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Sacral Neuromodulation: Does It Affect the Rectoanal Angle in Patients with Fecal Incontinence?

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Abstract
Background In the past decade numerous studies have been published on the successful treatment of fecal incontinence with sacral neuromodulation (SNM). The underlying mechanism of action for lower bowel motility disorders has been unclear. In the present study, the effect of SNM on the rectoanal angle in patients with fecal incontinence was investigated.
Patients and Methods In 12 consecutive patients who qualified for SNM an X-defecography study was performed before SNM and at 6 months after permanent implant. Three single lateral rectal views were taken: one during rest, one during squeeze, and one during Valsalva's maneuver, after which the patient was asked to evacuate as rapidly and completely as possible during lateral fluoroscopy. At 6 months two further defecography studies were performed, one during stimulation with the pacemaker on and one with the pacemaker off.
Results The defecography studies showed that the rectoanal angle decreased during rest, squeeze, and Valsalva's maneuver. A slight increase in rectoanal angle was seen during defecation. However, the differences did not reach statistical significance. Sacral neuromodulation improved fecal continence significantly in all patients at 6 months. Median incontinence episodes per week decreased from 6.2 to 1.0 ($P = 0.001$), and incontinent days per week decreased from 3.7 to 1.0 ($P = 0.001$) with SNM. There were no significant changes in the median resting and squeeze anal canal pressures, 46.5 versus 49.7 mmHg and 67.1 versus 72.3 mmHg, respectively. Median stimulation amplitude at follow-up was 2.7 V (range: 0.9–5.3 V).
Conclusions Rectoanal angle did not decrease significantly in patients with fecal incontinence during SNM.

Keywords Fecal incontinence · Sacral neuromodulation · Defecography · Sacral nerve stimulation

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Introduction

Sacral neuromodulation (SNM) has emerged as a successful treatment for urinary voiding disorders and lower bowel motility disorders in recent years [1, 2]. However, understanding and knowledge about the underlying mechanism of action for urological disorders as well as lower bowel motility disorders remains limited. Since 1995, numerous studies have been published on SNM as a potential treatment for fecal incontinence. Some authors have demonstrated a significant increase in both the maximum resting and squeeze anal canal pressures, whereas others have not [3, 4]. Consequently, a simple direct effect on the efferent motor nerve supply of the anal sphincters resulting in increased pressures is not likely to be the underlying mechanism of action. Several other hypotheses on the mechanism of action have been suggested: altered rectal sensation and motility, effects on the sensory and

autonomic function, modulation of anorectal reflexes and corticospinal pathways, and a central modulation effect [5–8]. The observed clinical effect of SNM is probably caused by a combination of all these mechanisms. Through stimulation or modulation of the sacral nerve plexus, all structures and pathways involved in continence and defecation are likely to be affected and altered.

Matzel et al. [9] performed a cadaveric dissection study of the neuroanatomy of the striated musculature of the anal continence mechanism. They found that the neural supply of the pelvic floor and external anal sphincter derives from S2–S4. However direct branches emerging proximal to the sacral plexus supply the levator ani and puborectal muscles. The remaining fibers of the sacral nerves form the sacral plexus, from which the pudendal nerve originates, supplying the external anal sphincter. The functional relevance of these findings was also investigated in five patients with lower urinary tract dysfunction in whom pudendal nerve stimulation resulted in a maximal rise in anal canal pressure. Stimulation of the root of S2 also increased anal canal pressure, probably through the pudendal nerve, its contribution of motor fibers deriving mostly from S2. Stimulation of S3 caused a contraction of the pelvic floor musculature with a slight increase in anal canal pressure, but primarily, and maybe more importantly, decreasing the rectoanal angle.

