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Urodynamic features of the pelvic pain patient and the impact of neurostimulation on these parameters

Pelvic pain is among the most challenging frontiers that remain in medicine. It is amorphously subjective and intrinsically difficult to measure, localize, or assess. Its disabling nature can be deceptive and vary enormously from patient to patient. That so many of the patients have overlapping or associated emotional problems which require a great deal of time and understanding does not make the task of a busy surgeon any easier.

In general, lower urinary tract (LUT) dysfunction may be attributable to any disease process or injury that affects the neural reflexes governing urine storage or release [1, 2, 7, 17, 19]. Many clinical conditions affecting the LUT are poorly understood. Chief among these is the problem of pain. The actual incidence of pelvic pain is elusive, but there can be little doubt that it affects a significant percentage of the urogynecological patient population. It is often overlooked as a component of other complaints, or included as part of any one of a number of labels. Thus syndromes described as recurrent cystitis, endometriosis, urgency frequency, urge incontinence, retention syndromes, chronic constipation, frequent loose stools, and abnormal periods often harbor significant components of pelvic pain. Along with the descriptive symptoms, there is commonly a global hypersensitivity of pelvic tissues, but in particular, the pelvic floor and bladder neck. This is typical of the conditions urethral syndrome, interstitial cystitis, endometriosis, vulvodynia, prostatodynia, dysparunia, and proctalgia fulgax.

The underlying cause of ''pain'' can be very difficult to discover, given the complexity of the central and peripheral neural system, and the fact that pathophysiological changes at one site in the nervous system, or target organ, are generally tied to changes in other sites or organs.

Pain is a symptom that historically has been difficult to quantify and hence difficult to treat. This can be frustrating for patient and physician alike. However, there is hope. The fact that pain is commonly associated with tangible behavior and physical findings provides an opportunity to quantify the degree of neuroregulatory disturbance which often is the source of pain. Effective management of the LUT dysfunction can provide a guideline path for managing pain. This concept follows the basic principle that sensory disturbances are generally mirrored in motor behaviors. Through quantifying and treating the motor dysfunction, relief of the sensory disturbance often is achieved.

The pelvic floor

The anatomy of the pelvic floor encompasses two functional layers. The deeper layer, or levator, and its various components, produces a bellows-like visible deepening and flattening of the buttock crease, in everyone, with voluntary tightening and relaxation efforts. Contraction of the levator can be induced via stimulation of the third and fourth sacral nerves. The pelvic organs are then lifted upward and anterior toward the pubis.

The second functional layer of the pelvic floor is composed of all of the muscles below the levator – the transversus perinea, ileo- and bulbo-cavernosus muscles, the urethral sphincter, and the superficial anal sphincter. These muscles contract in an anterior–posterior direction. They are innervated by the pudendal nerve, which is primarily derived from S2.

Although, the two motions, bellows and clamp, are distinct, they normally will blend together. However, in voiding dysfunction, the movements can become fractionated, absent, or dysphasic, relative to each other and to the bladder. This knowledge can be used for a more informative rectal examination.

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The rectal examination

Voluntary attempts to tighten and relax the perineum should be evaluated as part of the routine clinical assessment of all patients. This examination can be done with the patient lying on one side, or, while standing, flexed at the hips and leaning against an examination table.

Patients should be able to produce on request an immediate, unhesitating identity with the pelvic floor. Contraction and relaxation of the pelvic floor muscles should be clearly evident. The movement should be effortless and free of extraneous muscular activity (i.e., abdominal tightening, Valsalva, pelvic tilt, etc). There should be a visible deepening and flattening of the buttock groove (with contraction and relaxation of the levator). The movement should be repeatable with consistency, and the contraction should be sustainable for 2–3 s before relaxation is permitted. The buttocks should then be slightly separated and the anal skin observed for wrinkling with a hold attempt, along with a front to back squeeze of the superficial sphincter. (An easier way to check the functional integrity of these pudendal innervated perineal muscles is to observe contraction of the base of the penis during the hold/relaxation effort.)

