Pudendal Neuropathy and the Importance of EMG Evaluation of Fecal Incontinence

Anthony M. Vernava III, M.D., Walter E. Longo, M.D., Gail L. Daniel, R.N.

From the Department of Surgery, Section of Colon and Rectal Surgery, St. Louis University School of Medicine, St. Louis, Missouri

A prospective study was undertaken to evaluate pudendal neuropathy in fecal incontinence. Fifty-two patients (38 women and 14 men) with fecal incontinence underwent manometric and electromyographic evaluation (measurement of pudendal nerve terminal motor latency [PNTML] and sphincter muscle mapping). The average age of all patients was 54 ± 17 years. Fifty-two percent (27/52) were found to have a pudendal neuropathy (PNTML > 2.1 milliseconds). Seventeen of these 27 patients (63 percent) had a bilateral pudendal neuropathy. Patients with a pudendal neuropathy were older than those without a neuropathy (63.7 years *vs.* 51.9 years; $P = 0.01$). Women were significantly more likely than men to have a pudendal neuropathy ($P = 0.03$). Nine patients had an anatomic sphincter defect identified, and six of these (67 percent) had a neuropathy; 4/6 (67 percent) had a bilateral pudendal neuropathy. In the 43 patients who did not have an anatomic sphincter defect, there was no difference in resting pressure (69 mmHg *vs.* 60 mmHg; $P = 0.4$) or maximum voluntary contraction (95 mmHg *vs.* 86 mmHg; $P = 0.5$) when patients without a neuropathy were compared with those with a neuropathy. Patients with a pudendal neuropathy had a shorter sphincter length than those without a neuropathy (3.0 cm *vs.* 3.9 cm; $P = 0.01$). Bilateral pudendal neuropathy tended to occur more frequently in women ($P = 0.08$) and was not associated with poorer resting pressure, maximum voluntary contraction, or shorter sphincter length. We conclude that pudendal neuropathy is a common cause of fecal incontinence, particularly in older women, and frequently occurs in association with a sphincter defect. Manometric evaluation alone is not helpful in identifying the neuropathic patient. PNTMLs should be routinely measured in the evaluation of fecal incontinence. [Key words: Fecal incontinence; Anorectal physiology; Electromyography; Pudendal nerve terminal motor latency; Muscle mapping; Manometry]

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Fecal incontinence can be a disabling disorder that affects the lifestyle of a significant but unknown number of patients. The etiologies of incontinence are varied and include traumatic disruption of the external anal sphincter, neurogenic dysfunction of the pelvic floor musculature, alteration of rectal compliance, and a change in stool consistency, among other causes. Anatomic disruption of the external anal sphincter is the most common surgically correctable cause of fecal incontinence; however, pudendal neuropathy has been hypothesized to be an even more significant cause of incontinence.[1] In fact, many cases of incontinence thought, in the past, to be idiopathic are now felt to be due to a neuropathy.[2] As many as 80 percent of women with idiopathic fecal incontinence have evidence of nerve damage to the pelvic floor musculature.[2] Indeed, pudendal neuropathy can coexist with an obvious anatomic sphincter defect and is known to adversely affect the result of sphincter repair in such cases.[3,4] Over the past decade, the anorectal physiology lab has come to play an important role in the evaluation of patients with fecal incontinence. Despite the realization that fecal incontinence is frequently due to neuropathy, many patients have only manometry performed.[5] The relationship of manometric values (*i.e.*, resting pressure, maximum voluntary contraction, and squeeze pressure) in incontinent patients with neuropathy compared with those without neuropathy is unknown, and it is unclear whether there are any manometric values characteristic of pudendal neuropathy. The purpose of this study was to prospectively define the incidence of pudendal neuropathy in a consecutive group of incontinent patients and to determine whether there exist any manometric findings characteristic of neuropathy within this condition.

