

Prospective Comparative Study of Abnormal Distal Rectoanal Excitatory Reflex, Pudendal Nerve Terminal Motor Latency, and Single Fiber Density as Markers of Pudendal Neuropathy

Yash P. Sangwan, M.D., M.S. (Surg.), F.R.C.S.(Ed), F.R.C.S.(Glasg),
John A. Collier, M.D., Richard C. Barrett, B.S., John J. Murray, M.D.,
Patricia L. Roberts, M.D., David J. Schoetz, Jr., M.D.

*From the Department of Colon and Rectal Surgery, Lahey Hitchcock Medical Center,
Burlington, Massachusetts*

PURPOSE: This study was undertaken to determine the role of abnormal distal rectoanal excitatory reflex (RAER) as a marker of pudendal neuropathy and to compare results with pudendal nerve terminal motor latency (PNTML) and single fiber density (SFD) estimation. **METHODS:** Fifteen female patients (mean age, 47.1 (range, 20–70) years) referred to the pelvic floor laboratory with pelvic floor disorders (fecal incontinence, 13 patients; constipation, 2 patients) were evaluated prospectively with neurophysiologic tests and balloon reflex manometry for evidence of pudendal neuropathy. **RESULTS:** Pudendal nerve terminal motor latency provided evidence of pudendal neuropathy in ten patients (67 percent) and was normal in five patients (33 percent). Increased SFD confirmed denervation of the external anal sphincter in 12 patients (80 percent), being normal in 3 patients (20 percent). Distal RAER was abnormal in 13 patients (87 percent) and was normal in 2 patients (13 percent). In ten patients (67 percent), the three diagnostic modalities were in complete agreement, correctly identifying neuropathy in nine patients (60 percent) and excluding nerve damage in one patient (7 percent). Distal RAER was normal despite prolonged PNTML and increased SFD in one patient (7 percent). In two patients (13 percent), distal RAER was abnormal or absent despite normal PNTML and SFD. Pudendal nerve terminal motor latency was normal in the presence of abnormal distal RAER and increased SFD on electromyography in two patients (13 percent). **CONCLUSIONS:** Abnormal distal RAER compares favorably with current neurophysiologic tests used to diagnose pudendal neuropathy. [Key words: Pudendal neuropathy; Single fiber density; Pudendal nerve terminal motor latency; Balloon reflex manometry]

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Pudendal neuropathy, denervation damage to the external anal sphincter (EAS), is confirmed by documentation of delayed conduction in the pudendal nerves¹ or by electromyographic (EMG) evidence of denervation and reinnervation of the EAS.² Recently, we³ reported that an abnormal distal rectoanal excitatory reflex can also serve as an alternative qualitative index of pudendal neuropathy. We now report a prospective study evaluating the role of an abnormal distal rectoanal excitatory reflex (RAER) as a marker of pudendal nerve damage and compare results with single fiber density (SFD), the gold standard of neurogenic injury, and pudendal nerve terminal motor latency (PNTML).

METHODS

Fifteen female patients (mean age, 47.1 (range, 20–76) years) who were referred to the pelvic floor laboratory for diagnostic evaluation of pelvic floor disorders (fecal incontinence, 13; constipation, 2) were prospectively evaluated for evidence of pudendal neuropathy. Of the 13 patients with fecal incontinence, symptoms developed after obstetric injury in nine patients; in four patients, the cause was idiopathic. One patient in the idiopathic group had associated rectal prolapse. Mean number of vaginal deliveries was 2.3 (range, 0–4). Patients with constipation had symptoms of obstructed defecation and fecal soil-

Address reprint requests to Dr. Collier: Department of Colon and Rectal Surgery, Lahey Hitchcock Medical Center, 41 Mall Road, Burlington, Massachusetts 01805.

ing. Because some patients had both constipation and incontinence, patients were classified into fecal incontinence or constipation groups according to their chief presenting symptom. All patients were evaluated with anorectal manometry. Evidence of pudendal neuropathy was obtained with EMG and estimation of PNTML, and results were compared with the status of distal RAER on balloon reflex manometry.

