
Urological Neurology and Urodynamics

TREATMENT OF MOTOR AND SENSORY DETRUSOR INSTABILITY BY ELECTRICAL STIMULATION

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ABSTRACT

A new application of electrical stimulation to inhibit detrusor activity has been used in 15 patients with a variety of neural lesions. The results were astonishingly good and the device was well tolerated. In patients treated successfully for detrusor instability an absence of urgency occurred as a by-product of electrical stimulation. Therefore, stimulation was used to treat uncomfortable bladder urgency without detrusor instability and was successful in the majority of patients.

Anal sphincter, pelvic floor and direct pudendal neural stimulations have been used for many years in the treatment of incontinence.¹ These devices initially were used to improve urethral closing function. Anal and pudendal stimulation also can result in inhibition of reflex detrusor activity.² While Glen has reported good results with pelvic floor stimulators³ these devices have not been used widely by urologists. Patients tolerate anal plug electrodes poorly, and the operation and effectiveness of various battery-powered devices have been unsatisfactory. Operative placement of pelvic floor or pudendal nerve electrodes and use of a radiofrequency device, although apparently helpful, have not gained general popularity.⁴

In the course of experimental studies on the inhibition of detrusor activity in nonhuman primates with spinal cord injury, we noted that bipolar anal sphincter stimulation was effective only if current was delivered by needle electrodes to the sphincter muscle. It is possible to inhibit reflex detrusor activity with minimal current loss by applying a positive current to the anal sphincter with a negative electrode placed over the posterior tibial nerve. This electrode placement improves current draw and efficacy compared to a bipolar anal sphincter electrode. We then found that current could be applied via a positive electrode transcutaneously to the common peroneal or posterior tibial nerve, with a ground electrode placed over the contralateral common peroneal or posterior tibial nerve with similar results. Current losses were low, averaging 20 to 30 per cent, and efficacy was unchanged. Detrusor contractility was inhibited effectively. Clinical trials in patients yielded similar results. Patients reported absence of incontinence and noticed no feeling of urgency with the device. We also tried stimulation in a number of patients with severe sensory urgency and without incontinence associated with interstitial cystitis. Four representative cases are described.

CASE REPORTS

Case 1. M. C., a 54-year-old woman, presented with frequent uninhibited bladder contractions that occurred with no warning and that could not be prevented. The symptoms developed gradually after multiple operations for herniated intervertebral disks and were exacerbated by a subsequent lumbar fusion.

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These procedures left the patient with a permanent right lower extremity paresis. Treatment with a variety of anticholinergic agents did not result in improvement. Urodynamic evaluation showed a sudden, coordinate, detrusor contraction that occurred at 200 ml. bladder volume, which the patient could not inhibit. The detrusor response occurred with no warning. The patient initially was treated successfully by anal sphincter stimulation but she could not tolerate the anal plug electrode and, finally, refused to use it. Transcutaneous posterior tibial nerve stimulation was instituted, after which the patient has remained dry, although constant stimulation is necessary to achieve continence.

Case 2. D. M. C., a 36-year-old T6 paraplegic man, presented 7 years after injury with a condom catheter in place. An excretory urogram was normal and urodynamic evaluation showed a detrusor contraction at 200 ml. bladder volume opposed by vigorous sphincteric activity for 12 seconds, which was followed by sphincteric relaxation and voiding at a mean intravesical pressure of 32 cm. water. The residual urine was 100 ml. The patient voided by suprapubic tapping, which resulted in brief detrusor-sphincter dyssynergia followed by coordinate vesical sphincteric activity. Posterior tibial nerve stimulation was begun, which prevented detrusor activity for up to 4 hours. When awareness of vesical sensation occurred the stimulator was turned off and the patient voided by suprapubic tapping. A condom catheter was not required. A repeat cystometrogram with the working stimulator in place showed a flat vesical filling curve to 480 ml., at which time the patient became uncomfortable, turned the stimulator off and voided at a mean intravesical pressure of 32 cm. water. The residual urine was 84 ml.

Case 3. S. H., a 58-year-old woman, suffered intermittent total incontinence after a retropubic urethropexy. Incontinence occurred whenever the patient assumed the upright position in the morning and frequently during the day with no warning. Anticholinergic agents did not result in any change in the symptoms. Urodynamic evaluation showed a brisk, uninhibited bladder contraction elicited at 200 ml. bladder volume, which the patient could not control. The patient was unable to void voluntarily at volumes <200 ml. Common peroneal nerve stimulation was instituted, which controlled the bladder contractility.

Case 4. T. B., a 23-year-old woman, complained of daytime
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and nighttime urinary frequency. The patient voided 20 to 25 times daily and was always uncomfortably aware of a feeling of bladder distension. Cystoscopy showed typical interstitial cystitis confirmed by random biopsy, which revealed transmurial inflammatory changes associated with eosinophilic infiltration. Multiple urine cultures were negative for bacterial growth and the symptoms did not respond to anticholinergic medication, bladder overdistension, dimethyl sulfoxide and methylprednisolone acetate instillations. The patient was treated with a stimulator, which allowed her to tolerate bladder filling for 3 to 4 hours without discomfort.

