Sacral nerve stimulation for the treatment of refractory voiding and bowel dysfunction

Karen L. Noblett, MD; Lauren A. Cadish, MD

Sacral nerve stimulation, sometimes referred to as a "pacemaker for the bladder and bowels" delivers nonpainful, electrical pulses to the sacral nerves to improve or restore function. A relatively simple procedure works via a complex mechanism to modulate the reflexes that influence the bladder, bowels, sphincters, and pelvic floor. Current approved indications include urinary urge incontinence, urgency-frequency, nonobstructive urinary retention, and fecal incontinence. The history, mechanism of action, evolution, and landmark literature for this treatment modality are reviewed.

Key words: incontinence, interstim, neuromodulation, sacral nerve stimulation

Sacral nerve stimulation (SNS) delivers nonpainful, electrical pulses to the sacral nerves to modulate the reflexes that influence the bladder, sphincter, and pelvic floor to improve or restore function. SNS has been an available treatment for refractory voiding dysfunction in the United States since 1997, and in Europe since 1994. Over 125,000 implants have been performed worldwide, 53% of which have occurred in the last 3.5 years. Since its inception, the therapy has evolved to a minimally invasive procedure that can be performed as an outpatient under local anesthesia. At present, InterStim (Medtronic Inc, Minneapolis, MN) is the only implantable device approved for SNS therapy. Current Food and Drug Administration (FDA)-approved indications for SNS include urinary urge incontinence, urgency-frequency, nonobstructive urinary retention, and most recently (May 2011), fecal incontinence.

History of sacral nerve stimulation

M. H. Saxtorph, a Danish general surgeon, introduced the idea of electrical stimulation for the treatment of bladder dysfunction in 1878.[1] Since that time multiple iterations of bladder, spinal cord, pelvic and pudendal nerve, and pelvic floor electrical stimulation techniques have been introduced. Nashold and Jonas demonstrated direct spinal cord stimulation facilitated micturition.[2,3] However, their work was met with suboptimal outcomes because of concomitant stimulation and contraction of the external urethral sphincter resulting in high outlet resistance. To overcome the problem of simultaneous stimulation of the detrusor and striated urethral sphincter, investigators sought more peripheral targets. As early as 1979, Tanagho and his group performed multiple experiments using a canine model, and determined that stimulation of the ventral portion of the sacral nerve root was most effective in achieving micturition.[4] The finding that a detrusor contraction could be achieved separately from activation of the urethral sphincter allowed them to forsee a feasible electronic "bladder pacemaker" for use in humans.[5] This landmark research, ultimately lead to the technique for sacral nerve stimulation and initiation of clinical trials.[6,7]

Mechanism of action

Although the exact mechanism of action of SNS in the treatment of voiding and bowel dysfunction is not completely understood, we have gained more insight into the processes at work. One common question is: how does a single intervention remedy both storage and emptying disorders? Addressing this requires an understanding of normal voiding patterns.

For infants and toddlers who have not yet achieved voluntary control over bladder function, a critical level of bladder distention is required to stimulate the voiding reflex. This sensory input, on reaching the pontine micturition center, simultaneously allows for a coordinated detrusor contraction and concomitant urethral relaxation, thus facilitating urination. Gaining voluntary control over, and learning to suppress, this voiding reflex is a complex process that is mediated at a higher level in the cerebral cortex. Continence is also maintained via an intact guarding reflex, which is a progressive, involuntary increase in the activity of the external urethral sphincter during bladder filling resulting in increased outlet resistance. Voluntary voiding is facilitated through stimulation of the excitatory efferent pathway, resulting in inhibition of the sympathetic system and pudendal nerve, and activation of the sacral parasympathetics.[8]

Urgency-frequency and urge incontinence fall under the umbrella term of overactive bladder (OAB). A recent study estimated that one-third of American adults suffer from bothersome symptoms of OAB, and that prevalence increases with age.[9] Most cases of OAB are considered to be idiopathic, however, animal data exist suggesting there may be improper signaling from the bladder urothelium leading to subsequent voiding dysfunction.[10]

From the Division of Female Pelvic Medicine and Reconstructive Surgery, Department of Obstetrics and Gynecology, University of California Irvine, Orange, CA.

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Reprints: Karen L. Noblett, MD, 101 The City Dr., Building 56, Orange, CA 92868. knoblett@uci.edu.

