was known as an uncommon disorder, described in the early 19th century, and often attributed to psychological causation; i.e., a "functional" syndrome, an unsatisfactory concept that lacked clear definition. Fowler suggested that this voiding disorder was due to failure of relaxation of the urinary sphincter. Of the listed features,[1,2] in addition to retention, only three are positive. The onset has been associated with opiate therapy, with gynaecological or abdominal surgery, and other minor medical procedures and with childbirth. There is frequent psychological co-morbidity. The most striking of the three positive features, that affected women

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Received: 12 August 2020 **Accepted:** 27 October 2020

often note that withdrawal of their catheter is accompanied by a feeling of something gripping it, has been ascribed to spasm of the urethral rhabdosphincter (RS).[3] This is attributed to the finding of complex repetitive discharges (CRDs) and decelerating burst (DBs) activity in needle electromyographic (EMG) recordings from the external urinary RS.[1,3,4] However, in the initial report only one control subject was studied and in that subject these EMG features were also found.[4] Later, it was recognised that this EMG feature was present in less than 50% of women with non-obstructive urinary retention.[5]

In striated skeletal muscles, EMG activity of this type is almost always abnormal, and found in both neurogenic or myopathic disorders,[6] especially when a muscle fibre is injured during the insertion of the EMG needle. CRDs do not require motor endplate firing or voluntary neural activity but are due to ephaptic excitation occurring between adjacent striated muscle fibres.[7] It has been suggested that CRDs and DBs in the urinary sphincter may be due in some way to hormonally driven cross-talk between smooth muscle and striated muscle fibres in the urinary sphincter complex.[5] Fowler and colleagues assumed that this sphincteric EMG feature was pathological and caused urinary retention by preventing sphincter relaxation.[3] However, the extent of sphincter muscle involvement during the EMG discharge and the force generated by the discharge have never been studied. In addition, several subsequent investigators have found this sphincteric EMG feature in normal women.[5] Tawadros et al.[5] reported it in the luteal phase of the menstrual cycle in 5/15 women and in both follicular and luteal phases in 8/15, but not in the follicular phase alone. It is therefore not an abnormal finding and, therefore, cannot account for difficulty voiding. Indeed, radiologic, urodynamic and mathematical modelling studies have shown that urinary continence is achieved by musculo-elastic forces outside the urethra, rather than by contraction of the urethral sphincter muscle alone.[8] Fowler et al.[1] also suggested that difficulty voiding was associated with polycystic ovary syndrome, found in 14 of 22 affected women. However, subsequently neither polycystic ovary syndrome, nor endometriosis, were confirmed as associated features.[3,9] The atonic enlarged bladder is considered secondary to functional outflow tract obstruction. Although not at first specifically recognised, a variety of neuropsychiatric symptoms are common associated features and there is frequently chronic pelvic pain.[9,10]

In summary, the symptoms and investigative findings in young women presenting with idiopathic non-obstructive urinary retention extend inconstantly across multiple bodily systems, and the suggested principal feature, the EMG finding, occurs in normal women and varies according to the menstrual cycle.[5] A different explanation for non-obstructive urinary retention in young women must be sought, although it is possible that there is no unitary causation.

A different explanation for non-obstructive urinary retention in women

Lax or weakened uterosacral ligaments (USL) can lead to urinary retention and difficulty in voiding, a disorder termed the posterior fornix syndrome (PFS), first described in 1993.[8,11] PFS, like Fowler's syndrome (FS), presents with idiopathic non-obstructive urinary retention, associated with frequency, urgency, nocturia, pelvic pain and neuropsychiatric symptoms. It can be cured or improved by USL plication in younger women with good collagen[11] or, more effectively, especially in older women, with a specifically located tape insertion procedure which creates new collagen to reinforce the weakened USL's.[12,13] This experience has implications for management of women with this spectrum of voiding disorders, including FS and PFS.[8,14]

Feedback control of bladder storage and voiding

A feedback control model of urinary storage and voiding is recognised[15] (Figure 1). Urine storage (continence), the resting mode, is modulated by the closure reflex, that is also under conscious influence (Figure 1); white arrows). In the pelvic floor, bladder storage is controlled by the tone of three opposed vectors (Figure 1; large arrows).[16-18] The storage mode is interrupted by voiding, a brainstem reflex response under conscious control that is initiated by perceived bladder filling. This is described as follows:

Urethral closure: The pubococcygeus muscle (PCM) contracts forwards against pubourethral ligaments (PULs). levator plate (LP) contracts backwards against PUL and USL. LMA (conjoint longitudinal muscle of the anus) contracts downwards to rotate the bladder and close the proximal urethra by "kinking" at the bladder neck[18] (Figure 1). These actions increase urethral flow resistance, determined by Poiseuille's Law as proportional to the fourth power of the urethral radius.[19-21] At the same time, the urethral RS also contracts, further narrowing the urethral lumen. The closure response also causes all three vectors (arrows in Figure 1) acting oppositely, to tension the vagina, supporting the urothelial stretch receptors ("N" in Figure 1) from below. The resulting tension counteracts the hydrostatic pressure of urine in the bladder, suppressing increased afferent impulses to cortex which would otherwise cause activation of the voiding response.