Consistency and equitability of the muscular contraction should be noted. Any time separation between the levator and superficial sphincter contractions should be noted, as well as any deficiency in hold or relaxation effort. Also, inappropriate twitches or fibrillations in anal muscle fibers should be carefully noted.

A final assessment is then performed via the rectal examination. Before focusing on anatomical aspects of the examination, the tonus of the deep and superficial anal sphincter should be noted. The following questions might be addressed: (1) Is the tonus of the sphincter normal, weak or high? (2) Is there tenderness of the levator postero-laterally on either side? (3) Is there tenderness of the prostate apex, base, or of the external sphincter? A voluntary contraction effort against the examining finger is then requested. Muscle contraction is assessed for strength, consistency, and repeatability. Is there a distinct contraction as well as relaxation above and below the basal tone? Is the levator edge distinct or mushy? Is there movement in both the levator and pudendal sphincter components?

Clinically it is helpful to recognize degrees of inefficiency that effect the behavior of the pelvic floor between voids as well as during the void. Patients with pelvic pain syndromes often have hypertonic and hypersensitive sphincters. As the conscious aspects of pelvic floor recognition and control must be evolved, as with any coordinated muscle movement, efforts can be directed toward the learning and conditioning aspects underlying correct pelvic floor control. This is a natural complement to additional forms of therapy.

Urodynamic changes

Table 1 and Table 2 summarize the urodynamic findings from a group of 179 women with refractory pelvic pain. The urodynamic tracings in these patients were similar to those previous described [21. 24]. Using a dual microtip transducer recording method, high pressures, exaggerated sensitivity, and flagrant hyper-reflexic, unstable reflex activity were noted at the level of the external urethal sphincter during filling. Hyperalgesia and instability of the striated urethral sphincter was especially evident in pelvic pain patients. In addition, there were variations in many of the other urodynamic parameters, i.e., bladder sensitivity, compliance, and capacity, as well as the urinary flow and efficiency of emptying. As the vast majority demonstrated significant abnormalities either prior to, or during the void, dynamic recordings of urethral behavior were found to be highly useful in documenting the disturbance in LUT function [6]. Also, as a group, these patients were (are) very inefficient in the exercise of voluntary control over

Table 1 Urodynamic findings in 179 female pelvic pain patients. Note departure of bladder and urethral external sphincter from suggested normal ranges (*N*)

Variable	Average	Range	Standard deviation
Age (years)	40	16–80 years	12
Urethral sphincter pressure (N = 60–70 cm H_20)	87	153–17	26
Bladder capacity (N = 350–500cc)	297 cc	649–50	131
Urethral sensitivity (0 = Absent, Mild = 1, Severe = 2)	1.5	0–2	0.67

Table 2 Urodynamic findings in 185 female pelvic pain patients. Note departure of bladder and urethral external sphincter from suggested normal ranges (*N*)

Variable	Average	Range	Standard deviation
Age (years)	39	20–80 years	12
Urethral sphincter pressure (N = 60–70 cm H_20)	86	153–17	26
Bladder capacity (N = 350–500 cc)	298 cc	649–50	131
Urethral hypo-/hypersensitivity (0 = Absent, Mild = 1, Severe = 2)	1.5	0–2	0.67

Table 3 Peak urethral sphincter pressures by decade in female pelvic pain patients (recorded using a microtip transducer type catheter). *Pk Ur Pres* peak urethral sphincter pressures. Data Source: Initial urodynamic recordings from pelvic pain patients. Up-regulation in tone and sensation predominates in all age grouaps. There is a gradual loss of sphincter tone with each decade

Age range (years)	Average (cm H_2O)	Range (cm H_20)	Standard deviation	Number and percentage of patients (total = 179) or percentage of age group	
				(*n*)	(%)
16–50[a]				148	83
16–19	108	130–91	19	5	3
20–29	95	131–35	23	28	16
20–29 (Pk Ur Press > 70)	102	131–74	15	24	85 (of age group)
30–39	92	150–42	24	69	38
30–39 (Pk Ur Press > 70)	99	150–71	19	56	80 (of age group)
40–49	83	138–31	26	44	25
40–49 (Pk Ur Press > 70)	96	138–72	18	32	68 (of age group)
50–59	74	153–49	25	19	11
50–59 (Pk Ur Press > 70)	90	153–71	24	10	50 (of age group)
60–69	70	97–46	20	8	4
60–69 (Pk Ur Press > 70)	92	97–82	8	3	38 (of age group)
70–80	64	135–17	43	6	3
70–80 (Pk Ur Press > 70)	110	135–84	110	2	33 (of age group)

