The Impact of Sacral Neuromodulation on Sexual Dysfunction

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Introduction
Sacral neuromodulation (SNM) has become an established option in the treatment of refractory overactive bladder, frequency-urgency syndrome, non-obstructive urinary retention, chronic pelvic pain, and chronic fecal incontinence [1]. In 1980s Tanagho and Schmidt pioneered the development of an implantable sacral electrode which would provide the basis for the concept of SNM and the InterStim device (Medtronic, Minneapolis, MN) [2]. Although the mechanism of action remains unclear, the current leading hypothesis suggests that neuromodulation acts by stimulating peripheral somatic afferent pathways in the spinal roots, which in turn modulate voiding and continence pathways in the central nervous system [3]. Other studies evaluated alterations in brain activity during SNM. Blok et al. [4] using PET and, Gill et al. [5] using MRI, concluded that SNM is responsible for activation or deactivation of brain centers via afferent neural pathways and it is dependent of the strength of the stimulus.

SNM treatment is performed as a staged procedure. At the first surgical stage, a timed quadripolar lead is percutaneously implanted in the S3 foramen using bony landmarks and fluoroscopic guidance. The nerve root is stimulated electrically to assess correct placement, which is then connected to a pulse generator left subcutaneously over the buttocks. If the patient shows ≥ 50% improvement in voiding symptoms after screening evaluation during a 1- to 2-week trial period, the device is perma-
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nently implanted. SNM additionally appears to have additional positive benefit on sexual function [1]. Reports addressing this issue are scarce. Whether improvements in sexual function are independent of, or secondary to improvements in urinary function and overall improved quality of life remains unclear [1, 6].

In this study, we evaluate the impact of SNM on sexual function, and analyze any association between SNM and age at SNM, functional diagnosis and post-void residual (PVR) urine pre-SNM.

Methods

We performed a retrospective analysis of all patients who underwent SNM for urinary or bowel dysfunction, at a single tertiary center from May 2012 to August 2016. All patients underwent a staged procedure. After a trial test, patients who presented at least a 50% symptom improvement on bladder diary were permanently implanted with InterStim II® (Medtronic, Minneapolis, MN). A single tined, 4-electrode lead was placed into S3 foramen, under local anesthesia and fluoroscopy guidance. Patients who did not qualify for permanent implantation underwent explantation of the lead.

Sexual function was assessed in all sexually active individuals, before surgery and postoperatively. Men were assessed with the simplified International Index of Erectile Function (IIEF-5) and women with the Female Sexual Function Index (FSFI) adding all domains scores (desire, arousal, lubrication, orgasm, satisfaction and pain). A total IIEF-5 core < 22 indicates male erectile dysfunction and a total FSFI score ≤ 26 indicates female sexual dysfunction. Besides, sexual function questionnaires scores, other analyzed variables included gender, age at SNM, functional diagnosis [chronic urinary retention, overactive bladder (OAB), detrusor overactivity with impaired contractility (DOIC), detrusor-sphincter dyssynergia, and fecal incontinence], underlying pathology, and PVR. An increase in sexual function scores after SNM compared to pre-SNM was considered “improvement”.

The primary endpoint was any observed change in IIEF-5 or FSFI (total score and specific domains) associated with SNM. The secondary endpoint was the association of IIEF-5 or FSFI (total score and specific domains) with age at SNM, functional diagnosis, and PVR. Regarding age at SNM, patients were divided into 2 groups: age < 40 years and > 40 years. Regarding PVR, patients were also divided into 2 groups: PVR < 150 ml and PVR ≥ 150 ml. Adverse events were also assessed. Patients not sexually active or who were lost to follow-up were excluded.

