Pudendal Nerve Terminal Motor Latency Influences Surgical Outcome in Treatment of Rectal Prolapse

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PURPOSE: This study was undertaken to document the effect of pudendal nerve function on anal incontinence after repair of rectal prolapse. METHODS: Patients with full rectal prolapse (n = 24) were prospectively evaluated by anal manometry and pudendal nerve terminal motor latency (PNTML) before and after surgical correction of rectal prolapse (low anterior resection (LAR; n = 13) and retrorectal sacral fixation (RSF; n = 11)). RESULTS: Prolapse was corrected in all patients; there were no recurrences during a mean 25-month follow-up. Postoperative PNTML was prolonged bilaterally (>2.2 ms) in six patients (3 LAR; 3 RSF); five patients were incontinent (83 percent). PNTML was prolonged unilaterally in eight patients (4 LAR; 4 RSF); three patients were incontinent (38 percent). PNTML was normal in five patients (3 LAR; 2 RSF); one was incontinent (20 percent). Postoperative squeeze pressures were significantly higher for patients with normal PNTML than for those with bilateral abnormal PNTML (145 *vs.* 66.5 mmHg; $P = 0.0151$). Patients with unilateral abnormal PNTML had higher postoperative squeeze pressures than those with bilateral abnormal PNTML, but the difference was not significant (94.8 *vs.* 66.5 mmHg; $P = 0.3182$). The surgical procedure did not affect postoperative sphincter function or PNTML. CONCLUSION: Injury to the pudendal nerve contributes to postoperative incontinence after repair of rectal prolapse. Status of anal continence after surgical correction of rectal prolapse can be predicted by postoperative measurement of PNTML. [Key words: Prolapse; Pudendal nerve; Manometry; Rectopexy; Fecal incontinence]

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Fecal incontinence occurs commonly in patients with complete rectal prolapse. The mechanism of incontinence is multifactorial, and reports of improvement in continence after repair have been variable. Some conflicting information in the literature regarding postoperative sphincter function may be attributable to the inclusion of patients with internal prolapse (intussusception).[1, 2] Patients with intussusception generally have normal resting and squeeze pressures preoperatively and usually have no postoperative change in sphincter function.[2] Complete extrusion of the rectum through the anal sphincter may result in pudendal nerve injury from repetitive stretching during rectal prolapse. The resulting irreversible neurogenic injury may be an important factor in continued anal incontinence after rectal prolapse repair.

To define the role of pudendal nerve injury in anal incontinence in the setting of rectal prolapse, we evaluated patients with complete rectal prolapse using anal manometry, electromyography, and defecography to assess anal sphincter function before and after repair. The aims of this study were as follows: 1) assess the effect of surgical repair of rectal prolapse on anal sphincter function; 2) determine whether neurogenic injury contributes to postoperative incontinence.

PATIENTS AND METHODS

Anal manometry (AM), electromyography (EMG), and defecography were used to evaluate 24 patients with full rectal prolapse before and following surgical repair. Full rectal prolapse was demonstrated during office examination or documented with defecography. Patients with partial prolapse or internal intussusception were excluded from the study. Rectal prolapse was repaired using rectosacral fixation with Marlex™ (C.R. Bard, Inc., Billeries, MD) mesh in 11 patients and low anterior resection with suture rectopexy in 13 patients. Surgical treatment was the choice of the operating surgeon and not dependent on preoperative symptoms. Two men and 22 women were studied. Average age was 59 (range, 32–76) years.

Questions about incontinence and function were administered in person by LS. Detailed medical evaluation including medical, surgical, and obstetric history was obtained preoperatively. All medications, including over-the-counter medications, were re-

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corded. All patients were questioned regarding their past and current bowel function at each evaluation.

Anal manometry was performed in the left lateral decubitus position. A hydraulic capillary perfusion system (Arndorfer Medical Specialists, Greendale, WI) was used to perfuse a flexible, four lumen, 140 cm polyvinyl catheter. Radially positioned ports were located 4 cm from the tip at 90° from one another. Water was perfused at a rate of 0.5 ml/min, and the catheter was withdrawn 1 mm/s using a reverse geared Harvard pump system (Harvard Apparatus, Southnatick, MA). Pressure transducer was interfaced with an IBM computer, and recordings were interpreted by a commercially available software program (Synetics/Polygraph, Synetics Medical Inc., Irving, TX). Maximum resting pressure, maximum squeeze pressure, and sphincter length were measured in all four quadrants. Sensory threshold was recorded as the first perception of rectal filling using an air-filled rectal balloon.