[a] Average is 35 ± 8 years

the the sphincter and pelvic floor. The majority (83%) of the patients were under the age of 50. The average urethral pressures declined with each decade, as did the percentage of patients in each decade with pressures above 70 cm H20. The peak age group of patients was between 30 and 39 years. This observation is consistent with a progressive deterioration in sphincter tone over time, due to ongoing dysfunctional activity. However, longitudinal studies would be needed to confirm this.

Effect of modulation on urodynamic parameters

Urodynamic studies were available in 15 patients both prior to a sacral foramen implant and 6–12 months after activating the implanted stimulators (Tables 3, 4). All patients had significant voiding dysfunction and pelvic pain. All had received a better than 50% improvement in all symptoms during a screening trial. All subsequently benefited from their implant, though in varying degrees. Comparing the urodynamic studies among the patients as a group proved inherently problematic. Selected graphs before and after initiating neurostimulation therapy for individual patients could have been presented. However, this would not necessarily reflect the responses in the patients as a group and could not include all aspects of the studies. As such, the averages and standard deviations were used for the group as a whole. Extreme cases, such as the very good responder and the poor responder, could also have been broken out of this data pool. However, the point of presenting pooled data is to demonstrate the overall impact neurostimulation has on urodynamic parameters in many patients. Clearly, as a group there was a distinct improvement noted in overall LUT function. In females this was mostly demonstrated as an improvement in bladder capacity, while in the four males available, the improvement was seen more in the urethral closure pressures. The difference was most probably related to the prevalence of urgency/frequency type symptoms in the females and retention or hesitancy symptoms in the males.

Response to stimulation was most effective in those with urodynamically demonstrable pelvic floor dysfunction (instability, peak urethral pressures > 100 cm H_2O, or poor sphincter relaxation and dyssynergic voiding). This relief, as a rule, was sustained only with continued neurostimulation, hence the need for a permanently implanted electrode and pulse generator. The overall performance of the bladder was improved. This

Table 4 Peak urethral sphincter pressures by decade in female pelvic pain patients

Age range (Years)	Average	Range (cm H_2O)	Standard deviation	No/% of 185 Patients or Age Group
20–50	36 years	20–50 years	7 years	145/78%
	cmH_2O	cmH_2O	cmH_2O	
20–29	95	131–35	23	28/15%
20–29 (Pk Ur Press > 70)	102	131–74	15	24 (85% of Age Group)
30–39	91	150–42	24	70/38%
30–39 (Pk Ur Press > 70)	99	150–71	19	56 (80% of Age Group)
40–49	83	138–31	25	47/25%
40–49 (Pk Ur Press > 70)	96	138–72	18	32 (68% of Age Group)

was evident in the bladder capacities measured by natural fill (flow rates volumes) and routine cystometrogram recording, and by the capacity measured at the maximum compliance. By implication, there was an overall improvement in micturition reflex regulation. Bladder capacities moved toward a normal range. If they were high, they came down. If they were low, they increased. Similar observations were evident for the urethral sphincter pressures. The benefit of neuromodulation is the shift toward normal behavior. If a small shift is required, the changes in parameters would not be expected to be great (Figs. 1, 2). Much of the benefit would be on the sensory level or in information processing centrally. However, if the behavior was clearly anomalous, then benefits of modulation would be more apparent in the shift in urodynamic parameters (Figs. 3–5).

