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Abstract Reproduction Form B-1

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Title (type in CAPITAL LETTERS): **SACRAL ROOT TEST STIMULATION – OVERCOMING LEAD MIGRATION USING A NEW COILED LEAD**

Aims of Study:

Sacral nerve root neuromodulation is becoming accepted treatment in the management of refractory lower urinary tract dysfunction (1). Refractory urge incontinence, severe sensory/urgency syndrome and idiopathic chronic urinary retention are the usual indications for sacral nerve stimulation. The ease of insertion, pre-operative test stimulation and low complication rate makes sacral nerve stimulation an attractive therapeutic option when compared with more invasive surgical procedures (eg. Augmentation cystoplasty and urinary diversion).

Patients are assessed for suitability of a permanently placed sacral nerve stimulator by their response to a trial stimulation period (typically one week) using a temporary S3 electrode. The peripheral nerve evaluation (PNE) model 041830-002 lead (Medtronic, USA) is the usual lead used for the trial stimulation. Migration is the major problem with this lead. This frequently results in having to repeat the trial stimulation. The clinician is often faced with the dilemma of deciding whether a negative trial stimulation is due to lead migration or the patient being a non-responder. The PNE model 3057 lead (Medtronic, USA) was designed to eliminate lead migration. This new lead is coiled and consists of an insulated, multistranded wire with a stainless steel connector pin on one end and the exposed coiled wire electrode on the other end. The lead has a removable stylet to give stiffness to the lead, which allows for placement down a foramen needle.

The aim of this study was to compare the amount of lead migration of the PNE model 041830-002 lead with the PNE model 3057 lead.

Methods:

Ten women aged 23 to 79 years (median age 60) with refractory lower urinary tract dysfunction were studied. Diagnoses were: detrusor instability (6 cases) and bladder sensory urgency (4 cases). Patient assessment included history and physical examination, bladder diary, multichannel urodynamic studies and cystoscopy in patients with bladder sensory urgency.
Australian Federal Government Therapeutic Goods Authority approval was obtained to use the PNE model 3057 lead.
Under a combination of sedation and local anaesthesia, foramen needles were placed in both right and left S3 foramina. Suitable positioning of the foramen needles was assessed by the appropriate motor and sensory response to electrical stimulation. The leads were then passed down the foramen needles. The foramen needles were removed and the leads secured in position. The PNE model 041820-002 lead (old lead) was placed on one side and the model 3057 lead (new lead) placed on the contralateral side. The new lead was placed on the patient's right side unless a better motor response with stimulation of the foramen needle was

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Elicited on the left side. Patients remained in the prone position until a lateral sacral X-ray was performed immediately after lead placement. Leads remained in situ for up to 2 weeks. Repeat lateral sacral X-ray was performed just prior to lead removal.
The amount of migration of the old and new leads during the test stimulation period was measured by comparing the position of the leads on the X-rays at the start and end of the stimulation period. The distance measured was from the lead tip to the ventral aspect of the S3 foramen.

Results:

Lead migration

The new lead migrated a median distance of 5 mm (range 2-13 mm) and the old lead 11 mm (10-45mm) [$p < 0.05$].

Complications

No serious complication occurred during the test stimulation period. In one patient, the connector pin of a new lead became detached, requiring placement of another lead.

Conclusions:

The new peripheral nerve evaluation lead (model 3057-Medtronic, USA) was associated with minimal migration during the trial stimulation. Using this new lead will result in not having to place leads bilaterally and reduce the need for repeat of stimulation. The new lead will also reduce the uncertainty often caused by not knowing whether a negative trial stimulation is due to lead migration or a patient being a non-responder.

References:

I. J Urol 1998, 159: 1515-1519