

Clinical Case of the Month: Urology

Urinary incontinence in a male paraparetic patient (able to walk) with a mixed lower motor neuron lesion

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Case presentations

A 35 year old man sustained a fracture of L1 vertebra in a paraglider accident on July 14, 1990 in Alaska, sustaining an L5 sensory paraplegia with a neuropathic bladder and bowel, without the ability to empty his bladder. Initially urine drainage was achieved by a Foley catheter for 10 days, subsequently, intermittent catheterization (IC) was performed. On August 28, 1990, he was transferred to an Austrian rehabilitation centre and stayed there until December 21, 1990. During this time he learned to walk with crutches and orthoses.

Urological History on Admission to the Rehabilitation Centre: He had no sensation for the urge to void, but he experienced a dull pressure feeling in the lower abdomen when his bladder was full. Bladder emptying was by IC, sometimes with over-distensions, urinary tract infections and urinary incontinence.

Bowel Dysfunction: Irregular bowel emptying up to three-times daily with increasing fecal incontinence.

Sexual-Dysfunction: Loss of erection and ejaculation.

Clinical Findings: Sensation was normal to S1. In S2 pinprick was diminished bilaterally and light touch was diminished on the left. From S3 to S5, both pinprick and light touch were absent. No voluntary contraction of the external sphincter ani was felt and bulbocavernosus and anal reflexes were negative.

Urodynamic Evaluation (October, 1990): Detrusor hyperreflexia with hypocontractile detrusor contractions in combination with a more or less areflexic pelvic floor paresis.

Initial Treatment: Management of urinary detrusor and sphincter dysfunction consisted of IC, anticholinergics and low dose infection prophylaxis.

Due to increasing fecal incontinence, elsewhere a descending colostomy with closure of the distal sigmoid was performed (February 6, 1992).

When we saw the patient for the first time on February 13, 1992, urinary incontinence was the predominant complaint, the clinical findings were

unchanged compared to the previous ones, except that the bulbocavernosus reflex was weakly present.

Videourodynamics (February 13, 1992): Cystometric bladder capacity 200 cc, compliance 5, leak point pressure around 34 cm of water with continuous urinary dribbling, no real voiding, but increased EMG activity of the pelvic floor with increasing bladder filling. Videographically there was an incompetent, open bladder neck from the beginning, no reflux into the upper urinary tract, but reflux into the prostate was present (see Figure 1a and b).

Urological diagnosis 19 months after the injury was therefore of a low compliant detrusor (around 5) and a leak point pressure at 34 cm of water with continuous dribbling (urinary incontinence) in combination with a weak but hyperreflexic pelvic floor in an ambulatory paraplegic person.

Therapy: IC, anticholinergic therapy, and low dose infection prophylaxis were continued and alpha-adrenergic drugs added.

Follow-Up: Despite this treatment mostly afebrile urinary tract infections occurred, control-cystometry revealed a persisting low compliance bladder with slight improvement and a leak point pressure of 58 cm of water.

Urinary incontinence and recurrent urinary tract infections are still the predominant symptoms despite IC, anticholinergic therapy with Ditropan 5 mg, three times daily, low dosage infection prophylaxis and adrenergic medication in this ambulatory paraplegic person.

What further urological treatment would you recommend for this patient?

The opinions of four neuro-urological experts and the comments by Professor Madersbacher are:-

First presentation: Professor Iwatsubo, M.D.

