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Pelvic Floor Dysfunction A Treatment Update

New treatment modalities take advantage of the maturing relationship between pelvic floor dysfunction and chronic pelvic pain.

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The pelvic floor is comprised of muscles and fascia and has three functions: support of the pelvic organs, contraction, and relaxation. Their function is critical to proper micturition, defecation, and sexual intercourse. In the past, pelvic floor dysfunction (PFD) has been variously termed spastic pelvic floor syndrome, levator ani syndrome, proctalgia fugax, vaginismus, male chronic pelvic pain syndrome, non-neurogenic neurogenic bladder, and

coccydynia — all terms based upon the varied presenting features of the same phenomenon. Pelvic floor dysfunction may be defined as spasm or discoordination of the pelvic floor musculature. Spasm of these muscles commonly manifests with urological symptoms including poor urine stream, pelvic pain or pressure, urinary frequency and urgency, urge incontinence, and ejaculatory pain. These are the same complaints seen in patients with chronic pelvic pain (CPP) syndromes including interstitial cystitis (IC) and chronic prostatitis (CP). Other frequent co-existent symptoms include chronic constipation, lower back pain, penile, vaginal, peri-rectal pain, vulvodynia, dyspareunia, or generalized pain. Treatment of PFD, when present in IC or CP, is strongly recommended, along with bladder or bowel-directed therapy to achieve the optimal relief of symptoms. This article will review pelvic neuroanatomy, pathophysiology, PFD diagnosis, and treatment.

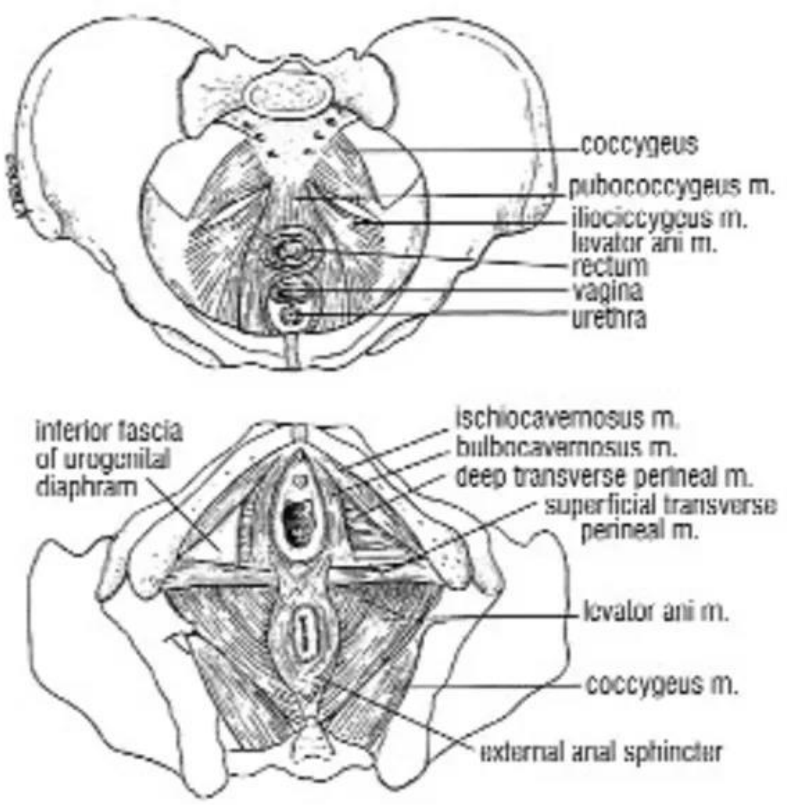

Figure 1. ***Muscles of the pelvic floor.***[24]

Anatomy

The pelvic floor muscles (PFM) include the levator ani (pubococcygeus, ileococcygeous, puborectalis), coccygeus, pyriformis, obturator and perineal muscles (see Figure 1). The levators derive circulation from the parietal branches of the internal iliac artery and innervation from sacral nerves S3 and S4, via the pudendal nerve. At rest, the PFM support the bladder and urethra in the anterior vaginal compartment, the anus and rectum in the

posterior compartment, and the cervix and uterus in the middle compartment. Like all skeletal muscles, resting tone is maintained by slow-twitch (type 1) efferent fibers, which contribute to the integrity of the proper anatomic positions of the pelvic organs, in addition to supportive fascia. These vary with hormone status, parity and body habitus.

