Published in final edited form as:

Int Urogynecol J. 2015 November ; 26(11): 1613–1617. doi:10.1007/s00192-015-2746-1.

Does preoperative anal physiology testing or ultrasonography predict clinical outcome with sacral neuromodulation for fecal incontinence?

Yarini Quezada¹, James L. Whiteside³, Tracy Rice², Mickey Karram¹, Janice F. Rafferty², and Ian M. Paquette^{2,4}

¹ Division of Female Pelvic Medicine and Reconstructive Surgery, The Christ Hospital Department of Obstetrics and Gynecology, Cincinnati, OH, USA

² Department of Surgery, University of Cincinnati College of Medicine, Cincinnati, OH, USA

³ Department of Obstetrics and Gynecology, University of Cincinnati College of Medicine, Cincinnati, OH, USA

⁴ 2123 Auburn Avenue, Suite 524, Cincinnati, OH 45219, USA

Abstract

Introduction and hypothesis—To determine the value of preoperative anal physiology testing and transanal ultrasonography in predicting clinical response to sacral neuromodulation for fecal incontinence.

Methods—We report a retrospective study of all patients treated with sacral neuromodulation for fecal incontinence in a single practice between 2011 and 2014 was performed. Patient demographics included age, gender, comorbidities, presence of an ultrasound-defined external sphincter defect, and history of prior anal sphincter repair. Cleveland Clinic Florida (CCF) scores were used to assess the severity of fecal incontinence at baseline, and at 3, 6 and 12 months. Pearson's correlation coefficient was used to evaluate the relationship between preoperative physiology testing and ultrasonography and patient outcome.

Results—Sacral neuromodulation was trialed in 60 patients, of whom 31 had anorectal physiology testing and 29 did not. Patients who were tested were younger (60.9 vs. 71.4 years, $p=0.013$) and more likely to have had a prior overlapping sphincteroplasty (40.5 % vs. 15 %, $p=0.043$). Among patients who progressed to complete system implantation, CCF scores at 3 and 12 months were similar whether they had physiology testing or not. Likewise, patient outcome did not correlate with the finding of an ultrasound-defined external sphincter defect. Pearson's correlation coefficient was used to evaluate the relationship between the test results and the 3-month CCF scores. CCF scores 3 months after full system implantation did not correlate with the presence or size of an external sphincter defect, resting or squeeze pressure, pudendal nerve terminal motor latency, rectoanal inhibitory reflex, or minimum detectable volume.

Ian M. Paquette ian.paquette@uc.edu.

Conflicts of interest Dr. Paquette and Dr. Karram report consulting relationships with Medtronic (Minneapolis, MN) for Interstim therapy.

Conclusions—Anal physiology testing and ultrasonography were not predictive of clinical outcomes among patients treated with sacral neuromodulation for fecal incontinence.

Keywords

Fecal; Incontinence; Neuromodulation

Introduction

Fecal incontinence (FI) is a complex problem with a prevalence ranging between 0.4 % and 18 % [1, 2]. It predominantly affects women as a result of either direct damage to the anal sphincter or stretch injury to the pudendal nerves during childbirth [3, 4]. The Mature Women's Health Study indicated that 18.8 % of women aged ≥ 45 years experience FI at least once per year, while 9.5 % have at least one episode per month [1]. FI has a profound impact on self-esteem and overall quality of life [5].

Over the past several years, many new treatment modalities have emerged, and among the more promising options is sacral neuromodulation (SNM) [6]. This minimally invasive technique modulates nerve impulses to the S3 nerve root, and has been shown to be very effective at improving FI symptoms in patients who have failed conservative measures [7–11]. Since mainstream acceptance as a treatment option for FI, it is unclear which patients may most benefit from treatment with SNM and there is no consensus as to the required work-up for patients prior to proceeding with SNM.

The work-up of patients with FI often includes anorectal physiology testing and transanal ultrasonography [12]. Though the goals of these testing modalities are to correlate objective findings with selection for particular treatment modalities, data from these studies have not been consistently shown to correlate with the severity of FI or prediction of outcomes following treatment with traditional management algorithms [12–16]. The purpose of this study was to investigate whether anorectal physiology testing or transanal ultrasonography may predict patient response to treatment with SNM.

Materials and methods

Eligible patients were identified from a prospectively maintained, single-institution database approved by the Institutional Review Board (IRB) of all patients with FI who had undergone test stimulation for SNM for FI from August 2011 to August 2014. Inclusion criteria were as follows: at least two episodes per week of FI, failure of conservative treatments, discussion of all available options, and written informed consent. The study was approved by our IRB.

