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Sacral Nerve Stimulation for the Overactive Bladder

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The refractory overactive bladder (OAB) represents one of the most challenging problems in general urology practice. Although rarely a life-threatening condition, the impact on individual quality of life in the physical and psychosocial domains is undeniable [1]. Furthermore, recent epidemiologic surveys confirm our clinical sense that prevalence of the OAB condition is escalating as our population ages [2]. Pharmacotherapy is most appropriately first-line treatment for OAB, but the several drugs currently available do not cure the condition for most patients. Many patients discontinue drug therapy because of intolerable side effects, expense, or lack of long-term adherence. Alternative treatments are needed for patients who are unable to tolerate pharmacotherapy or who do not derive the desired benefits.

Historical context

It is hard to believe that little more than 5 to10 years ago, clinicians had few treatment options to offer refractory OAB patients. Generic oxybutynin and the tricyclic antidepressant class of drugs were the typical agents of choice. Tolterodine arrived on the market only in 2000. When the available pharmacotherapy failed to offer benefit, some clinicians offered augmentation enterocystoplasty as a last resort. Given the scope of potential short-term and long-term complications after such major intestinal reconstructive surgery, most patients opted for diapers and catheters instead [3].

Meanwhile, animal model research during the latter half of the twentieth century helped to explain the neurophysiologic wiring of lower urinary tract control [4,5]. Some early clinical experimentation with functional electrical stimulation proved promising for control of urinary urge incontinence. As nonsurgical modalities, such techniques have used surface electrodes, anal and vaginal plug electrodes [6–8], and dorsal penile nerve electrodes [9,10]. Overall, however, such noninvasive modalities of stimulation have been limited by the intensive nature of the multiple treatment sessions and have proved less reliable at achieving and maintaining response.

The anatomic dissections and work of Tanagho and Schmidt [11] in the 1980s led to the development of a more invasive in situ modality of direct electrical stimulation of the sacral nerve root. This technique was the progenitor of the present-day sacral neuromodulation technique in widespread use. This technology is synonymously referred to in the literature as "sacral nerve stimulation" (SNS).

SNS capitalizes on the same principles as functional electrical stimulation; however, the close contact with the nerve root and the continuous electrical stimulation appear to offer the distinct advantage of more durable, consistent control of lower urinary tract dysfunction. This minimally invasive technology, which requires subcutaneous implantation of the electrode and pulse generator, is described later in the article.

How does sacral nerve stimulation work?

Pilot clinical trial data of the early 1990s led to US Food and Drug Administration (FDA) approval of the sacral neuromodulation device implantation in 1997. SNS has proved to be an

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effective, minimally invasive urologic surgical technique for the treatment of diverse lower urinary tract disorders; namely, refractory urinary urge incontinence and idiopathic voiding dysfunction or retention. Naturally, one must question how a single electrode stimulation of the third sacral foramen nerve root can serve such disparate clinical indications. Indeed, how can the same technique be used to control urge incontinence in one patient and to restore micturition in another patient who has idiopathic urinary retention?

Although this article is focused on the ability of SNS to treat OAB symptoms, it is helpful to revisit the pertinent neuroanatomy and neurophysiology of lower urinary tract functions. Understanding the fundamental blueprint of the central nervous system controls of micturition is essential to appreciating how SNS can treat a seemingly wide range of lower urinary tract dysfunctions. More details regarding the theorized range of mechanisms of action of SNS can be found elsewhere [12].

The micturition blueprint

Normal micturition is dependent on multiple overlapping neural pathways in the central nervous system. These pathways coalesce to perform three major functions: amplification, coordination, and timing [13]. The nervous system control of the lower urinary tract must be able to amplify weak smooth muscle activity to provide sustained increases of bladder contractility sufficient to empty the bladder. Likewise, the bladder and urethral sphincter function must be coordinated to allow the sphincter to open during micturition but to remain closed at all other times. Timing reflects the volitional nature of control over voiding that occurs with toilet training in human development. This control affords us the ability to initiate voiding over a wide range of bladder volumes.

In this regard, the bladder is a unique visceral organ that exhibits predominately voluntary rather than involuntary (autonomic) neural regulation. A number of important reflex mechanisms contribute to the storage and elimination of urine and modulate the voluntary control of micturition [14]. As an autonomically regulated organ, the bladder is also unusual in the sense that it remains in a ''turned off'' mode for most of the time. Thus, it behaves in a distinctively different manner than other visceral organs such as the heart, blood vessels, and gastrointestinal tract—all of which receive tonic autonomic regulation. When volitional desire to urinate occurs, the bladder ''turns on'' in an ''all-or-none'' manner to eliminate urine.