Voluntary contraction of the pelvic floor arises from a conscious impulse, while reflex contractions occur to close the urethra, anus and vagina, to prevent urine and stool loss, and as a vaginal protective mechanism. Phasic recruitment of large motor units propagated by fast twitch (type 2) fibers occurs in response to abdominal pressure increases such as coughing. Feedback inhibition ("guarding reflex") of the detrusor muscle will result in diminution of detrusor pressure, preventing bladder contraction. For an efficient contraction, the PFMs must have strength (via recruitment) and endurance (over time). In addition, during sexual arousal and orgasm, pelvic floor muscle contraction facilitates vasocongestion and contract involuntarily, respectively.

Coordinated floor relaxation must occur before, or in conjunction with, bladder or bowel contraction and is a result of inhibition of tonically active motor units, and is needed for proper micturition, defecation, and intercourse.

Overall, PFD and chronic pelvic pain are poorly managed because they are poorly understood. They can include bladder, bowel and sexual dysfunction, as well as be associated with depression, anxiety, and drug addiction. The prevalence of PFD is not well known, yet CPP affects 1 in 7 women[1] and accounts for 10% of all outpatient visits to gynecologists,[2] while CP accounts for 8% of all visits to urologists.[3] CPP is most common among reproductive age women and men between 18-50 years. It is defined as non-menstrual pain for three months or longer, that localizes to the anatomic pelvis and is severe enough to result in disability requiring medical or surgical treatment.

Pelvic Floor Dysfunction

PFD is secondary to muscle overactivity or underactivity. Underactive PFMs contract poorly, resulting in incontinence of urine and stool, and is commonly attributed to birth trauma. Overactive PFMs can result from a variety of causes, and develops over time. They can be urologic, gynecologic, gastrointestinal, musculoskeletal, neurologic, or psychologic in nature (see Table 1). Overactive PFMs do not relax appropriately when they should, resulting in increased outlet resistance. This leads to strained voiding and incomplete emptying with poor flow, constipation, and dyspareunia. Postponing voiding or defecation is done by PFMs contraction, however chronic postponement or "rushed voiding" heightens PFMs activity. When voiding is attempted, often detrusor contraction is poor and, when abdominal straining is used to assist elimination, the guarding reflex results in PFM contraction.[4] A muscle that is constantly contracting or in spasm will generate pain. Any nerve or vessel that travels through such muscle may be compressed,[5] and may, in turn, lead to pain. Constant afferent pain

signals to the sacral cord, pons and cerebral cortex can result in efferent activity that can aggravate the pain even further.[6]

Extrauterine disorders	Endometriosis, Adhesions, Adnexal cysts, PID, Ovarian remnant syndrome, Pelvic congestion syndrome, Residual accessory ovary, Subacute salpingitis, Post-op peritoneal cysts, endosalpingiosis, Ovarian retention syndrome, Ovulatory pain, Chlamydial endometritis or salpingitis, Chronic ectopic pregnancy
Uterine disorders	Adenomyosis, Endometritis, Atypical dysmenorrheal, Cervical stenosis, Endometrial or cervical polyps, Leiomyomata, Genital prolapse, IUD
Urologic disorders	Bladder cancer, Chronic UTI, Interstitial cystitis, Radiation cystitis, Recurrent cystitis, Recurrent urethritis, Stone disease, Detrusor overactivity, Detrusor-sphincter dyssynergia, Urethral diverticulum, Chronic urethral syndrome, Urethral caruncle, Chronic prostatitis, Chronic orchalgia
Musculoskeletal disorders	Abdominal wall myofascial pain, Verterbral compression fractures, Fibromyalgia, Mechanical lower back pain, Chronic coccygeal pain, Levator ani myalgia, Piriformis syndrome, Rectus tendon strain, Hernias, Muscular strain/sprain
Gastrointestinal disorders	Colon cancer, Chronic intermittent bowel obstruction, Colitis, Chronic constipation, Diverticular disease, Inflammatory Bowel Disease, Irritable Bowel Syndrome
Neurologic disorders	Neuralgia or nerve entrapment post-op, Herpes zoster (shingles), Degenerative joint disease, Disc herniation, Spondylosis, Neoplasm of spinal cord/ sacral nerve, Abdominal epilepsy, Abdominal migraine
Psychologic disorders	Personality disorder, Depression, Anxiety, Sleep disorder, Sexual or physical abuse

