



Treatment of Urinary Incontinence in Men with Electrical Stimulation: Is Practice Evidence-Based?

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Electrical stimulation is frequently recommended for the treatment of urinary incontinence in men. However, few randomized, controlled trials allow practitioners to evaluate the evidence base for this practice. The purpose of this article is to determine, based on a review of the literature, whether adequate evidence exists to support the use of electrical stimulation as a treatment of male urinary incontinence. Urge, stress, and overflow incontinence are evaluated separately. This review led to 3 conclusions: (1) theoretical and urodynamic evidence exists to support the use of electrical stimulation for urge incontinence, (2) conflicting evidence exists in the use of electrical stimulation for stress urinary incontinence, and (3) treatment of overflow incontinence in men has not been evaluated in a systematic way. For both stress urinary or overflow incontinence, practitioners should consider the existing research before recommending electrical stimulation as a first line of treatment. For urge incontinence, electrical stimulation may be an effective first-line treatment strategy. (J WOCN 2000;27:20-31)

Urinary incontinence has a significant effect on quality of life, leaving men feeling discouraged, disappointed, and distraught. In a study of men recovering from radical prostatectomy,¹ the depth of distress was clear; as one respondent desperately described it, "Gives thoughts of suicide—had never thought of it before, always attacked and solved a problem or dealt with it. But now I am out of control; there is nothing I can do. It affects my life totally." Quality of life for men with urinary incontinence may be improved if effective treatment is available.

Electrical stimulation is one strategy used to manage urinary incontinence. This article reviews the current literature on the use of electrical stimulation; the articles reviewed are believed to be representative of the current literature describing electrical stimulation as a treatment of male urinary incontinence. MEDLINE, CINAHL, the Cochrane Collaboration Group, and conference proceedings of the Wound, Ostomy and Continence Nurses, Society of Urologic Nurses and Associates, International Continence Society, Urology Nurses of Canada, American Urological

Association, and the International Conference on the Pelvic Floor (Montreal, September 1998) were searched. The reference lists of published articles were also checked. Search terms were incontinence, post prostatectomy, male incontinence, electrical stimulation, therapy, pelvic muscle exercises, and pelvic floor exercises. Published articles and abstracts in English were included if the focus involved treatment of urinary incontinence in men and if the intervention was noninvasive electrical stimulation. One foreign-language paper (in Spanish) with an English abstract was found but was not included as it appeared to be a case series only.² Studies evaluating men with spinal cord injury were excluded. Several case series reports were found as conference abstracts.³⁻⁶ Abstracts with incomplete information or absence of a control group also were excluded. A brief review of the neurophysiology of voiding is followed by a discussion of the conservative applications of electrical stimulation to urge, stress, or overflow incontinence. Therapies discussed include transcutaneous electrical nerve stimulation (TENS), bladder neck stimulation, and transrectal

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stimulation. I conclude with recommendations for practice and suggestions for future investigation.

NEUROPHYSIOLOGY OF VOIDING

The neurophysiology of voiding is a complex topic that is not well understood. Current knowledge has been hypothesized from anatomic studies, knowledge of relevant neurotransmitters involved, and a few physiologic experiments using animal (but not human) models. Numerous connections are suggested but not proven and some that have been demonstrated (like the sympathetic pathways) have an unclear function. Although the following review of neurophysiology of voiding is based on the current research, it is expected to change as further studies add to our incomplete knowledge.

Cerebral Control of Voiding

In infants, voiding may be governed by sacral or brain stem reflexes. Maturation of the nervous system, including learned behavior, gradually results in central reflex pathways under voluntary control. In adults, synchronized, voluntary voiding is highly dependent on intact brain, brain stem, and spinal cord centers. Central organization of the voiding reflex is located in the periaqueductal grey area (PAG)^{7,8} and a center in the pons, termed the “M” center⁹ (pontine micturition center). Based on positron emission tomography (PET) scans of healthy right-handed men, normal voluntary voiding or the ability to suppress the urge to void appears to be primarily associated with 4 areas in the brain: the pons, the PAG, hypothalamus, and the right inferior frontal gyrus.¹⁰ Control of the pelvic floor, perineal musculature, and rhabdosphincter originates mainly from the precentral and postcentral gyri (the paracentral lobe).¹¹ Input is also received from the basal ganglia and the anterior vermis of the cerebellum. Figure 1 outlines the primary cerebral areas on which normal voiding depends.