Table 1. Baseline characteristics

| Characteristic                  | Median (range) | n  | %   |
|--------------------------------|----------------|----|-----|
| Total number                   |                |    |     |
| Patients stage-1               | 49             |    |     |
| Patients stage-2               | 34             |    |     |
| Patients excluded, no active sexual life | 5  |    |     |
| Patients excluded, lost to follow-up | 5  |    |     |
| Patients included              | 24             |    |     |
| Gender                         |                |    |     |
| Female                         | 15             | 62.5|     |
| Male                           | 9              | 37.5|     |
| Age at SNM                     |                |    |     |
| < 40 years                     | 10             | 41.7|     |
| > 40 years                     | 14             | 58.3|     |
| Functional diagnosis           |                |    |     |
| CUR                            | 12             | 50  |     |
| OAB                            | 4              | 16.7|     |
| DOIC                           | 6              | 25  |     |
| DSD                            | 1              | 4.2 |     |
| FI                             | 1              | 4.2 |     |
| Underlying pathology           |                |    |     |
| Idiopathic                     | 4              | 16.7|     |
| Multiple sclerosis             | 4              | 16.7|     |
| Spinal cord injury             | 3              | 12.5|     |
| Fibromyalgia                   | 1              | 4.2 |     |
| CPPS                           | 1              | 4.2 |     |
| Endometriosis                  | 3              | 12.5|     |
| Compressive discopathy         | 1              | 4.2 |     |
| Radic myelitis                 | 1              | 4.2 |     |
| Lumbosacral plexopathy         | 1              | 4.2 |     |
| Syringomyelia                  | 1              | 4.2 |     |
| Cerebrovascular accident       | 1              | 4.2 |     |
| Benign prostatic hyperplasia   | 1              | 4.2 |     |
| Fecal incontinence             | 1              | 4.2 |     |
| Encephalitis                   | 1              | 4.2 |     |
| Follow-up, months              | 20.7(2–53)     |    |     |

CUR = Non-obstructive chronic urinary retention; DSD = detrusor-sphincter dyssynergia; FI = fecal incontinence; CPPS = chronic pelvic pain syndrome.
Clinical data was analyzed using IBM SPSS Statistics, version 24.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were reported as frequencies for categorical variables, and median (first quartile – third quartile) for continuous variables. Comparison between preoperative and postoperative sexual function scores was performed using the Wilcoxon Signed-Rank test. \( \chi^2 \)-test (two-sided Pearson \( \chi^2 \)-test with two degrees of freedom) was used between sexual function scores and patient parameters (age at SNM, functional diagnosis, PVR). Fisher’s exact test was used when the expected frequency was of five or less. Statistical significance was considered for p-values < 0.05.

### Results

Of the 49 patients who underwent stage-1 SNM, 34 were permanently implanted. Ten patients were excluded from the study (5 not sexually active and 5 lost to follow-up). Twenty-four patients met the inclusion criteria, 15 females and 9 males, with a median age of 41 years (range 26–72 years). Ten patients were < 40 years and 14 were > 40 years. Twelve patients had chronic urinary retention, 4 OAB, 6 DOIC, 1 detrusor-sphincter-dyssynergia and 1 fecal incontinence. Median follow-up was 20.7 months (range 2–53 months). Underlying pathologies are described in table 1.

| Domains      | Pre-operation [median (Q1–Q3)] | Post-operation [median (Q1–Q3)] | Improvement, n (%) | p   |
|--------------|--------------------------------|---------------------------------|--------------------|-----|
| Desire       | 3.6 (3.0–4.2)                  | 3.6 (3.0–4.2)                  | 2 (13)             | 0.18|
| Arousal      | 3.6 (2.7–4.8)                  | 4.5 (3.0–4.8)                  | 3 (20)             | 0.11|
| Lubrication  | 4.2 (3.0–5.4)                  | 4.8 (4.2–6.0)                  | 5 (33)             | 0.04|
| Orgasm       | 4.4 (2.8–4.8)                  | 4.4 (4.0–5.6)                  | 4 (27)             | 0.13|
| Satisfaction | 4.8 (2.4–5.2)                  | 4.8 (2.8–6.0)                  | 3 (20)             | 0.46|
| Pain         | 4.4 (2.8–6.0)                  | 5.2 (3.6–6.0)                  | 3 (20)             | 0.67|

Regarding FSFI, 2 patients < 40 years and 3 > 40 years had improvement (p = 1.0), showing no correlation between FSFI and age. The only detrusor-sphincter-dyssynergia patient showed significant improvement in FSFI (p = 0.05).