Referred Pain

In response to persistent nociceptive stimulation, whether the source is visceral or somatic in origin as pain is perceived, the efferent transmission of a sympathetic response can either take one of two paths. From the intermediolateral cell column from spinal levels T1-L2, the efferent signal travels via the spinal nerve to the paravertebral sympathetic ganglion. From here efferent signals can continue on a somatic path via spinal nerves to the skeletal muscle end terminal, or continue along the visceral path via splanchnic nerves to a pre-aortic ganglion, and from there to the visceral end organ.

Visceral afferent fibers travel along the same routes as pre- and post-ganglionic fibers of both sympathetic and parasympathetic nerves. Visceral afferents are long, and do not synapse, traveling from the viscus wall to the dorsal root ganglion. They consist of A-delta and C-fibers, which are minimally or not myelinated, respectively. There is convergence or “cross-talk" of visceral and somatic afferents in the dorsal horn of the spinal cord.[7] Chronic increased afferent output can confound the local source of the pain. Visceral-somatic convergence of noxious stimuli generates “referred pain" (see Table 2) to a perceived somatic source and, hyperalgesia, a decreased threshold to painful stimuli, occurs as well.

A “wind-up" of pain develops from spinal neurons that, from continuous noxious input, become perpetually self-stimulated, which can lead to an exaggerated reflex output with resultant bladder (end-organ) dysfunction, muscle spasticity, and spontaneous firing of dorsal horn neurons. Referred pain to other viscera, dermatomes, or skeletal muscle with decreased thresholds can develop. Noxious stimuli “kick-off" the loop that becomes a self-perpetuating cycle (see Figure 2).

Somatic Nerve	Dermatome	Component	Sensory Zone	Visceral Field
Iliohypogastric	T12-L1	Motor, Sensory	Groin, symphysis pubis	Ovary, Distal Fallopian Tube
Ilioinguinal	L1-2	Sensory	Groin, mons, labia, inner thigh	Proximal tube, Uterine Fundus
Genitofemoral	L1-2	Sensory	Mons, labia, anterior thigh	Proximal tube, Uterine Fundus
Lateral Femoral Cutaneous	L2-3	Sensory	Upper & inner thigh	Fundus, Lower Uterus
Pudendal	S2-4	Motor, Sensory	Inner thigh, introitus, perineum	Lower Uterus/Cervix, Bladder, Distal Ureter, Upper Vagina, Rectum

History and Physical

Not infrequently, the pelvic floor muscles cannot be isolated or identified in controls, yet patients, specifically with PFD, often display a lack of pelvic floor awareness and have poor relaxation with tender pelvic floor muscles. A history of dysfunctional voiding and/or defecation is often present and should signal further in-depth history taking. Urinary and fecal symptoms should be elicited, which can include urgency, frequency, incomplete emptying, hesitancy, pressure or pain. Pain can be pubic, peri-anal, vaginal, or lower back. Pain can be intermittent, constant, or peri-menstrual. Dyspareunia or vaginismus should prompt suspicion of PFD. Standing or sitting can aggravate pelvic pain, so that patients will frequently sit off-centered on one buttock to relieve direct abdominal pressure on the pelvic floor. Lying down will alleviate pelvic floor pain within 10-20 minutes, while pain from spondylosis is conversely exacerbated by recumbency.