Injury to any of the cerebral voiding areas causes some disruption to normal voiding function. For example, lesions in the frontal or paracentral areas, most often caused by a stroke, result in an inability to initiate voiding, involuntary voiding at low capacity, or reduced or absent sensation of the need to void. In elderly patients,

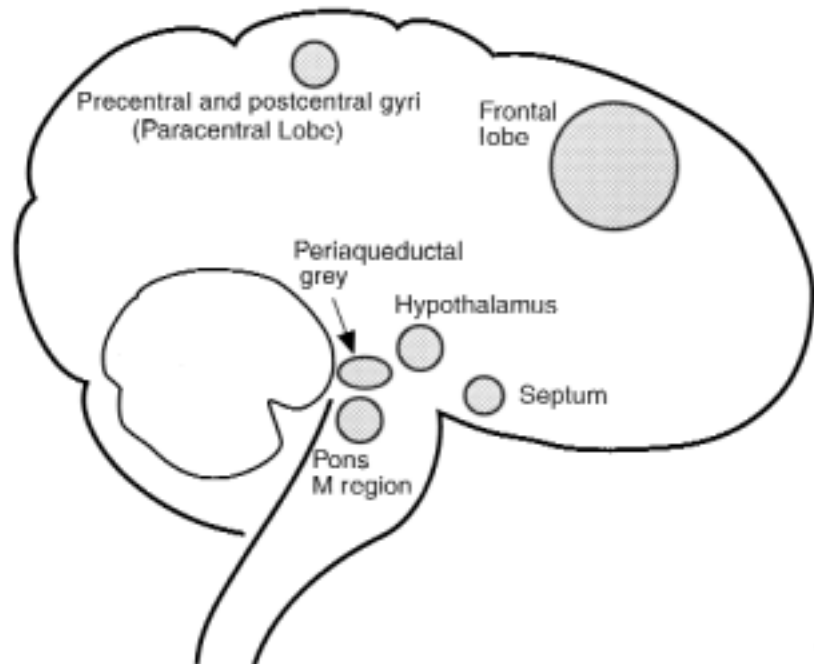


Figure 1. Cerebral voiding centers.

specific changes within the frontal lobe may contribute to incontinence.¹² Two notable changes have been observed in patients with both urge incontinence *and* a reduced sensation of bladder filling: (1) hypoperfusion of the right superior frontal lobe and (2) global underperfusion of the cortex, particularly the left side. These changes are less apparent in older people with incontinence who have normal sensation of bladder filling or in people with similar degrees of cognitive impairment but no urinary incontinence. Lesions involving the extrapyramidal area (basal ganglia, hypothalamus, septum, and limbic system) result in tremors, ataxia, and bladder overactivity associated with conditions such as Parkinson's disease. Finally, lesions at or below the pons cause loss of coordination between the detrusor and sphincter (dyssynergia).¹¹ Table 1 outlines the function of each area.

Spinal Cord Control of Voiding

In addition to cerebral control, intact neural pathways via the spinal cord play a critical role in normal detrusor filling and voluntary emptying. Mundy and Thomas¹¹ suggest viewing the lower urinary tract as a single, functional, coordinated unit innervated by sympathetic, parasympathetic, and somatic nerves. Figure 2 shows a simple schematic of the innervation of the lower urinary tract.

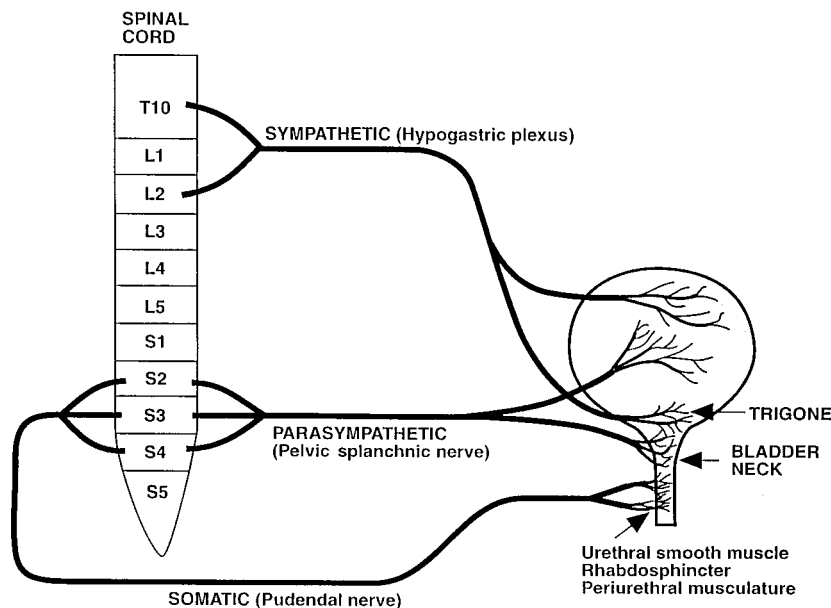


Figure 2. Innervation of the lower urinary tract

Electrical stimulation is applied to the somatic nervous system in noninvasive treatment for urinary incontinence.