Manometry

We used an eight-channel, water-perfused flexible probe 5.5 mm in external diameter. A computerized system (Narco Bio-Systems, Houston, TX) with menu-driven software was used to aid in the acquisition of data. A pressure recorder was interfaced with a microcomputer to facilitate analysis, graphic presentation, and generation of numeric reports. Protocol for performing manometry and analysis of manometric pressure has been detailed previously.^{4, 5}

Pudendal Nerve Terminal Motor Latency

Pudendal nerve terminal motor latency was determined using St. Mark's pudendal electrode using the technique described by Kiff and Swash.¹ In our laboratory, PNTML in normal individuals is 2 ± 0.2 ms.

Single Fiber Electromyography

The single fiber EMG recording was obtained using a Dantec counterpointTM (Dantec Elektronik Medicinsk, Skovlunde, Denmark) EMG apparatus and a

single fiber EMG electrode, 0.45 by 40 mm, with a platinum lead-off surface $25 \mu\text{m}$ in diameter. Low-frequency and high-frequency filter settings of 500 Hz and 10 kHz were used. Recordings were made from both sides of the sphincter, necessitating two separate skin penetrations. Mean number of single fiber potentials recorded from 20 different electrode positions provided the SFD.

Interpretation of Neurophysiologic Testing and Balloon Reflex Manometry

Patients were considered to have pudendal neuropathy when PNTML was prolonged for more than 2 standard deviations above normal (2 ± 0.2), that is, more than 2.4 ms. On single fiber EMG, neurogenic damage to the EAS was assumed when single fiber density was greater than 1.7 (normal, 1.5 ± 0.1).

Distal excitatory reflex was considered abnormal when it exhibited one of the four abnormal responses reported previously from this laboratory,³ namely, absent response, diminished amplitude, delayed response, or excitation at high volume of inflation only. In the absence of extensive traumatic damage to the EAS, an abnormal RAER was taken as evidence of neurogenic damage. The term distal RAER was used to denote excitatory response in the distal anal canal and to distinguish it from excitation observed in the proximal anal canal, which is often intact despite pudendal neuropathy.⁶

Table 1.
Results of Anorectal Manometry

Patient	Age, years	No. of Deliveries	Diagnosis	Initial Rectal Sensation, ml	Maximum Tolerable Volume, ml	Resting Pressure, mmHg	Squeeze Pressure, mmHg	Sphincter Asymmetry, %
1	59	3	Idiopathic	30	130	72.4	66.4	9.7
2	61	3	Obstetric injury	175	450	92.1	146.4	11
3	76	1	Idiopathic	80	120	28.8	44.1	19.9
4	46	2	Constipation	60	110	74.5	130.5	23.4
5	50	4	Obstetric injury	20	70	22.4	41.7	16.8
6	30	3	Obstetric injury	50	80	28.7	56.2	26.8
7	32	0	Constipation	40	120	84.6	92.4	33.6
8	29	2	Obstetric injury	40	90	40.2	35.9	22.6
9	20	1	Obstetric injury	40	90	15.6	36.8	39.7
10	49	2	Obstetric injury	40	240	52.7	69.9	18.4
11	64	3	Obstetric injury	50	85	16.3	18	25.9
12	23	1	Idiopathic	100	120	39.8	43.3	22.5
13	47	3	Obstetric injury	90	110	75.7	96.8	24.5
14	70	4	Obstetric injury	70	120	30.4	42.3	16.9
15	51	3	Idiopathic	40	60	20.7	27.3	22.3