The ability to ''turn on'' micturition in a switch-like fashion is facilitated by positive feedback loops in the micturition reflex pathway. During the micturitional amplification stage, bladder afferent activity stimulates sufficient efferent excitatory input to the bladder, which in turn initiates a bladder contraction. This positive feedback system, mediated in part by supraspinal parasympathetic pathways to the pontine micturition center, is a very effective mechanism for promoting efficient bladder emptying and for minimizing residual urine.

This positive feedback mechanism, however, can also pose as a potentially significant liability. In the presence of neuropathology, the positive feedback system may escape central inhibitory controls or may excessively sensitize bladder afferent signaling. The overall result of such a loss of ''checks and balance'' is the emergence of bladder hyperactivity and random urge incontinence.

Afferent and efferent pathways

Efferent outflow to the lower urinary tract can be activated by spinal afferent pathways and by input from the brain. Afferent signaling input from the lower urinary tract is key to modulating voiding function. Such afferent signaling arises from two main sources: (1) the pelvic visceral organs and (2) somatic afferent pathways by way of the pudendal nerves from the perineal muscle and skin. Although micturition control is commonly perceived as a primarily autonomic-driven circuit, somatic afferent pathways transmit important feedback from the genital organs, urethra, prostate, vagina, anal canal, and skin, which can modulate voiding function [13–15].

Bladder afferent nerves are critical for sending signals of bladder fullness and discomfort to the brain to initiate the micturition reflex. The bladder afferent pathways are composed of two types of axons: small myelinated A-delta fibers and unmyelinated C-fibers. A-delta fibers transmit signals mainly from mechanoreceptors that detect bladder fullness or wall tension. The C-fibers, on the other hand, mainly detect noxious signals and initiate painful sensations. The bladder C-fiber nociceptors perform a similar function and signal the central nervous system whenever an infection or irritative condition exists in the bladder. C-fiber bladder afferents also have reflex functions to facilitate or trigger voiding [16,17], which can be

viewed as a bodily defense mechanism to eliminate irritants or bacteria from the lower urinary tract. The C-fiber bladder afferents have been implicated in the triggering of reflex bladder hyperactivity associated with neurologic disorders such as spinal cord injury and multiple sclerosis.

Bladder hyperactivity and urinary incontinence presumably occur as the consequence of loss of voluntary control of voiding and the reemergence of primitive voiding reflex circuitry. Such primitive voiding reflexes are hypothesized to have been normal neonatal reflex patterns that in time became suppressed with postnatal development. Alternatively, new reflex circuits could arise as a consequence of abnormal C-fiber afferent sensitization [18].

Under normal conditions, the latter are thought to be mechanoinsensitive and unresponsive to bladder distension (hence the name "silent" C-fibers). As a consequence of neurologic or inflammatory diseases or possibly during the aging process, however, the silent C-fibers may become sensitized to bladder distension and thus trigger unwanted micturition reflexes [17]. This type of bladder hyperactivity could theoretically be suppressed by blocking C-fiber afferent activity or by interrupting reflex pathways in the spinal cord by SNS.

Latent inhibitory pathways

To serve as a balance to the micturition blueprint design, nature has provided other latent mechanisms for inhibitory modulation of the micturition reflex. These more primitive mechanisms reside in the spinal cord and can be awakened by stimulation of various somatic and visceral afferent nerves [5,19]. The spinal organization of these inhibitory mechanisms has been elucidated by electrophysiologic studies in animals [20,21]. The authors hypothesize that these modulatory mechanisms can be reactivated by SNS in the treatment of the OAB condition (Fig. 1).

Experimental data from animals [22] have shown that sacral preganglionic outflow to the urinary bladder receives inhibitory inputs from various somatic and visceral afferents and from a recurrent inhibitory pathway [4,5,19]. The experiments have also provided information about the organization of these inhibitory mechanisms [20,21]. Electrical stimulation of somatic afferents in the pudendal nerve elicits inhibitory mechanisms [18]. This result is supported by the finding that interneurons in the sacral autonomic nucleus exhibit firing correlated with bladder activity and demonstrate inhibition by activation of somatic afferent pathways. This electrical stimulation of somatic afferent nerves in the sacral spinal roots could inhibit reflex bladder hyperactivity mediated by spinal or supraspinal pathways.