Sympathetic dominated reflexes. Sympathetic dominated reflexes allow detrusor relaxation and sphincter contraction during bladder filling. Sympathetic innervation arises from neurons in spinal segments T10 to L2 and reaches the lower urinary tract through the hypogastric plexus. β -Adrenergic neurons in the body of the detrusor and α -adrenergic neurons at the bladder neck, proximal urethra, and rhabdosphincter contribute to detrusor relaxation and sphincter closure by the release of noradrenaline from the nerve endings.¹¹ With respect to electrical stimulation, the sympathetic system is of secondary importance. Instead, the parasympathetic and somatic systems are thought to be more directly modified by the application of an electrical current.

Parasympathetic dominated reflexes. When the M region of the pons is stimulated by impulses from detrusor stretch receptors, signals are sent to sacral parasympathetic neurons, resulting in a synchronized micturition reflex characterized by detrusor contraction and sphincter relaxation.⁹ This input reaches the bladder wall by means of the parasympathetic neurons located at spinal levels S2 to S4. Parasympathetic stimuli reach the detrusor via the sacral plexus, and the stimuli

are transmitted to cholinergic nerves; they release acetylcholine from muscarinic receptors in the body of the detrusor.

Somatic reflexes. The somatic nerves (*somā*, from the Greek, meaning body) control voluntary pelvic muscle contractions and arise from the pudendal and perineal nerves. Alpha motor neurons originating from S3-S4 (occasionally S5) innervate the upper surface of the pelvic floor, whereas those arising from S2-S4 innervate the perineal aspect of the pelvic floor. As Mundy and Thomas¹¹ note, these subtle variations in somatic innervation may have an impact in injury or neurologic disease. Electrical stimulation is applied to the somatic nervous system in noninvasive treatment for urinary incontinence. Stimulation of these somatic nerves may cause sphincter activation, bladder inhibition,¹³ and detrusor activation. The selective effect of electrical stimulation for the treatment of voiding dysfunction is achieved with specific parameters. Table 2 summarizes the innervation of the system.

Stimulation Parameters for Electrical Stimulation

The recommended parameters are based on the work of Fall and Madersbacher¹³ and other Scandinavian researchers who have been leaders in the theoretical application of electrical stimulation for stress and urge incontinence. Fall and Madersbacher recommend the following for the treatment of stress urinary incontinence: square pulses to optimally activate nerve fibers, bipolar or alternating stimulation to reduce or eliminate tissue damage, and intermittent pulses to reduce muscle fatigue. A frequency between 20 Hz and 50 Hz produces a tetanic contraction. To allow muscle relaxation between contractions and prevent muscle fatigue, there should be at least the same number of seconds of "nonstimulation" as there are seconds of stimulation. A typical pattern might be 5 seconds of stimulation alternating with 10 seconds of no stimulation.¹⁴ For the treatment of urge incontinence (detrusor instability), a frequency of 5 to 6 Hz and pulse duration of 0.2 to 0.5 ms is recommended.¹⁵ Because a 5- to 6-Hz frequency may be uncomfortable when the intensity is increased, 10 Hz is generally preferred. Intensity for both stress and urge incontinence should be at the maximum tolerable (but not painful) level. Intensity will vary both with individual threshold and with the position of

Table 1. Centers involved in normal vesicourethral function

Center	Anatomic areas	Controls	Impact of injury/disease
Frontal lobe	Superior and inferior frontal gyrus Anterior cingulate gyrus, genu of the corpus callosum	Voluntary control of detrusor contraction until socially appropriate Suppression of sensory output	Involuntary voiding, often at low bladder capacity; loss of sensation or awareness of urge to void
Paracentral lobe	Superior aspect of precentral and postcentral gyri	Pelvic floor and perineal muscles	Inability to initiate voiding
Dorsomedial pons	Also called pons micturition center, Barrington's nucleus, or the M region L region	Coordination of the sacral cord parasympathetic motor neurons and the nucleus of Onuf Controls contraction of the pelvic floor musculature; may maintain closure of the rhabdosphincter during filling	Urinary retention Detrusor overactivity
Periaqueductal gray		Plays a central role in the control of emotion and pain receptors; activates premotor interneuron in the M region to trigger voiding	Not known
Hypothalamus	Preoptic area	Projects directly to M center; may play role in initiation of voiding; exact role unknown but may be the final common pathway from the upper brain to the M region	Detrusor overactivity
Septum, amygdala, basal ganglia, anterior vermis of cerebellum		Exact mechanism unclear	Detrusor overactivity (as in Parkinson's disease)

