

Sacral Neuromodulation for Urinary Retention Induced by Sexual Abuse

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Abstract

Objective: To report that urinary retention as a result of sexual abuse can respond to sacral neuromodulation. **Method:** In a 38-year-old woman in whom chronic, complete urinary retention developed as a result of psychological and sexual abuse in childhood, sacral neuromodulation was added to her therapeutic regimen in an interdisciplinary approach. **Results:** The combination of psychotherapy and neuromodulation restored the ability to void, whereas psychotherapy alone had not. **Conclusion:** Neuromodulation can effectively treat urinary retention in patients with a conversion disorder as a result of sexual abuse (German J Psychiatry 2001;4:29-32).

Key words: Neural conduction, urinary retention, sexual abuse, epilepsy.

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Introduction

Sexual and psychological abuse may cause urinary retention (1,2). Somatic treatment will be difficult as long as the psychological origin is not recognized; likewise, psychotherapy alone may be ineffective. We report a patient with urinary retention as the late result of sexual and psychological abuse in whom an interdisciplinary approach proved successful.

Case Report

In 1993, a 33-year-old nun presented with recurrent urinary tract infections caused by an inability to empty her bladder completely (residual volume, 150 to 250 ml). The

voiding difficulties had begun in 1979 with no apparent cause. In 1989, she had been instructed at another institution in self-catheterization, which she performed 3 times/day. Also in 1989, fasciculation of the right arm and leg had prompted a neurologic examination. The reflex status was normal and no paralysis was seen. Computed tomography and magnetic resonance imaging of the brain ruled out encephalitis disseminata. The electroencephalogram showed no specific focus. As the results of a liquor examination two years previously had been normal, this invasive test was not repeated.

At presentation to our institution in 1993, the urologic work-up was inconclusive. Urethral calibration and cystoscopy were normal. Radiographic imaging showed no neurogenic bladder deformation or reflux of contrast medium into ureters or kidneys. Bladder position was normal and no diverticuli were seen. The sensation of bladder filling was impaired, with a slight sensation beginning at a bladder volume of 500 ml. Measurement of bladder and urethral pressure

during filling and emptying revealed an enlarged hypotonic bladder with residual volumes of up to 750 ml and increased muscle tone of the urethral sphincter and pelvic floor. The urinary flow was interrupted, requiring abdominal straining and accompanied by pelvic floor dyssynergia. Attempts to strengthen the bladder with bethanechol (3 x 25 ml) or to relax the pelvic floor with baclofen (3 x 10 mg) and phenoxybenzamine (3 x 5 mg) did not restore micturition.

Five years later, in 1998 at age 38, she returned to our institution with the development of a complete inability to void. Her social history, related by her psychotherapist, suggested a psychological cause.

Psychological Background

The patient was one of seven children of a farm family. At the age of 2 years, she began experiencing nocturnal epileptic seizures, which she tried to conceal from her family. Between ages 4 and 6, she was sent to live with her grandmother, where she was treated as a household servant. When she was 11, her 15-year-old brother began to abuse her sexually. She failed elementary school and learned sewing. At 18 years, she fell from a tractor and did not regain consciousness for 2 days. The sexual abuse by her older brother continued until she left home at the age of 20 to enter a convent.

As a nun, she received her high-school diploma and successfully completed training as a tailor and instructor for disabled children, both professions she practiced. Despite these achievements, her self-esteem remained low and symptoms of a post-traumatic personality disorder developed. Insomnia with nightmares, chronic nervousness, distrust, feelings of estrangement, emptiness and paranoia resulted in social retreat. She gained weight, and gradually an inability to empty her bladder completely developed.

At age 32 years, she started drinking. Aggravated by her epilepsy, alcohol addiction developed within months. At age 34, an epileptic seizure occurred in public. The frequency of these attacks increased and her alcohol addiction worsened. Medical treatment for epilepsy failed, as she did not take her medication regularly. When she lost her instructor's position, her convent urged her to seek psychotherapy, for which she was hospitalized from August to November 1997.

In the beginning she was distrustful, almost hostile; only gradually could her trust be gained. Initially she discussed her alcohol abuse and later her epileptic seizures, which her superiors had incorrectly attributed to intoxication. The Order had taken her driver's license and put her under constant surveillance, both in- and outside the convent. She did not contest these restrictions, withdrawing to the passive role of victim. She mentioned her inability to void only as a mechanical impediment,

inability to void only as a mechanical impediment, and when she spoke of her psychological and physical abuse it was superficially and without emotion.

