Treatment of refractory urge urinary incontinence with sacral spinal nerve stimulation in multiple sclerosis patients

J L H Ruud Bosch, Jan Groen

Summary

Background Urinary incontinence in multiple sclerosis (MS) patients is usually due to detrusor hyperreflexia. Patients who do not respond to conservative measures such as anticholinergics, with or without clean intermittent catheterisation, are difficult to manage.

Methods We applied electrical stimulation to the S3 sacral spinal nerves with the aim of activating afferent somatic nerve fibres. Stimulation of these fibres can inhibit the micturition reflex. An S3 electrode coupled to a subcutaneously placed pulse generator was implanted in four women who had shown a good response during temporary stimulation via a percutaneously placed wire electrode. All patients were followed for at least 2 years.

Findings The number of leakage episodes decreased from a mean of 4 to 0·3 per 24 h. Two patients were completely dry. The hyperreflexia disappeared in one, improved in two, and got worse in one patient. The urodynamic result in the last patient may be explained by clinical progression of the multiple sclerosis.

Interpretation Chronic stimulation of the S3 sacral spinal nerve by an implantable neuroprosthesis is a promising treatment option for selected multiple sclerosis patients with refractory urge incontinence.

Lancet 1996; 348: 717–19

Introduction

Incontinence in multiple sclerosis (MS) patients is usually due to detrusor hyperreflexia. The commonest cause of hyperreflexia in MS is spinal pathology. In cats, when a spinal lesion interrupts the spinobulbospinal pathways of the micturition reflex, a new sacral segmental reflex arc may become functional as a result of neuroplasticity. The afferent neurons of this reflex are unmyelinated C-fibres which serve as the afferent arc for detrusor hyperreflexia. Incontinent MS patients, who are refractory to conservative treatment with anticholinergic drugs, with or without clean intermittent catheterisation, are difficult to manage. Of the sacral spinal nerves, S3 is the most practical nerves to activate for bladder inhibition. The use of low amplitude adjustable within a range of 0·5–20 mA). The use of low amplitude and short (210 ms) stimuli ensures preferential depolarisation of larger myelinated nerve fibres. The thin and unmyelinated pain fibres and autonomic fibres are not activated. During stimulation the patients perceive a slight pulling sensation near the rectum which extends towards the labia. The muscle response associated with S3 stimulation is a bellows-like inward

Department of Urology, Erasmus University and Academic Hospital, Rotterdam, the Netherlands (J L H R Bosch MD, J Groen MD)

Correspondence to: Dr J L H R Bosch, AZR-Dijkzigt, Department of Urology, H-1073, Dr Molewaterplein 40, 3015GD Rotterdam, the Netherlands

Vol 348 • September 14, 1996 717
THE LANCET

Symptoms recurred after removal of the test electrode. Patients were considered eligible for a permanent implant provided the patient showed a successful result during the test. The stimulation can be adjusted telemetrically.

Subchronic test stimulation

An electrode (3-0 flexon pacer wire, Becton and Dickinson) was placed in the S3 foramen giving the best pelvic floor muscle response on one or both sides and of nerve integrity. Apart from the efferent somatic (sensory) nerve fibres in the pudendal nerve, and muscle afferents from the limbs,6,7,12 these fibres are stimulated (invisibly) with the external nerve stimulator and left in situ for 5 days. The movement of the levator ani muscle and plantar flexion of the great toe. This response acts as a biological indicator of correct needle position and of nerve integrity. Apart from the efferent somatic (motor) nerve fibres innervating the pelvic floor muscles, afferent somatic (sensory) nerve fibres are stimulated (invisibly) with the aim of inhibiting the micturition reflex.

Subchronic test stimulation

An electrode (3-0 flexon pacer wire, Becton and Dickinson) was placed in the S3 foramen giving the best pelvic floor muscle response to stimulation. Good muscle responses on one or both sides were found in all patients. The electrode was connected to the external nerve stimulator and left in situ for 5 days. The patients were instructed to keep the stimulation on continuously and to keep another voiding/incontinence diary. Patients with a non-muscular somatic (sensory) needle placed in the S3 foramen that showed a successful result during the test. The lead was fixed in the foramen and urodynamic studies which were done 6 months after implantation with electrical stimulation on.

Results

All six patients were women. In patient 5 (table 1) it was not possible to place a temporary electrode due to obesity. Of the five women who completed the test period, four responded with a greater than 50% reduction and one with a 20% reduction in pad use and/or leakage episodes. A permanent electrode was not implanted in this last patient (patient 6). Although, on occasion, she had voided volumes of up to 160 mL, her cystometric capacity was only 115 mL. Her more severe hyperreflexia than patients 1-5 may explain her limited response. The duration of follow-up in the patients with permanent electrodes is shown in table 2.

The symptomatic results of S3 sacral spinal nerve stimulation are summarised in table 2. The number of leakage episodes decreased from a mean of 4 to 0.3 per 24 h. Two patients were completely dry. In all patients the improvements in the number of leakage episodes were stable at long-term follow-up and even slightly better than that found during the test period.