An analysis by our own group of all studies published showed that in those studies reporting an increase in anal canal pressures the maximum stimulation amplitude comfortable to the patient was used. In those studies using voltages just above the sensory threshold for stimulation, no increases in anal canal pressures were found [10]. The same publication showed that a therapeutic effect is obtainable with stimulation below the sensory threshold while the resting and squeeze anal canal pressures remained unaffected during stimulation [10]. As expected, the motor threshold was significantly higher than the sensory threshold. Thus a chronic low-grade stimulation at sensory or therapeutic (lower) threshold is insufficient to raise anal canal pressures, providing evidence against a role for increased resting and squeeze anal canal pressures as the mechanism of action of SNM in fecal incontinence. Against this background and with the notion that the neural supply of the pelvic floor musculature is distinct from that of the external anal sphincter, we hypothesized that the mechanism of action of SNM might involve an effect on the rectoanal angle in patients with fecal incontinence.

The aim of the present study was to analyze the effect of SNM on the rectoanal angle in patients with fecal incontinence. To that purpose, data were obtained before and during SNM in patients with fecal incontinence.

Materials and methods

Twelve consecutive patients with fecal incontinence who qualified for permanent SNM were asked to undergo a defecography study before and 6 months after permanent pacemaker implantation. Patients completed a three-week bowel habits diary before and during SNM, objectifying incontinence episodes and incontinent days. Fecal incontinence was defined as involuntary loss of stool at least once a week. Conventional treatment consisting of both conservative (drug) and biofeedback therapy had failed in all patients. Exclusion criteria were a history of anorectal malformation; rectal surgery within the preceding 12 months; presence of a rectal prolapse, rectocele or intussusception; inflammatory bowel disease; and chronic diarrhea.

Resting and squeeze anal canal pressures were recorded with a Konigsberg catheter (Konigsberg Instrument Inc., Pasadena, CA) that was connected to a computer-assisted polygraph (Synectics Medical, Stockholm, Sweden). Pudendal nerve terminal motor latency (PNTML) was measured with a St. Marks's glove electrode (PNTML <2.4 ms on both sides was considered normal). Endoluminal ultrasound (SDD 2000, Multiview, Aloka, Japan, 7.5 MHz endo-anal transducer) was used to assess the external anal sphincter.

The preoperative defecography study was part of the preoperative work-up to exclude intussusception or a rectocele. To perform the X-defecography study, the rectum was filled with approximately 250 ml of barium sulfate gel, and the patient was then seated on a special commode. Three single lateral rectal views were taken, one during rest, one during squeeze, and one during Valsalva's maneuver, after which the patient was asked to evacuate as rapidly and completely as possible during lateral fluoroscopy. Oral barium solution was given as part of the standard examination. At 6 months two defecography studies were performed, one with the pacemaker on and one with the pacemaker off. Both the same radiologist and the same surgeon evaluated the defecography studies independently of each other. The rectoanal angle was measured on the study film by drawing two straight lines, one at the level of the posterior rectal wall and one at the central longitudinal axis of the anal canal.

The surgical procedure and equipment for SNM were as described extensively in previous reports [11]. To confirm electrode position, an X-ray film was taken on the day of surgery.

Data were analyzed with the Wilcoxon's signed-rank test in SPSS version 16.0 (SPSS, Chicago, IL). Results are given by their median values and range unless stated otherwise; $P < 0.05$ was considered significant.

Results

Twelve patients (11 women) with a mean age of 51.2 years (range: 26.1–68.4 years) were included in the study. Patients had been incontinent for feces for an average of 6.7 (1.5–20) years. All patients had idiopathic incontinence; additionally, one patient had a mild form of multiple sclerosis. All patients had structurally intact external sphincters on endoluminal ultrasound (two after an anal repair). Six patients also suffered from urinary incontinence. Prolonged PNTML was found in 10 patients, nine had bilateral pudendopathy; median PNTML values were 2.4 (2–5.4) ms on the right side and 2.2 (2–2.8) ms on the left side. Ten permanent electrodes were placed in the foramen of S3 (six on the right side and four on the left side) and two in the foramen of S4 (one on each side). There were only minor complications: two patients experienced wound leakage that was managed conservatively with antibiotics, and in two patients the pacemaker was re-implanted in the abdominal wall because of pain at the original implantation site in the buttock.