Table 2.
Results of Neurophysiologic Testing and Balloon Reflex Manometry

Patient	Left PNTML, ms	Right PNTML, ms	SFD	Pudendal Neuropathy	Status of Distal RAER
1	2.5	1.72	2	Yes	Absent
2	NR	NR	2.1	Yes	Normal
3	NR	NR	2.1	Yes	Absent
4	2.66	3.28	2	Yes	Absent
5	2.03	2.03	2.3	Yes	Absent
6	2.5	2.5	2.3	Yes	Absent
7	2.03	2.03	1.42	No	Normal
8	2.19	2.03	2	Yes	Diminutive
9	1.56	1.88	1.39	No	Absent
10	2.34	2.5	1.95	Yes	Diminutive
11	2.5	2.03	2.35	Yes	Absent
12	3.13	1.88	1.85	Yes	Absent
13	2.03	2.34	1.5	No	Present at high inflation volume only
14	2.97	NR	1.92	Yes	Present at high inflation volume only
15	2.97	1.72	2	Yes	Diminutive/present at high inflation volume only

PNTML = pudendal nerve terminal motor latency; SFD = single fiber density; RAER = rectoanal excitatory reflex; NR = no response.

RESULTS

Patient age, diagnosis, and results of anorectal manometry are given in Table 1. Results of neurophysiologic testing and balloon reflex manometry are listed in Table 2. Using the criterion described earlier, PNTML provided evidence of pudendal neuropathy in ten patients (67 percent) and was normal in five patients (33 percent). Increased SFD confirmed denervation of EAS in 12 patients (80 percent), and was normal in 3 patients (20 percent). Distal RAER was abnormal in 13 patients (87 percent) and normal in 2 patients (13 percent).

In ten patients (67 percent), the three diagnostic modalities were in complete agreement (Table 3). In nine patients (60 percent), pudendal neuropathy was confirmed by both prolonged PNTML and increased SFD, with an abnormal distal RAER on balloon reflex manometry. In one patient (7 percent), pudendal neuropathy was excluded by normal PNTML and SFD values, and balloon reflex manometry also displayed a normal distal RAER. Correlation coefficient, phi coefficient, between abnormal distal RAER and PNTML or SFD was not significant (PNTML *vs.* distal RAER, phi coefficient = 0.14; SFD *vs.* distal RAER, phi coefficient = 0.29).

Table 3.

Comparative Analysis of Results of Neurophysiologic Testing and Balloon Reflex Manometry

Distal RAER	PNTML	SFD	No. of Patients
Abnormal	Prolonged	Increased	9
Normal	Normal	Normal	1
Normal	Prolonged	Increased	1
Abnormal	Normal	Increased	2
Abnormal	Normal	Normal	2

RAER = rectoanal excitatory reflex; PNTML = pudendal nerve terminal motor latency; SFD = single fiber density.

In one patient, distal RAER was normal and failed to corroborate evidence of pudendal neuropathy, which was suggested both by prolonged PNTML and increased SFD. In two patients, distal RAER was abnormal or absent despite normal PNTML and SFD. However, in one of these patients (Patient 9), extensive traumatic damage to external sphincter (Fig. 1) and not pudendal neuropathy accounted for the absence of the distal RAER. In two patients, PNTML was normal, despite abnormal distal RAER and increased SFD on EMG (Table 3).