Electromyography was performed using a finger electrode coupled with an electrical stimulator (Dantec Neuromatic 2000, Dantec Medical Inc., Santa Clara, CA). Measurements were done in the left lateral decubitus position after AM was completed.

Defecography was performed in the Department of Radiology at Barnes-Jewish Hospital of St. Louis, Washington University Medical Center. Contrast material thickened with carboxymethylcellulose was instilled in a retrograde manner through a large-bore catheter. The patient was seated on a cushioned plastic bedpan and videotaped in the upright position. Videotapes were reviewed by a single reviewer (Dr. Oh) to maintain uniform observation. Internal Review Board-approved consent was obtained for follow-up testing (defecography, anal manometry, and EMG).

Comparison between manometric values was performed with the unpaired Student's *t*-test and Welch's approximate *t*-test for comparison of means. Comparison between continence and pudendal nerve function was performed with Fisher's exact test with Yates' correction.

RESULTS

There were no adverse reactions to manometry, electromyography, or defecography, and there were no major surgical complications. Follow-up examinations and repeat testing were performed 3 to 76 months after surgical repair. There was no evidence of recurrent rectal prolapse in the group studied.

Continence

Fecal incontinence was defined as the inability to control liquid (Grade II) or solid (Grade III) stool. Patients who were incontinent of gas (Grade I) but who had control of liquid and solid stool were considered to be continent for the purpose of this study. Preoperatively, 12 patients (50 percent) were incontinent of liquid or solid stool, and 12 patients had complete control or were only incontinent of gas (Fig. 1). Improvement was noted in four patients (33 percent) after surgical repair (2 low anterior resection (LAR); 2 retrorectal sacral fixations (RSF)). Nine of the 12 patients who were continent preoperatively remained so after surgery (5 LAR; 4 RSF). Incontinence developed in three patients after surgery (3 LAR). Thus, 13 patients (54 percent) improved or remained continent, and 11 patients (46 percent) were incontinent after repair of rectal prolapse.

Defecography

Defecography was performed on 13 patients (7 LAR; 6 RSF) at time of diagnosis and was repeated an average of 33 (range, 12–76) months later (Table 1). Radiologic evidence of rectal prolapse or intussuscep-

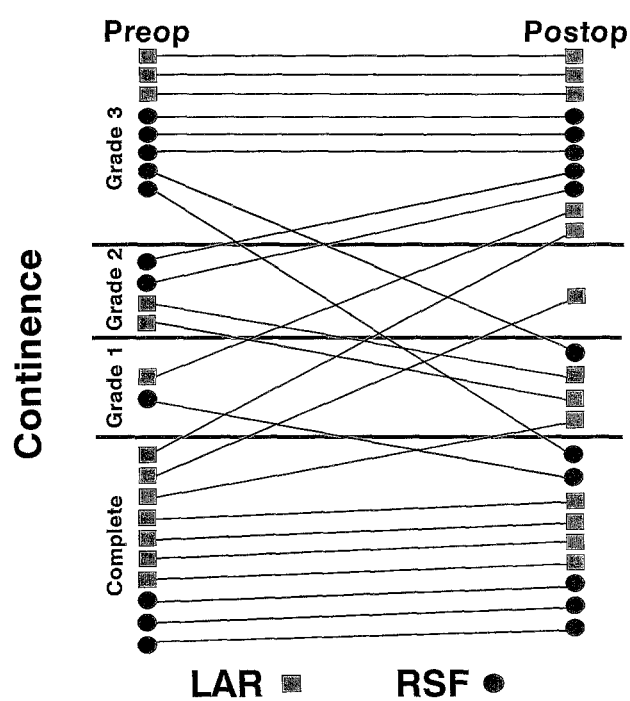

Figure 1. Effect of prolapse repair on continence. Preop = preoperative; Postop = postoperative; LAR = low anterior resection; RSF = retrosacral fixation; Grade I = incontinent of gas; Grade II = incontinent of liquid stool; Grade III = incontinent of solid stool.

Table 1.
Defecography

	Mobility		Evacuation Normal	
	Abnormal Preoperatively	Improved Postoperatively (%)	Preoperatively	Postoperatively (%)
LAR	7	7 (100)	7	2 (28)
RSF	6	6 (100)	5	3 (60)

LAR = low anterior resection; RSF = rectosacral fixation.