MATERIALS AND METHODS

Fifty-two consecutive patients who were referred for fecal incontinence underwent evaluation in the anorectal physiology laboratory. Physiologic evaluation consisted of anorectal manometry and elec-

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Address reprint requests to Dr. Vernava: Section of Colon and Rectal Surgery, St. Louis University School of Medicine, 3635 Vista at Grand, P.O. Box 15250, St. Louis, Missouri 63110.

tromyography. Manometry measured resting anal canal pressure, maximum voluntary contraction pressure, and sphincter length. Anal canal pressures were recorded in mmHg. Sphincter length was measured in cm. Electromyography consisted of measurement of both pudendal nerve terminal motor latencies (PNTMLs) and muscle mapping of the external anal sphincter. Patients were placed in the left lateral decubitus position, and the perineum was inspected and gentle digital examination performed. Manometry was performed on all 52 patients using a flexible, polyethylene, eight-channel, water-perfused catheter with a radial head design (Arndorfer, Inc., Greenvale, WI) connected to an eight-channel hydraulic microcapillary perfusion pump (Arndorfer, Inc.). Water was infused through each channel at a constant rate of 0.5 cc per minute. The water perfusion apparatus was connected to Synectics (Synectics, Inc., Irving, TX) manometry hardware interfaced with an IBM (International Business Machines, Inc., Armonck, NY) computer on which data were viewed and stored; the Synectics software for anorectal manometry was used. Calibration of the system was performed prior to each study. Manometry was performed using a station pull-through technique beginning at 10 cm from the anal verge. Measurements for resting pressure and maximum voluntary contraction were obtained at each centimeter interval from 10 cm to 0 cm.

Anorectal muscle electromyography was done using the Dantec Neuromatic N 2000 (Dantec, Skovlund, Denmark). PNTML was measured on all 52 patients using the technique described by Kiff and Swash[1] and Swash and Snooks.[6] Normal PNTML was defined as ≤2.1 milliseconds.[1, 6] Mapping of the external anal sphincter was performed in those patients suspected of having an anatomic sphincter defect, using a concentric needle as described by Kiff[7] and Swash and Snooks.[6]

Statistical analysis was performed using chi-squared with Yates continuity correction and the unpaired *t*-test. Statistical significance was defined at $P < 0.05$.

RESULTS

Among the 52 incontinent patients, the average age was 54.2 ± 17.1 years. There were 38 females and 14 males. The mean resting pressure of the entire group was 63 ± 33.8 mmHg, and the mean maximum voluntary contraction was 90.3 ± 42.3 mmHg. The average sphincter length was 3.5 ± 1.1 cm. Fifty-two percent (27/52) of patients referred for evaluation of fecal incontinence were found to have a pudendal neuropathy. Patients with pudendal neuropathy were older (58.5 *vs.* 49.5 years; $P = 0.056$). Overall, pudendal neuropathy tended to occur more frequently in women than in men; 23 of 27 patients with pudendal neuropathy were women ($P = 0.08$). Seventeen of the 27 (63 percent) neuropathic patients had a bilateral pudendal neuropathy. Women were significantly more likely than men to have a bilateral pudendal neuropathy; 16 of 17 patients with bilateral neuropathy were women ($P = 0.04$). Nine patients (one male and eight females) were found to have an anatomic sphincter defect, and they were evaluated separately.

Incontinent Patients Without an Anatomic Sphincter Defect (n = 43)

Forty-three patients without an anatomic sphincter defect were evaluated. Of these 43 patients, 21 (49 percent) had a pudendal neuropathy. Patients with a pudendal neuropathy were significantly older than those without (63.7 *vs.* 51.9 years; $P = 0.01$). Females were significantly more likely than males to have a pudendal neuropathy; 18 of 30 women had one, but only 3 of 13 men did ($P = 0.03$). Manometric values were assessed. There was no significant difference in resting pressure (60.8 ± 34.6 mmHg *vs.* 69.4 ± 36.6 mmHg; $P = 0.4$) or maximum voluntary contraction (86.2 ± 34.1 mmHg *vs.* 94.8 ± 50.2 mmHg; $P = 0.5$) when patients with a pudendal neuropathy were compared with those without. Patients with a pudendal neuropathy had a shorter sphincter length than those with normal nerve function (3.0 cm *vs.* 3.9 cm; $P = 0.01$). Bilateral neuropathy, which occurred in 13 of the 21 patients with pudendal neuropathy, was not associated with poorer resting pressure (61.8 ± 29.7 mmHg *vs.* 58.9 ± 43.5 mmHg; $P = 0.8$), maximum voluntary contraction (80.4 ± 21.7 mmHg *vs.* 95.7 ± 48.5 mmHg; $P = 0.3$), or shorter sphincter length (2.9 ± 0.8 cm *vs.* 3.3 ± 1.1 cm; $P = 0.4$).