Study procedures

Baseline patient assessment included a complete history and physical examination. The selection strategy for testing reflects a change in our practice pattern over time. Our previous practice pattern was to obtain physiology and ultrasound testing in all patients with FI. Once we developed experience with SNM, we stopped ordering routine testing. The two cohorts described here represent consecutive periods of time in which the first group of patients were tested and the second group of patients were not. When performed, physiology testing

consisted of resting and squeeze pressures, rectoanal inhibitory reflex (RAIR), minimum sensory threshold, and pudendal nerve terminal motor latency. Anorectal manometry records the pressure exerted by the anal sphincters and puborectalis during rest and during contraction. Diminished pressures are often seen in the setting of FI. The RAIR is an involuntary internal anal sphincter relaxation in response to rectal distension, which allows some rectal contents to descend into the anal canal. Absence of RAIR implies a functional disorder such as Hirschprung's disease. Pudendal nerve terminal motor latency evaluates for damage to the pudendal motor nerves on the basis of nerve conduction. Prolonged conduction implies damage to the pudendal nerves. There are no standardized values for normal, as each institution uses different types of equipment for measurement and thus, normal values are specific to each institution.

Transanal ultrasonography was performed using a three-dimensional diagnostic ultrasound system (Hawk type 2050; B-K Medical, Naerum, Denmark) with a rotating endoprobe at 13 MHz. Anorectal manometry was performed using a four-microtip transducer and a water-perfused catheter (Mui Scientific Type SR4B-5-0-0-0; Mississauga, Ontario, Canada). The water-perfusion method was performed using a pull-through technique with the patient lying in the left lateral position.

Disease severity was measured using the Cleveland Clinic Florida (CCF) FI scale, a validated scoring system, which assigns scores from 0 indicating perfect continence to a maximum of 20 indicating complete incontinence (Table 1). The CCF FI scale combines loss of flatus, liquid and solid stool, as well as impact on quality of life and the use of a pad to assess the severity of FI [17]. Patients were followed up at 3, 6, and 12 months, and annually thereafter. Patients received either a staged test evaluation with a quadripolar lead (model 3889, Interstim; Medtronic® Inc., Minneapolis, MN) for 14 days as previously described [18], or a basic evaluation for 7 days with a nontined lead (model 3057; Medtronic® Inc. Minneapolis, MN). The method used for test stimulation reflects a shift in our practice pattern to the basic evaluation with the nontined lead as our experience with the technique increased. Patients who experienced 50 % improvement in FI episodes during the test period were offered implantation of an Interstim generator (model 3023).

Statistical methods

The baseline characteristics of patients who had preoperative physiology testing and those who did not were compared using Student's *t* test for continuous data and Fisher's exact test for categorical data. Changes in CCF scores from baseline to after implantation were evaluated using Student's *t* test. Correlations between testing parameters and posttreatment CCF scores were assessed using Pearson's correlation coefficient. The primary endpoint was the postoperative CCF score. All *p* values are two-tailed and we considered a *p* value <0.05 as statistically significant. Stata version 13.1 (Statacorp, College Station, TX) was used for statistical analysis.

Results

SNM was trialed in a total of 60 patients of whom 55 (91.6 %) progressed to complete implantation. Among the 60 patients, 31 had anorectal physiology testing and 29 did not.

Patients who were tested were younger (60.9 vs. 71.4 years, $p=0.013$) and more likely to have had a prior overlapping sphincteroplasty (40.5 % vs. 15 %, $p=0.043$). There were no differences in gender, diabetes, smoking status, or baseline CCF score (Table 2). Test stimulation was successful (defined as 50 % improvement in weekly incontinent episodes during test stimulation) in 89.5 % of patients who underwent physiology testing and in 95.5 % of those who did not undergo physiology testing, but the difference was not statistically significant ($p=0.64$). Table 3 shows the results of physiology testing in the 31 patients who were tested. Notably, 40.3 % of patients assessed with ultrasound had a complete external sphincter defect (mean defect size 113°).

Among patients who progressed to complete system implantation, CCF scores at 3 and 12 months were similar whether they had physiology testing or not. Likewise, patient outcome did not correlate with the finding of an ultrasound-defined external sphincter defect (Fig. 1).

Pearson's correlation coefficient was used to evaluate the relationship between the test results and the 3-month CCF scores. CCF scores 3 months after full system implantation were not correlated with the presence or size of an external sphincter defect, resting or squeeze pressure, pudendal nerve terminal motor latency, RAIR, or minimum detectable volume (Table 4).