The patient's gait and stance are examined. Exams done early in the day may not be as pronounced as one done late in the day after the patient has been on their feet or at work for long periods. Once in lithotomy, general anatomy, light touch sensation and reflexes are evaluated. Muscle tone, sensation, and tenderness at rest are assessed by a gentle examining finger. Spasm and tenderness may be unilateral or bilateral. The inability to isolate or squeeze the pelvic muscles around the finger may be indicative of already tensed floor muscles that cannot contract any further. Despite being neurologically intact, the patient may not demonstrate anal wink, perineal lifting, or closure of the genital hiatus. Relaxation may only be partially demonstrated, in a step-down fashion. Muscle fasciculations may be palpated or visualized by the examiner but not perceived by the patient. The examiner's finger is used to palpate pelvic floor muscles transvaginally or anally. Tone, tenderness and referred pain sensations should be assessed per muscle group. The patient should be asked to squeeze against the finger in the vagina and anus. Strength and duration of squeezing is observed. Slow lifting of the levator, indicative of poor recruitment is characteristic of PFD. The ability, speed and duration of muscle relaxation are equally important. Pyriformis muscle palpation is easier on rectal than vaginal exam, and can be isolated if the patient is asked to abduct the thigh against resistance which will generate pain if tense.

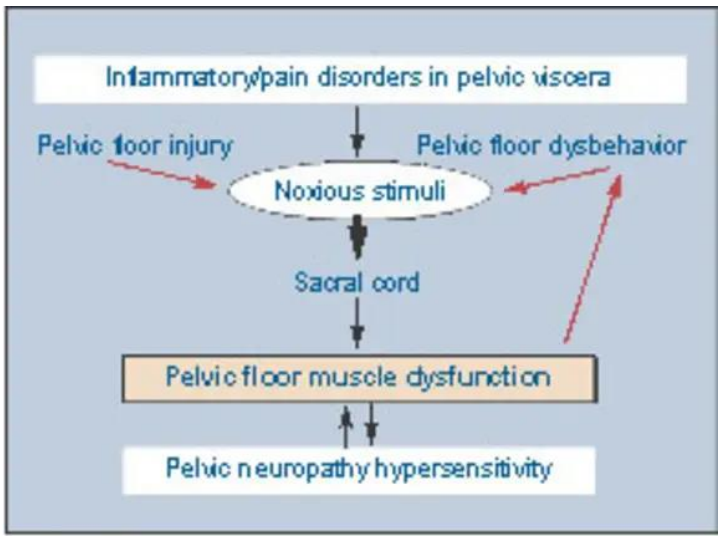

Figure 2. ***Origins of Pelvic Floor Muscle Dysfunction. Adapted from Rovner E, Propert KJ, Brensinger C, et al.*** [9]

Two common patterns on pelvic exam are seen. Patients with a 'frozen pelvis' demonstrate thickened and immobile pelvic muscles, with an increased baseline tone, inability to squeeze or recruit muscles, and poor ability to relax on command. These patients, when asked to contract their pelvic floor, often will valsalva while holding their breath. Patients with less severe PFD can, to some degree, contract the floor muscles despite increased tone, but are unable to relax on command. Physical exam may reveal the thickened levator shelf with a softer more proximal coccygeus muscle. Application of pressure is performed from medial to lateral, in order to attempt to reproduce local pain, referred pain or pelvic visceral symptoms. The location of trigger points is also evaluated.

Though not required, pressure-flow urodynamic testing with EMG is often done on these patients as part of a voiding dysfunction work up. Uroflow often demonstrates an interrupted, obstructed or valsalva augmented flow pattern. High pressure-low flow curves may be seen with a simultaneous increase in EMG activity indicative of dys-synergic pelvic floor/external sphincter activity.