Proximal Sphincter

Distal Sphincter

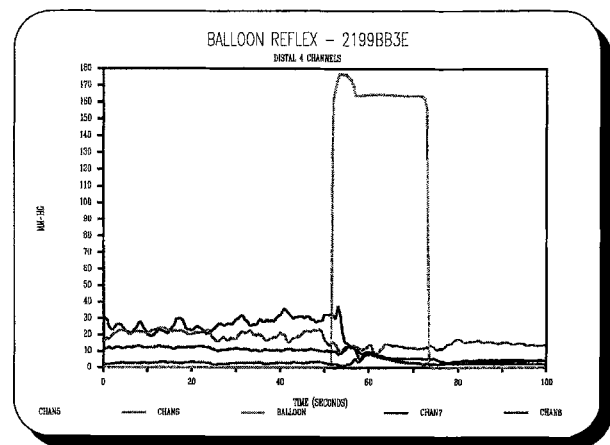
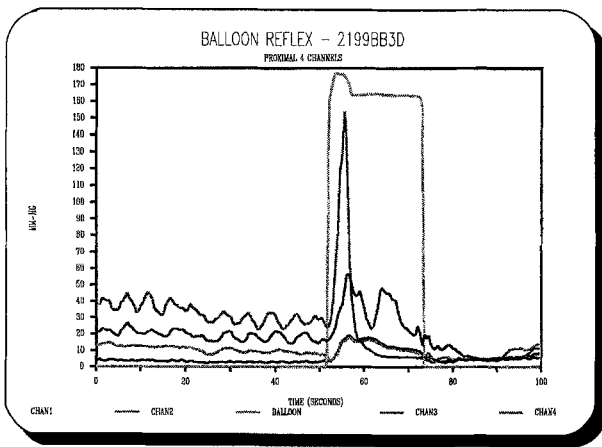
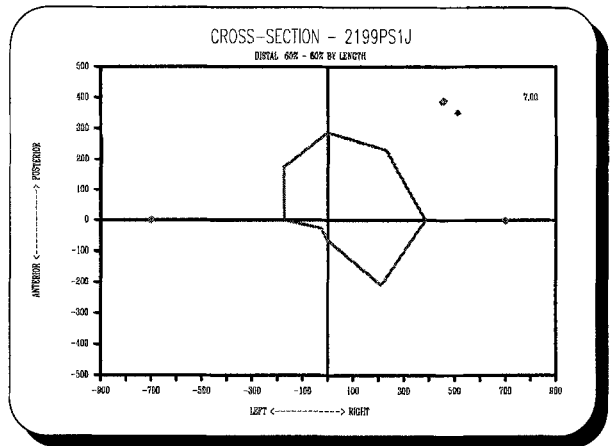
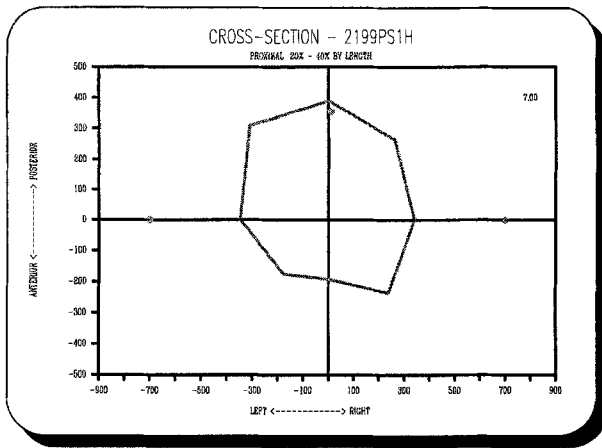
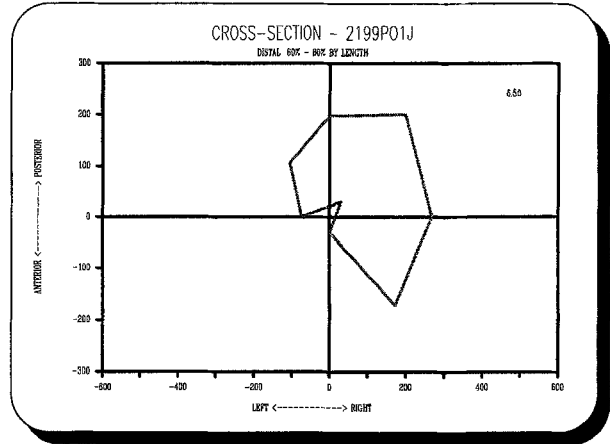
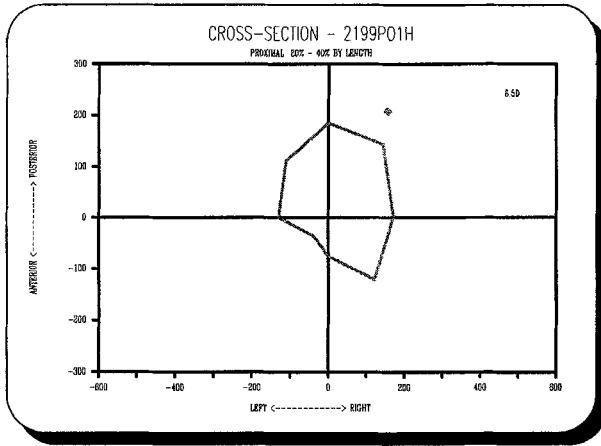