When FSFI domains where correlated with age at SNM, functional diagnosis and PVR, statistical significance was observed for arousal (p = 0.03), lubrication (p = 0.05) and satisfaction (p = 0.03) and functional diagnosis only. Patients with DOIC showed the best results (table 3).

Adverse events occurred in 9 patients (37.5%). The most common was lead migration (12.5%), followed by loss of battery (8.3%), no response (8.3%), partial response (4.2%) and “pocket” syndrome (4.2%), defined as pain at the buttock where the pulse generator was placed.

### Discussion

SNM is a clinically effective therapy for several urinary and bowel dysfunctions, suggesting a common control mechanism [7]. Additionally, SNM appears to have a positive effect on sexual function [1]. However, its mechanism of action on sexual function remains poorly understood. Sympathetic fibers innervate the urinary bladder through the hypogastric nerve, inhibiting bladder contraction, and exciting smooth muscle at the bladder base and urethra [8]. In men, contraction of seminal vesicles and the prostate with expulsion of sperm and seminal fluid into the posterior urethra is also mediated by sympathetic nerves which also close the bladder neck preventing retrograde flow [9]. Sympathetic nervous system opposes sexual responses by vasoconstriction but plays an important role in pelvic contractions with orgasm [10]. Parasympathetic nerves reach the pelvic plexus and

### Table 2. Improvement in FSFI scores by domain after surgery

| Domains      | Pre-operation [median (Q1–Q3)] | Post-operation [median (Q1–Q3)] | Improvement, n (%) | p   |
|--------------|--------------------------------|---------------------------------|--------------------|-----|
| Desire       | 3.6 (3.0–4.2)                  | 3.6 (3.0–4.2)                  | 2 (13)             | 0.18|
| Arousal      | 3.6 (2.7–4.8)                  | 4.5 (3.0–4.8)                  | 3 (20)             | 0.11|
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| Satisfaction | 4.8 (2.4–5.2)                  | 4.8 (2.8–6.0)                  | 3 (20)             | 0.46|
| Pain         | 4.4 (2.8–6.0)                  | 5.2 (3.6–6.0)                  | 3 (20)             | 0.67|
the bladder wall via pelvic nerves, causing bladder wall contraction [8]. Both in men and women, parasympathetic nerves are responsible for smooth muscle relaxation, vasodilation and high blood inflow resulting in genital structures tumescence such as penile erection and vulvar and clitoral swelling [10, 11]. Somatic innervation is provided by the pudendal nerve to the lower urinary tract [8] and is responsible for transmission of sensation