Defecography showed no significant change in rectoanal angle during stimulation at 6 months. However, a clear tendency was observed toward a decreased or sharpened rectoanal angle during rest, squeeze, and Valsalva maneuver, and, as shown in Figs. 1 and 2, an increased or blunted rectoanal angle was recorded during defecation with the SNM. Interobserver agreement was assessed by the Bland–Altman plot; 98% of the rectoanal angle measurements of both raters lie within ±2 SD of the mean difference (Fig. 3).

In all 12 patients the initial significant continence reduction of more than 50% during trial screening was reproduced after permanent implant (Fig. 4). The mean improvement 6 months after implantation of continence for episodes and days was, respectively, 86% (60–100%) (95% confidence interval [CI] 78–95%) and 77% (43–100%) (95% CI 63–90%). Anal manometry showed no significant difference in mean anal canal resting pressures (baseline 46.5 vs. 6 months 49.7 mmHg) and squeeze pressures (baseline 67.1 vs. 6 months 72.3 mmHg) before and during stimulation (Fig. 5). Median stimulation amplitude at 6 months follow-up was 2.7 (0.9–5.3) V (Fig. 6). Three of six patients with urinary incontinence also noticed a subjective improvement in urinary continence.

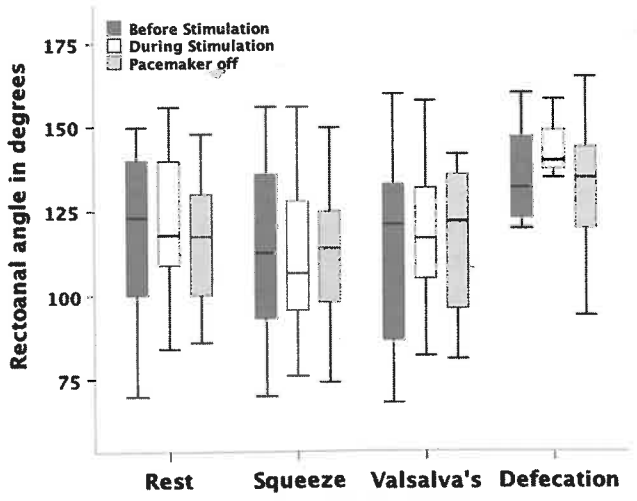

Fig. 1 Median rectoanal angles measured by the radiologist during rest, squeeze, Valsalva's maneuver, and defecation

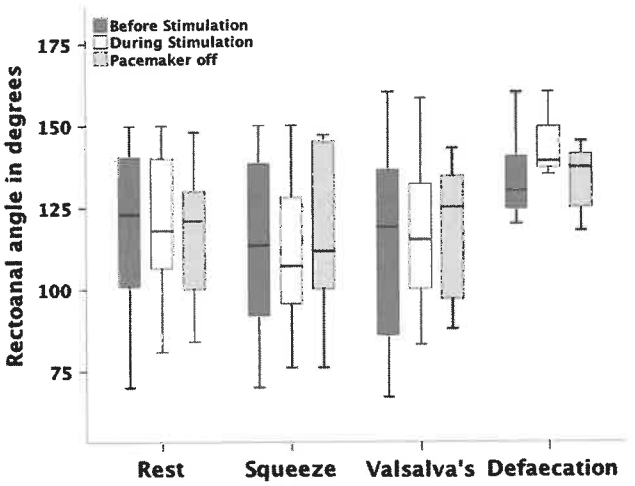

Fig. 2 Median rectoanal angles measured by the surgeon during rest, squeeze, Valsalva's maneuver, and defecation

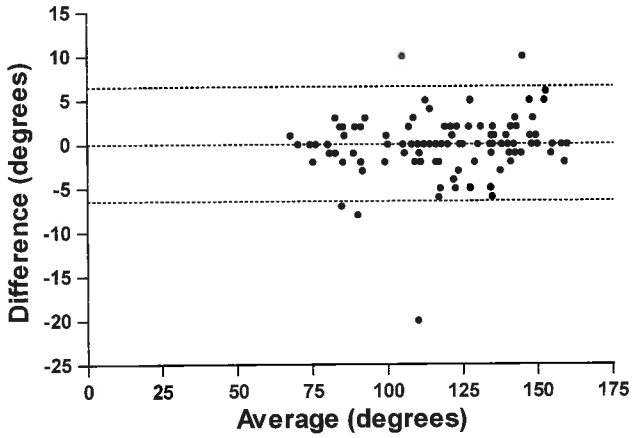

Fig. 3 Bland–Altman plot; comparison of the rectoanal angles measured by the radiologist and the surgeon