Prior to diagnosing PFD, it is important to rule out organic causes of pelvic pain related either to the bladder, rectum or genital tracts (see Table 1). Frequently other pain syndromes will co-exist. Central or peripheral neurologic lesions and post-surgical nerve entrapment are other conditions within the differential diagnosis as well. All sites of pain should be identified and all factors that can incite inflammation must be addressed. This includes oral or intravesical agents known to alleviate organ-directed pain, such as intravesical dimethyl sulfoxide (DMSO).

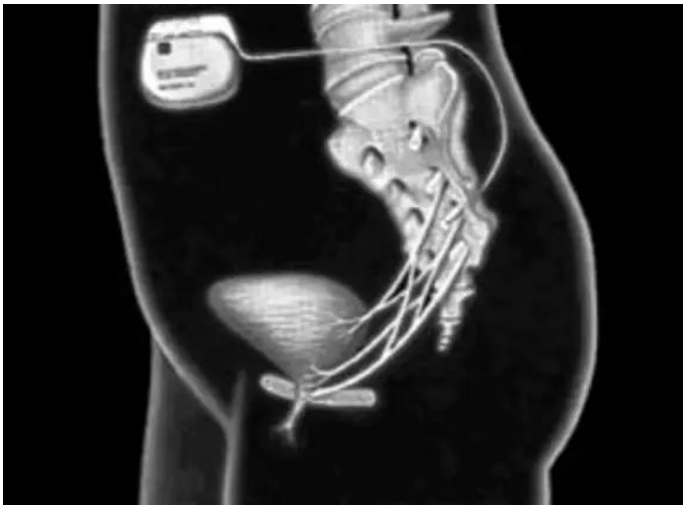

Figure 3. ***Interstim®, Medtronic, Minneapolis, MN***

Treatment

The best approach to treatment of PFD is multimodal. Urology, gynecology, gastroenterology, psychiatry, physical therapy, and pain management all can contribute their specialty expertise to address the patient who frequently carries multiple diagnoses and is often managed with a multitude of medication. However, the specific symptoms of the viscera involved with pelvic pain should be addressed first in order to determine the level of responsiveness at the primary organ level, while simultaneously addressing pelvic floor muscle dysfunction. A simple but successful guideline the authors use for treatment of PFD associated with pelvic pain syndromes is summarized in Table 3 as the “6 P’s". Gaining perception of the pelvic floor muscles can be easily done by instructing the patient to perform ‘reverse Kegels,’ consciously relaxing the perineum as if to release flatus, and to avoid long periods of sitting or tight-fitting clothes. Our approach is to ensure adequate control of pain as well as constipation. Simultaneous use of warm sitz baths assists in facilitating pelvic relaxation. Constipation must be avoided even to the point of producing loose stools early on. Osmotic agents are preferred to cathartics, and we often employ polyethylene glycol (MiraLax, Braintree Laboratories, Braintree, MA). This approach is contraindicated with bowel obstruction.

Using interstitial cystitis as an example, bladder-directed therapies include antibiotics, pentosan, NSAIDs and intravesical DMSO, and often impart partial or short-term relief. Intravesical dimethyl sulfoxide (DMSO) acts as an anti-inflammatory agent, a local anesthetic, and a scavenger for intracellular hydroxy free radicals.[8] It is instilled twice as 50 mL of 50% solution. It may be given with a cocktail of gentamicin, lidocaine, sodium bicarbonate, and heparin. Various non-organ directed oral therapies are listed in Table 4. Amitriptyline has been a common treatment for visceral pain syndromes, and has many pharmacologic affects, including anticholinergic, anti-serotonergic, antiadrenergic, antihistaminergic, and analgesic affects.9 The exact mechanism of action though remains unknown.