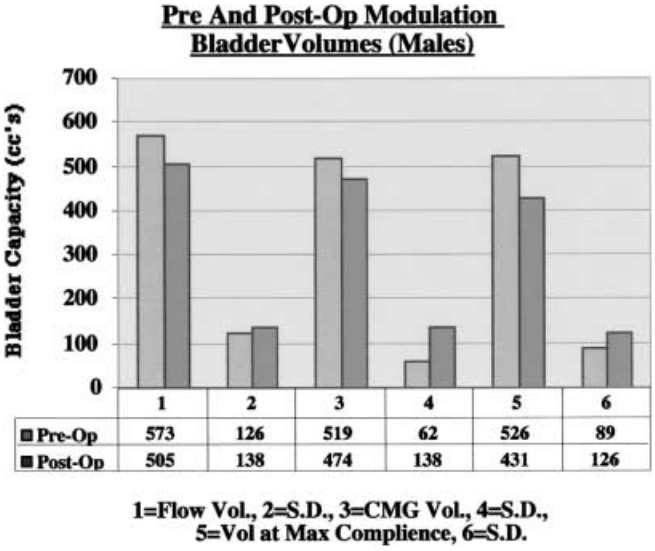

Fig. 2 Female urethral pressures and respective standard deviations, recorded before and after 6–12 months of modulation therapy achieved via a foramen electrode implant

Discussion

The LUT is a sensitive indicator of any gross or occult deficiency in neuroregulation. This is because of a rather extensive representation of the LUT within several brainstem nuclei. As such, virtually any neurological compromise, whether of metabolic or anatomical nature, related to injury, disease or developmental origins, can compromise behavior of the LUT [5, 8, 14, 15, 25]. The type of dysfunction that occurs as a rule reflects a loss of inhibitory gating. There is an up-regulation in peripheral reflex activity with consequent spasticity and/or hypersensitivity.

A denominator common to almost all patients with dysfunctional voiding, with or without a pelvic pain complaint, is an inability to selectively contract or relax the pelvic floor on command [21]. Furthermore, voiding difficulties can often be linked to troublesome symptoms in childhood. There is therefore a question of toileting habit and how it may impact the integrity of the LUT over time. Even if there are subtle, causal neurological deficiencies, learned behavior remains an issue. Learned toileting behavior, or perhaps the lack of learning approaches, could profoundly influence the integrity of the LUT in later life. The basis for this thinking comes from rules of behavior that influence all tissues in the body.

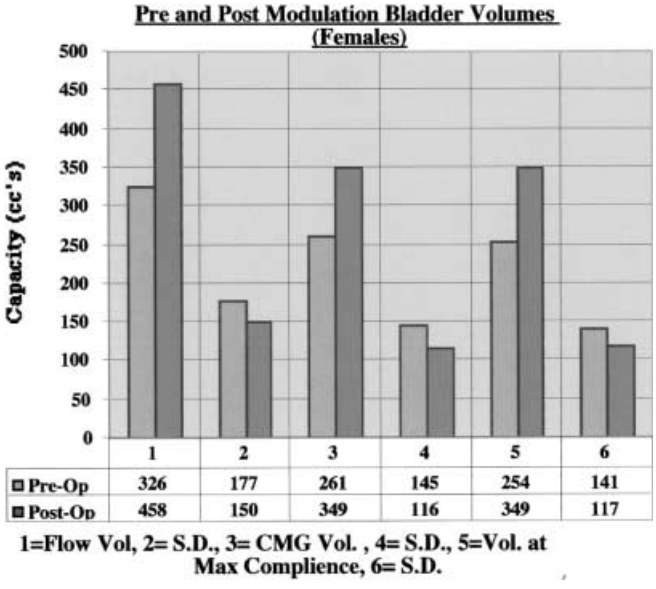

Fig. 1 Male bladder capacities and respective standard deviations, recorded via flow rate (natural fill) routine cystometrogram and the recorded volume with maximum compliance