Data from reference 9-11.

the probe. Table 3 summarizes the parameters for electrical stimulation.

ELECTRICAL STIMULATION FOR THE TREATMENT OF URINARY INCONTINENCE IN MEN: AN OVERVIEW

In 1895 Griffiths¹⁶ described relaxation of the detrusor with stimulation of the proximal end of a transected pudendal nerve. Over the years, and particularly in the past 2 decades, stimulation has been reported as an effective treatment for men with urge, stress, or overflow incontinence. It has been delivered by TENS, surface electrodes (transrectal probe or transrectal patch), and transurethral bladder neck stimulation. Many case series have supported the use of electrical stimulation in male incontinence therapy. However, recent randomized controlled studies have challenged the findings, particularly with respect to post-prostatectomy incontinence. The following

discussion reflects representative articles on the treatment of urge, stress, and overflow incontinence and the effectiveness of the therapies considered in terms of the known physiologic impact of stimulation. Table 4 summarizes all known English-language articles in which electrical stimulation was used as the primary treatment of male incontinence.

Detrusor Instability (Urge Incontinence)

The symptoms of urgency, urge incontinence, and frequency associated with detrusor instability have been successfully relieved or improved by the use of electrical stimulation. Theoretical underpinnings support the use of electrical stimulation for this purpose. Detrusor inhibition for urge incontinence is achieved through the pudendal afferent nerves (*afferens*, Latin for carrying to or inward), which transmit impulses to the brain from the sensory nerves. Stimulation of the pudendal affer-

Table 2. Neurologic circuits affecting detrusor and pelvic muscle function

Innervation (efferent)	Origin	Innervate	Neurotransmitters
Sympathetic	T10-L2 predominantly via the hypogastric nerve arising from synapses in the inferior mesenteric plexus	Bladder and rhabdosphincter; stimulation causes detrusor relaxation and some sphincter contraction	α -Adrenergic receptors at the bladder neck, proximal urethra, and rhabdosphincter receive sympathetic input contributing to closure β -Adrenergic fibers contribute to detrusor relaxation and bladder filling
Parasympathetic	S3-S4 via pelvic (splanchnic) nerves or nervi erigentes; synapse either with postganglionic branches or with ganglia in the wall of the bladder	Stimulation causes detrusor contraction and sphincter relaxation during voiding	Acetylcholine released from cholinergic fibers is commonly accepted as the neurotransmitter; neuropeptides and nitric oxide are thought to play a role in detrusor contraction and rhabdosphincter relaxation; exact transmitters not understood
Somatic	Anterior horn of S2-S4 originating from Onuf's nucleus; arise via the pudendal nerve and possibly the pelvic nerves; innervate upper surface of pelvic floor (S3, S4, and S5); perineal aspect of pelvic floor via pudendal and perineal nerves (S2-S4)	Rhabdosphincter; upper surface of pelvic floor via S3- S5; perineal aspect via S2-S4; under voluntary control so can relax/ contract as appropriate	α Motor neurons; specific neurotransmitter is not known; afferent signals to cerebral cortex and extrapyramidal system via the pelvic floor skeletal muscles and periurethral striated muscles

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Both studies suggest that TENS plays an inhibitory role in detrusor instability.

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ent nerves leads to central inhibition of the parasympathetic output via the pelvic nerve and reflex hypogastric nerve activation (sympathetic stimulation). Although systematic research in men is lacking, encouraging results in women with detrusor instability¹⁷ should hold true for men.