| Table 3. | Sexual index score association with age at SNM, functional diagnosis and PVR |
|-----------|---------------------------------------------------------------------------|
|           | Improvement, n (%) | p     |
|           |                   |       |
| IIEF-5    |                   |       |
| Age at SNM|                   |       |
| < 40 years| 2 (67)            | 0.52  |
| > 40 years| 2 (33)            |       |
| Functional diagnosis |   |       |
| CUR       | 1 (20)            | 0.12  |
| OAB       | 1 (100)           |       |
| DOIC      | 2 (100)           |       |
| DSD       | 0 (0)             |       |
| FI        | 0 (0)             |       |
| PVR       |                   |       |
| < 150 ml  | 2 (67)            | 0.52  |
| ≥ 150 ml  | 2 (33)            |       |
| FSFI: total |                 |       |
| Age at SNM|                   | 1.00  |
| < 40 years| 2 (29)            |       |
| > 40 years| 3 (38)            |       |
| Functional diagnosis |   | 0.05  |
| CUR       | 1 (14)            |       |
| OAB       | 0 (0)             |       |
| DOIC      | 3 (75)            |       |
| DSD       | 1 (100)           |       |
| FI        | 0 (0)             |       |
| PVR       |                   |       |
| < 150 ml  | 3 (50)            | 0.54  |
| ≥ 150 ml  | 1 (17)            |       |
| FSFI: desire |                |       |
| Age at SNM|                   | 1.00  |
| < 40 years| 1 (14)            |       |
| > 40 years| 1 (13)            |       |
| Functional diagnosis |   | 0.09  |
| CUR       | 0 (0)             |       |
| OAB       | 0 (0)             |       |
| DOIC      | 1 (25)            |       |
| DSD       | 1 (100)           |       |
| FI        | 0 (0)             |       |
| PVR       |                   | 1.00  |
| < 150 ml  | 1 (17)            |       |
| ≥ 150 ml  | 1 (17)            |       |
| FSFI: arousal |               |       |
| Age at SNM|                   | 0.56  |
| < 40 years| 2 (29)            |       |
| > 40 years| 1 (13)            |       |
| Functional diagnosis |   | 0.03  |
| CUR       | 0 (0)             |       |
| OAB       | 0 (0)             |       |
| DOIC      | 2 (50)            |       |
| DSD       | 1 (100)           |       |
| FI        | 0 (0)             |       |
| PVR       |                   | 1.00  |
| < 150 ml  | 2 (33)            |       |
| ≥ 150 ml  | 1 (17)            |       |
| FSFI: lubrication |             |       |
| Age at SNM|                   | 0.11  |
| < 40 years| 4 (57)            |       |
| > 40 years| 1 (13)            |       |
| Functional diagnosis |   | 0.05  |
| CUR       | 1 (14)            |       |
| OAB       | 0 (0)             |       |
| DOIC      | 3 (75)            |       |
| DSD       | 1 (100)           |       |
| FSFI: orgasm |              |       |
| Age at SNM|                   | 1.00  |
| < 40 years| 2 (29)            |       |
| > 40 years| 2 (25)            |       |
| Functional diagnosis |   | 0.07  |
| CUR       | 0 (0)             |       |
| OAB       | 0 (0)             |       |
| DOIC      | 3 (75)            |       |
| DSD       | 1 (100)           |       |
| FI        | 0 (0)             |       |
| PVR       |                   | 1.00  |
| < 150 ml  | 2 (33)            |       |
| ≥ 150 ml  | 1 (17)            |       |
| FSFI: satisfaction |             | 0.56  |
| Age at SNM|                   |       |
| < 40 years| 2 (29)            |       |
| > 40 years| 1 (13)            |       |
| Functional diagnosis |   | 0.03  |
| CUR       | 0 (0)             |       |
| OAB       | 0 (0)             |       |
| DOIC      | 2 (50)            |       |
| DSD       | 1 (100)           |       |
| FI        | 0 (0)             |       |
| PVR       |                   | 1.00  |
| < 150 ml  | 2 (33)            |       |
| ≥ 150 ml  | 1 (17)            |       |
| FSFI: pain |                 | 1.00  |
| Age at SNM|                   |       |
| < 40 years| 1 (14)            |       |
| > 40 years| 2 (25)            |       |
| Functional diagnosis |   | 0.32  |
| CUR       | 1 (14)            |       |
| OAB       | 0 (0)             |       |
| DOIC      | 1 (25)            |       |
| DSD       | 1 (100)           |       |
| FI        | 0 (0)             |       |
| PVR       |                   | 1.00  |
| < 150 ml  | 1 (17)            |       |
| ≥ 150 ml  | 1 (17)            |       |

CUR = Non-obstructive chronic urinary retention; DSD = detrusor-sphincter dyssynergia; FI = fecal incontinence; CPPS = chronic pelvic pain syndrome.

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and control of motor neurons to the pelvic floor [10] and, in men is also responsible for penile sperm expulsion [9]. Afferent innervation carries information from bladder and internal pelvic organs through hypogastric and pelvic nerves to the dorsal horns of T11–L2 and, from bladder neck and sphincter through pelvic and pudendal nerves to S2–4 [8] as well as from dorsal nerve of penis/clitoris, perineal nerve, posterior labial nerve and inferior rectal nerve via the pudendal nerve to the central nervous system [11–13]. Vagal fibers may also convey sensory information from pelvic organs to sensory nuclei in the brainstem, as seen in women with complete spinal cord transection with maintained menstrual cramping, analgesia and orgasm, in whom vagal pathway remains functional and may be capable of mediating sexual responses [13].