Incontinent Patients with an Anatomic Sphincter Defect (n = 9)

Nine patients (eight women and one man) had an anatomic sphincter defect. Patients with a

sphincter defect were significantly younger than the rest of the group (37.5 years *vs.* 57.7 years; $P = 0.0008$). The average resting pressure for the group with a sphincter defect was 53 ± 23 mmHg, which was lower than but not significantly different from the resting pressure in the group of patients without a sphincter defect (65.2 ± 35.5 mmHg; $P = 0.3$). The average maximum voluntary contraction was 88.9 ± 42.7 mmHg and, again, was lower but not significantly different compared with the group of incontinent patients without a sphincter defect (90.6 ± 42.8 mmHg; $P = 0.9$). Patients with a sphincter defect had a shorter sphincter length, but this was not significant (3.0 cm *vs.* 3.6 cm; $P = 0.2$).

Six patients (66 percent) with a sphincter defect had a concomitant pudendal neuropathy. The presence of neuropathy did not significantly affect resting pressure (58.3 mmHg *vs.* 42.3 mmHg; $P = 0.4$), maximum voluntary contraction (99.7 mmHg *vs.* 67.4 mmHg; $P = 0.3$), or sphincter length (3.3 cm *vs.* 2.6 cm; $P = 0.6$).

DISCUSSION

The precise incidence of fecal incontinence in the general population is unknown. Thomas *et al.*[8] conducted a postal survey of two London boroughs involving 14,844 patients and discovered a prevalence of fecal incontinence of 4.2 per 1,000. Tobin and Brocklehurst[9] estimate a prevalence of fecal incontinence of 10.3 per 100 in elderly, institutionalized patients. The incidence increases with age, and women are eight times more likely to suffer fecal incontinence than men.[2] The etiology of fecal incontinence is multifactorial and includes traumatic sphincter disruption and damage to the pelvic nerve supply (S2,3,4), among other causes, and the specific etiology has substantial implications for successful management. Successful operative restoration of fecal continence owing to traumatic division of the external anal sphincter has been reported by many authors and is the treatment of choice for this condition.[3, 4, 10, 11] However, the coexistence of pudendal neuropathy with a sphincter defect appears to diminish the chance for successful restoration of continence with the plication sphincter repair.[3, 4]

The true incidence of neurogenic fecal incontinence as compared with idiopathic incontinence, and whether or not all idiopathic fecal incontinence has a neurogenic etiology, is not clear. Snooks *et al.*[2] estimate that as many as 80 percent of women with idiopathic fecal incontinence have evidence of nerve injury to the pelvic floor musculature. A similar estimate for men is not available. Beersiek[12] studied the histopathology of 16 patients who had undergone surgery for "idiopathic" fecal incontinence and found histologic evidence of a neurogenic disorder in 12 of the 16 patients (75 percent).[12] Parks *et al.*[13] first hypothesized in 1977 that idiopathic fecal incontinence was due to damage to the nerve supply of the muscles of the pelvic floor. This hypothesis was confirmed in 1984 by Kiff and Swash,[1] who found prolonged pudendal nerve conduction in 30 patients with idiopathic fecal incontinence compared with 28 normal controls. The etiology of pudendal neuropathy in women is proposed to be due to a combination of direct trauma to the nerve and stretch injury to the nerve, both of which occur when the infant descends into the pelvis during a vaginal delivery.[2] Snooks *et al.*[2] suggest that this pudendal nerve injury is reversible in 60 percent of women and further suggest that those patients who have persistently prolonged pudendal nerve conduction two months after delivery may become incontinent later in life. Operative management of neurogenic fecal incontinence had comprised postanal repair, as pioneered by Sir Allan Parks, but this operation has had variable results and is not widely practiced in the United States.[14] Currently, many patients with neurogenic fecal incontinence are managed nonoperatively with a combination of stool bulking, slowing agents, a bowel regimen, and biofeedback.