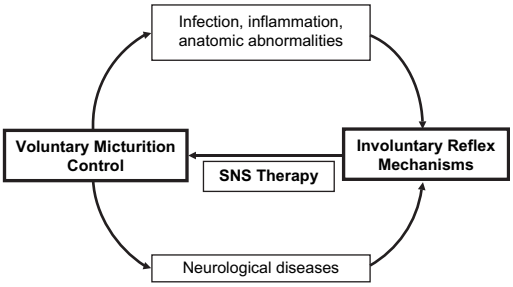

Fig. 1. Conceptual diagram shows that SNS modulates the balance of volitional and reflex pathways controlling micturition.

Pelvic floor electrical stimulation

In addition to the strong evidence from animal research that identified somatic afferent modulation of bladder and urethral reflexes, there are also data from clinical physiologic studies. As previously mentioned, functional electrical stimulation offers a favorable nonsurgical treatment for many patients who have detrusor instability. Stimulation techniques typically use surface electrodes, anal and vaginal plug electrodes, and dorsal penile nerve electrodes.

Such clinical research reinforces the view that stimulation of sacral afferent circuits can modify bladder and urethral sphincter reflexes. The success of pelvic floor electrical stimulation therapy relies on convergence of common visceral and somatic sensory innervation pathways in the central nervous system [23]. By stimulating somatic afferent pathways, it is possible to block the processing of visceral afferent signals being delivered to the same region of the spinal cord. Another example of this principle is the technique of posterior tibial nerve stimulation. With percutaneous electrical stimulation of this nerve or its dermatome, it is possible to block sensory afferent inputs from the bladder [24]. Ohlsson and associates [8] reported encouraging success using electrical somatic nerve stimulation with transvaginal probes in women and transrectal probes in men. Despite a documented average 45% increase in bladder capacity, only one half of their patients

reported a 30% decrease in the frequency of micturition. Fall [6] also reported favorable long-term results of vaginal electrical stimulation in the treatment of refractory detrusor instability and stress urinary incontinence. Seventy three percent of the women who had detrusor instability became asymptomatic during treatment, whereas 45% remained free of symptoms after discontinuation of therapy. Many patients, however, required up to 6 months of therapy before benefit was apparent.

Sacral nerve stimulation suppression of bladder hyperactivity

Several reflex mechanisms may be involved in the SNS suppression of bladder hyperactivity. Afferent pathways projecting to the sacral cord can inhibit bladder reflexes in animals and humans by way of two pathways: (1) inhibition of the sacral interneuronal transmission and (2) direct inhibition of bladder preganglionic neurons of the efferent limb of the micturition reflex circuit (Fig. 2). The source of afferent input may be somatic, visceral, or both; namely, sphincter muscles, distal colon, rectum, anal canal, vagina, uterine cervix, and cutaneous innervation from the perineum. Of the two aforementioned mechanisms responsible for somatic and visceral afferent inhibition of bladder reflexes, the most common mechanism at play in SNS is believed to be the suppression of interneuronal transmission in the bladder reflex pathway [16,25].

It is assumed that this inhibition occurs, in part, on the ascending limb of the micturition reflex and therefore blocks the transfer of signaling input from the bladder to the pontine micturition center. This action prevents involuntary (reflex) micturition but does not necessarily suppress voluntary voiding. This is the clinical scenario typically observed in SNS therapy of OAB. The preservation of volitional voiding function suggests that the descending excitatory efferent pathways from the brain to the sacral parasympathetic preganglionic neurons are not inhibited.

Targeting the descending excitatory efferent pathways is much more effective in turning off micturition reflexes because it directly suppresses firing within the spinal cord motor outflow. This suppression can be induced by electrical stimulation of the pudendal nerve or by mechanical stimulation of the anal canal and distal bowel. As alluded to, however, such stimulation is also expected to nonselectively block voluntary and involuntary voiding. Therefore, this inhibitory pathway appears to play a lesser role in the explanation of SNS mechanism of action. As a large body of experience has shown, SNS performed for voiding dysfunction or OAB syndrome typically allows patients to retain normal voiding mechanisms.