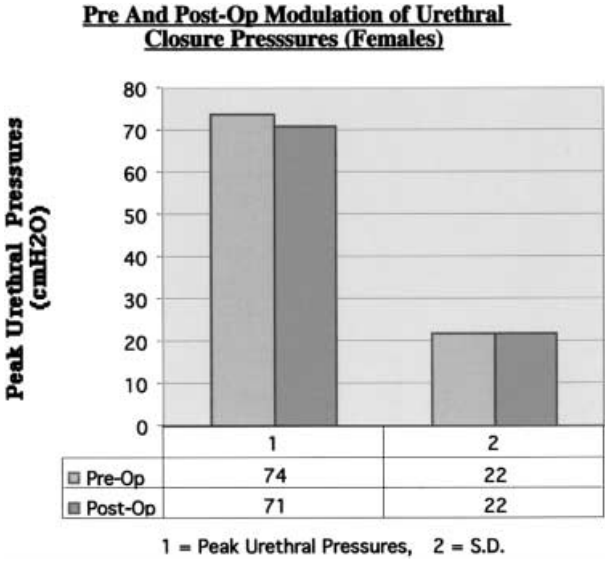

Fig. 3 Female bladder capacities and respective standard deviations recorded via flow rate (natural fill) routine cystometrogram and the recorded volume at maximum compliance

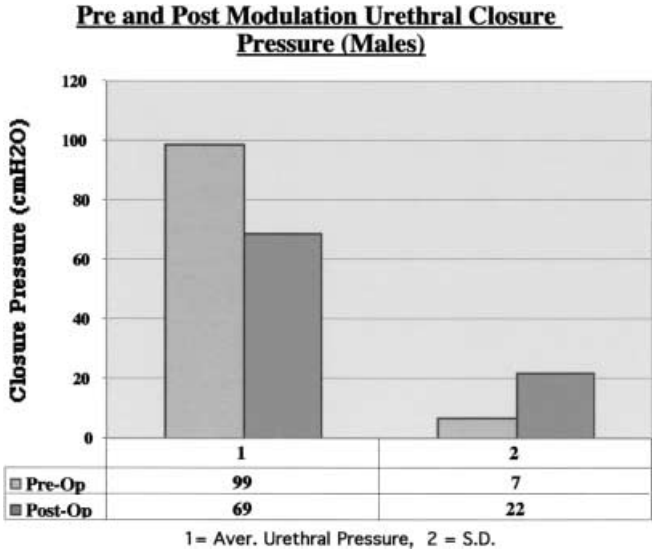

Fig. 4 Male urethral pressures and respective standard deviations, recorded before and after 6–12 months of modulation therapy achieved via a foramen electrode implant

In this regard, it is known that repetitive inappropriate afferent feedback to the CNS can be extremely harmful to delicate central regulatory circuits [4, 11, 12, 18, 27, 28]. Persistent CNS exposure to noxious neural events, e.g., any repetitive noxious use of the muscle with exercise (straining with each void effort or chronic holding), especially when coupled with chronic anxiety, can trigger a breakdown of the normal inhibitory gating within CNS regulatory pathways. This is a phenomena known as ''wind-up''. This term stems from neurophysiological studies on C-fibers exposed to a repetitive noxious stimulus [18]. These studies have shown that C-fiber responses progressively augment (i.e., the degree of membrane depolarization) with each noxious stimulus. Eventually the C-fibers no longer repolarize back to baseline and they remain in a ''brittle'' activity state. Permanent changes in the nociceptor occur and, clinically, tissues become tender to touch or are associated with chronic pain. A similar consequence, known as ''sensitization'', can occur after actual injury to a nerve [28].

Communicative changes within the CNS can subsequently occur [6, 8, 13, 26, 29], or, if pre-existing, may be aggravated. Some of the more commonly described events include: (a) architectural changes in spinal circuits, such as an increase in dendritic synaptic communication with other spinal cells, (b) an increased amount of neurotransmitter release into a synaptic space, which then spills over to neighboring synapses causing activity or ''crosstalk'' in neural pathways that would normally be silent [26], (c)sensory inputs from visceral organs can ''converge'' with central pathways carrying somatic information [6], resulting in referred pains to somatic dermatomes, and (d) wind-up can occur within the dorsal root laminations, as well as at other levels within the CNS [18]. There is an overall decrease in the usual filtration and central gating of sensory inputs. Instead of afferent sensory information being contained within a particular dermatome, the sensory inputs now trigger responses in adjacent pathways, potentially activating neural pathways two or four segments above or below the primary cord input level. This observation is appropriately referred to as expansion of a receptive field. It is most obvious in the presence of CNS injury, but can occur without actual anatomic neural injury as well.