Biofeedback involving EMG has demonstrated success in multiple studies in treating dysfunctional voiding, urinary symptoms, pelvic pain, and functional bowel symptoms. Intravaginal, anal, or patch surface electrodes are used in a typical 20-minute session for at least 12 weeks duration. Re-education of pelvic floor muscle activity and function has a reasonably high success rate in two-thirds to three-fourths of patients. It requires a high level of patient and caregiver motivation, yet is minimally morbid and relatively inexpensive.

Internal massage (Theile massage) by myofascial manipulation and myofascial release can be performed in order to lengthen or stretch shortened and tense pelvic muscles. This can be done by careful transvaginal or transanal massage of the various muscle groups by a specially trained physical therapist. Slow progressive massage over at least a 12 week period can help increase the range of motion of the pelvic floor and, in severe cases, even proceed to biofeedback.

Relaxation exercises and heat application can augment release of trigger points that are commonly found at the lateral attachment of the levators to the arcuate line. Trigger points represent an increase in tone of muscle after chronic strain or fatigue that may be a self-generating source of pain through local release of lactic acid or other noxious substances. Treatment for trigger points usually involves hyperstimulation analgesia such as stretch, local injection of anesthetic agents, transcutaneous electrical stimulation (TENS), and acupuncture. All of these treatments act as counter-irritants that alter the central gate or threshold control and result in the prolonged response.

The action of an injected local anesthetic has the effect of blocking the central response.[10] Trigger point injection, along with subsequent massage, can be applied to difficult cases not responsive to simple massage and heat and is usually performed transvaginally. If the trigger point injection was successful, then massage becomes painless, and a series of usually 3 trigger point injections can be given 2-3 days apart to prolong analgesic response.

Treatment with muscle relaxants, predominantly diazepam (2-10 mg, BID- QID), can help control acute pain, spasm, and maintain an adequate level of muscle relaxation in order to proceed with biofeedback or massage. Lidocaine 5% patches have been used as topical therapy for chronic lower back pain, and 5% ointments have also been used for application upon the vulva,[11] and, anecdotally, on the vaginal apex for pain syndromes. In addtion, 2% ointment have been used for application on the urethral meatus for pain, or prior to intercourse.

Electric Neuromodulation

Neuromodulation is defined as any treatment process that alters the function of the nervous system, and thus secondarily alters the end organ. Neuromodulation has been used for decades in the form of biofeedback, drug therapy, and physical therapy. Neurostimulatory devices include acupuncture, sacral nerve stimulation, and pudendal nerve stimulation, among other modalities.

The proposed action of sacral nerve stimulation (SNS) for refractory urge incontinence, urgency, and frequency, involves inhibition of sensory afferents and spinal tract interneurons involved in spinal segmental reflexes, as well as facilitation of voiding by suppressing the guarding reflex in cases of idiopathic urinary retention.[12] Pudendal nerve afferents are believed to have an important inhibitory role on the voiding reflex. Although mapping studies have shown S2 to carry nearly twice as many pudendal afferents than S3, S3 stimulation cause less motor efferent stimulation of the lower extremity. However in 7-18% pudendal afferent distribution may be absent from S3 and confined to a different root (S2) altogether,[13] leading to a potential lack of response in a small, but discrete, percentage of cases.

“P"	Therapy Description
Perception	Teach self-awareness of pelvic muscles
Pain	Adequate analgesia: medications, behavior modification, yoga
Poop	Avoid constipation
Pool	Hot sitz baths/soaks twice a day
Pills	Diazepam for pelvic muscle relaxation
Pressure points	Digital massage of pelvic muscles, trans-vaginally or anally

Agent	Class	Visceral Pain Syndrome
Amitriptyline	TCA	IC
Fluoxetine	SSRI	IC
Gabapentin	Anti-epileptic	IC
Pentosan	GAG substitute	IC
Hydroxyzine	H-1 antagonist	IC
L-Arginine	Essential amino acid	IC
Quercetin	Anti-inflammatory	IC
Ibuprofen	NSAID	IC
Alosetron	5-HT agonist	IBS
Tegaserod	5-HT-4 agonist	IBS
Fedotozine	K-opioid agonist	IBS