This paraparetic male appears to have a mixed neurological lesion of the sacral spinal cord and the cauda equina at sensory L5 level. The sphincters in

flaccid paralysis initially may have partially recovered later since BCR became weakly positive on February 13, 1992. However, a leak point pressure of about

34 cm of water is too low to prohibit his stress type of incontinence. On the other hand, the detrusor was described to be spastic with an upper motor neuron

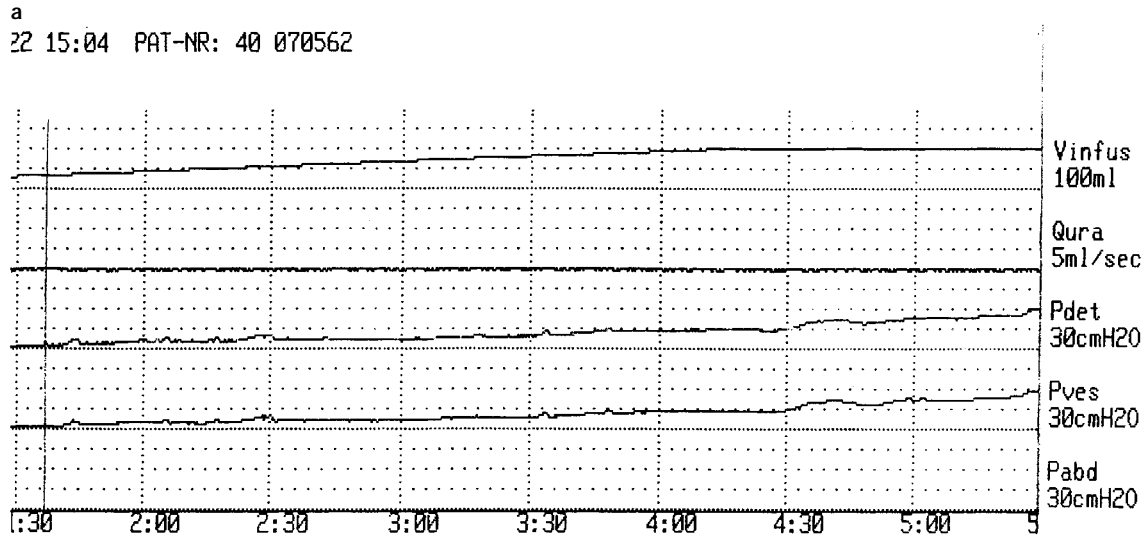


Figure 1 Videourodynamics (Feb. 13, 1992). (a) Urodynamics (filling cystometry): cystometric bladder capacity 200, compliance 3. (b) Voiding cystourethrogram: irregular bladder outline due to trabeculation and pseudodiverticula, wide open bladder neck and proximal posterior urethra, massive reflux into the prostate, constant narrowing of the urethra at the level of the pelvic floor

lesion in October, 1990, which is now in low compliance of 5 ml/cmH₂O under the anticholinergic medication. High intravesical pressure being suggested by the open bladder-neck with prostatic reflux of contrast medium is another cause of urinary incontinence. His bladder pressure at less than 200 ml capacity already exceeds the leak point of 34 cmH₂O, thus continuous urinary leakage could not be controlled.

Therapy proposed to control his urinary incontinence should firstly be conservative. (1) An insufficient dose of an anticholinergic agent may not eliminate hyperreflexia, and the bladder may remain in low compliance. A cystometrogram under 10 mg Scopoline butylbromide IV injection may differentiate low compliance from the fibrotic bladder. If this test improves the compliance, a sufficient amount of Oxybutinine or Terodiline hydrochloride may be effective. If he cannot tolerate an increased dose of such drugs, instillation of Oxybutinine hydrochloride into the bladder at each catheterization may help. (2) To increase leak point pressure, Gax Collagen periurethral injection may be required since the leak point pressure of 58 cm of water obtained by alpha-adrenergic drug may be insufficient for the stress incontinence of this paraparetic walker. As far as CIC is continued, accidental urinary infection cannot always be avoided. (3) Electrical stimulation with an anal plug is sometimes effective for stress incontinence, increasing sphincter pressure (UP_{max}) with reflex inhibition of detrusor spasticity, especially in patients with increasing EMG activity of the pelvic floor with increasing bladder filling. If these conservative measures do not help, (4) Sacral anterior root stimulation with posterior rhizotomy using the Finetec electrical stimulator may be effective for controlled voiding, with elimination of urinary infection, and may save the patient from erectile impotence. However, we have no experience of this surgical procedure in Japan.