The authors hypothesize that SNS effects depend on electrical stimulation of somatic afferent axons in the spinal roots, which in turn modulate voiding and continence reflex pathways in the central nervous system. The afferent system is the most likely target because beneficial effects can be elicited at intensities of stimulation that do not activate movements of striated muscles [26–28].

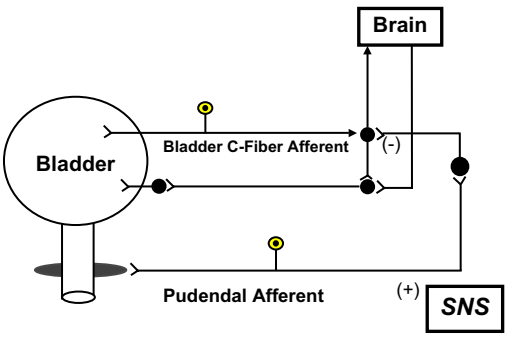

Fig. 2. Stimulation of multiple afferent nerve pathways can inhibit the micturition reflex.

Indications for sacral nerve stimulation

Currently, the InterStim (Medtronic, Minneapolis, MN) device has approval from the FDA for the following indications: (1) refractory urge incontinence, (2) refractory urgency and frequency, and (3) idiopathic urinary retention. As a minimally invasive, outpatient urologic procedure, SNS has demonstrated long-term efficacy and safety. Later in this article, the reported outcomes and complications data are summarized.

In addition to the previously mentioned FDA-approved indications, there is a growing body of clinical experience that suggests the value of SNS technology for other ancillary applications. For example, small case series have shown that SNS can improve lower urinary tract symptoms associated with multiple sclerosis and incomplete

spinal cord lesions [29,30] in addition to interstitial cystitis and pelvic floor pain [31].

Patient selection

It is common practice to begin empiric conservative treatment of clinical OAB symptoms. Typically, conservative therapy for this condition encompasses dedicated trials of combination anticholinergic pharmacotherapy, pelvic floor physiotherapy, and behavioral modification. When it becomes apparent that conservative therapy has failed, the patient should undergo urodynamic testing to objectively characterize the lower urinary tract symptoms.

A thorough bladder diary completed by the patient adds another dimension of documentation regarding the severity of urgency/frequency symptoms and the number of leak episodes. The diary also offers insight into the amount and timing of fluid intake. Because the patient's subjective symptom reporting is central to the success or failure of SNS testing, a well documented bladder diary is critical to patient selection.

Not every appropriate candidate for SNS will derive benefit. It is unfortunate that no current, reliable predictors are available to determine which subset of candidates may achieve response. The InterStim procedure, therefore, must be conducted with a preliminary test phase.

Contraindications

The usual contraindications encompass any patient who fails to achieve an appropriate symptom response to test stimulation. Likewise, clinical judgment must dictate whether a patient is capable of operating the neurostimulator device. Other potential urologic contraindications include conditions of bladder outlet obstruction such as benign prostatic hypertrophy, urethral stricture, or cancer.

Another important contraindication, not often mentioned in the urologic literature, pertains to any subsequent use of diathermy. This therapeutic modality involves the generation of local heat in targeted body tissues by high-frequency electromagnetic radiation, electric currents, or ultrasonic waves. Traditionally, diathermy has been used by a range of health care providers including physical therapists, chiropractors, dentists, and sports therapists in efforts to promote wound healing and to relieve muscle pain and spasms.

This modality is now being used more and more in minimally invasive surgery. Any mode of diathermy can theoretically transfer energy through the implant device and cause severe local tissue injury due to heating at the tissue/device interface. Such tissue injury could lead to permanent injury or even death. Regardless of whether the implanted neurostimulation device is turned off, its presence still poses a risk of injury to surrounding tissue and of device failure. Further details can be found at the manufacturer's Web site: http://www.medtronic.com/neuro/interstim/interstim_warning.html.

Techniques

Percutaneous nerve evaluation

Traditionally, a test trial period known as percutaneous nerve evaluation (PNE) was performed in the office setting using local anesthesia. After the temporary test electrode was placed in the third sacral foramen, the lead was then secured to the skin and connected to an external pulse generator. Three to 5 days of test stimulation followed, with the completion of another bladder diary. Based on this short-term follow-up evaluation, the patients who achieved sufficient symptom improvement had the temporary lead removed and proceeded to a scheduled operative permanent neuromodulator device implantation. With refinement of the operative techniques over time, what once involved an open surgical incision and exposure of the paramedian sacrum has evolved into a minimally invasive, percutaneous procedure [32].