Table 2.
Manometry: Low Anterior Resection (LAR) *vs.* Rectosacral Fixation (RSF)

	Mean Maximum Resting Pressure (mmHg)	Mean Maximum Squeeze Pressure (mmHg)	Average Sphincter Length (cm)
LAR (n = 13)			
Preoperative	42.2 ± 19.4	70.3 ± 29.5	3.0 ± 0.7
Postoperative	48.1 ± 22.4	85.1 ± 42.9	2.7 ± 0.4
RSF (n = 11)			
Preoperative	49.1 ± 26.5	87.5 ± 44.3	3.5 ± 0.6
Postoperative	49.0 ± 24.6	103.5 ± 66.4	2.7 ± 0.9

tion was demonstrated in all 13 patients before repair. After repair, the sigmoid colon appeared fixed to the sacrum, and rectosigmoid mobility was minimum. Preoperative evacuation of rectal contrast was good in 12 of the 13 patients evaluated with defecograms. Postoperative improvement in rectal evacuation was seen in the one patient who had poor evacuation preoperatively. Evacuation of rectal contrast worsened in eight patients (5 LAR; 3 RSF) after prolapse repair. No specific bowel complaint could be attributed to poor evacuation in these eight patients; five patients were continent, and three patients were incontinent.

Anal Manometry

All patients underwent AM at time of diagnosis, and the test was repeated an average of 25 (range, 3–76) months after surgical procedure. Mean maximum resting pressure, maximum squeeze pressure, and sphincter length for all patients before and after surgical prolapse repair are seen in Table 2. Preoperative resting pressures, squeeze pressures, and sphincter lengths were similar between surgical groups ($P > 0.1$). There was no significant difference in postoperative sphincter function between surgical groups ($P > 0.1$). Mean maximum resting pressures were unchanged after surgical repair and did not differ between the two surgical groups (Table 2). Mean maximum squeeze pressures improved in both groups and reached normal values after surgical repair, but this improvement was not statistically significant. Although mean maximum squeeze pressures were slightly higher in RSF patients, the difference was not significant compared with the LAR group. Average sphincter length was similar between the two groups preoperatively; it was unchanged by LAR and decreased after RSF ($P < 0.03$).

Initial resting and squeeze pressures were not significantly higher in patients who achieved postoperative continence (Table 3). An increase in resting and squeeze pressures after rectal prolapse repair was seen in the group of patients that was continent postoperatively. This improvement was not seen in incontinent patients, whose mean values for resting and squeeze pressures remained below normal. Increase in resting and squeeze pressures after prolapse repair in continent patients, however, did not achieve statistical significance. Continent patients had postoperative resting and squeeze pressures within the normal range (>40 mmHg, >80 mmHg, respectively), whereas postoperative values for incontinent patients remained below normal expected values. No change in sphincter length was seen in the group that was continent postoperatively. A significant decrease in sphincter length was seen in patients who were incontinent postoperatively ($P < 0.0001$).

Table 3.
Manometry: Rectal Prolapse

Postoperative	Mean Maximum Resting Pressure (mmHg)	Mean Maximum Squeeze Pressure (mmHg)	Average Sphincter Length (cm)
Continent (n = 13)			
Preoperative	48.5 ± 28.8	84.1 ± 40.8	2.9 ± 0.7
Postoperative	51.6 ± 28.5	114.8 ± 61.2	2.9 ± 0.7
Incontinent (n = 11)			
Preoperative	41.7 ± 13.6	71.2 ± 22.9	3.6 ± 0.4*
Postoperative	37.7 ± 16.1	68.4 ± 34.4	2.4 ± 0.5*

* $P < 0.0001$.

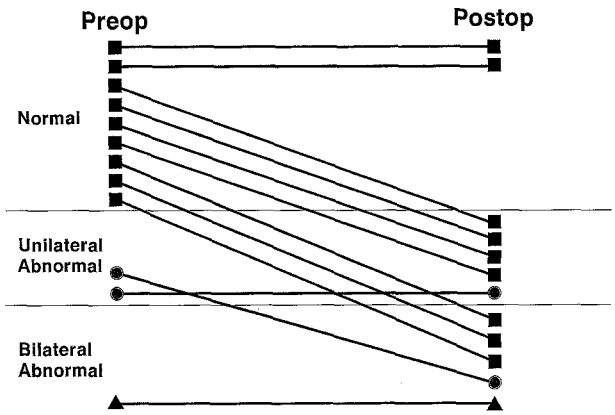

Figure 2. Pudendal nerve latency. Preop = preoperative; Postop = postoperative.