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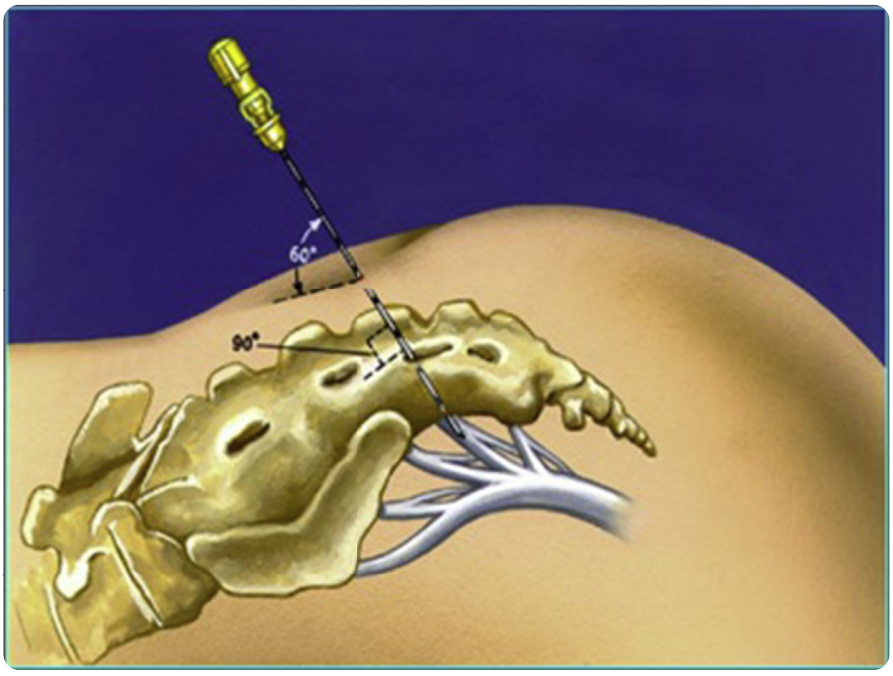
FIGURE 1
Proper placement of needle at the S3 foramen



Optimal placement of the needle is at approximately a 60 degree angle, into the medial and superior portion of the S3 foramen.

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Extensive work to help understand the neurophysiology of voiding reflexes has been accomplished by deGroat and colleagues.[11-15] They demonstrated that sacral preganglionic outflow to the bladder receives inhibitory input from both somatic and visceral afferents. deGroat et al[16] also showed that stimulation of the somatic afferents in the pudendal nerve induces inhibitory mechanisms. This is supported by the finding that interneurons in the sacral autonomic nucleus exhibit firing during bladder activity and are inhibited by stimulation from somatic afferents.

Because of the low level of stimulation delivered with SNS, it most likely functions via the somatic afferents because these nerves are depolarized at much lower levels than the autonomic nerves. For patients with urinary retention, SNS is believed to activate the pudendal nerve afferents originating from the pelvic organs into the spinal cord. At the level of the spinal cord, pudendal afferents may turn on voiding reflexes by suppressing exaggerated guarding reflexes, thus relieving symptoms of patients with urinary retention. In patients with OAB, it is theorized that these same pudendal afferents can activate the inhibitory reflexes that promote bladder storage by inhibiting the afferent limb of an abnormal voiding reflex. This blocks input to the pontine micturition center, thereby restricting involuntary detrusor contractions without interfering with normal voiding patterns. Research comparing the positron emission tomography (PET) scans of urge incontinent individuals with long-term SNS use to those who had activation of stimulation for the first time in the PET scanner found notable differences. In the group with SNS for at least 6 months, blood flow to areas involved in sensorimotor learning was decreased, suggesting that SNS influences brain areas involved in bladder alertness and awareness.[17]

In patients with fecal incontinence, limited information is available to explain the mechanism of action. A small study demonstrated the use of SNS was associated with higher tolerance of rectal distention, but the neurologic mechanism behind this is unclear.[18] Similar to their action in the urinary system, the pudendal afferent somatic fibers are believed to be the primary player, working by inhibiting colonic propulsive activity and activating the internal anal sphincter.[19] A mechanism relating to colonic motility may explain why patients with significant anal sphincter defects may still derive benefit from SNS. Notably, chronic constipation is also an approved indication for Interstim in Europe. Dinning and colleagues have demonstrated increased colonic peristalsis and frequency of bowel movements in slow-transit constipation participants with SNS stimulation.[20,21] As with the bladder, we are faced with the complexity of understanding how SNS may be effective against abnormalities in both bowel storage and emptying.

Procedure

SNS involves a 2-stage procedure. The initial phase is considered the test stimulation period where the patient is allowed to evaluate whether the therapy is effective. There are 2 techniques that exist to perform the test stimulation.

1. The first is an office-based procedure termed the *percutaneous nerve evaluation* (PNE). This involves placing a temporary electrode wire through the S3 sacral foramen under local anesthesia, with or without fluoroscopic guidance. The wire is connected to an external generator worn for a trial period of 3-7 days (Figures 1 and 2). Those with at least 50% improvement in symptoms during the test phase are candidates for chronic implant of the lead and implantable pulse generator (IPG). The advantage of the PNE is that it is an incision free procedure performed in the office using local anesthesia. The disadvantage is that the wire is not securely anchored in place and has the propensity to migrate away from the nerve with physical activity.