ACUTE AND CHRONIC USE OF A SACRAL POSTERIOR AND ANTERIOR NERVE ROOT STIMULATOR TO INCREASE BLADDER CAPACITY IN SPINAL CORD INJURY

Kirkham APS, Knight SL, Casey ATM, Avenell S, Creasey G, Shah PJR, Craggs MD
Royal National Orthopaedic Hospital, Stanmore, Middlesex, UK
and Institute of Urology, University College London, UK

Abstract
Neuromodulation by stimulation of pudendal afferents has been shown to increase bladder capacity in spinal cord injury. It may be an alternative to the posterior rhizotomy of the sensory roots from S2 to S4 that normally accompanies a Finetech-Brindley Sacral Anterior Root Stimulator, resulting in the loss of reflex erections. We describe two patients in whom continuous low level stimulation of the mixed sacral nerves via an extradural Finetech-Brindley device resulted in effective suppression of detrusor hyperreflexia. Neuromodulation in the laboratory increased bladder by up to 300% using either continuous or conditional stimulation, and neuromodulation at home resulted in a bladder capacity comparable to that achieved with oxybutinin. Although these results suggest that neuromodulation may be a replacement for posterior rhizotomy, persisting detrusor-sphincter dyssynergia may affect stimulator-driven emptying and is probably the reason for incomplete voiding in the patients described in this study.

Introduction/Background
Detrusor hyperreflexia (DH) and detrusor-external urethral sphincter dyssynergia (DSD) are common consequences of suprasacral spinal cord injury and may cause incontinence, reduced bladder capacity and high intravesical pressure leading to vesicoureteric reflux and renal impairment.

The Finetech-Brindley Sacral Anterior Root Stimulator (SARS) is an effective and established way of managing the bladder in spinal cord injury. It is normally accompanied by a rhizotomy of the posterior (sensory) roots between S2 and S4 in most cases abolishing DH, increasing bladder capacity and preventing DSD during stimulator-driven emptying. Although rhizotomy is effective, it has a number of disadvantages - it is destructive, abolishes reflex erections in men and may worsen stress urinary incontinence. These drawbacks discourage many patients from considering a SARS device.

We are currently investigating the use of implant-driven neuromodulation as an alternative to rhizotomy. Instead of cutting the posterior roots, they are stimulated at a low level to suppress detrusor hyperreflexia. When necessary, the anterior roots are stimulated at a higher level for bladder emptying.

Neuromodulation is effective - over the last thirty years pudendal afferent stimulation (whether by electrical or magnetic stimulation of the sacral roots, or electrical stimulation of the dorsal penile/clitoral nerve or anus) has been shown to suppress DH and increase bladder capacity in humans. However, there is limited experience of using it chronically in spinal cord injury.

We hypothesised that continuous low-level mixed sacral nerve root stimulation applied via the conventional extradural Finetech-Brindley device without rhizotomy (this configuration is better termed a Sacral Anterior and Posterior Root Stimulator Implant – SPARSI) would increase bladder capacity to a clinically useful degree. Below we describe our experience with such a device in two patients.

Methods
Patient details: Patient PG (38y) sustained a complete T10 spinal cord injury in 1995. Patient DL (35y) sustained a complete T4 spinal cord injury in 1995. Both were previously managed by high dose anticholinergic medication and intermittent self-catheterisation.

Preoperatively, anticholinergics were stopped and continuous surface stimulation of the dorsal penile nerve at 15Hz and twice the current necessary to produce a pudendo-anal reflex was used to check the response to neuromodulation during slow bladder filling. Bladder capacity was increased from 210ml to 360ml and from 171ml to 512ml in PG and DL respectively.

Both patients underwent implantation of a 2 channel extradural Finetech-Brindley sacral nerve stimulator. In each case, the electrodes of one channel were applied to the mixed S3 and S4 roots bilaterally, and those of the other channel to S2 bilaterally.

After stopping anticholinergics, neuromodulation was tested in the laboratory a) with provocation of hyperreflexic contractions by rapid instillation of 60ml saline and b) during slow-fill cystometries with a filling rate of 10ml/min. In the provocation experiments, neuromodulation was conditional – activated once bladder pressure rose by 10cm water, and stopped when...
intravesical pressure fell to baseline. In the slow fills, it was either continuous – applied throughout filling, or conditional – applied for one minute each time intravesical pressure rose by 10cm water. Filling was stopped when there was incontinence or a sustained pressure rise above 35cm water, and bladder volume measured by aspiration. A four-channel microtip transducer catheter (Gaeltech, Isle of Skye, UK) was used to measure bladder and urethral pressure, and a one channel microtip catheter used for measurement of anal sphincter pressure.