The application of electromyography to physiologic disorders of the anorectum has been difficult, and the equipment is expensive. Interpretation of concentric-needle electromyography and single-fiber activity requires a significant amount of prior experience with the technique. Measurement of PNTML likewise requires experience in order to perform the test and to provide accurate interpretation. These factors have probably limited its application in the anorectal physiology laboratory. In a recent survey of members of The American Society of Colon and Rectal Surgeons (ASCRS) and the Coloproctology Section of the Royal Society of Medicine on the availability and perceived utility of 19 various anorectal physiologic tests, Karulf *et al.*[5] found that the ability to measure pudendal nerve latency was available in only 15 percent of

American anorectal physiology laboratories and in only 34 percent of English anorectal physiology laboratories. Further, members of the ASCRS ranked the utility of the pudendal nerve latency examination 17th out of the 19 methods surveyed. The English surgeons felt that measurement of pudendal nerve latency was somewhat more important and ranked the test the 10th most helpful out of 19 different methods.[5]

The results of our study indicate that pudendal neuropathy, as determined by the measurement of PNTML, is commonly associated with both idiopathic fecal incontinence and incontinence due to an anatomic sphincter defect. Women are more likely than men to suffer a pudendal neuropathy, and older individuals are also more likely to suffer a pudendal neuropathy. These findings are consistent with those of other investigators.[1, 2, 5-7] We further found that there is no characteristic manometric signature for pudendal neuropathy. Incontinent patients with pudendal neuropathy have squeeze pressures and resting pressures similar to those incontinent patients without neuropathy. Even bilateral pudendal neuropathy did not worsen squeeze or resting pressures to lower than those of patients with "idiopathic" incontinence. Neuropathic patients did have a shorter sphincter length than others, and the reason for this may be muscle atrophy and shortening. Anorectal manometry was not helpful in predicting the presence or absence of pudendal neuropathy.

Several other investigators have attempted to evaluate the relationship between anorectal manometry and electromyography. Felt-Bersma *et al.*[15] performed a study evaluating the relationship between anal electromyography (maximum contraction pattern and signs of denervation) and maximum squeeze pressure. In their study, normal squeeze pressure always predicted a normal maximum contraction pattern, but an abnormal maximum contraction pattern only predicted normal squeeze pressure 43 percent of the time. The authors found no correlation between maximum squeeze pressure and evidence of denervation of the external anal sphincter.[15] These authors did not measure pudendal nerve latency, but their findings are consistent with ours in that they found no significant relationship between maximum squeeze pressure and evidence of sphincter denervation. Felt-Bersma *et al.*,[15] on the basis of this lack of relationship, concluded that the clinical utility of anal electromyography was limited and that it should not be routinely performed. We strongly disagree and feel that pudendal nerve latencies should be measured in all patients who present with fecal incontinence. Precisely because there is no relationship between maximum voluntary contraction and the presence of pelvic neuropathy in incontinent patients, anal manometry is useless in predicting neuropathy.

Pinho *et al.*[16] evaluated the efficacy of surface electrode electromyography as an indicator of anal sphincter function and found a positive correlation between intra-anal electromyography and resting pressure ($r = 0.62$) and maximum squeeze effort ($r = 0.73$). Although the authors did not perform pudendal nerve latency, these results contrast with ours but may be explained by the varied patient population in Pinho *et al.*'s[16] study and the uniform patient population in ours. Obviously, factors other than neuropathy can affect striated muscle function. A muscle that functions poorly, for whatever reason, will be incapable of generating "normal pressures" even in the absence of neuropathy. Therefore, we may not have been able to demonstrate a relationship between neuropathy and squeeze pressure because there were no normal patients in our study. This is important because it helps to demonstrate the limitation of utilizing manometry alone in the evaluation of incontinence. Patients with normal squeeze pressures are likely to have normal pudendal neurologic function, but the converse is not true; patients with poor squeeze pressures do not necessarily have a pudendal neuropathy, and the only way to determine this is to measure the pudendal nerve latency.

Bartolo,[17] in a review of anorectal physiologic tests, agrees that anal sphincter electromyography is useful in the evaluation of fecal incontinence. Wexner *et al.*,[18] in a study of the neurophysiologic assessment of the pelvic floor for a variety of pelvic floor disorders including incontinence, concluded that electromyography and measurement of PNTML provided complementary information to manometry alone. Moreover, they stated that PNTML cannot be ascertained by any method other than direct transanal stimulation of the nerve.

CONCLUSIONS

Pudendal neuropathy is a common finding in patients with fecal incontinence. Women are more likely to suffer a pudendal neuropathy than are

men, and the condition increases in frequency with advancing age. There does not appear to be a significant relationship in incontinent patients between maximum squeeze pressure or resting pressure and unilateral or bilateral pudendal neuropathy. Electromyographic examination specifically measuring PNTML is the best way to tell whether the patient has a neuropathy. Routine measurement of PNTMLs is recommended in the physiologic evaluation of all patients with fecal incontinence.

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