Figure 1. Figure depicts cross-section of proximal (left half of figure) and distal (right half of figure) anal sphincter. Cross-sectional asymmetry of the sphincter at rest (top), on squeeze (middle), and results of balloon reflex manometry (bottom) are represented from above downward. Cross-section of proximal sphincter (left half of figure) shows a left anterior defect at rest (top), which is well compensated during squeeze (middle), indicating good EAS function. On balloon reflex manometry, RAER is good (bottom). Cross-section of distal sphincter (right half of figure) of the same patient (Patient 9) shows a defect in the left anterior quadrant at rest (top) not compensated on squeeze (middle). On balloon reflex manometry, distal RAER is absent (bottom).

Various types of abnormalities of distal RAER identified included absence of reflex in eight patients, diminished amplitude of reflex excitation in two patients, excitation only at high volume (120 ml) of balloon inflation in two patients, and, in one patient, a diminished excitation amplitude only at high volume (120 ml) of inflation. Three patients had high (>25 percent) cross-sectional sphincter asymmetry. In one of these patients (Patient 9) with normal PNTML and SFD, high sphincter asymmetry (39.7 percent) accounted for the absence of distal excitation.

DISCUSSION

Determination of PNTML and estimation of SFD are the most commonly used means of confirming pudendal neuropathy. In a previous study, we³ reported that an abnormal distal RAER may serve as an alternative index of pudendal neuropathy. Although correlation coefficient between abnormal distal RAER and PNTML or SFD is not significant in the present series, agreement among the three investigative methods in confirming or excluding pudendal neuropathy in ten patients (67 percent) confirms that abnormal distal RAER is a useful marker of pudendal neuropathy. These findings suggest that the distal abnormal RAER compares favorably with other currently used neurophysiologic tests (PNTML estimation and SFD measurement). Lack of statistical correlation is a function of the small sample size, and a larger sample series may reveal statistical significance.

A drawback of using abnormal distal RAER as an index of pudendal neuropathy is that, in patients with EAS defects, distal RAER may be absent despite intact pudendal nerves. This is well exemplified in Patient 9 (Fig. 1) in whom the high cross-sectional asymmetry secondary to a distal sphincter defect led to an absence of distal RAER. These findings reaffirm the observations of an earlier study⁶ from this institution, which found that traumatic EAS defects may lead to isolated absence of distal excitatory response.

Our results reiterate that PNTML is not an infallible index of neurogenic injury because, in two patients,

PNTML was normal despite increased SFD. That the distal RAER was abnormal in these two patients suggests that, in the absence of extensive traumatic injury to the EAS, an abnormal distal RAER should arouse suspicions of neuropathy even when PNTML is normal. An abnormal distal RAER may also have a role in selecting patients for EMG. We suggest that, in patients with suspected pudendal neuropathy, a normal PNTML in the presence of an abnormal distal RAER is an indication for SFD estimation. However, when both PNTML and distal RAER are abnormal, patients may be spared the painful experience of the invasive procedure of SFD estimation.

CONCLUSION

Neuromuscular abnormalities of the EAS may result in absence of distal RAER. In the absence of extensive EAS defects or EAS involvement in myopathic disorders, absence of distal RAER indicates pudendal neuropathy. As a marker of pudendal neuropathy, abnormal distal RAER compares favorably with other currently available techniques used to document pudendal neuropathy.

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