TENS. One noninvasive method of providing electrical stimulation in men is TENS. Hasan and associates¹⁸ applied TENS to 30 men with detrusor instability. During initial urodynamic evaluation, TENS was randomly evaluated in 3 active and inactive positions at the sacral and thoracic dermatomes. After initial testing to confirm a treatment effect, 3 weeks of continuous TENS was applied to the sacral dermatomes S2 and S3. Self-adhesive patch electrodes delivered 50 Hz with a pulse width of 200 μ s; the current was strong enough to provoke a "tickling" sensation. After treatment subjects experienced a significant improvement in the

number of voids over 24 hours, the use of incontinence pads, and episodes of urge incontinence. Replication of the study by another research team confirmed that, during urodynamics, TENS increased the first desire to void and decreased unstable contractions.¹⁹ Both studies suggest that TENS plays an inhibitory role in detrusor instability.

Alternate TENS placement. TENS has been applied to nontraditional positions such as the dorsal penile nerve,²⁰ the hamstring and quadriceps muscles,²¹ or the posterior tibial or peroneal nerves.²² Bladder capacity, increased voided volume, improved bladder compliance, and improved symptom control have been reported, but these studies are case series and lack control groups or precise outcome measures. Such improvement rates must be cautiously interpreted, as a placebo effect of incontinence therapy as high as 39% has been reported.²³

Surface anal electrodes. Surface anal electrodes are the most common mode of delivering electrical stimulation to men. In a large case series, 24 men and 51 women with idiopathic detrusor instability, sensory urgency, or detrusor hyperreflexia caused by multiple sclerosis received 15 twenty-minute stimulation sessions over a 3-week period.²⁴ Data were not analyzed separately by gender but the authors reported a significant improvement in symptoms and in bladder capacity. Ten or more sessions were required before a therapeutic effect was noted. Moreover, the authors suggest that subjects with detrusor instability at low volumes were unlikely to benefit from electrical stimulation. In subjects with idiopathic instability, the treatment effect lasted more than 2 years. However, for those with multiple sclerosis, the treatment effect lasted only 2 months. Of note is that 100% of subjects with sensory urgency were reported cured, whereas Bower and colleagues¹⁹ reported no effect of electrical stimulation on sensory urge symptoms.

Stress Urinary Incontinence

Electrical stimulation of the pudendal efferent nerves (*effuo*, Latin for outward flow), which carry impulses from the central nervous system to the bladder, causes a reflex response in the pelvic floor striated muscles. It is believed that the artificial activation of the pelvic muscles may simulate a voluntary pelvic muscle contraction and therefore be effective for the treatment of stress urinary incontinence. Adequate urethral and pelvic muscle strength to maintain continence relies on both fast and slow twitch muscle fibers. Fast twitch fibers promote continence by closing the urethral sphincter during sudden rises in abdominal pressure and the latter promote continence by maintaining urethral closure pressure over extended time periods. Because electrical stimulation is known to cause a conversion from fast twitch to slow twitch fibers,²⁵ it is postulated that stress urinary incontinence also may be effectively treated with stimulation. Unfortunately, the results of randomized controlled studies in both men and women with stress urinary incontinence have been variable. Although some report a significant improvement,^{26,27} others have reported no difference compared with a control group,²⁸ pelvic muscle exercises, vaginal cones,²⁹ or after radical prostatectomy.^{30,31}

Table 3. Recommended parameters for electrical stimulation

Square pulse shape	Optimally activates nerve fibers
Bipolar or alternating stimulation	Reduces electrochemical reactions at the electrode site and reduces or eliminates tissue damage
Frequency	5-10 Hz for detrusor instability; 20-50 Hz for stress urinary incontinence
Pulse duration	0.2-0.5 ms; inhibits detrusor activity
Intermittent pulse	For stress urinary incontinence; reduces muscle fatigue
Intensity	Adjust to individual tolerance; should be the maximum possible without being painful

Data from references 14 and 15.