Comment by Professor Madersbacher:

I agree that the treatment of neuropathic urinary incontinence should first be conservative. To increase the leak point pressure by periurethral injection of collagen is an option, however, if it is not successful and the implantation of a Scott-Sphincter is necessary, a previous collagen injection makes the operation more difficult and hazardous. Electrical stimulation of the pelvic floor in this patient (with an incomplete lesion) may increase sphincter pressure together with reflex inhibition of detrusor spasticity, however, from our experience, the effect on the pelvic floor may not be sufficient to render a weak neuropathic sphincter competent. Sacral deafferentation in a patient with an incomplete lesion must be considered carefully as he may lose useful sensation and erection; moreover, the electrical stimulation may be painful. The operation may be technically difficult due to the traumatic conus-cauda-lesion.

Second presentation: Professor I Perakash, M.D.

This patient has an unusual lesion with a weak pelvic floor, but active bladder and external urethral sphincter. He has detrusor-sphincter dyssynergia which is obvious from the increased EMG activity of the sphincter during cystometrographic study. This is also confirmed by the voiding cystourethrogram which shows a non-related external urethral sphincter with prostatic-ejaculatory reflux and widely dilated proximal urethra (bladder neck). The bladder wall also shows marked trabeculations. This patient therefore has a neuropathic bladder with detrusor-sphincter dyssynergia. The presence of urine in the dilated proximal urethra leads to constant dribbling. Conservative management with intermittent catheterization is not appropriate for detrusor-sphincter dyssynergia associated with prostatic-ejaculatory reflux and significant bladder wall changes. One could think of transurethral sphincterotomy and a leg bag to prevent future upper tract complications. Another choice would be to surgically close the bladder neck, auto-augmentation or loop augmentation and intermittent catheterization to prevent constant leakage.

Comment by Professor Madersbacher:

I agree that conservative management in this patient is not appropriate and that transurethral sphincterotomy may be necessary. In order to achieve continence further surgical procedures are necessary: the closure of the bladder neck, subsequent auto-augmentation or loop-augmentation with consequent intermittent catheterisation is one of several options, however, this would mean a second stoma on the abdominal wall and two stomas may be difficult to handle.

Third presentation: Mr TP Stephenson FRCS

I would view that this patient has been quite unsuitable for a Brindley procedure as he has an incomplete lesion. My probable policy would be to have done a sphincterotomy and then 6 weeks later a myectomy and an artificial urinary sphincter of the 800 AMS model. The alternative would be to do a substitution cystoplasty instead but I think that myectomy would be so successful in the neuropath that this would be the procedure of choice.

Comment by Professor Madersbacher:

The suggestion made is precisely the therapy we have undertaken in this patient. The alternative, a substitution cystoplasty, has the disadvantage that the patient would have two stomata at the abdomen.

Fourth presentation: Dr M Stohrer, MD

A purely theoretical therapeutic decision presents a problem without communication with the patient, if

only because the remaining options have varying effects on the quality of life. Therefore, the *patient* should consider all of the options regarding possible repercussions for his conduct of life, life expectancy and quality of life.

The least invasive choice would be to attempt sterile intermittent catheterisation (5 times a day) and renounce from the antibiotic prophylaxis. If freedom from infections could be attained in this way, compliance and storage function might improve greatly. Supplementary, the conservative treatment might be optimised and its efficacy improved by the intravesical introduction of anticholinergics or spasmolytics, possibly combined with iontophoresis (Physionizer).