Progressive chaos in sensory processing would eventually result in the emergence of irritative voiding symptoms and dyssynergic voiding efforts. Chronic LUT dysfunction could become permanently disrupted

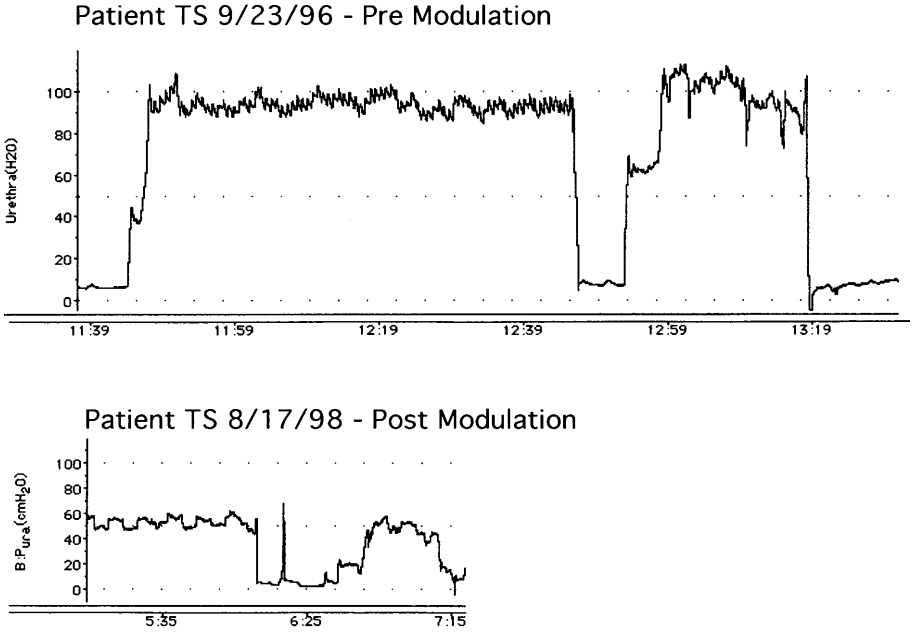

Fig. 5 The urethral pressure profile from a female pelvic pain patient at two points in time. The first was performed prior to implant of an S3 foramen electrode, at the age of 35 years. The second study was performed 3 years later, after an intensive period of modulation and medication. The profile at presentation was of a high pressure. The second profile reflects a normalization of sphincter tone and more appropriate central regulation of sphincter tone

by up-regulated sacral reflex arcs. Morphologic changes in tissue would eventually result. With this scenario, the important consideration in care is not the physical alteration of tissue, but the underlying aberrant afferent – afferent interactions within the cord.

As with any striated muscle system that is overused in a repetitive manner, time becomes a factor in the generation of chronic pain. Based on the potential of known physiological consequences associated with repetitive dysfunctional striated muscle behavior, a patient with pelvic muscle dysfunction retained in the memory and chronic from a young age, can be considered at greater risk of pelvic organ failure later in life. Even neural repair from physical injury could be compromised. These are simple common sense principles that suggest an early approach to management of the dysfunctional voider.

Sensitization, convergence projection, aberrant C-fiber (sympathetic) activity or wind-up, and expansion of receptive fields are classic response properties of the CNS to nociceptive input [6]. A vicious cycle of pain and dysfunction, involving organs within a dermatome and an expanded receptive field, then results. All of this can be very erosive to the integrity of peripheral tissues. The symptoms of urethral allodynia, referred pain, frequency, incontinence, and urgency are consistent with these CNS events, as are the signs of sphincter hyperalgesia (local pain), pelvic floor tenderness and rigidity of movement, and dyssynergic voiding. The progressive loss of tone with each decade may reflect ''burn out'' of the urethral sphincter. This ''burn out'' in turn may explain the rapid drop off in presentation of pelvic pain patients after the age of 50. It is also a concept that is consistent with rising incidence of incontinence in the post 60 age group. Unchecked spasticity will negatively impact the integrity of any tissue over time.