Neuromodulation was always at a frequency of 15Hz, which has been found to be optimum in previous experiments using dorsal penile nerve stimulation. It also results in a less jerky skeletal muscle response than frequencies below 10Hz. The threshold for successful neuromodulation was determined using provocations and stimulation of either channel. In both the laboratory and at home, we used pulse widths between 1.5 and 5 times this threshold level to compensate for changes in stimulation intensity due to transmitter and receiver block position. This level of stimulation was always below that which resulted in bladder contraction.

In one patient (PG), neuromodulation was used at home. This was initially continuous, with stimulation on both channels at between 2 and 4 times the threshold for neuromodulation determined during provocation experiments. Bladder capacity was determined using self-catheterisation, and where incontinence occurred before catheterisation, an attempt was made to estimate its volume. Continuous neuromodulation was then compared with a) a control period with no stimulation, b) intermittent stimulation: 51 seconds on, 51 seconds off (these are the maximum periods allowed by the stimulator box) and c) anticholinergic suppression with oxybutinin 30mg a day. Because the patient often self catheterised before his bladder was full, and because the estimates of volume leaked were conservative, the mean volume at self catheterisation underestimated the bladder capacity and the maximum values in each period gave the best indication of the achievable bladder capacity. Non-parametric statistical tests were used to compare the results in each period.

Results

Both patients had preserved reflex erections and persistent detrusor hyperreflexia. It was difficult to reliably provoke contractions with a single 60ml instillation in PG, and although neuromodulation was usually effective, it was not always so. Clearer results were obtained in DL (figure 1). Neuromodulation was effective via either channel.

Figure 1 Intravesical pressure trace showing the method for determination of the threshold pulse width for suppression of provoked hyperreflexic contractions, using each channel.

In DL, continuous stimulation increased bladder capacity from a mean of 290ml in 3 control fills to a mean of 390ml in three fills with neuromodulation. In PG, control capacity of 100-170ml was increased to over 500ml using either continuous or conditional neuromodulation (figures 2,3).

Figure 2 Sample intravesical pressure traces with continuous and conditional neuromodulation.

Figure 3 Bladder capacity during two days’ testing with neuromodulation.
Volumes at self catheterisation were obtained in PG during 4 periods in the year 2000, a total of 26 days (figures 4,5).

**Figure 4** Serial bladder volumes at self catheterisation in four separate periods. Where there was leakage of urine before catheterisation, an attempt was made to estimate its volume: the clear boxes on the top of some histogram bars represent this estimation.

The median volume at ISC with continuous stimulation, intermittent stimulation and oxybutinin was in each case 250ml; the median control volume was 150ml. Because the catheterisation interval was determined by the patient, these results underestimate bladder capacity. Even so, the volumes with i) continuous and ii) intermittent stimulation were each significantly higher (p<0.01, Mann-Whitney test) than in the control period. Skeletal muscle contraction during use at home was small and not inconvenient.

**Figure 5** Median values, 25th percentiles and ranges of the volumes in figure 4.

In both patients, strong intermittent stimulation of the S3 channel produced a rise in bladder pressure of >80cm water and interval voiding. However, neither patient voided more than 50% of bladder volume, probably due to persisting detrusor-external urethral sphincter dyssynergia. We are currently investigating strategies to minimise this.

**Discussion/Conclusions**

These results demonstrate that neuromodulation via a SPARS is an effective way of increasing bladder capacity both in the laboratory and during everyday use at home. Percutaneous sacral root stimulation has previously been shown to increase capacity in spinal cord injured patients, but the great virtue of the approach described here is that one device has the potential to be used both for bladder emptying and for neuromodulation. The volumes achieved at home are comparable with the effects of high dose anticholinergics, and it is notable that in this study intermittent stimulation was as effective as continuous. This is probably due to the ‘carry over’ effects of neuromodulation, which may last several minutes. The efficacy of conditional stimulation in the laboratory suggests that an event-driven system for neuromodulation is feasible, and such a system has already been demonstrated in anaesthetised cats.

Although these results suggest that neuromodulation may be an alternative to posterior rhizotomy, persisting detrusor-sphincter dyssynergia may compromise stimulator-driven bladder emptying and further studies are needed to establish how significant this problem will be.

**Acknowledgments:** This project was funded by a Clinical Fellowship from the Special Trustees of the Royal National Orthopaedic Hospital (and associated Culyer funds from the UK Department of Health). Generous financial assistance has also been provided by Neurocontrol Corporation, USA and Finetech Medical Limited, UK.

**References**

stimulation. To be published in *Neurourology and Urodynamics (accepted)*