A second, relatively minor invasive option would be partial myectomy. This can improve the compliance with the condition that the infection can be controlled. For the presented patient, a fascial sling procedure would make sense to achieve continence. An alloplastic sphincter is contra-indicated in this patient who has no bladder sensation, because he

would then be at increased risk for extraperitoneal perforation of the created diverticulum, should strict adherence to the catheterisation regimen not be warranted. The fascial sling is safer in this respect, because the risk of perforation will be reduced by overflow incontinence. When the autoaugmentation proves that it is not effective (to be judged 1–2 years after surgery), it can be replaced by enterocystoplasty.

Comment by Professor Madersbacher:

Conservative treatment can be further optimised, however, it is unlikely that a patient who can walk with a neuropathic weak sphincter will achieve continence. Partial myectomy (auto-augmentation of the bladder) can restore an adequate filling phase. With regard to sphincter incompetence the proposed fascial sling procedure may result in difficult catheterisation for a male patient. We feel that the Scott Sphincter is not a contra-indication for such a patient but it should be combined with a previous sphincterotomy.

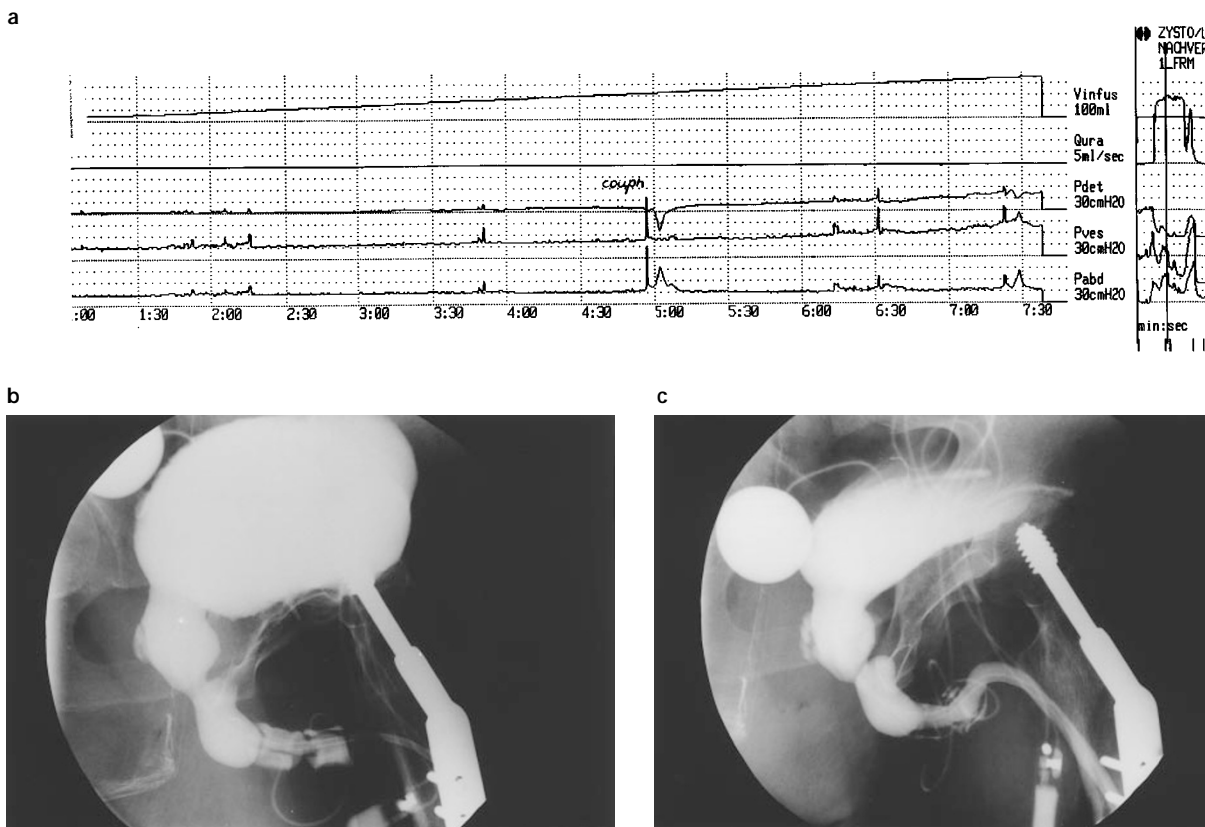


Figure 2 Videourodynamics (Jan. 28, 1997). (a) Urodynamic investigation: filling cystometry: cystometric bladder capacity 380cc, compliance 12 Flow (recorded separately for technical reasons), Fmax 30cc/sec. (assisted by abdominal straining), no residual urine. (b) Voiding cystourethrogram (after anteriomedian sphincterotomy, autoaugmentation of the bladder, Scott-sphincter implantation around the bulbous urethra and additional implantation of a second cuff): on the left side (2b) with the cuffs inflated smooth bladder outline, dilated urethra proximal to the cuffs, wide open bladder outlet; on the right side (2c) with the cuffs deflated, micturition along a normal anterior urethra reflux into the prostate, note that also the augmented part of the bladder empties well

Professor Madersbacher's personal treatment of the patient:

Our therapeutic strategy: As the patient remained incontinent despite IC and anticholinergics and has recurrent urinary tract infections despite low dosage infection prophylaxis and proper technique of IC, we decided (1) to eliminate the functional obstruction present at the level of the external sphincter (which was also causing massive reflux into the prostate) by sphincterotomy and (2) to treat his reflex and neurogenic stress incontinence by a bladder augmentation procedure (as anticholinergics were not sufficiently effective) and by implantation of an artificial Scott-sphincter.