These neural changes would also be reflected in tone, reflex excitability and tissue sensitivity that are evident on the physical exam, void diary urodynamic studies, cystoscopy, etc.

Treatment

It is especially important to appreciate that inadvertent stress, or inefficient conditioning of reflexes concerned with LUT function, could result in serious voiding dysfunction. This is especially true during the time the nervous system is maturing, i.e., early childhood or even in utero. Therefore prevention of dysfunctional voiding and/or urogenital pain syndromes should begin by addressing toileting habits of children ages 2–8 years. Relaxed timely voids should be gently encouraged and prolonged holding discouraged. Conscious regulation of voiding requires a degree of maturity achieved with the age of reason, not simply with the age at which myelination of neurons occurs. This is a logical and critically important step to the long-term prevention and management of refractory pelvic pain disorders.

Most biofeedback programs ultimately rely on the patient to consciously relearn dynamic pelvic floor regulation. If, however, there are permanent changes in the CNS, something more than intermittent short-term therapy will be necessary. Lifestyle adjustments, daily medications, modulation methods such as pelvic floor stimulation, acupuncture and implanted neurostimulators, along with periodic long-term follow-up care, all have an important role in the management of pelvic pain syndrome. The immediate goal is to restore the behavior dynamic of pelvic floor striated muscle to a level more in line with Mother Nature's design. This hypothesis is supported by the consistent improvement rates reported for modulation based therapies.

Implanted devices

Treatment of the pelvic pain prior to implant of a foramen electrode, in the patients represented in Figs. 3 4, despite the variety of pharmacological and surgical options used, was typically disappointing. With neurostimulation, the degree of pain relief was reflected in the degree of shift toward normalcy in the respective urodynamic studies. Those patients who did best were those who had identifiable pain (and dysfunction) in the pelvic muscles. Those patients reporting pelvic pain in the absence of demonstrable pelvic floor dysfunction and levator tenderness did poorly.

Those patients who benefit from neurostimulation can be helped in a cosmetically acceptable manner, that is, regardless of the time, place, need, or dependence on the neurostimulation [3, 20, 22, 23]. The modulating effect must be in the same dermatome of pain to be effective. Direct nerve stimulation is the most efficient way to elicit the necessary pelvic muscle contraction, which in turn, provides the best hope of relieving pain. Usually, with stimulation of a sacral nerve, the patient feels a pleasant pulling or tugging in the perineum. The better the muscle contraction the better the prognosis for relief. It follows that healthier nerves provide more hope for effective relief. With poor nerve coupling of the electrode, or weak pelvic muscle contraction because of poor neural integrity (e.g., post radiation) stimulation tends to produce more of a stinging, uncomfortably sensation.

A period of testing is required to assess neural integrity, to map muscle recruitment obtained with stimulation of the various sacral nerves and to determine if stimulation can be applied effectively on a continuing basis. In this procedure, an insulated needle is positioned in one or more of the sacral foramen. Stimulation is applied, gradually by increasing (ramping) the current to a comfortable intensity. Stimulation intensities will be much higher and more comfortable when good muscle contraction is obtained. If it is not, the stimulation will be unpleasant. When it is effective, tenderness will be markedly reduced in tissues during and after application

of the stimulation. A temporary electrode is then inserted into the foramen through the test needle and left in place for 3–7 days. If the patient does well during this period of trial stimulation, then there is the option of placing the electrode permanently or of waiting and repeating the trial. As a rule, very few patients who benefit from the stimulation will remain in remission from their pain once the stimulation therapy is withdrawn.

Neuromodulation is a nonspecific stimulus that causes both afferent and efferent impulses to be carried in the somatic nerves. Once they reach the spinal cord, the impulses are routed via interneurons to the detrusor motor nuclei and the pudendal nuclei in the cord and brain stem. Here, neurostimulation has an inherent down-regulating effect on CNS excitability. Mechanisms by which this occurs are several. They include a positive conditioning effect on nerve conduction, improved neural metabolism, down-regulation of facilitating peptide expression, and more efficient neural reflex regulation (i.e., gating principles).