Discussion and conclusions

The results of our study indicate that preoperative anal physiology testing and ultrasonography do not correlate with patient outcome following treatment with SNM. Although these studies may have some merit in selecting patients for treatment with other modalities such as sphincteroplasty or biofeedback, they appear to be unnecessary if SNM is the therapy choice. The prognostic value of preoperative physiology testing in FI is still controversial.

Some studies support anal manometry and endoanal ultrasonography in guiding treatment options in patients with FI [19, 20]. However, other studies suggest that findings on physiology testing do not consistently correlate with severity of FI or prediction of outcome [14, 15]. Endoanal ultrasonography may be a useful tool for identifying patients with complete sphincter disruption and allow the surgeon to choose between the options of sphincter repair or SNM. However, the findings on ultrasonography do not correlate with the degree of success with SNM. Studies by Tjandra et al. corroborated this finding, showing equivalent results with SNM in patients with intact sphincters and those with complete sphincter disruption [21].

Anorectal physiology testing consists of a number of simple noninvasive tests. While the goal would be to correlate objective findings with response to various treatments, significant variability has been reported especially regarding anal manometry [14]. Lam et al. studies 600 patients who underwent anorectal manometry and transanal ultrasonography between 2003 and 2009. They concluded that incontinent patients tended to have lower pressures and smaller rectal capacity, and were more likely to have a sphincter defect than controls. However, incontinent and continent patients showed a substantial overlap in anorectal

manometry and transanal ultrasonography results [12]. Liberman et al. studied 90 patients referred to their anorectal physiology laboratory who underwent either medical or surgical management on the basis of transanal ultrasonography, pudendal nerve terminal motor latency and anorectal manometry as well as physical examination by a colorectal surgeon. They concluded that anorectal physiology testing is useful in the evaluation of patients with FI with transanal ultrasonography being the most likely to change their management [15]. Overall, the results of physiology testing have yet to be consistently correlated with prediction of outcomes following various treatments for FI [12–16].

The value of pudendal nerve terminal motor latency testing in FI remains unclear. Though some studies have shown a correlation [21–23], more recent studies suggest that the presence or absence of pudendal neuropathy does not predict outcome after sphincter repair [24, 25]. Our study supports the findings of Brouwer and Duthie who demonstrated no correlation between pudendal nerve latency testing and outcomes following SNM [26]. Studies looking at the mechanism of action of SNM have shown that in addition to improvement in muscle function, SNM also modulates sacral reflexes that regulate rectal sensitivity and contractility, anal motility and brain activity relevant to the continence mechanism [27–29]. It may be possible that current testing modalities are simply not able to detect which of the stages in these complex interactions are predictive of success with SNM. Our findings are in agreement with those of other studies showing that the presence of a complete external defect does not predict patient outcome after SNM implantation. Tjandra et al. studied patients with external sphincter defects up to 120° and demonstrated 100 % continence in 41.5 % and 75 – 99 % improvement over baseline [11]. The neurophysiologic basis underlying this correlation is likely the proposed sensory neuromodulatory effects of SNM rather than a direct effect on the external anal sphincter [9].

The limitations of our study include the fact that it was a retrospective review in which patients were not randomized. This leads to the possibility of selection bias among the groups. A randomized trial would be needed to eliminate this type of bias. It is also possible that there are other factors predictive of success with SNM which we did not study and have yet to be identified. Additionally, no quality of life instrument was used in this study. It is also possible that a larger sample size would be needed to truly detect a negative result. Although a more interesting question may be which factors are predictive of failure with a stage 1 test, the failure rate is so low (8.4 %) that a very large multi-institutional study would be needed to achieve adequate statistical power.

SNM patient outcomes do not correlate with preoperative physiology testing values. Currently, the best predictor of success is patient response to test stimulation. Although endorectal ultrasonography may aid in the selection of patients for treatments such as sphincteroplasty, it does not predict response to SNM.

References

1. Brown HW, Wexner SD, Segall MM, Brezoczky KL, Lukacz ES. Accidental bowel leakage in the mature women's health study: prevalence and predictors. *Int J Clin Pract.* 2012; 66:1101–1108. [PubMed: 23067034]