More than one theory tries to explain the mechanism of SNM, either by stimulating the afferents and thereby restoring the correct balance between excitatory and inhibitory impulses from and to the pelvic organs at a sacral and supra-sacral level [14], or by inhibition of the afferent somatic pathways [3]. Studies assessing brain activity during SNM showed changes in activity in other areas but not in the pons or periaqueductal grey matter (well-known centers that coordinate bladder control) suggesting SNM exerts its effect through afferent neural pathways and involves a more intricate network of cortical centers [4, 5]. However, urinary and sexual functions share the same stimulated neural pathways.

Most of published studies evaluate the impact of SNM only on female sexual function. In our study both males and females were analyzed. Our male cohort demonstrated an improvement on IIEF-5 that corresponded to a change in erectile dysfunction from moderate to mild. Lombardi et al. [15] showed that 45% of men with incomplete spinal cord injury who were submitted to SNM reached and maintained a normal IIEF-5 score for >3 years. Erection depends mainly on pudendal somatic afferent innervation, through which tactile stimuli enters the central nervous system and, parasympathetic efferent innervation, through which motor signals generate an erection response, comprising a reflex arc [11, 16]. Both of these neural pathways are intrinsically modulated by SNM and probably this explains the improvement on erection domain.

Sexual response is a complex mechanism that includes physical and subjective mental responses to certain stimuli. Sexual response cycle comprises 4 stages: desire, arousal, orgasm and resolution [9, 17] and is controlled by central neural mechanisms: psychogenic and reflex. Reflex stimuli originate from local stimuli like the skin, which produces a motor response by a spinal sacral reflex [9].

Although sexual reflex mechanisms in women have not been investigated as well as in men, female sexual response starts with arousal phase, characterized by vulvar and clitoral swelling, vaginal lubrication and lengthening and subjective pleasure and excitement. Arousal is followed by the plateau phase during which sexual excitement continues. Orgasm may follow a variable period of arousal [10].

In our study, 5 women showed improvement in overall FSFI score, from a median baseline score of 24.1 to 26.3 after SNM. Lombardi et al. [18] reported that 4/11 neurogenic females and 2/8 idiopathic with sexual dysfunction, obtained positive effects on sexual response after SNM. Similarly, Zabihi et al. [12] reported a 53% improvement in all FSFI domain scores except desire and pain in a cohort of 36 women. Our study also showed some improvement in all domains, mostly in lubrication and orgasm. Nevertheless, only lubrication showed statistically significant improvement (p = 0.04). Zabihi et al. [12] reported significant improvement in all domains except desire and pain. Pauls et al. [19] reported significant improvement in total FSFI and all domains except arousal. In contrast, Parnell et al. [3] reported significant improvement only in desire domain. Gill et al. [6] reported significant improvement in total FSFI and arousal and satisfaction domains. Banakhar et al. [1] reported significant improvement in total FSFI and desire and orgasm domains. These inconsistent results are likely due to the small sample sizes of the studies, and improvement in total FSFI scores was related to improvement in specific domains.

Desire disorder is the deficiency of sexual fantasies/thoughts, and/or receptivity to sexual activity that causes personal distress. It may result from psychological, emotional or endocrine disorders [20–22]. It is unlikely that SNM can have a direct impact on this domain, once it is a local treatment. Nevertheless, improving overall quality of life and self-esteem may have an indirect impact.

Arousal disorder is an inability to attain or maintain adequate sexual excitement causing personal distress. It can be experienced as lack of subjective excitement or lack of genital (lubrication/swelling) responses. These conditions may occur secondary to psychological factors or medical conditions such as diminished vaginal/clitoral blood flow, prior pelvic trauma, pelvic surgery, medications, etc. [20–22]. There is a correlation between arousal and erection in the male, and there is a potential
for a disconnect between subjective and objective sexual arousal in women, that may explain why some women experience sexual response such as vaginal lubrication in the context of non-consensual sexual activity (e.g., rape) [10]. Thus, SNM may have its principal role in objective arousal domain, once parasympathetic stimulation results in relaxation of vaginal wall smooth muscle, vasodilation and high blood inflow resulting in vulvar and clitoral engorgement. Additionally, pudendal nerve stimulation can improve arousal response by carrying sensory information and modulating the reflex arc.