Voiding reflex: Bladder afferents "N" to Barrington's nucleus in the pons (Figure 1) which activates the micturition reflex, subject to cortical modulation (white arrows, Figure 1). The subject feels a sensory urge, and the forward vector due to PCM muscle activity relaxes (broken circle, Figure 1).[16-18] This causes the proximal

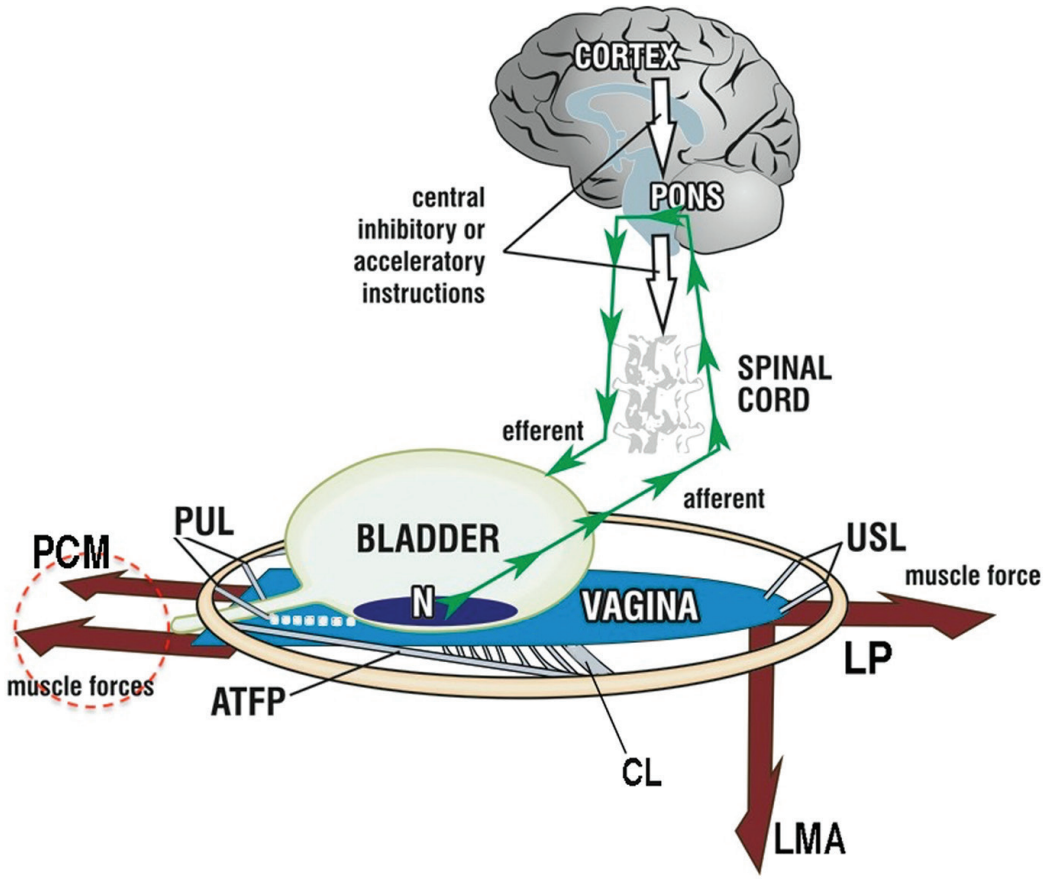

Figure 1. Feedback control of closure and micturition Schematic 3D sagittal view of bladder and other anatomical structures involved in micturition: 3 opposite vector forces (large arrows) pubococcygeus muscle (PCM) contracts forwards against pubourethral ligaments (PUL); levator plate (LP) contracts backwards against PUL and uterosacral ligaments (USL); conjoint longitudinal muscle of the anus (LMA) contracts downwards against USL; small upwards green arrows = afferent impulses; downward green arrows = efferent impulses; large white arrows = central suppression of both. (broken circle) = relaxation of PCM prior to micturition. If PUL or USL are loose, the muscles contracting against them (arrows) weaken. Urethra cannot be closed (incontinence), opened (emptying problems) or organs stretched to support "N", (urge incontinence)