Discussion

In the present study the aim was to evaluate the effect of SNM on the rectoanal angle, as the neural supply of the pelvic floor musculature is different from that of the

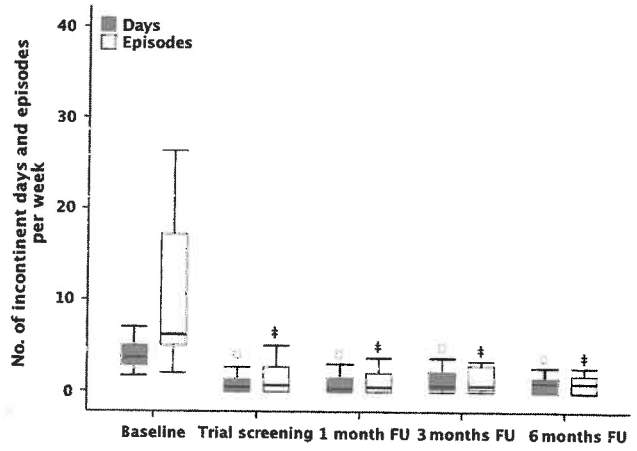

Fig. 4 Box-and-whisker plot with results of median incontinence days and episodes per week. □ $P = 0.002$; ‡ $P = 0.003$

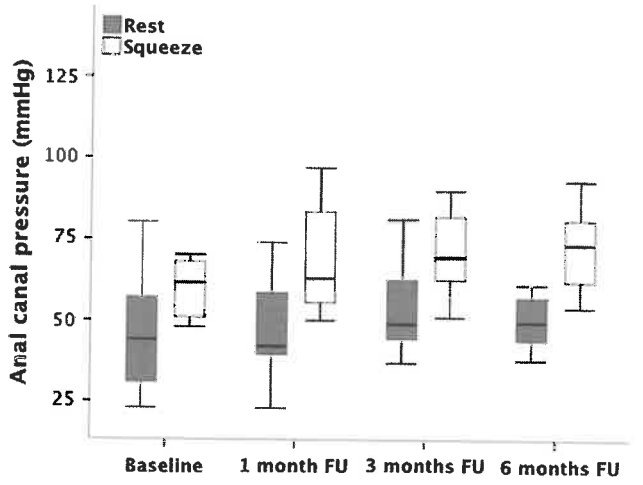

Fig. 5 Box-and-whisker plot with results of median anal canal resting and squeeze pressures

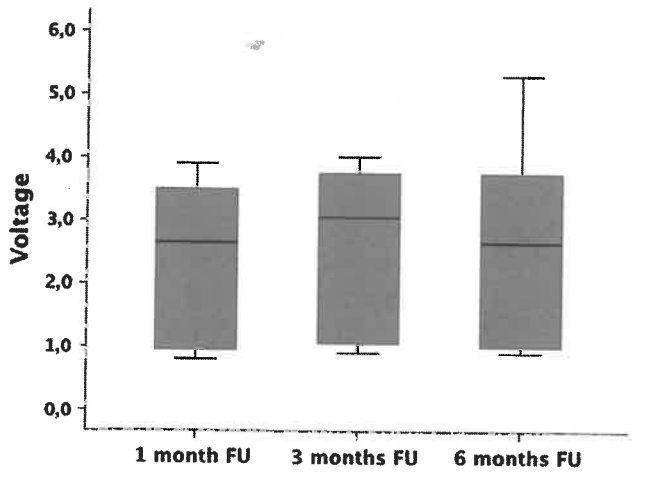

Fig. 6 Box-and-whisker plot with results of median stimulation voltages after permanent implant

external anal sphincter. A clear tendency was observed toward a decreased or sharpened rectoanal angle during rest, squeeze, and Valsalva's maneuver and an increased or blunted rectoanal angle during defecation when the SNM was activated. Although, this would be a logical action of SNM, which could at least partially explain the therapeutic effects, the differences were not statistically significant, likely because of the small population size.