The PNE test stimulation, however, offers a sizeable degree of false-positive and false-negative responses in up to 40% of patients [33]. Because of inconsistent test responses and theoretic temporary lead migration, the development of a tined lead permanent electrode offers another option.

Tined lead electrode

The advent of the tined lead modification (Fig. 3) in 2002 brought a number of technical improvements: (1) a sutureless anchoring system that allowed for a minimal surgical incision; (2) minimal incisions allowed for the procedure to be performed under a combination of intravenous sedation and supplemental local anesthesia, and (3) short-acting sedation allowed for intraoperative testing of patient sensory response to

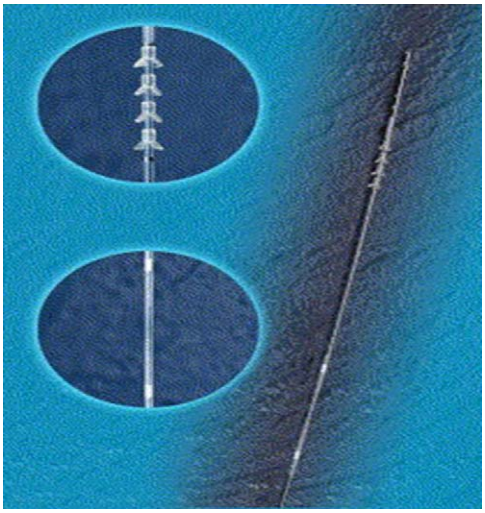

Fig. 3. Tined leads of the permanent electrode. (Reprinted with the permission of Medtronic, Inc. © 2006.)

stimulation. These advantages have led to the increasing popularity of the staged permanent electrode placement. Instead of the usual PNE described earlier, many clinicians are opting to perform the stage 1 test stimulation in the outpatient operating room setting using the tined lead permanent electrode. Proper positioning with the percutaneous approach can be readily confirmed with fluoroscopic guidance (Fig. 4).

Aside from the logistic advantages, the tined lead staged procedures also offer the ability to prolong the testing interval from a few days to a few weeks. With less chance of electrode migration and a longer test interval, one can anticipate a higher likelihood of a positive response to SNS. A recent prospective randomized trial compared the outcomes of one- versus two-stage techniques [34]. Although this study was relatively small, the findings suggested that the two-stage method had a higher short-term and long-term success rate. Although the two-stage implant added an additional direct cost of 1941 Euro per patient, the investigators contended that the lower revision and failure rates rendered the two methods cost-equivalent.

If a satisfactory motor and sensory response to stimulation is achieved (Table 1) with stage 1 testing, then it is appealing to use the same successful electrode as part of the definitive implantation device. If the patient does not achieve any benefit from SNS, then the tined lead electrode and its temporary percutaneous wires can be easily disconnected and removed at a second brief operating room visit.

The final implantable InterStim system is comprised of a battery-powered neurostimulator, an extension cable, and the tined lead with quadripolar electrodes (Fig. 5). At the second stage of permanent implantation, the pulse generator is placed within a subcutaneous pocket of the superior buttock. Subsequent adjustments of the stimulator impulse settings can be accomplished easily and noninvasively with the use of a remote electronic programming device [11,35–37].