CX: Cervix; CL: Cardinal ligament; ATFP: Arcus tendineus fascia pelvis

urethral pressure to fall, and is followed by detrusor contraction, and voiding of urine. PCM relaxation (red broken circle, Figure 1) allows the posterior muscles (large arrows) to funnel out the posterior wall of the urethra (broken white lines, Figure 1) just before the bladder detrusor muscle contracts.[18] This proximal urethral funnelling exponentially reduces internal urethral resistance to flow (Figure 2), so reducing the work required to void by bladder contraction.[18] It also allows urine to enter the proximal urethra, augmenting the urge to void by a sensory reflex loop. The opposing muscle forces must be balanced, or instability is introduced into the system (see Figures 1 and 3).

Video-urodynamics and uterosacral ligamentous laxity: Obstruction in the region of the striated sphincter (i.e., distal half of urethra) is a feature of idiopathic non-obstructive urinary retention; for example, in FS and PFS.[3,8,11] In FS this has been ascribed to striated urethral sphincter overactivity but USL laxity[8], as described above, is a more plausible explanation. When the USLs are lax (Figure 3), the posterior muscle vectors (arrows) are weak and the resultant altered balance of forces (lower illustration in Figure 3) shifts to the anterior muscle vectors. Increased input from sensory receptors at the bladder neck "N" will then induce activation of the micturition reflex, but compensatory (or socially defensive) activation of the closure reflex maintains continence (white arrows, Figure 1). The already unbalanced forward vector, due to uncompensated activity of the PCM, over-tensions the distal vagina, closing the distal urethra more tightly (Figure 3). This causes a raised maximum urethral pressure, "catheter grabbing", and consequently an increased post-void urine volume (see Table 1), since a higher hydrostatic pressure is then needed to activate sufficient sensory afferent input to trigger the voiding response. Because the PCM relaxes immediately before micturition starts, excess distal closure tone (Figure 3) now resolves. The posterior vectors (Figure 1; large arrows) remain weak because the USLs, against which they

contract, (Figure 1) are lax. The posterior urethral wall cannot open sufficiently to reduce resistance to voiding (Poiseuille's Law; see Figure 2) before bladder contraction (exactly as shown by video urodynamics[1,3]). The detrusor then contracts against an only partially opened urethra, causing difficulty voiding or "obstructed micturition". The exponential relationship between urethral narrowing and urine flow described by Poiseuille's theorem[18-20] is core in understanding the abnormal urinary storage pattern in FS and PFS and also, probably, with regard to the more recent concept of "underactive bladder" disorder (UAB).[21-23] Similar video-urodynamic features define FS and PFS, further supporting the proposed unity of the two syndromes.

Urodynamics in idiopathic non-obstructive urinary retention

Urinary volumes in FS may exceed 1,000 ml.[2,3] Upregulated activation of the closure reflex causes increased tone in the forward vector (large arrow, Figure 3) so that afferent impulses cannot trigger the voiding reflex. The same process activates the urethral RS causing a raised maximum urethral pressure. Although only four of our patients with PFS had maximal urethral closure pressures (MUP) >90cm water, the mean MUP in these was reduced from 93 cm to 75 cm water following USL reconstruction.[8] Detrusor contraction was reduced in three women with PFS.[8] These findings are similar to those in FS.[3] In PFS, following USL reconstruction, the mean resting bladder volumes were reduced from 598 ml to 301 ml and mean emptying time from 50 seconds to 20 seconds[8] (Table 1).