The solution for fecal incontinence is probably as multifactorial as the etiology of the problem. Since the introduction of SNM, the traditional focus on anal sphincter dysfunction [12–14] has shifted toward a more complex approach. As stated by Melenhorst et al., the anal sphincter injury itself is an indication of existing damage and not the sole cause of fecal incontinence [15]. Altered rectal sensation and adaptation, as well as traction and damage to the pudendal nerve and pelvic floor, are also major contributing factors [16, 17]. This finding is supported by the observation that both biofeedback therapy and SNM can improve fecal incontinence in patients with sonographic evidence of sphincter disruption [15, 18]. This finding provides additional evidence that integrity of the sphincter is not the most important factor in fecal continence. Because the working mechanisms of treatment options targeting multiple contributing factors for fecal incontinence, like biofeedback therapy and SNM, are completely different, the positive effects are difficult to explain. Although biofeedback therapy has been commonly used as the next step in conservative treatment with an overall success rate of 72% (range: 29–92%) [19], quantification in terms of physiologic parameters of this improvement has been difficult. Some studies have shown improvements in rectal filling sensations and anal canal pressures, while other studies did not confirm these findings. Moreover, there is a lack of correlation between symptomatic improvement and manometric parameters [19–22], which is also true for SNM.

In our search of the literature we could not find a similar study comparing rectoanal angle before and after biofeedback therapy. A comparison between both therapies concerning the rectoanal angle would be of interest and might provide more insight into the respective mechanisms of action.

Pudendal nerve dysfunction is found in 38% of women with fecal incontinence after childbirth [23]. Less is known about the damage to the pelvic splanchnic nerves (S2–S4) during childbirth and the subsequent dysfunction of these nerves. The neural control of continence and evacuation mechanisms is likely to be mediated at different levels of the nervous system as combined sensory and motor dysfunctions are found in patients with fecal incontinence [24, 25]. Disruption at any level as a result of stroke, multiple sclerosis, and spinal or peripheral nerve injury can give rise to incontinence as a result of abnormalities in

visceral afferent nerve sensitivity, motor efferent nerve activity, and/or central processing.

Despite advances in our knowledge of the pathophysiology of incontinence, it is still largely unclear at what level SNM has an effect on regaining continence. The effect is probably not a simple direct efferent stimulation, as the anal sphincter contractions seen at the trial screening stage for SNM during peripheral neural evaluation are mediated by afferent input [26]. Data from biofeedback therapy suggest that both an enhanced use of the residual functional capacity and cortical awareness may be involved [27]. The latter mechanisms also seem to be true for SNM, as patients treated with SNM not only show an improvement in rectal sensation [6] but also attribute their improvement to an increased awareness to evacuate. In urological studies it has been shown that SNM modulates sensorimotor learning areas in the brain during the acute phase, as well as brain areas implicated in the awareness of bladder filling, the urge to void, and timing of micturition during the chronic phase [7]. These findings suggest that areas involved in alertness and awareness may play a role. The effect on the cortical sensory area in urological patients was also seen through somatosensory evoked potentials (SEPs) of the pudendal nerve, which showed a significant decrease in threshold voltage and SEP latency during SNM [28]. In patients with fecal incontinence SNM reduces corticoanal excitability, which is reversible, as a rebound effect is seen after termination of SNM [5]. Whether these dynamic brain changes directly influence the functional improvement in continence or whether they are a reactive result of the patient's being continent again remains to be elucidated.

Conclusions

Although the neural supply of the pelvic floor musculature is different from that of the external anal sphincter, this study did not show a statistically significant change in rectoanal angle during SNM. However, a clear tendency was observed toward a decreased or sharpened rectoanal angle during rest, squeeze, and Valsalva's maneuver. An increased or blunted rectoanal angle during defecation a small population size may account for these findings. Further research is necessary for a better understanding of the mechanism of action of SNM.

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