Orgasm disorder is the difficulty of attaining orgasm following sufficient sexual stimulation and arousal that causes personal distress [20–22]. Orgasm is the result of cerebral processing of sensory nerve stimuli. Again, it seems that the mechanism of SNM in orgasm is more indirect by improving quality of life and self-esteem.

Pain disorders are the recurrent or persistent genital pain. It can develop secondary to psychological disorders or physiological like vaginal atrophy and decreased lubrication, damage to the pelvic nerves, inflammatory or anatomical conditions, etc. [20–22].

All phases of sexual cycle are interconnected and improving one may have a positive impact in the others. In our opinion, improving lubrication may be the most important, as it can resolve some pain disorders as well as improves arousal that can result in ameliorating orgasm disorders and an overall improvement in sexual function, including desire.

Our study demonstrated a modest positive impact of SNM on IIEF-5 and FSFI scores, yet not statistically significant, except for a few FSFI domain scores. An advantage of our study is that we have not only assessed the impact of SNM on sexual function, but also have looked for a relation between sexual function and other variables, in particular, age at SNM, functional diagnosis and PVR. Younger men (< 40 years) apparently produced better results, although not statistically significant. These results were expected as ageing correlates negatively with erectile dysfunction with worldwide prevalence varying 1–10% for men < 40 years to as high as 50–100% for men > 70s and 80s [23]. Also, Banakhar et al. [1] did not find any correlation between the FSFI scores and age, due to the small sample size, while Jarrett et al. [24] reported that the percentage of improvement was inversely correlated with age. Similarly, we could not find any significant association between sexual function and PVR, although patients with a PVR < 150 ml showed improvement in sexual function. Interestingly, we did find a statistically significant association between FSFI and functional diagnosis (p = 0.05), specifically in arousal (p = 0.03), lubrication (p = 0.05) and satisfaction (p = 0.03). Patients with a more complex functional diagnosis, specifically DOIC, showed the most significant improvement on the mentioned sexual function domains with small sample size limitations. Conversely, Zabihi et al. [12] reported that patients with voiding symptoms enjoyed significant improvement (166% improvement on FSFI total score and in all domains except desire). Patients with more complex micturition disorders have the worst overall quality of life and small improvements in their condition may represent a bigger subjective impact, which can be reflected in better sexual quality of life. On the other hand, more complex dysfunctions may be associated with modulation of several non-clear neural pathways leading to a more effective result.

In our study, adverse events were mostly minor with only 4 patients (16.7%) requiring surgical revision. The remaining adverse events were dealt with reprogramming. In a study involving 407 patients, Peters et al. [25] reported a reoperation rate of 32.9%. Reasons for reoperation included lack of efficacy (21.4%), pain (10.3%), lead breakage (6.6%), device removal for MRI (2.7%), lead migration (1.7%), infection (2%) and battery depletion (8.6%). Noblett et al. [26], in the InSide study involving 272 patients, reported higher rates for both complication and reoperation rates, 47 and 32%, respectively. In a similar study, Siegel et al. [27] reported that the most frequent adverse events were undesirable change in stimulation (18%), implant site pain (13%), no response (6%), lead migration (4%), infection (4%), and similar reasons for reoperation. Although lead migration rate was higher in our study, we think this may be attributable to the small sample size. Lead migration has become less common now with the tined lead than in the past [28].

Although SNM is undoubtedly efficacious in micturition and bowel dysfunctions, few studies have addressed the role of SNM in sexual function. However, most of these studies have reported good results and shown evidence of its benefit on sexual dysfunction.

Hassouna et al. [29] showed that SNM is a cost-effective option in the management of patients with refractory OAB when compared to either botulinum toxin or medical therapy, potentially assuming in the future a first-line treatment role for refractory OAB with superior long-term outcomes. Similar evidence is earnestly awaited for sexual dysfunction.

Our study reveals some weaknesses, mainly for being a retrospective analysis and a small cohort size. However, it has the advantage of scrutinizing the confounding
impact of other factors, such as avoiding pattern, age and functional diagnosis, which were lacking in the previous literature.

Conclusion

Our results suggest that SNM can play a potential role in sexual dysfunction, especially young patients with complex functional pathology, for whom sexual function is of utmost importance for their quality of life. The predictive factors for improvement are unknown and should be the target of further prospective studies.

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