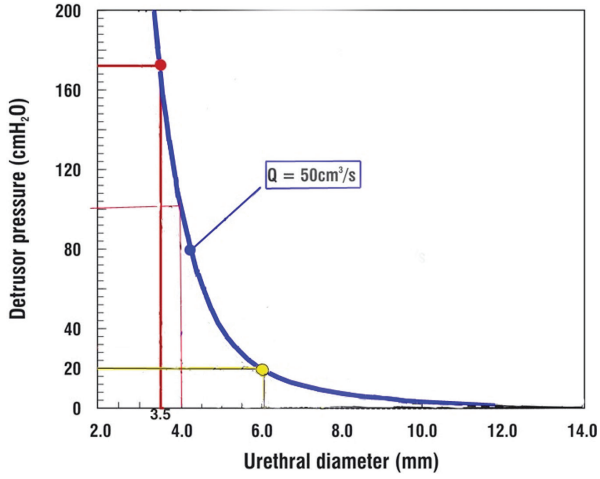

Figure 2. Exponential nature of urine flow is related to urethral diameter. For a flow rate of 50 ml/sec (thick blue line), closing the urethral diameter from 4 mm to just 3.5 mm increases the head of pressure required by the detrusor to expel urine from the bladder exponentially, from 100 cm H_2O to 172 cm. Conversely, expanding urethral diameter to 6 mm (yellow lines), reduces the head of pressure required to evacuate to only 20 cm H_2O. The blue line represents the total urethral resistance to flow, including dynamic and frictional flow components
Q: Flow

Table 1. Pre-operative and post-operative results (after TFS reinforcement of USLs) in 24 women with posterior fornix syndrome

	Pre-op	Post-op	p value
PVR	272 ml (100-630)	34 ml (0-150)	<0.0001
Abnormal bladder emptying (24 patients)	100%	9 100% cured 9 80% cured 2 50% improved 3 failed	<0.0001
Natural bladder volume	Mean 593 mi	Mean 301 ml	0.0001
Voiding time	Mean 50 sec	Mean 20 sec	0.006
Peak urine flow rate	42 ml/sec	37 ml/sec	NS
Chronic pelvic pain VAS scale assessment	18/24	14 improved 80%	<0.0001
Max urethral closure pressure (4 patients)	>93 cm water	75 cm water	NS
Urge	17/24 3-4 wet episodes/day (range: 1-8/day)	13/17 cured 3 episodes/day (range: 1-8) in 4 patients	<0.0015
Frequency	Mean 12/day (range: 5-21) 14 voided >14 times/day	Mean 8.5/day (range: 5-13) 8 voided >8/day	0.015
Nocturia	2 or more/night in all 24 patients. Mean 4.6 (range: 2-9)	13 cured 10 failures mean 2.7/night (range: 2-6) Host to F/U	<0.0001
Excess detrusor activity	4 patients	1 patient	-

TFS: Tissue fixation system; USL: Uterosacral ligaments; PVR: Post-void residual; VAS: Visual anolog scale; NS: Not significant; sec: Second, Pre-op: Preoperative; Post-op: Postoperative, Max: Maximum, F/U: Follow-up

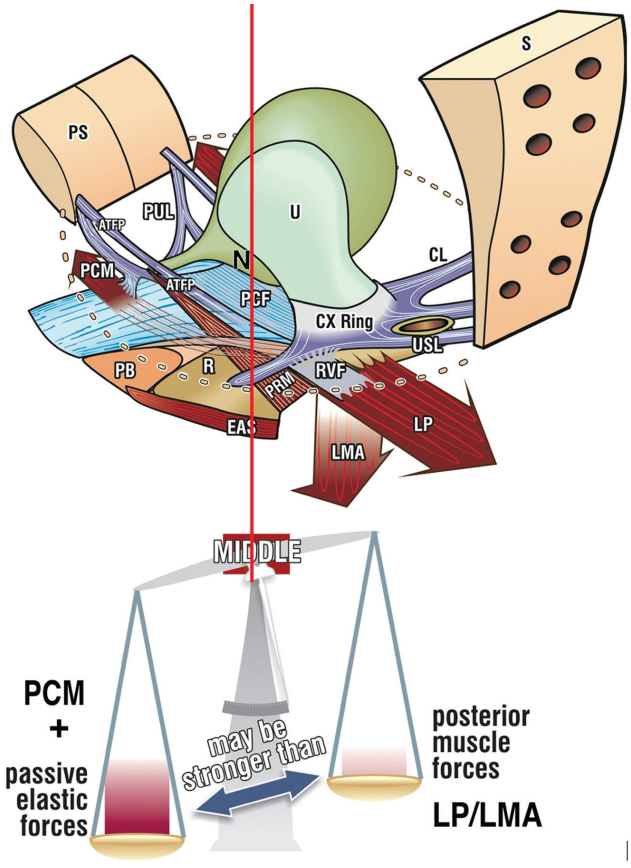

Figure 3. The opposing muscle vectors are normally always in balance in the pelvic floor (red vertical line). The posterior vectors require a firm uterosacral ligamentous (USL) insertion point. If USLs are lax, the posterior vectors weaken and the system loses balance, moving forward (to the left of the red vertical line). PCM compensates by contracting forwards.
N: Bladder base stretch receptors; PCM: Pubococcygeus muscle; LP: Levator plate; LMA: Longitudinal muscle of the anus; PUL: Pubourethral ligament (After Petros PE, The Female Pelvic Floor); PS: Pubic symphysis; S: Sacrum, U: Uterus