2. Ditah I, Devaki P, Luma HN, Ditah C, Njei B, Jaiyeoba C, Salami A, Ditah C, Ewelukwa O, Szarka L. Prevalence, trends, and risk factors for fecal incontinence in United States adults, 2005–2010. *Clin Gastroenterol Hepatol*. 2014; 12:636–643. e2. [PubMed: 23906873]
3. Johnson JK, Lindow SW, Duthie GS. The prevalence of occult obstetric anal sphincter injury following childbirth –literature review. *J Matern Fetal Neonatal Med*. 2007; 20:547–554. [PubMed: 17674269]
4. Mellgren A. Fecal incontinence. *Surg Clin North Am*. 2010; 90:185–194. [PubMed: 20109642]
5. Brown HW, Wexner SD, Segall MM, Brezoczky KL, Lukacz ES. Quality of life impact in women with accidental bowel leakage. *Int J Clin Pract*. 2012; 66:1109–1116. [PubMed: 23067035]
6. Kaiser AM, Orangio GR, Zutshi M, Alva S, Hull TL, Marcello PW, Margolin DA, Rafferty JF, Buie WD, Wexner SD. Current status: new technologies for the treatment of patients with fecal incontinence. *Surg Endosc*. 2014; 28:2277–2301. [PubMed: 24609699]
7. George AT, Kalmar K, Panarese A, Dudding TC, Nicholls RJ, Vaizey CJ. Long-term outcomes of sacral nerve stimulation for fecal incontinence. *Dis Colon Rectum*. 2012; 55:302–306. [PubMed: 22469797]
8. Hull T, Giese C, Wexner SD, Mellgren A, Devroede G, Madoff RD, Stromberg K, Collier JA. Long-term durability of sacral nerve stimulation therapy for chronic fecal incontinence. *Dis Colon Rectum*. 2013; 56:234–245. [PubMed: 23303153]
9. Johnson BL 3rd, Abodeely A, Ferguson MA, Davis BR, Rafferty JF, Paquette IM. Is sacral neuromodulation here to stay? Clinical outcomes of a new treatment for fecal incontinence. *J Gastrointest Surg*. 2015; 19:15–19. [PubMed: 25115325]
10. Matzel KE, Lux P, Heuer S, Besendorfer M, Zhang W. Sacral nerve stimulation for faecal incontinence: long-term outcome. *Colorectal Dis*. 2009; 11:636–641. [PubMed: 18727721]
11. Tjandra JJ, Chan MK, Yeh CH, Murray-Green C. Sacral nerve stimulation is more effective than optimal medical therapy for severe fecal incontinence: a randomized, controlled study. *Dis Colon Rectum*. 2008; 51:494–502. [PubMed: 18278532]
12. Lam TJ, Kuik DJ, Felt-Bersma RJ. Anorectal function evaluation and predictive factors for faecal incontinence in 600 patients. *Colorectal Dis*. 2012; 14:214–23. [PubMed: 21689265]
13. Bharucha AE. Pro: Anorectal testing is useful in fecal incontinence. *Am J Gastroenterol*. 2006; 101:2679–2681. [PubMed: 17227512]
14. Bharucha AE. Update of tests of colon and rectal structure and function. *J Clin Gastroenterol*. 2006; 40:96–103. [PubMed: 16394868]
15. Liberman H, Faria J, Ternent CA, Blatchford GJ, Christensen MA, Thorson AG. A prospective evaluation of the value of anorectal physiology in the management of fecal incontinence. *Dis Colon Rectum*. 2001; 44:1567–1574. [PubMed: 11711725]
16. Wald A. Con: Anorectal manometry and imaging are not necessary in patients with fecal incontinence. *Am J Gastroenterol*. 2006; 101:2681–3. [PubMed: 17227513]
17. Jorge JM, Wexner SD. Etiology and management of fecal incontinence. *Dis Colon Rectum*. 1993; 36:77–97. [PubMed: 8416784]
18. Wexner SD, Collier JA, Devroede G, Hull T, McCallum R, Chan M, Ayscue JM, Shobeiri AS, Margolin D, England M, Kaufman H, Snape WJ, Mutlu E, Chua H, Pettit P, Nagle D, Madoff RD, Lerew DR, Mellgren A. Sacral nerve stimulation for fecal incontinence: results of a 120-patient prospective multicenter study. *Ann Surg*. 2010; 251:441–449. [PubMed: 20160636]
19. Fowler L, Mills A, Virjee J, Callaway M, Durdey P, Thomas MG. Comparison of ultrasound and manometric sphincter length and incontinence scores. *Dis Colon Rectum*. 2003; 46:1078–1082. [PubMed: 12907902]
20. Chan MK, Tjandra JJ. Sacral nerve stimulation for fecal incontinence: external anal sphincter defect vs. intact anal sphincter. *Dis Colon Rectum*. 2008; 51:1015–25. [PubMed: 18484136]
21. Chen AS, Luchtefeld MA, Senagore AJ, Mackeigan JM, Hoyt C. Pudendal nerve latency. Does it predict outcome of anal sphincter repair? *Dis Colon Rectum*. 1998; 41:1005–1009. [PubMed: 9715157]
22. Gilliland R, Altomare DF, Moreira H Jr, Oliveira L, Gilliland JE, Wexner SD. Pudendal neuropathy is predictive of failure following anterior overlapping sphincteroplasty. *Dis Colon Rectum*. 1998; 41:1516–1522. [PubMed: 9860332]