Antiepileptic drugs were instituted, but, with the recurrence of her alcohol addiction, treatment was interrupted for an alcohol withdrawal regimen and was reinstated on an ambulatory basis in May 1998. Contact with the psychotherapist was maintained through regular telephone conversations.

Psychotherapy aimed at restructuring her depressive recognition and building up social competence. She learned to reject the role of victim, and she developed self-reliance. She took responsibility within the Order and garnered respect. However, her epilepsy, voiding dysfunction, and looming alcohol addiction continued to undermine her position. She suffered from occasional epileptic seizures, as she refused to take her medication for fear of addiction, and this refusal was interpreted by her superiors as disobedience. Intermittent self-catheterization (3 to 4 times per day), accompanied by recurrent urinary tract infections, evoked the memory of the chronic sexual abuse of her youth. This was the psychological background when, at the end of 1998, she presented with a complete inability to void and sacral neuromodulation was offered by the urologist.

Sacral Neuromodulation

Sacral neuromodulation has been reported to restore micturition in patients with idiopathic urinary retention (3). Square pulses of 12 Hz are applied to one of the sacral spinal nerves S2, S3 or S4 on one or both sides, depending on the results of test stimulation. If voiding is restored during this subchronic stimulation period, a permanent stimulator can be implanted. The stimulator is switched off by the patient to initiate voiding and is switched on again thereafter. How permanent stimulation, interrupted only to void, can restore micturition is as yet unknown.

At the time that a test of neuromodulation was offered to the patient, her creatinine was normal (0.7 mg/dl). Cystography showed an initial sensation at 500 ml, an urge to void at 650 ml, and a capacity of 700 ml with no reflux. Coughing did not provoke a bladder contraction, and the bladder configuration was normal. When another urodynamic examination was planned, the patient asked her psychotherapist to urge the urologist not to repeat unnecessary tests that might enhance her memory of the sexual trauma.

With the patient under full anesthesia, a marked contraction of the levator ani muscle at 2 V indicated the integrity of the peripheral nerves and the correct position of the stimulating wire at S3 bilaterally (12 Hz, Medtronic test stimulator, Düsseldorf, Germany). As is standard during test stimulation, the wire was not fixed near the sacral nerve but only covered with adhesive pads on the skin. The patient (now weighing 90 kg with a height of 168 cm) had a subcutaneous fat layer

Figure 1. Implanted neuromodulator (ventral position) with sacral foramen electrode (dorsal) at right



of 7 cm, and care was taken to maintain the wire's position when she was transferred back into her bed from the operating table. To confirm the correct position, a low-voltage stimulus (approximately 2 V) was delivered with the patient awake—a test that should evoke sensation in the perineum and vagina. When no such sensation occurred at 10 V on either side, X-ray confirmed bilateral displacement. The procedure was repeated two days later. This time the wires were looped in a subcutaneous pocket before exit. Despite this precaution, complete displacement occurred on the left side. The right wire was partially displaced (12 mm) and evoked a sensation at 10 V in the right gluteus muscle with projections to the perineum and vagina. Every 3 to 4 hours the patient was asked to switch off the stimulator and void on a specially equipped commode that documented the urinary flow. One to two minutes after the cessation of stimulation, the patient was able to empty her bladder partially. Several flows showed a voided volume ranging from 340 to 485 ml with residual volumes between 200 and 500 ml, while maximal and average flow rates were normal. Despite a residual of 50%, which would necessitate self-catheterization, the patient requested a third test stimulation. The partially displaced right wire was removed, and complete inability to void recurred.

In March 1999, the third test stimulation was done. This time the wire was fixed at the fascial layer with absorbable 3-0 suture. The wire did not dislodge and reliably provoked sensation at an amplitude of 1 to 2 V in both labia and the perineum with an intensity the patient had not experienced before. When the stimulation was switched off, voiding began without delay. Initial voiding volumes ranged from 400 to 800 ml with residuals of 90 to 250 ml. The patient was asked to void more often to keep the voiding volume below 500 ml, and the residuals remained below 100 ml. The flow characteristics reflected an intact bladder muscle.