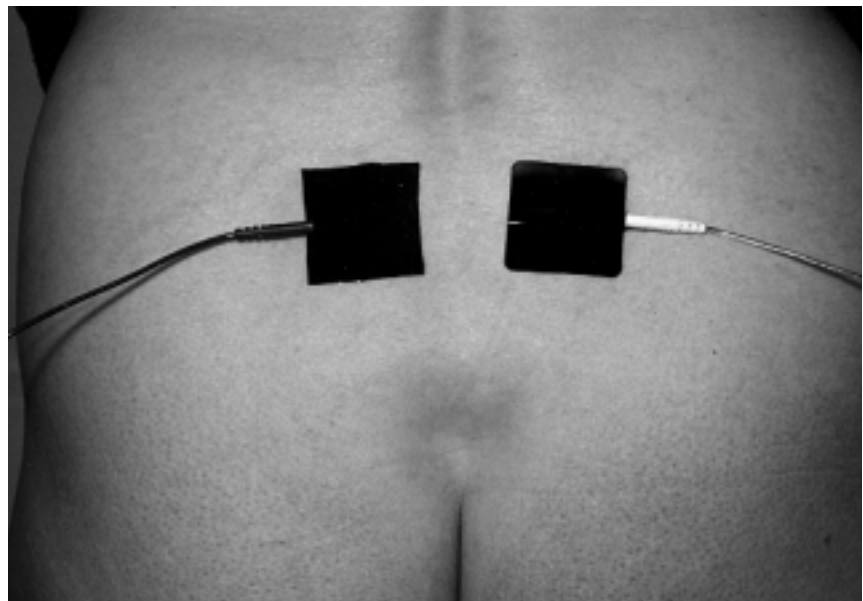


Figure 3. TENS electrodes placed at the Sacral Dermatomes (S2-S3) for the treatment of detrusor instability.

Despite weak evidence for electrical stimulation in the treatment of stress urinary incontinence in men, such treatment remains the area that has received the most critical attention. This may be because of the high number of relatively young men with stress incontinence after radical prostatectomy.

Sotiropoulos and colleagues³² were among the first to suggest electrical stimulation as a treatment for incontinence after prostatectomy. Sixteen men received treatment with a surface anal electrode, including 3 who had undergone radical prostatectomy, 6 after suprapubic prostatectomy for benign enlargement, and 7 after posttransurethral resection of the prostate. Only 3 men who had undergone radical prostatectomy noted improvement, including one whose surgery was 11 years earlier. The remaining subjects either stopped treatment or their condition remained unchanged.

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Table 4. Electrical stimulation for treatment of male incontinence

Author(s) (year); study design	Subjects	Method	Parameters and equipment	Outcome measures	Results
Hasan and colleagues ¹⁸ (1996); pretest/posttest; random location of TENS patches	N = 71; 30 men and 41 women underwent TENS; all had DI, and the following other therapies had failed: bladder distension, medications, bladder transection, and trigonal injection	TENS of sacral dermatomes (S2-S3) using sticky patch electrodes; 50 Hz, pulse width of 200 µm; current was strong enough to produce a "tickling" sensation; TENS conducted under 3 conditions in random order: no TENS, at the S2-3 dermatomes, and at the T12 dermatome	TENS: treatment lasted 3 wk (authors do not state if TENS was once a day or continuous)	Urodynamics on 1 occasion but with TENS in 3 locations; after TENS: patient completed diary, pad use before and during the trial for 3 wk	TENS: 12 subjects dropped out; 18% reported some skin irritation; no clinically significant objective changes occurred on urodynamics (bladder capacity increased by 50 mL); subject diary showed a decrease in number of day and night voids, increase in voided volume, decrease in urgency, and decrease in symptom scores
Kraus & Lilien ³³ (1981); pretest/posttest	N = 8; post TURP; 5 mo to 17 y after operation	Continuous stimulation for 6-21 d with 2 perianal patches at 3 and 9 o'clock positions; no other details provided	No description of parameters or equipment	Subject report of improvement	6 of 8 subjects "improved;" authors suggest that stimulation may have acted as a reminder to do PMEs rather than having a therapeutic effect per se
Moore & Estey ¹ (1999); randomized controlled trial	N = 63; post RP; median 8 wk after operation	PME, ES, + PME or standard treatment (written + verbal instruction preoperatively) Treatment: 2 x/wk for 12 wk PME + ES or PME with physiotherapy; strength, endurance, speed, and control assessed by DRE; home exercises on nontreatment days; follow-up at 12, 16, and 24 weeks after enrollment	InCare* PRS system with surface anal electrode; ES delivered at 50 Hz, biphasic pulse shape with 1-second bursts, 1-second pulse width, and 1-second pulse trains; ES for 10 min alternated with PME for total of 30 min (adjusted if patient was fatigued)	24-hour pad test, F/V chart, number of pads, 2 QOL questionnaires	1. No significant difference between groups; all subjects improved rapidly up to 12 wk after surgery: rapid improvement affected any potential treatment response 2. Authors recommend waiting until at least 6 mo after surgery before starting intensive treatment 3. Quality of life was severely affected by incontinence and patients needed support in the early weeks of recovery