23. Sangwan YP, Collier JA, Barrett RC, Roberts PL, Murray JJ, Rusin L, et al. Unilateral pudendal neuropathy. Impact on outcome of anal sphincter repair. *Dis Colon Rectum*. 1996; 39:686–689. [PubMed: 8646958]
24. Buie WD, Lowry AC, Rothenberger DA, Madoff RD. Clinical rather than laboratory assessment predicts continence after anterior sphincteroplasty. *Dis Colon Rectum*. 2001; 44:1255–1260. [PubMed: 11584195]
25. Ricciardi R, Mellgren AF, Madoff RD, Baxter NN, Karulf RE, Parker SC. The utility of pudendal nerve terminal motor latencies in idiopathic incontinence. *Dis Colon Rectum*. 2006; 49:852–857. [PubMed: 16598403]
26. Brouwer R, Duthie G. Sacral nerve neuromodulation is effective treatment for fecal incontinence in the presence of a sphincter defect, pudendal neuropathy, or previous sphincter repair. *Dis Colon Rectum*. 2010; 53(3):273–278. [PubMed: 20173472]
27. Gooneratne ML, Scott SM, Lunniss PJ. Unilateral pudendal neuropathy is common in patients with fecal incontinence. *Dis Colon Rectum*. 2007; 50:449–458. [PubMed: 17279299]
28. Lundby L, Moller A, Buntzen S, Krogh K, Vang K, Gjedde A, et al. Relief of fecal incontinence by sacral nerve stimulation linked to focal brain activation. *Dis Colon Rectum*. 2011; 54:318–323. [PubMed: 21304303]
29. Michelsen HB, Thompson-Fawcett M, Lundby L, Krogh K, Laurberg S, Buntzen S. Six years of experience with sacral nerve stimulation for fecal incontinence. *Dis Colon Rectum*. 2010; 53:414–421. [PubMed: 20305440]

Fig. 1.
(3 and 12 month CCF score stratified by physiology testing and ultrasound results)

Table 1

Cleveland Clinic Florida fecal incontinence scale from 0 (perfect continence) to 20 (complete incontinence)

Type of incontinence	Frequency of incontinence episodes ^a				
	Never	Rarely	Sometimes	Usually	Always
Solid	0	1	2	3	4
Liquid	0	1	2	3	4
Gas	0	1	2	3	4
Pad usage	0	1	2	3	4
Quality of life impact	0	1	2	3	4

^a *Never* no episodes, *Rarely* <1 per month, *Sometimes* >1 per month, <1 per week, *Usually* >1 per week, <1 per day, *Always* >1 per day

Table 2

Patient demographics

Variable	Testing (<i>n</i> = 31)	No testing (<i>n</i> = 29)	<i>p</i> value
Age (years)	60.9	71.4	0.013
Female gender	96.7 %	90.9 %	0.13
Diabetes	21.1 %	27.3 %	0.77
Smoker	7.9 %	13.6 %	0.66
Prior repair ^a	40.5 %	15 %	0.043
Baseline CCF score	15.1	15	0.91
Successful test ^b	89.5 %	95.5 %	0.64

^aDefined as previous overlapping sphincteroplasty

^bDefined as 50 % decrease in weekly incontinent episodes during test stimulation

Table 3

Physiology testing results in the 31 patients tested (29 patients were no tested)

Parameter	Test result
Sphincter defect	
Presence	40.3 %
Mean size	113°
Rectoanal inhibitory reflex	85.2 %
Pudendal nerve terminal motor latency (ms)	
Left	2.0 ms
Right	2.0 ms
Resting pressure (mean)	34.8 mmHg
Squeeze pressure (mean)	53.1 mmHg
Minimum detectable volume	25 cm ³

Table 4

Correlation between test results and 3-month CCF scores

Parameter	Pearson's coefficient	<i>p</i> value
Presence of sphincter defect	0.25	0.07
Size of sphincter defect (if present)	0.31	0.35
Rectoanal inhibitory reflex	0.16	0.23
Pudendal nerve terminal motor latency	0.16	0.42
Resting pressure (mean)	0.15	0.48
Squeeze pressure (mean)	0.1	0.98
Minimum detectable volume	0.31	0.14