Comorbidities in idiopathic non-obstructive urinary retention (FS)

Hoeritzauer et al.[9] studied comorbidities in a chart review of 62 patients with voiding difficulty or retention all of whom had clinical and sphincteric EMG features considered at that time to be diagnostic of FS.[1-3] Of these, 31 (50%) had unexplained chronic pain syndromes. Of the latter, 12 (19%) were taking opiates for unexplained, predominantly abdomino-pelvic, pain syndromes. Fifteen (24%) had other unexplained neurological symptoms, such as non-epileptic attacks and non-organic leg weakness. Abdomino-pelvic surgery seemed to have triggered FS onset in 21 (35%). Ongoing psychological symptoms were recognised in 31%. Only 19 (30%) had no psychological co-morbidity, or pelvic pain. In patients presenting with functional neurological disorders, most of whom are female, lower urinary tract symptoms, including overactive bladder (OAB) syndrome and low flow rate, are frequent.[9] These complex comorbidities once again raise the unresolved issue as to whether difficulty voiding in young women is an entity due to pelvic floor instability, or whether psychological stress is primarily causative. It seems more likely to us, however, that these non-organic features are secondary to the stress of the dysfunctional bladder symptoms.[9,10,24]

Chronic pelvic pain

Pelvic pain was not part of the original description by Fowler et al.[1] However, Hoeritzauer et al[9] found that 50% of FS patients at Queen Square had chronic pelvic pain. Idiopathic pelvic pain, sometimes termed "perineal neuralgia", has long been a difficult clinical problem. Heinrich Martius,[25] writing in the German literature in 1938, suggested that chronic pelvic pain was due to laxity in the USLs causing loss of support to the ganglion of Frankenhauser (T11-L2), located near the cervix,[26] and of the sacral plexus (S2-4) (Figure 4). The anatomy of the ganglion of Frankenhauser[26] and the sacral plexus, important components of the innervation of the uterus and other pelvic organs, was described by Langley and Anderson[27] in 1896. Pelvic pain is common in PFS. In 1996, Petros reported a 70% cure rate of chronic pelvic pain 12 months after USL plication with native tissue USL repair.[28] Since then immediate relief has been reported with a posterior sling procedure that reinforces the USLs.[12,28-31]

Unifying Posterior Fornix Syndrome and Fowler's syndrome

Abnormal bladder emptying, chronic pelvic pain and high post-void residual urine, symptoms described in FS, whether or not defined by the now redundant EMG finding, are also features consistent with PFS.[11] The characteristic symptoms of PFS are urge, nocturia, abnormal bladder emptying with high post-void residual urine, and a frequent association with chronic pelvic pain. We have previously argued that PFS is indistinguishable from urinary retention in young women, as described in FS.[8] Indeed, the two syndromes are likely one and the same; both are relieved following restoration of pelvic muscle balance by surgical reconstruction of the USLs utilising a posterior sling procedure (Figure 2). In our patients diagnosed with PFS the fundamental diagnostic criteria for FS and PFS were normalized, even curing self-catheterizing retention.[14,18]

Implications

FS and PFS are two of a group of disorders of urine storage and voiding of uncertain pathogenesis; others include OAB syndrome, UAB syndrome, detrusor underactivity (DU), urgency/incontinence, and urgency/frequency. We consider that FS as