Table 4. Continued

Author(s) (year); study design	Subjects	Method	Parameters and equipment	Outcome measures	Results
Nakamura & Sakurai ²⁰ (1984); pretest/posttest	N = 22; frequency, urgency, UI that had failed to respond to medication; etiology: Parkinson's disease, prostate hyperplasia, or idiopathic	TENS on dorsum of penis (penile branch of pud- endal nerve)	Continuous stimulation through bipolar surface electrodes to dorsal nerve of penis; square-wave pulses; 0.5 ms duration, 10-20 Hz, amplitude 5 to 50 V	CO ₂ cystometry, UPR, EMG of anal sphincter	Increased capacity, decreased spasticity in 10/22 patients; DI suppressed in 7/10; EMG activity increased in anal sphincter; no significant change in urethral pressure
Okada and colleagues ²¹ (1998); pretest/posttest	N = 19 (10 men, 9 women); 14 with DH, 5 with DI	TENS applied over the hamstring and quadricep muscles of 1 or both legs with surface electrodes; hamstring and quadriceps were stimulated alternately for 4 sec each with 4-second rest; treatment 20 min daily for 14 d	Neurotech (Ireland); [†] pulse duration 0.2 ms, frequency 30 Hz, maximum intensity just below pain threshold	Cystometry, voiding chart	Maximal cystometric capacity increased 50% from pretreatment (in 8/14 with DH and 3/5 with DI); increase in capacity was approximately 70 mL (range 30-130 mL); UI and frequency improved and lasted up to 3 months after treatment; bilateral TENS did not improve outcomes
Primus and colleagues ³⁶ (1996); pretest/posttest	N = 75; all with urgency or UI; 51 women (30 had MS) and 24 men; DI; sensory urgency or DH; in most, prior conservative treatment had failed	Rectal or vaginal surface electrode; all subjects received 5 sessions of ES for 20 min each for 3 wk in the OPD; no other treatment information provided	Stimulation device made by Biegler, (Austria); 1 ms monophasic square waves at 20 Hz, on for 1.5 sec, off for 3 sec; maximum tolerable amplitude	Cystometry, patient supine; symptom assessment	43% cured of DI; in the remaining 57%, DI occurred at higher volumes; all patients with sensory urge were cured; mean maximum bladder capacity increase was 50 mL; 12/30 patients with DH improved objectively and 17 subjectively; symptoms recurred in approximately 2 mo; 29/45 patients with idiopathic DI remained improved at 2-year follow-up

continued on page 28

Table 4 Continued

Author(s) (year); study design	Subjects	Method	Parameters and equipment	Outcome measures	Results
Primus and colleagues ³⁶ (1996); pretest/ posttest	N = 48 (22 men, 26 women); hypocontractile detrusor (40); acontractile- detrusor (8)	Intravesical stimulation	Transurethral stimulation catheter with a silver electrode on the tip connected to a stimulator; bladder filled with saline solution to half of maximum capacity; current 10 mA, 20 Hz, pulse duration 4 ms, stimulation 20 sec, rest 4 sec; rise time 2 sec; stimulation lasted 90 minutes and was done 5 times during 1 wk	Pretreatment and posttreatment urodynamics (cystometry, pressure flow, and uroflow)	Pretreatment mean PVR was 460; post- treatment 32/48 voided with PVR of 57 mL; 39% had detrusor contrac- tion; 75% had improved bladder sensation; SEs: epididymitis (1), cystitis (7), 8 relapsed but improved after repeated stimula- tion; there were no clear predictors of success
Sotiopoulos and colleagues ³² (1976); posttest	N = 16; 3 post RP; 13 post TURP; do not state how many years after surgery	Rectal surface stimulation; ES ranged between 1 and 15 mo; do not state No. of times/d nor who administered the treatment	"Portable pulse generator" with anal ring electrode positioned at the levator ani; bi- phasic pulse, frequency 20 Hz increase to 200 Hz in a few cases (rationale not stated)	None stated— only state that subjects were cured, controlled, improved, or unchanged	100% cure for 3 post-RP subjects; only 2/13 post-TURP were controlled; the remaining 11 stopped treatment or did not improve

DH, Detrusor hyperreflexia; DI, detrusor instability; DRE, digital rectal examination; EMG, electromyography; ES, electrical stimulation; F/V, filling/voiding; OPD, outpatient department; PME, pelvic muscle exercises; PVR, postvoid residual; QOL, quality of life; RP, radical prostatectomy; SE, side effects; TENS, transcutaneous electrical nerve stimulation; TURP, transurethral resection of prostate; UI, urge incontinence; UPP, urethral pressure profile.

*InCare Medical Products, Libertyville, Ill.

†Neurotec, Bio-medical Research Ltd, Shannon, Co. Clare, Ireland.