The SNS device most commonly used by urologists is InterStim® manufactured by Medtronic, Minneapolis, MN (see figure 3). A trial stimulation test is performed initially as an outpatient procedure. Using fluoroscopic guidance, the percutaneous lead is placed within the S3 sacral foramen to stimulate the S3 nerve and is connected to an outside generator resembling a pager. Placement is verified in the operating room with a bellow of the gluteus and/or a pulling sensation in the genitalia. If the patient demonstrates at least a 50% improvement in symptoms over the 1-2 week trial period, the temporary lead is replaced with a permanent internal pulse generator that is implanted in the upper buttock. It is FDA-approved for use in refractory urinary frequency and urgency and urge incontinence, as well as idiopathic non-obstructive urinary retention. Approximately 60-70% of patients "graduate" from the first to second stage, and several thousand devices have been placed in the past seven years.

In the original multi-center trial in the United States,[14] 64% of patients sustained at a greater than 50% reduction in their symptoms. Discontinuation resulted in a return to baseline of symptoms. Approximately 50% of initial non-responders will respond to a "salvage" second attempt at lead placement. In the original series, lead migration in those with permanent devices was 8.4%, and wound infection was 6%. However, the original series involved a formal incision down to the sacrum where the lead was sutured to the sacral periosteum. Percutaneous lead placement, and subcutaneous generator placement minimize infection risk and make surgical revision less morbid.

Multiple studies of SNS and IC/pelvic pain patients have shown significant reduction in visual analog pain scales, reduction in urinary frequency[15–18], decreases in severity and duration of pain, improved quality of life scores, and reduced urge fecal incontinence.[19] In another study by Everaert[20] of patients with refractory pelvic pain treated with SNS, 60% went on to permanent impulse generator implantation, and all patients had a >50% sustained reduction in pain at 36 months follow up. However, when pelvic pain was the only treatment goal of SNS, the results were not as successful.[21]

Transcutaneous electrical nerve stimulation (TENS) involves placing two electrodes suprapubically about 10-15 cm apart. Stimulation is given at maximal tolerable intensity for up to 2 hours twice a day. It is believed to inhibit detrusor contraction by influencing the anterior cutaneous branch of the iliohypogastric nerve, or by inhibiting pelvic splanchnic afferents that join the inferior hypogastric plexus.[22] Though it is easy to apply, there is conflicting data on whether it results in demonstrable urodynamic changes, and it must be used for long periods.

Direct pudendal nerve stimulation is another new alternative option. The Bion® (rechargeable) microstimulator device, from Advanced Bionics Corp., Valencia, CA, is implanted percutaneously next to the pudendal nerve in Alcock's canal. A percutaneous screening test is performed to gauge responsiveness. A positive test will result in a more than 50% increase in bladder volume at first involuntary detrusor contraction, or maximum bladder capacity. After the permanent wireless device is implanted, it is programmed through

radiofrequency telemetry signals. The internal lithium battery is recharged daily while the patient sits on a specially designed chair pad powered by a wall outlet. Still in trial phase, early 6 months results revealed a greater than 50% reduction in daily incontinence episodes, and increases in voided volume and bladder capacity.[23]

Neuromodulation with SNS is currently considered the standard of care therapy for medically refractory urgency/frequency and urge incontinence and idiopathic urinary retention. Follow up analyses and larger trials are needed to ensure long term efficacy of both SNS and Bion, however these technologies represent serious, viable, and promising options for refractory voiding dysfunction — with or without — concomitant PFD or pelvic pain.

Pelvic floor dysfunction, associated with pelvic pain or voiding dysfunction, represents a clinical disease entity that only recently has gained wider appreciation. Multi-specialty involvement is important for an optimal patient-focused approach. Newer treatment advances are available for patients with more refractory symptoms, yet the majority of patients will respond favorably to the less invasive, simplified protocol (see Table 3) the authors have been utilizing.

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