At the end of March 1999, a permanent stimulator was implanted at S3 on the right (Medtronic IPG stimulator model 3023; see Figure 1). The radiotransmitter enabled the patient to turn the stimulator on and off to change the amplitude within a programmed range. The stimulation parameters were 0.5 - 1.7 V amplitude, 13 Hz frequency, monopolar stimulation with the stimulator positive and the electrode position No. 1 negative. Six days after implantation the voided volume ranged from 320 to 730 ml, with residual volumes at or below 30%. Wound healing occurred uneventfully.

In February 2000, eleven months after implantation, the patient voided 3 to 4 times per day, slept well at night, and felt no pain. Indeed, she claimed to be so familiar with the stimulator that she ceased regarding it as unusual. Her work with disabled children was no longer hampered by intermittent catheterization or bladder infections. She even took up a sport, although her choice (the trampoline) was not fortuitous. It was during this activity that her electrode dislodged, and she was again unable to void. Stimulation caused muscle pain in the sacral area on the right. When the electrode was surgically repositioned at the end of February 2000, it became evident that the fixation threads were all in place, but all three eyes in the silicon mantle designed for electrode fixation were torn. This time the thread was looped and tied around the electrode to make further displacement less likely. The ability to empty the bladder of volumes around 500 ml with a residual of less than 100 ml resumed within days.

Bladder neuromodulation boosted the patient's progress in psychotherapy. For the first time, this demonstrated to her that an "organic" impairment could be "healed," and this encouraged her to comply with her prescribed antiepileptic drug regimen. The work-up of her past sexual trauma could finally be integrated, as the need to catheterize (which had revived the trauma) was abolished. Her relationships with the members of her family and her Order changed owing to her growing self-confidence, and she began to dress with greater care as her attitude towards her body improved as well. She is now contemplating a college education.

Discussion

In children, the "lazy bladder syndrome" is well known. Excessive toilet training or fear of strange bathrooms (4) may cause infrequent voiding, resulting in gradual overdistension of the bladder with loss of the filling sensation (5). Residual urine with recurrent urinary tract infections may ensue. A similar mechanism may be postulated in our patient in whom sexual abuse resulted initially in an inability to relax the pelvic floor properly during voiding and later in a complete inability to void. In a previous report by Kroll et al. (1), a 14-year-old girl from a pathologic family setting presented in urinary retention with a bladder capacity of 1300 ml. Medication could not restore micturition, but, after 10 days' rest in a sanatorium, spontaneous voiding resumed. It is interesting to note that, in our patient, when the ability to void was partially restored during test stimulation, the residual volume correlated with the malposition of the stimulating electrode. This finding indicates the unlikelihood of a placebo effect. Thus, the indications for neuromodulation can be broadened to include not only idiopathic (3) or iatrogenic (6) urinary retention but also that caused by sexual or psychological abuse in the absence of permanent physical trauma. Prerequisites for success appear to be the presence of intact nerves running from the pelvic plexus to the bladder and pelvic floor and a viable bladder muscle that has not been overdistended.

From a psychotherapeutic point of view, the urinary retention in this case appears to be a special form of post-traumatic vaginism. Although both the urologist and patient were initially skeptical of neuromodulation~seeing

it as a mechanistic approach to a psychological problem~the outcome proved that it was worth a try. Although it cannot be predicted whether further progress in psychotherapy will make neuromodulation unnecessary, as it is now the patient's quality of life has significantly improved since the introduction of sacral neuromodulation in an interdisciplinary approach.

References

1. Kroll P, Martynski M, Jankowski A: The role of psychogenic factors as a cause of urinary retention in a patient with lazy bladder syndrome. *Wiad Lek* 51 (Suppl 3):102-105, 1998
2. Khaled K, Vause S: Genital mutilation: a continued abuse. *Br J Obstet Gynaecol* 103:86-87, 1996
3. Shaker HS, Hassouna M: Sacral root neuromodulation in idiopathic nonobstructive chronic urinary retention. *J Urol* 159:1476-1478, 1998
4. Bauer SB, Retik AB, Colodny AH, Dyro FM: The unstable bladder of childhood. *Urol Clin North Am* 7:321, 1980
5. Webster GD, Koefoot RB, Sihelnik S: Urodynamic abnormalities in neurologically normal children with micturition dysfunction. *J Urol* 132:74, 1984.
6. Heyden B von, Bothe HW, Hertle L: Urinary retention after hysterectomy: sacral neuromodulation to restore micturition. *J Urol* 162:2094-2095, 1999.