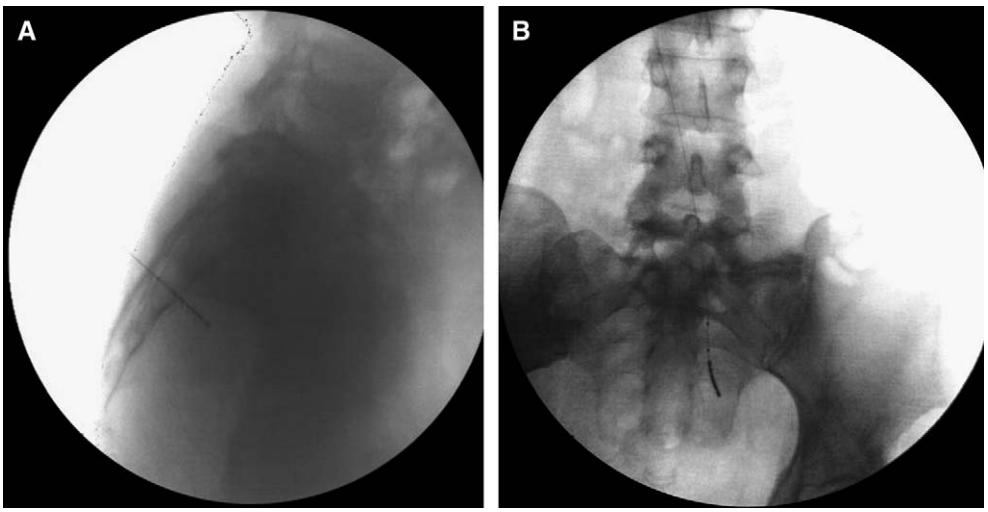

Fig. 4. Lateral (*A*) and anteroposterior (*B*) sacral radiographs showing tined lead electrode placement.

Table 1
Reflex responses to sacral nerve root stimulation

Nerve Root	Pelvic Floor	Ipsilateral Lower Extremity	Sensation
S2	Anal sphincter contraction	Lateral leg rotation, contraction of toes and foot	Vaginal or proximal penile contraction
S3	"Bellows" response of pelvic floor, bladder and urethral sphincter contraction	Great toe plantar flexion	"Pulling" in rectum, variable sensations in labia, tip of penis or scrotum
S4	"Bellows" response of pelvic floor	None	"Pulling" in rectum

Fig. 5. SNS InterStim permanent implantable device components. (Reprinted with the permission of Medtronic, Inc. © 2006.)

Reported outcomes and complications

Large case series and randomized controlled trials of the SNS device have been ongoing since the early 1990s. The collective efficacy data demonstrate that approximately 70% of patients who undergo SNS testing for urgency, frequency, or urge incontinence achieve success compared with the 4% within comparable control groups (Tables 2–4) [38–41]. It is important to clearly define "success" for the patient as "greater than 50% improvement of symptoms." Certainly, there will be occasions where a patient cannot readily judge the degree of symptom relief attained with the SNS device. In such a situation, a 24-hour trial with the stimulator device turned off makes the determination clearer for the patient and the physician. Multiple studies have shown that deactivation of the stimulator device results in rapid return to baseline symptoms for most patients. Similarly, reactivation of the same device leads to prompt return of symptom control [39,41,42].

In an overall review of adverse events reported from 27 studies in the literature, Brazzelli and colleagues [38] noted an overall surgical revision rate of 33%. Most commonly, reoperation was

Table 2
Sacral nerve stimulation therapy outcomes for urgency frequency

Author	Type of study	N=	Technique	F/U (mo)	Reported outcome variables	Overall conclusion
Everaert 2002 abstract	RCT	22	PNE vs staged implant	12	• +129 ml bladder capacity • −3 daily voids	
Hassouna 2000	RCT	25	PNE	6–24	• +91 ml bladder capacity • −7.6 mean daily voids • +101 ml mean void volume • −0.4 urgency rank	• 56% improved at least 50 percent at 6 months
Siegel 2000	Prospective cohort	29	PNE	24	• −7.1 mean daily voids • +92.5 ml mean void volume	• 56% improved at least 50 percent • Urge severity improved 69%

Table 3
Sacral nerve stimulation therapy outcomes for urge incontinence

Author	Type of study	N=	Technique	F/U (mo)	Reported outcome variables	Overall conclusion
Everaert 2004	Randomized trial	8	One-stage	12–24	• +101 ml void volume • 0 leaks/day at 24 mo • −3 mean daily voids	Failures positively related to one-stage implant, and negatively related to age
		9	Two-staged	24	• + 126 ml void volume • 0 leaks/day at 24 mo • −3 mean daily voids	
Weil 2000	RCT	38	PNE	6–36	• −88% mean leak episodes • −90% mean pad use • + 39% bladder capacity	• 56% dry in implant group • 4% dry in controls • 33% improved at least 50 percent
Schmidt 1999	RCT	58	PNE	6–36	• −7.1 mean daily leak episodes • −5.1 mean daily pad use • + 143 ml bladder capacity	• 47% dry • 29% improved at least 50 percent
Siegel 2000	Prospective cohort	41	PNE	36	• −5.6 mean daily leak episodes • −2.3 mean heavy leak episodes • −3.3 mean daily pad use	• 46% dry • 13% improved at least 50 percent
Spinelli 2001	Registry	84	PNE			
	Retrospective	42		41	• 42% had <8 voids daily • 42% had 8–12 voids daily • 18% had > 12 voids daily	• 39% dry • 23% less than one daily leak episode
	Prospective	42		6–18	• −4.3 mean daily leak episodes • 84% had < 8 voids daily at 6 months	• 65% dry at 6 mo • 43% dry at 18 mo
Bosch 2002	Prospective cohort	44	PNE	47	• –4.2 mean daily pad use • −5.8 mean daily leak episodes • −4.9 mean daily voids • +47 ml void volume • +76 mean bladder capacity	• 40% dry • 20% improved at least 50 percent