Further Therapy: On April 4, 1992 an anteromedian transurethral sphincterotomy was performed. Afterwards, bladder emptying was without residual urine and the urine cultures remain sterile.

On November 10, 1992 implantation of an artificial sphincter and auto-augmentation of the bladder was performed. Due to remarkable scars in the area of the posterior urethra, first the auto-augmentation was performed and subsequently the artificial sphincter was implanted around the bulbous urethra. The operations were without complications despite the colostomy.

Follow-Up: A return visit on December 14, 1992, revealed a functional bladder capacity up to 380 cc bladder emptying without residual urine when deflating the cuff and with slight abdominal straining.

Despite a Scott-sphincter system and a bladder capacity up to 500 cc, during walking, however, urinary dribbling occurred occasionally. Further evaluation showed that the filled cuff did not entirely occlude the bulbous urethra.

Further Therapy: On May 8, 1993, a second cuff was placed around the bulbous urethra ('tandem-sphincter') without any postoperative complication.

Follow-Up: With a tandem sphincter the patient now remains dry, low dosage infection prophylaxis is continued intermittently.

At the last visit, on January 29, 1997, including a videourodynamic study, the bladder was seen to be emptying every 4–5 h after deflating the cuff. He had sterile urine without any residual, and was continent both day and night.

Videourodynamics: Cystometric bladder capacity was 350 cc, compliance 12. Videographically a wide open bladder outlet with an incompetent bladder neck due to the neuropathic lesion and after the anteromedian sphincterotomy was noted as well as a smooth walled bladder outline, slight persistent reflux into the prostate, good position of the two cuffs, which occluded the urethra, no reflux to the upper urinary tracts which were normal as seen on echography (see Figure 2a and b).

Further therapy:

- 1 Regular bladder voiding every 4–5 h, as described above, after having deflated the cuffs.
- 2 Intermittent low dosage infection prophylaxis (4 weeks TMPS, 4 weeks Nitrofurantoin and 4 weeks interval).
- 3 Next check-up in 1 year.

In summary, the management of this patient's reflex and neurogenic stress incontinence was only possible by operative procedures. The prerequisites for the treatment of neurogenic stress incontinence by the implantation of the Scott-sphincter was adequate management for the low compliance bladder which was achieved by auto-augmentation in a satisfactory way. Nevertheless, this patient deserves regular follow-up to check his bladder compliance and the intravesical pressures that are required for bladder emptying.