Table 3: Urodynamic data before implant (pre) and 6 months after implant

Permanent electrode implant

The implant consists of a Pisces Quad 3886 foramen lead connected, via a 7495 extension lead, to an Itrel II 7424 pulse generator (Medtronic, Kerkrade, the Netherlands), placed subcutaneously in the lower abdomen. The lead is fixed in the foramen. The lead was fixed in the foramen which showed a successful result during the test. The stimulation can be adjusted telemetrically.

Follow-up tests included regular voiding/incontinence diaries and urodynamic studies which were done 6 months after implantation with electrical stimulation on.

Results

All six patients were women. In patient 5 (table 1) it was not possible to place a temporary electrode due to obesity. Of the five women who completed the test period, four responded with a greater than 50% reduction and one with a 20% reduction in pad use and/or leakage episodes. A permanent electrode was not implanted in this last patient (patient 6). Although, on occasion, she had voided volumes of up to 160 mL, her cystometric capacity was only 115 mL. Her more severe hyperreflexia than patients 1-5 may explain her limited response. The duration of follow-up in the patients with permanent electrodes is shown in table 2.

The symptomatic results of S3 sacral spinal nerve stimulation are summarised in table 2. The number of leakage episodes decreased from a mean of 4 to 0.3 per 24 h. Two patients were completely dry. In all patients the improvements in the number of leakage episodes were stable at long-term follow-up and even slightly better than that found during the test period.

The urodynamic data at 6 months follow-up are summarised in table 3. The hyperreflexia disappeared in patient 3. In patient 1 the volume at first and at maximum hyperreflexic contraction as well as the bladder capacity increased; in patient 2 the amplitudes of the hyperreflexic contractions clearly decreased. In patient 4 the urodynamic situation deteriorated. There have been no complications.

Discussion

Our results show that treatment of refractory urge incontinence by chronic S3 sacral spinal nerve stimulation is feasible in selected MS patients.

An explanation for the effectiveness of this treatment in detrusor hyperreflexia is based on animal experiments and electrophysiological studies in human beings. Spinal inhibitory systems capable of interrupting a detrusor contraction can be activated by electrical stimulation of afferent anorectal branches of the pelvic nerve, afferent non-muscular somatic (sensory) fibres in the pudendal nerve, and muscle afferents from the limbs.6,12 Tense fibres pass through the sacral spinal nerves. Afferents from the pelvic floor muscles are without an inhibitory effect on the bladder, at least in the cat.12 All six patients were women, who generally have more to gain from being dry than men since there are no good collection devices for women. In three of our four patients the urodynamic results were in agreement with the voiding diary results, although complete disappearance of detrusor hyperreflexia does not seem to be a prerequisite for an excellent symptomatic result. The provocative nature of

Table 2: Results of voiding/incontinence diary measures comparing pre treatment (pre) with subchronic test period (test), 6 months, and last follow-up (last)

Table 3: Urodynamic data before implant (pre) and 6 months after implant

<table>
<thead>
<tr>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bladder volume (mL) at 1st hyperreflexic contraction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>440</td>
<td>195</td>
<td>175</td>
</tr>
<tr>
<td>6 mo</td>
<td>405</td>
<td>328</td>
<td>—</td>
</tr>
<tr>
<td>Amplitude (cm H2O) of 1st hyperreflexic contraction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>32</td>
<td>22</td>
<td>48</td>
</tr>
<tr>
<td>6 mo</td>
<td>18</td>
<td>46</td>
<td>—</td>
</tr>
<tr>
<td>Bladder volume (mL) at maximum hyperreflexic contraction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>460</td>
<td>201</td>
<td>175</td>
</tr>
<tr>
<td>6 mo</td>
<td>452</td>
<td>331</td>
<td>—</td>
</tr>
<tr>
<td>Amplitude (cm H2O) of maximum hyperreflexic contraction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>56</td>
<td>35</td>
<td>48</td>
</tr>
<tr>
<td>6 mo</td>
<td>25</td>
<td>46</td>
<td>—</td>
</tr>
<tr>
<td>Bladder capacity (mL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>478</td>
<td>201</td>
<td>177</td>
</tr>
<tr>
<td>6 mo</td>
<td>452</td>
<td>331</td>
<td>492</td>
</tr>
</tbody>
</table>

— No hyperreflexia present. Follow-up urodynamic studies were done with electrical stimulation on.
standard medium-fill urodynamic tests as opposed to natural filling could be an explanation for this discrepancy. In patient 4 symptomatic improvement was good although urodynamically the situation had deteriorated. The symptomatic improvement in this patient can be explained by a decreased 24 h voided volume which shows that she had changed her drinking habits. This may have been due to increasing apathy, which characterised the clinical progression of the MS in this patient. The progression started about 6 months after the implant.

Patients should be cautioned that the clinical picture of MS often evolves in an unpredictable way and that progression of the disease may jeopardise the long-term result. The low complication rate and the urodynamic and durable clinical effects achieved support the soundness of this approach and show that it is worthwhile to continue to explore this treatment option in selected MS patients. The fact that no irreversible changes to the bladder or nerves occur is an advantage of this treatment option over destructive alternatives. Before general acceptance, longer follow-up is needed as well as studies comparing groups of patients randomly assigned to implantation or no treatment to assess possible spontaneous improvements.

This study was supported by a grant from the Health Insurance Board of the Netherlands (Ziekenfondsraad; OGP 90-005).

References