performed for the following device-related reasons: (1) to relocate the pulse generator because of pain, (2) to revise lead placement because of inadvertent lead migration, or (3) to remove the entire device due to local infection.

The introduction of the tined lead implantation system has simplified the overall operative procedure. What was once a paramedian incision and dissection down to fascial anchoring of the electrode has evolved into a percutaneous lead insertion guided by fluoroscopy. In addition, the tines theoretically help to secure the desired lead positioning within the soft tissues. The reported incidence of lead migration with the earlier technique was significant (range, 11.8%–16%) and would often lead to loss of stimulation efficacy [43]. In addition, relocating the implantable pulse generator from its original position within the lower abdomen to an upper buttock site has decreased the incidence of pain and infection at the device location from 42% to 16% [44]. Reported adverse events, however, appear to be

Table 4
Sacral nerve stimulation implant: reported complications rate

Authors	Type of study	N=	F/U (mo)	Incid (%)	Reported complication	Notes
Hassouna 2000	Prospective cohort	219	12			Based upon pooled data
				15.3	• Pain at implant site	
				9	• New pain	
				8.4	• Lead migration	
				6.1	• Infection	
				5.5	• Transient electrical shock	
				5.4	• Pain at lead site	
				3	• Adverse change in bowel function	
				1.7	• Technical problems	
				1.6	• Device malfunction	
				1	• Change in menses	
				0.6	• Adverse change in voiding	
				0.5	• Persistent skin irritation	
				0.5	• Suspected nerve injury	
				0.5	• Device rejection	
				9.5	• Other	
				33.3	• Surgical revision	
Schmidt 1999	RCT	157	6–36			Based upon pooled data
				15.9	• Pain at implant site	
				19.1	• Pain at lead site	
				7	• Lead migration	
				5.7	• infection	
				32.5	• Surgical revision	• Surgical revision did not prevent favorable outcome

readily corrected by device revision or, as a last resort, device explantation. To date, no major neurologic complications have been reported [41].

Quality-of-life impact

The quality-of-life research in the area of the OAB condition continues to burgeon. Questionnaires, generic (Short-Form 36 Health Survey) and condition specific (Incontinence Impact Questionnaire), are being used more and more in the evaluation of OAB therapies. The scrutiny of potential benefits of sacral neuromodulation for overall patient well-being is no exception. Some investigators have recently reported significant global improvement of patient perception of quality of life with respect to SNS outcomes [45–47].

Summary

Overall, efficacy data from a collective body of global clinical experience supports the conclusion that an estimated 70% of patients who receive SNS therapy become dry or show substantial (>50%) improvement of their otherwise refractory OAB symptoms. The cited randomized controlled trials [38–42] further support this efficacy, given that the control groups (usual conservative therapy) experienced only a 4% benefit [42]. The safety profile of the implantation procedure remains consistent over the increasing length of follow-up since the device's introduction to the clinical market. The relatively high revision rate of 33% has been a relative concern; however, since the late 1990s, the revision rate appears to have dropped significantly [48]. One can speculate that device modifications and growing clinician experience with the technology and procedure have played important roles.

SNS therapy has evolved into one of the most widely accepted treatment modalities in the arena of neurourology. The authors believe that SNS activates or ''resets'' the somatic afferent inputs that play a pivotal role in the modulation of sensory processing for micturition reflex pathways

in the spinal cord. Lower urinary tract symptoms of the OAB syndrome can be suppressed by one or more pathways (ie, by direct inhibition of bladder preganglionic neurons or by inhibition of interneuronal transmission in the afferent limb of the micturition reflex). When conservative treatments for OAB symptoms fail, this minimally invasive technology offers a safe, reliable, and durable treatment for lower urinary tract dysfunction.

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