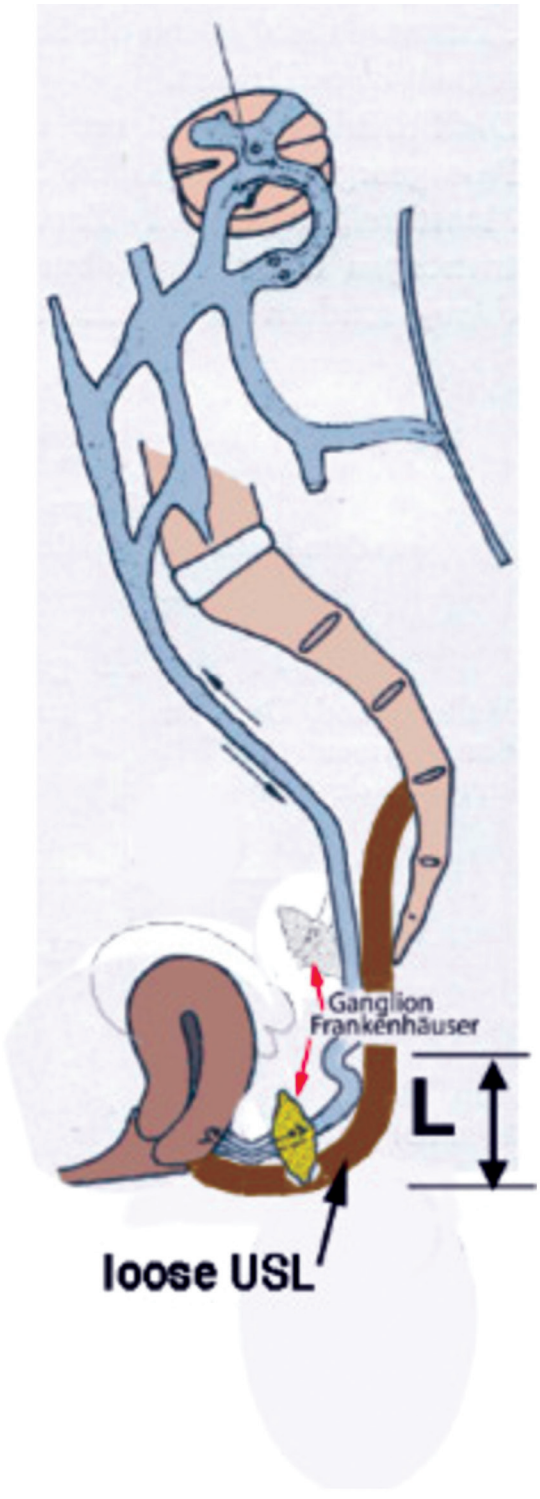

Figure 4. Pathogenesis of chronic pelvic pain of unknown origin After Martius[25] The Ganglion of Frankenhauser and the Sacral Plexus are supported by the uterine insertion of the uterosacral ligaments (USL). When the USLs are lax (L), these sympathetic and parasympathetic nervous structures are vulnerable to stimulation by gravity, prolapse or intercourse causing pain. Applying the bottom blade of a bivalve speculum to stretch the lax USLs usually provides sufficient mechanical support to the USLs to relieve urgency or chronic pelvic pain

currently defined,[3,9] is identical to the previously reported PFS, of organic causation, and usually due to USL laxity. Not surprisingly PFS, like FS, is frequently associated with psychological distress. All the features of PFS are sustainably cured or much improved after simple USL reconstruction,[8] preferably with the relatively non-invasive posterior sling procedure.[8,12] A multicentre trial of various procedures involving 616 patients[24] reported that USL reconstruction greatly improved pelvic pain in 79% of 197 patients. Other PFS symptoms were also improved.[31,32] For example, urge incontinence was improved in 86%, frequency in 84%, nocturia in 69%, and the infrequent associated faecal incontinence in 65%. This marked improvement in comorbidities of chronic pelvic pain after restoration of the uterosacral ligamentous anatomy by USL reconstruction implies that these symptoms are likely to be due to the underlying anatomical disorder.[9] Whether it is helpful to classify FS and PFS in the category of recent descriptions of the bladder/detrusor underactivity (UAB/DU) complex,[22,23] in which OAB is regarded as an essential component of UAB, but presenting with urinary retention, is open to research and discussion.

In PFS/FS, USL laxity is mechanical, as demonstrated by improvement in urinary retention and emptying after surgery, even in women with little or no prolapse.[8,14,15] The mechanical origin of USL laxity can be demonstrated by inserting a cylindrical or rubber ring pessary into the posterior fornix; symptoms of chronic pain, urge, nocturia and emptying will then immediately improve. In young nulliparous women with FS/PFS ligamentous laxity may be congenital although, so far, this has not been studied. If it occurs following pregnancy, the cause is most likely overstretching of component collagen of USL due to relaxin-induced depolymerisation.[33] or, if after delivery, overstretching by the fetal head. After the menopause, it may result from leaching of collagen from pelvic floor ligaments.