RESULTS

Patients were judged to be either wet or dry by urodynamic testing with cystography. Two patients judged wet were, in fact, improved and refused to give up the stimulators. In 2 patients with stress incontinence and detrusor instability, which were established clearly urodynamically, we considered urodynamic evidence of persistent stress urinary incontinence but total absence of detrusor instability as dry with respect to the condition treated. Over-all results with the underlying clinical diagnoses are given in the table.

DISCUSSION

The mechanism by which electrical stimulation inhibits detrusor activity is not defined clearly. In nonhuman primates effective inhibition of detrusor reflex responses to filling in animals sedated with ketamine hydrochloride can be achieved by stimulation of the sacral roots (S1 to S4), anal sphincter, pudendal nerve, posterior tibial nerve and common peroneal nerve. In normal animals reflex responses also can be delayed by acupuncture applied over the posterior tibial nerve. Indeed, the acupuncture points for inhibition of bladder activity are over the common peroneal and posterior tibial nerves. This traditional Chinese practice was the source of our idea to use transcutaneous stimulation at those points.

The method is not essentially different from prior techniques aimed at anal sphincter stimulation in an effort to improve continence. We found that approach unrewarding for a number of reasons. The typical anal plug electrode consists of positive and negative stainless steel rings mounted on a specially designed retention base and coupled to a high frequency battery-powered stimulator. Large current losses occur along the rectal mucosa between the electrodes, which may result in mucosal injury and loss of battery power. Delivered current to the anal sphincter musculature with this technique is only about 20 to 40 per cent of current draw. In addition, high frequency stimulation is unnecessary and frequently ineffective in inhibiting bladder activity.

Reproducible, effective inhibition of reflex detrusor activity requires 5 to 8 volts, 2 to 10 cycles per second and a pulse width of 5 to 20 msec. Current losses with a single positive intra-anal electrode and a negative ground electrode placed over the posterior tibial nerve are only about 10 to 15 per cent. The working electrical energy in watts is miniscule and the mean current draw in a stimulated nerve that results in detrusor inhibition is in the neighborhood of 50 to 100 μ Amp. Therefore, the major clinical problem is the design and application of suitable electrodes that permit sacral neural stimulation without massive current loss. It would appear that this result can be achieved by transcutaneous electrical stimulation applied by a stick-on electrocardiograph-type electrode with a foam backing over the common peroneal or posterior tibial nerve. This electrode placement, coupled with a commercially available transcutaneous electrical nerve stimulator used for the control of pain, provides a reasonable method to inhibit detrusor activity.

Pt. No.	Diagnosis	Result
1	Detrusor instability	Dry
2	Detrusor instability	Dry
3	Detrusor instability	Dry
4	Detrusor instability	Dry
5	Detrusor instability	Possible improvement
6	Detrusor instability	Dry
7	Detrusor instability	Dry
8	Detrusor instability	Dry
9	Parkinsonism, detrusor instability	Dry
10	Multiple sclerosis with detrusor instability	Improved
11	Multiple sclerosis with detrusor instability	Improved
12	C4-C5 spinal cord injury	Dry
13	T6 spinal cord injury	Dry
14	T6 spinal cord injury	Dry
15	C5-C6 spinal cord injury	Dry
16	Sacral cordoma	Improved
17	Interstitial cystitis	Improved
18	Interstitial cystitis	Improved
19	Interstitial cystitis	No change
20	Interstitial cystitis	Improved
21	Interstitial cystitis	Improved
22	Radiation cystitis	No change

The stimulator should have a variable output in voltage, frequency and pulse width so that the lowest effective intensity can be selected by the patient.

As a result of experimental and clinical findings others have established that the center for micturition is supraspinal.⁵⁻⁷ However, there is considerable evidence that a sacral pathway exists that results in inhibition of the detrusor as a by-product of pudendal neural activity. Approximately 30 per cent of spinal cord injury patients have inadequate, apparently inhibited detrusor responses to filling associated with hyperreflexic external sphincteric activity.⁸ The demonstrated ability to inhibit reflex detrusor activity effectively in our patients with spinal cord injury supports the latter concept of an intrasacral cord pathway for inhibition of detrusor activity. Measured recovery of 75 to 80 per cent of the induced current from the ground electrode, placed on the contralateral lower extremity, is a simple kind of H reflex. The current obviously traverses the sacral cord. Afferent as well as antidromic efferent activity induced in the posterior tibial nerve can be associated with effective inhibition of detrusor activity. Use of electrical stimulation to treat bladder discomfort has not been reported previously. The results in this small series were good enough so that the patients did not want to give up the stimulators. The technique is worth trying before augmentation cystoplasty.

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