Neuromodulation for Voiding Dysfunction and Fecal Incontinence: A Urology Perspective

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KEYWORDS

• Neuromodulation • Overactive bladder • Fecal incontinence • Sacral nerve • Pudendal nerve • Tibial nerve

KEY POINTS

- Neuromodulation is an effective, minimally invasive technique for the management of urinary urgency and frequency, urgency incontinence and nonobstructive urinary retention.
- Neuromodulation has recently been approved and shown efficacy in the treatment of fecal incontinence.
- This article reviews the physiology, indications, implantation methods and outcomes of neuromodulation.

INTRODUCTION

Neuromodulation uses electrical or chemical modulation to affect the physiologic response of an organ. Using electrical stimulation to control voiding dysfunction was first described by Tanagho and colleagues[1] in 1989. Those initial reports of success in treating voiding dysfunction refractory to traditional methods have led to significant research over the past 2 decades. This article discusses the physiology, indications, methods, and results of available neuromodulation techniques for the treatment of bladder and bowel dysfunction.

Bladder dysfunction in the form of urinary urge, urinary frequency, and urgency incontinence are commonly described as overactive bladder (OAB). The International Continence Society defines OAB as a symptomatic syndrome suggestive of lower urinary tract dysfunction.[2] It is estimated that 33.3 million adults suffer from OAB in the United States and as the population of aging adults continues to grow this number is likely to increase.[3] Treatment modalities typically begin with noninvasive measures, like behavioral modification, pelvic floor physical therapy, and pharmacologic therapies. In the past, surgical options, including augmentation enterocystoplasty, detrusor myectomy, bladder denervation, and urinary diversion, were commonly performed.

FI is defined as the involuntary loss of flatus or stool. This experience can be a humiliating and life-altering event for patients. The exact prevalence of this condition is unknown, but published rates have ranged from 1% to 2%[4] to as high as 11% to 15%.[5] The problem is multifactorial, and current treatments result in modest overall success. FI may be secondary to many causes, categorized by having structurally intact but weak anal sphincters (such as rectal prolapse, constipation, neuropathy, and inflammatory bowel disease) or structurally defective sphincters (congenital malformations and obstetric, surgical, and traumatic injury). Traditional nonsurgical

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treatment options have included dietary and pharmacologic stool modification, antimotility agents, biofeedback, injectable bulking agents, and radiofrequency application to the anal sphincter, all with results falling short of desired goals.[6–9] The initial surgical management of FI secondary to anal sphincter trauma traditionally has been either direct sphincter repair or, more commonly, overlapping sphincter repair. Long-term success rates are poor, ranging from 35% to 50%.[10] Advanced options have included placement of an artificial bowel sphincter, dynamic graciloplasty, and fecal diversion. These methods are invasive, technically challenging, and fraught with complications, limiting their widespread use.[11,12]

For 15 years, sacral neuromodulation (SNM) has been Food and Drug Administration (FDA) approved for the treatment of urinary urgency and frequency, urgency incontinence, and nonobstructive urinary retention. During that time, many investigators have observed improvement in bowel dysfunction in patients with sacral neuromodulators. These observations and further studies have resulted in the recent FDA approval of SNM for FI. Neuromodulation has gained acceptance as a treatment modality for bladder and bowel dysfunction. It offers a minimally invasive, reversible method with low morbidity when other first-line treatment options have failed.

THE PHYSIOLOGY OF NEUROMODULATION

The exact neural mechanisms responsible for the effects of electrical neuromodulation on the lower urinary tract and bowel are unknown. Prior to discussing how neuromodulation works, the normal micturition pathway is reviewed briefly. Normal detrusor function relies on a balance between excitatory and inhibitory pathways to maintain continence and the ability to volitionally void. Baseline activity of the sympathetic system provides storage and continence by inhibiting detrusor contractions and maintaining sphincter tone. Parasympathetic activation stimulates detrusor contraction, sphincter relaxation, and ultimately micturition. This balance between sympathetic and parasympathetic nervous systems is under suprasacral control. Bladder afferent signaling relays information about fullness, pressure, stretch, and pain, initiating voiding through multiple reflex pathways. Supraspinal input from the pontine micturition center and cerebral cortex on these sacral reflex pathways control voiding in a voluntary manner. The pontine micturition center provides negative feedback to inhibit voiding and promote continued storage and positive input leading to the induction of voiding. This complex system to maintain control of voiding can be altered by loss of supraspinal inhibitory control or increased sensitization to bladder afferent signals, both contributing to involuntary voiding.[13]

The control of sensory input to the central nervous system (CNS) is thought to work through a gate-control mechanism.[14] The gate-control theory states that noxious stimuli perception does not entirely depend on the A-delta and C-fiber sensory nerves transmitting information to the CNS but on the pattern of peripheral nerve activity.[15] A-delta bladder afferent nerve fibers project to the pontine nuclei to provide inhibitory and excitatory input to reflexes controlling bladder and sphincter function. Afferent C-fibers within the bladder are normally thought to be mechanoinsensitive and unresponsive and are thus referred to as silent C-fibers. These normally inactive C-fibers may be sensitized by neurologic diseases, inflammation, infection, or normal bladder functions, such as distention, thus causing activation of involuntary micturition reflexes and OAB.[16] Sensory input from large myelinated pudendal nerve fibers may modulate erroneous bladder input conveyed by A-delta or C-fiber afferents at the gate control level of the spinal cord. OAB may then be attributed to a deficiency of the inhibitory control systems involving pudendal afferent nerves.

A significant amount of research has focused on the effect of SNM on afferent sensory nerve fibers with the dominant theory that electrical stimulation of these somatic afferent fibers modulates voiding and continence reflex pathways in the CNS.[14] The success of electrical neuromodulation for OAB may result from the restoration of the balance between bladder inhibitory and excitatory control systems.[17] Electrical stimulation modulates the afferent sensory input of the bladder on the pontine center, thereby inhibiting involuntary contractions. Neuromodulation may also remedy OAB by the alteration of afferent signals delivered to the spinal cord that effect activity and basal tone of the pelvic floor.[18]

SACRAL NEUROMODULATION

The InterStim Therapy System (Medtronic, Minneapolis, MN, USA) is the only FDA-approved device for sacral nerve stimulation as a means to treat refractory urinary urgency, frequency, incontinence, and nonobstructive urinary retention. The device has also received recent FDA approval for treatment of FI. This device consists of a tined quadripolar lead that is inserted percutaneously through the S3 sacral foramen and attached to a permanent implantable pulse generator (IPG).

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