Temporary sacral nerve stimulation in patients with fecal incontinence owing to rectal hyposensitivity: A prospective, double-blind study

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Background. *Rectal hyposensitivity (RH) can lead to fecal incontinence (FI). Sacral nerve stimulation (SNS) is known to modulate rectal sensation, but no data about affecting FI owing to RH are available. This prospective study aimed to assess the therapeutic effect of temporary SNS on patients with FI owing to RH.*
Methods. *Twenty-four patients with FI owing to RH had temporary SNS (4 weeks on followed by 1 week off). Before SNS (baseline), after 4 weeks of stimulation (on), and at the end of the off week we recorded first constant sensation (FCS), defecatory desire volume (DDV), maximum tolerated volume (MTV), anal pressures, bowel diaries, Wexner incontinence score, and FI quality-of-life score (FIQOL).*
Results. *There were significant decreases in DDV and MTV during the on-treatment period ($P < .0001$); this decrease was not significant during the off period. FCS was not significantly affected by SNS. FI episodes significantly improved during the on period in 22 patients (from 5.3 to 1.1 per week; $P < .0001$) and mean Wexner incontinence score improved from 13.3 to 1.7 ($P < .0001$). Anal pressures (resting and squeeze) significantly increased during the on period but not during the off period. There was significant improvement in FIQOL during the on period only.*
Conclusion. *SNS can be effective in restoring continence and improving QOL in patients with FI owing to RH. Improved continence might be related to improvement of rectal sensation and/or increased anal pressure. The washout effect of SNS on the continence score, DDV, and MTV after cessation of stimulation needs to be explained. (Surgery 2015;157:56-63.)*

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Intact anorectal sensation is fundamental to normal anorectal functions, including defecation and continence. Normally there is a complex interaction between sensory and motor function,[1] and abnormalities of either component may result in disorders of evacuation or continence. Rectal hyposensitivity (RH) is a physiologic abnormality that relates to a diminished perception of rectal distension. It is present in 16% of all patients attending for physiologic assessment of anorectal dysfunction, with an equal prevalence among males and females.[2]

The causes of RH are unknown. Traditionally, RH might reflect impaired afferent nerve function peripherally or centrally. Direct injuries to the pelvic or sacral nerves, diabetes mellitus, multiple sclerosis, and cerebrospinal disease have been implicated. Still we have a considerable percentage of idiopathic RH.[3]

RH has frequently been reported in patients with chronic constipation and idiopathic fecal incontinence (FI). Moreover, it has been reported to be a predictor of poor outcome in the treatment of FI with biofeedback techniques and surgery.[4] However, despite these observations, the presence of RH is not often considered when clinical

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decisions are made regarding the selection of patients with FI for surgery.[4]

Rectal sensory function is commonly quantified by recording the threshold volumes required to elicit first constant sensation (FCS), urge to defecate volume, and maximum tolerated volume (MTV). RH is defined as an increase in the sensory thresholds beyond the normal range.[3,5]

In patients with RH, the precise pathophysiologic mechanisms involved in the development of symptoms are unclear; however, in constipation, sensorimotor dysfunction of the rectum,[6] reflex (viscero-visceral) inhibition of proximal gut function, and secondary colonic dysmotility (rectocolonic inhibitory reflexes) are all possible contributing factors.[7] Although FI might be the result of internal anal sphincter relaxation, owing to a chronic rectoanal reflex inhibition without sufficient compensation by external anal sphincter contraction.[5]

Sacral nerve stimulation (SNS) has been proven repeatedly to be an effective treatment for patients suffering from FI.[8,9] Abdel Halim et al[10] reported that temporary SNS does not change rectal compliance, but is associated with significant changes to the pressure thresholds of rectal distension. Therefore, SNS may be effective in the treatment of FI owing to RH. Therefore, we have evaluated the efficacy of SNS in patients of FI owing to RH.

PURPOSE

The aim of this study was to assess prospectively the effect of SNS on FI owing to RH.

PATIENTS AND METHODS

The current study prospectively included 24 adult patients with FI owing to RH operated in the period from February to October 2013. Patients were recruited consecutively from those failing conservative biofeedback treatments and attending the units of Colon and Rectal Surgery at two tertiary referral centers (Health Insurance Institute and Alexandria University Hospital) to undergo investigation for FI a >4-year period from June 2009 to December 2013.

FI was defined as patients with a Wexner incontinence score of >10.[11] RH was defined as ≥2 of 3 abnormal values of FCS, defecatory desire volume (DDV), and MTV compared with departmental control ranges for normal persons. Ethics approval was granted by the Alexandria University Ethics Committee and all patients provided written consent to participate before any intervention. The study was entered on to the International Clinical Trials Database before starting operations in 2013 (Trial ID: ACTRN12613000099729).

Exclusion criteria included age (<16), diabetic neuropathy, pudendal nerve neuropathy (pudendal nerve terminal motor latency >2.2 ms), multiple sclerosis, Parkinson disease, stroke, bleeding disorders, and cardiac pacemakers. In addition, patients were excluded if they had external anal sphincter injury, sphincter denervation, rectal prolapse, Hirschsprung disease, inflammatory bowel disease, were pregnant, or had severe cardiac disease or chronic renal failure.

All patients undergoing SNS were symptomatic for ≥2 years, and all conservative measures, including dietary, pharmacologic, and biofeedback treatments, had failed.

SNS methods. Temporary stimulation was performed with unilateral percutaneous nerve evaluation performed by insertion of a stimulating electrode (3065USC; Medtronic, Minneapolis, MN) into the S3 sacral foramen under local anesthesia.[12] Default stimulation parameters were set at 210-microsecond pulse width, 15-Hz frequency, and a subsensory amplitude ranging between 0.5 and 10 V. Stimulation was continuous for 4 weeks followed by 1 week off treatment. Successful percutaneous nerve evaluation (on basis of clinical reduction of incontinence episodes by ≥50% as judged by physician and patient) was followed by the offer of permanent stimulation.

Anal manometry and assessment of rectal sensory function. Anorectal manometry using station pull-through perfusion catheter systems by the use of an 8-channel, water-perfused manometry catheter (Synectics, Stockholm, Sweden)[13] was done for all patients with evaluation of mean anal resting pressure (MARP) and maximum anal squeezing pressure (MSP).

Rectal sensory thresholds to distension were assessed using the intrarectal bag. Patients were asked to report when they had FCS, DDV, and MTV. The volume at each threshold point was recorded for each patient.

Patient evaluation. All patients were evaluated preoperatively by clinical interview and examination. The clinical interview included a detailed questionnaire with special reference to frequency of incontinence, stool consistency, and past history of pelvic or anorectal surgery. Preoperative assessment included anorectal physiology studies (measuring MARP, MSP, FCS, DDV, and MTV). Endoanal ultrasonography (10-MHz transducer; Hitachi Medical, Tokyo, Japan) was done by an experienced radiologist to categorize the internal and external anal sphincters as intact or disrupted. Electromyography for anal sphincters was done to exclude patients with sphincter denervation. Grading of incontinence severity was performed