Krauss and Lilien³³ reported improvement from electrical stimulation in 6 of 8 men with stress incontinence between 5 months and 17 years after transurethral resection of the prostate. Previous treatment with pelvic muscle exercises had not been effective. After 1 to 3 weeks of continuous stimulation with 2 perianal patch electrodes, 3 men reported that they were completely dry and 3 stated they were much improved. Nonetheless, the authors suggested that the stimulation may simply have worked as a reminder to the patients to contract their pelvic floor muscles, and that this effect alone may be significant in patients achieving continence.

In a recent randomized, controlled trial, electrical stimulation was combined with biofeedback, pelvic muscle exercises, with or without bladder retraining for men

with incontinence after radical prostatectomy.³⁴ The results were positive; those in the treatment group achieved continence earlier than the control group. However, it is not possible to state whether it was the stimulation, the pelvic floor muscle exercises, or the combinations of strategies that led to improvement.

The positive benefit of electrical stimulation reported above is challenged by 2 randomized, controlled trials evaluating men treated with electrical stimulation after radical prostatectomy. The first, a conference abstract only, studied 39 men assigned to a control or active treatment group with an electrical stimulation portable home unit 15 minutes daily for 12 weeks.³¹ In the second study, 63 men after radical prostatectomy were randomized to twice-weekly therapy sessions

and home exercises with either electrical stimulation plus pelvic floor muscle exercises or pelvic floor muscle exercises alone.²¹ In both studies, after 12 weeks of treatment, no differences existed between the treatment or control groups. Larger sample sizes in both studies may have resulted in treatment group differences, and a group receiving only electrical stimulation may have clarified the role of stimulation. Despite these limitations, both reports challenge the accepted belief that electrical stimulation is an effective modality for the treatment of incontinence after prostatectomy.

Overflow Incontinence

A further application of electrical stimulation involves detrusor activation in acontractile or reduced contractility bladders. Stimulation of the bladder neck activates afferent nerves causing depolarization of receptors in the bladder wall and local muscle contraction. It is theorized that the afferent stimuli transmitted to the brain first restore the sensation of bladder filling and then activate the efferent nerves, in turn resulting in a bladder contraction. Myelinated A fibers are used for this transmission.³⁵ The largest case series of bladder neck stimulation was presented by Primus and colleagues,³⁶ who treated 22 men with acontractile or hypocontractile bladder of neurogenic origin. Men received stimulation by a catheter-mounted ring electrode for 90 minutes for 5 consecutive days. Significant improvement occurred in detrusor contractions and bladder emptying. Subjects who relapsed improved after a second course of stimulation. These promising results may offer hope to some people with poor emptying who otherwise use intermittent catheterization.

RECOMMENDATIONS FOR PRACTICE

The primary role of electrical stimulation appears to be in the treatment of urge incontinence in both men and women. The post-treatment urodynamic changes in individuals with detrusor instability are indicators of some physiologic response to therapy.¹⁸⁻²² Stimulation, in combination with other behavioral strategies, may be an important option for individuals seeking treatment for urge incontinence, although randomized trials are needed to determine the full benefit. Second, a therapeutic option in the form of bladder neck stimulation may be avail-

able to individuals with decreased or absent contractility, although further study is required to clarify for whom this approach would be most effective. Finally, the evidence for electrical stimulation in treatment of stress urinary incontinence is weak. The controlled studies that have been conducted suggest that electrical stimulation alone or combined with pelvic muscle exercises does not significantly enhance treatment outcomes in men with incontinence after radical prostatectomy.^{30,31} However, a role as adjunct therapy may be possible when sensory awareness or urge symptoms are also present, as Van Kampen and colleagues³⁴ illustrated. The equivocal results indicate that practitioners need to consider carefully before recommending electrical stimulation as the first-line treatment for stress urinary incontinence.

CONCLUSION

Although the studies presented in this review should be interpreted with some caution and further research is needed, these results suggest that electrical stimulation is a noninvasive and potentially useful tool for the treatment of urinary incontinence in men, particularly when caused by detrusor instability. If care of the incontinent individual is to be based on the best evidence available, health care professionals need to be prepared to question, challenge, and test current practice. Further research on neurophysiology, with both nurses and physiologists working together, may heighten an understanding of the voiding process by combining both a practical and theoretical approach. Finally, different methods of discovery with PET scans and isotopes may replace the use of animal models. Continence care professionals are ready to address the issues of electrical stimulation and to develop guidelines for future collaborative work and enhanced patient care.

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