Other methods of management

Non-surgical management by pelvic floor physiotherapy using a squatting protocol[34] has achieved >50% improvement in OAB, nocturia, bladder emptying symptoms and reducing post-void residual bladder volume. However, uncontrolled observations such as these are difficult to interpret. Psychotherapy, similarly, is difficult to assess, but is generally considered unhelpful. Certainly, relief of associated depression and anxiety is an important aspect of overall management of non-obstructive voiding problems.[9,24]

Sacral neuromodulatory therapy has a long history in management of voiding dysfunction.[35] It has been reported effective in urge incontinence (68%), urgency/frequency (56%)

and non-obstructive retention (71%), whether or not defined by the previously accepted EMG feature of FS.[3,36,37] This treatment has also been used in OAB, interstitial cystitis, chronic pelvic pain, fecal incontinence, constipation and neurogenic bladder disorders. Since sacral neuromodulatory therapy appears helpful for such a wide variety of disordered bladder disorders, it has been suggested that it somehow resets brainstem autoregulation of bladder emptying and storage. It is not a specific therapy, but it is clinically indicated when the functional disorder is refractory to other therapies. Its use in non-obstructive urinary retention and other voiding disorders is not informative with regard to pathogenesis, although it does imply a disorder of neural network coordination, whether primary or secondary.

CONCLUSION

FS/PFS are part of a spectrum of related urinary voiding and retention syndromes. The relationship between USL laxity and cure or improvement of urinary retention by ligament-based surgery, native tissue ligament repair or posterior sling procedures is well established. Physiotherapy and other conservative strategies also have a place. Fowler's reports, 20-30 years ago, were important in stimulating awareness and research into non-obstructive urinary retention in young women. However, it is likely there are both local pelvic floor and central neurological causations.

Ethics

Peer-review: Externally peer-reviewed.

DISCLOSURES

Conflict of Interest: There are no conflicts of interest.

Financial Disclosure: The authors of the publication did not receive financial support from any grant/research sponsor or commercial organisation.

REFERENCES

1. Fowler CJ, Christmas TJ, Chapple CR. Parkhouse HF, Kirby AS, Jacobs HS. Abnormal electromyographic activity of the urethral sphincter, voiding dysfunction, and polycystic ovaries: a new syndrome? BMJ. 1988;297:1436-1438.
2. Swinn MJ, Wiseman OJ, Lowe E, Fowler CJ. The cause and natural history of isolated urinary retention in yong women. J Urol 2002;167:151-156
3. Panicker JN, Pakzad M, Fowler Cl. Fowler's syndrome: a primary disorder of urethral sphincter relaxation. The Obstetrician and Gynaecologist 2018;20:95-100.DOI:10.1111/tog.12448
4. Fowler CJ, Kirby RS, Harrison MJG. Decelerating bursts and complex repetitive discharges in the striated muscle of the urethral sphincter, associated with urinary retention in women. J Neurol Neurosurg Psychiatry 1985;48:1004-1009
5. Tawadros C, Burnett G, Derbyshire LF, Tawadros T, Clarke NW, Betts CD. External urinary sphincter electromyography in asymptomatic women and the influence of the menstrual cycle. BJU Int. 2015;116:423-431
6. Fellows LK, Foster BJ, Chalk CH. Clinical significance of complex repetitive discharges: a case-control study. Muscle Nerve 2003;28:504-507
7. Trontelj J, Stålberg E. Bizarre repetitive discharges recorded with single fibre EMG. J Neurol Neurosurg Psychiatry 1983;46:310-316
8. Petros PE, Abendstein B, Swash M. Retention of urine in women is alleviated by uterosacral ligament repair: implications for Fowler's syndrome, Cent European J Urol. 2018; 4:436-443
9. Hoeritzauer I, Stone J, Fowler C, Elneil-Coker S, Canon A, Panicker J. Fowler's syndrome of urinary retention: a retrospective study of co-morbidity. NeuroUrol Urodyn. 2016; 35: 601-603.
10. Stone J, Hoeritzauer I, Joyce E, Pakzad M, Selai C, Panicker J. Chronic urinary retention; Fowler's syndrome (v1 Aug 2018). pp1-10. www.neurosymptoms.org (accessed June 2020)
11. Petros PE, Ulmsten U. The posterior fornix syndrome: a multiple symptom complex of pelvic pain and abnormal urinary symptoms deriving from laxity in the posterior fornix. Scand J Urol Nephrol. 1993;27(Suppl 153):89-93.
12. Petros P, Richardson PA. TFS posterior sling improves overactive bladder, pelvic pain and abnormal emptying, even with minor prolapse. A prospective urodynamic study. Pelviperineology. 2010;29:52-55.
13. Petros P, Lynch W, Bush M. Surgical repair of uterosacral/cardinal ligaments in the older female using the tissue fixation system improves symptoms of obstructed micturition and residual urine. Pelviperineology 2015;34:112-116
14. Petros P, Richardson PA. TFS posterior sling improves overactive bladder, pelvic pain and abnormal emptying, even with minor prolapse. A prospective urodynamic study. Pelviperineology. 2010;29:52-55.
15. Petros PE. Detrusor instability and low compliance may represent different levels of disturbance in peripheral feedback control of the micturition reflex. Neurourol and Urod 1999;18:81-91.
16. Petros PE and Ulmsten U. Role of the pelvic floor in bladder neck opening and closure: I muscle forces. Int J Urogynecol and Pelvic Floor 1997;8:74-80.
17. Petros PE and Ulmsten U. Role of the pelvic floor in bladder neck opening and closure: II vagina. Int J Urogynecol and Pelvic Floor 1997;8:69-73
18. Swash M, Petros P. The pelvic floor: neurocontrol and functional concepts. In GA Santoro et al (eds). Pelvic Floor Disorders. Zurich, Springer Nature AG. 2020 Chapter 4; in press. https://org/10.1007/978-3-030-40862-6_4