The fact that a group of patients with pelvic pain relatively refractory to most present day therapies, could be substantially benefited with neurostimulation, is impressive. It suggests a central role for pain and inflammatory change in peripheral tissue, and a need to globally down-regulate, central and peripheral, aberrant neural activity. Consistent with the plasticity rules of the CNS, this down-regulation or suppression needs to be in place for a ''prolonged'' period of time, perhaps permanently. Properly applied, implantable neuroprosthetics are a safe, low risk approach to the management of perineal/pelvic pain.

A classification scheme for the dysfunctional, painful lower urinary tract

As ''overactive'' terminology is descriptively limited, a more precise scheme is needed to describe physical findings. Bladder and urethral sphincter (pelvic floor) behavior have three measurable components: (1) tone, (2) reflex excitability, and (3) sensitivity (TRS). The sum of the three would appropriately be described as ''normal versus the hyper- or hypopathic conditions''. Hence a TRS classification scheme is logical, using the added degrees of severity – normal (N), absent (0), moderate ($+/-1$) or severe ($+/-2$) to quantify the various components. A dysfunctional prostatodynia sphincter, mildly hypertonic (T_1), severely hyperreflexic (R_2) and severely hypersensitive (S_2) could therefore be accurately described by $T_1R_2S_2$ terminology. If the bladder is included, sensory and motor integrity could be noted as being absent (−1), normal (N) mildly abnormal (1) or severely abnormal (2). Hence, D-$T_2R_0S_2$ would appropriately describe the poorly compliant, severely hypersensitive interstitial cystitis bladder. Classifying both the bladder and outlet logically follows. Thus a painful, hypertonic, noncontracting interstitial cystitis bladder could be summed up as a Det-$T_2R_0S_2$–Sph-$T_2R_1S_2$. The relatively normal bladder, but painful urethra could be summed up as Det-$T_NR_NS_N$/Sph-$T_2R_2S_2$ (e.g., prostatodynia).

This is a suggested classification approach. As it is a form of communication, it does not necessarily have to be validated in the same fashion as do instruments geared toward diagnosis. It is relatively simple, yet the scheme is an attempt to quantify severity of patient complaints. It could be used to summarize observations made on either the physical examination or with urodynamic studies. However, time will tell if it will have value, as that value will be determined by both applicability and ease of use. Classification schemes like this are necessary as a way of accurately describing the status of the LUT and facilitating discussion regarding presentation, pathogenesis, and prognosis for individual patients. The above approach would complement the symptom score indexes presently in use.

Conclusion: a changing philosophy towards pelvic pain conditions

The concept of plastic alteration in CNS regulation of the LUT should be considered when there is an obvious lack of conscious control of pelvic floor behavior and no explainable cause of chronic pain. The physical examination and/or urodynamic studies can be used to classify the status of the LUT. Therapeutically, the goal is to ''wind down'' CNS excitability first and foremost, before moving to surgically based treatments. Pre-emptive anesthesia [10] is recommended for any and all surgeries performed where there is evidence of pelvic neural dysregulation. This approach should limit the risk of aggravating symptoms [1], and result in a marked improvement in patient care and satisfaction.

The approaches to management of pain follow four lines of care: (1) The management of behavioral and psychological stress, i.e., biofeedback programs [16], (2) the use of medications to reduce pain, anxiety, and conditioned muscle tone, (3) neuromodulation approaches [3, 8, 13, 29], and (4) surgery for identifiable abnormalities. All four are important. Surgery has its place but should not be viewed as the primary therapy. It should always be preceded by the more conservative options, as there are inherent risks in wind-up and metabolic disturbance of peripheral nerve integrity. As the pelvic organs share most of their peripheral innervation and central regulation, symptom scores, voiding diaries and urodynamic studies are recommended in all pelvic pain patients. These are relatively easy to perform and will help to quantify the motor/sensory disturbances in neural regulation of pelvic organs. They will also provide a baseline guide for response to therapy.

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