19. Bush MB, Petros PEP, Barrett- Lennard BR. On the flow through the human urethra. Biomechanics 1997;30:967-969.

20. Petros PE and Bush MB. A mathematical model of micturition gives new insights into pressure measurement and function. Int J Urogynecol 1998;9:103-107

21. Wagenlehner F, Liedl B, Petros P. A finite element model validates an external mechanism for opening the urethral tube prior to micturition in the female. World J Urol 2015;33:1151-1157

22. Osman NI, Esperto F, Chapple CR. Detrusor Underactivity and the Underactive Bladder: A Systematic Review of Preclinical and Clinical Studies. Eur Urol. 2018;74:633–43.

23. Uren AD, Cotterill N, Harding C, Hillary C, Chapple C, Klaver M, et al. Qualitative exploration of the patient experience of underactive bladder. Eur Urol. 2017;72:402–7.

24. Goeschen K. Role of uterosacral ligaments in the causation and cure of chronic pelvic pain syndrome. Pelviperineology 2015;34:2-20

25. Martius H. Über einen häufigen gynäkologischen Symptomkomplex. Archiv Gynäkol Obstet 1938;166:332-335.

26. Frankenhausen F. Die Nerven der Gebaermutter und ihre Endigungin de glatter Muskelfasern. Ein Beitrag zur Anatomie und Gynaekologie. Ed Mauke F, Jena. 1967;1-82

27. Langley JN, Anderson HJK. The innervation of the pelvic and adjoining viscera. Part 7: anatomical olbservations. J Physiol (Lond) 1896;20:372-406

28. Petros PE Severe chronic pelvic pain in women may be caused by ligamentous laxity in the posterior fornix of the vagina. Aust NZ J Obstet Gynaecol 1996;36:3:351-354.

29. Wagenlehner F, Muller-Funogea I, Perletti GP, Abendstein B, Goeschen K et al Vaginal apical prolapse repair using two different sling techniques improves chronic pelvic pain, urgency and nocturia – a multicentre study of 1420 patients Pelviperineology 2016;35:99-104

30. Goeschen K. Posterior Fornix Syndrome: Comparison of original (2004) and modified (2015) post-PIVS anatomic and symptomatic results: a personal journey. Pelviperineology 2015;34:85-91.

31. Goeschen K. Role of uterosacral ligaments in the causation and cure of chronic pelvic pain syndrome. Pelviperineology 2015;34:2-20

32. Liedl B, Goeschen K, Yassouridis A, Inoue H, Abendstein B et al. Cure of Underactive and Overactive Bladder Symptoms in Women by 1,671 Apical Sling Operations Gives Fresh Insights into Pathogenesis and Need for Definition Change. Urol Int 2019;103:228-234

33. Downing SI, Sherwood OD. The physiological role of relaxin in the pregnant rat. IV the influence of relaxin on cervical collagen and glycosaminoglycans. Endocrinology 1986;118:471-479

34. Skilling PM, Petros PE Synergistic non-surgical management of pelvic floor dysfunction: second report. Int J Urogynecol 2004;15:106-110

35. Steele SS. Commentary. Sacral nerve stimulation: 50 years in the making. Can Urol Assoc J. 2012;6:231-232

36. De Ridder D, Ost D, Bruyninckx F. The presence of Fowler's syndrome predicts successful long-term outcome of sacral nerve stimulation in women with urinary retention. Europ Urol 2007;51:229-234

37. Van Kerrebroeck PEV, van Voskullen AC, Heesakkers JPFA, Lycklama A, Nyjholt AAB, Siegel S et al. Results of neuromodulation therapy for urinary voiding dysfunction: outcomes of a prospective, worldwide clinical study. J Urol 2007;178:2029-2034