Electromyography

Twelve patients had both preoperative and postoperative EMG (Fig. 2). None of the three patients with abnormal (either unilateral or bilateral) preoperative pudendal nerve terminal motor latency (PNTML) returned to normal in the postoperative period. All three patients were incontinent on postoperative evaluation. Seven patients with normal preoperative PNTML developed prolonged PNTML in the perioperative period (4 LAR; 3 RSF); four were incontinent postoperatively.

Postoperative PNTML was tested in a total of 19 patients, which included 12 patients tested preoperatively. PNTML was prolonged bilaterally (>2.2 ms) in six patients (3 LAR; 3 RSF); five were incontinent (83 percent). Postoperative PNTML was prolonged unilaterally in eight patients (4 LAR; 4 RSF); three were incontinent (38 percent). Postoperative PNTML was normal bilaterally in five patients (3 LAR; 2 RSF); one was incontinent (20 percent; Table 4). Postoperative resting pressures did not correlate with PNTML. Squeeze pressures, however, correlated well with pudendal nerve function. Patients with bilaterally normal PNTML had significantly higher postoperative squeeze pressures than patients with bilaterally abnormal PNTML (145 *vs.* 66 mmHg; $P < 0.01$). Patients with a single normal pudendal nerve had higher postoperative squeeze pressures compared with those with bilaterally abnormal PNTML, but the difference was not significant (95 *vs.* 66 mmHg; $P < 0.32$).

DISCUSSION

Fecal incontinence occurs in approximately two-thirds of patients presenting for surgical correction of rectal prolapse. Many studies have reported an improvement in continence after surgical correction.[1, 3] The mechanism for improved control is not clear, and to date there is no way of predicting who will remain incontinent postoperatively. Persistent incontinence after rectal prolapse repair may be attributable to injury of pudendal nerves stretched during rectal prolapse or chronic stretching of the anal sphincter by the prolapsing rectum. Surgical removal of the rectal reservoir or correction of the "obstructing" rectal prolapse may also contribute to patients' symptoms of urgency and postoperative incontinence.

Anatomic control of rectal prolapse has been demonstrated using defecography in patients treated with rectopexy alone and with sigmoidectomy.[4, 5] We demonstrated that anatomic correction of rectal prolapse occurs with either surgical repair (LAR or RSF) but that rectal emptying decreased in both groups. Cause of abnormal emptying is unclear. In patients studied, mesh rectopexy fixed the rectum to the sacrum; folding of redundant sigmoid over the rectopexy was not seen, and there was no evidence of obstruction attributable to stenosis of the lumen by mesh. Mobilization or division of the anterolateral ligaments may injure the autonomic innervation of the rectum and has been proposed as one mechanism contributing to poor rectal emptying.[6] In this study,

Table 4.
Postoperative Pudendal Nerve Status

PNTML	Incontinent (%)	Pressure (mmHg)			
		Rest		Squeeze	
		Preoperative	Postoperative	Preoperative	Postoperative
Normal (n = 5)	1 (20)	45.2 ± 18.4	56.0 ± 24.7	86.8 ± 19.0	145.0 ± 58.1*
Unilateral abnormal (n = 8)	3 (38)	51.2 ± 31.6	53.7 ± 32.4	79.3 ± 50.8	94.8 ± 62.1
Bilateral abnormal (n = 6)	5 (83)	44.3 ± 14.2	43.2 ± 20.8	84.7 ± 37.8	66.5 ± 26.0*

PNTML = pudendal nerve terminal motor latency.
* $P < 0.015$.

the rectum was completely mobilized posteriorly and anteriorly for both LAR and RSF, but lateral ligaments were not routinely divided. It is possible that adhesion formation around the rectum postoperatively may decrease mobility required for normal evacuation.

Using manometry to predict which patients will have improved postoperative continence has not been conclusive. Preoperative resting pressures below 10 mmHg and maximum squeeze pressures below 50 mmHg have been shown in one study to be associated with persistent postoperative incontinence.[1] Other investigators have not found this to be true.[7,8] In the current study, we did not find profoundly low preoperative resting or squeeze pressures, and we did not find low preoperative resting or squeeze pressures to be predictive of postoperative outcome.

Studies using manometry in the preoperative and postoperative period have had conflicting results. In the current study, postoperative continent patients had improvement in voluntary contraction (squeeze pressure) to a level that was higher than those who remained incontinent (Table 3). Resting pressure, indicative of internal sphincter function, did not change. Some investigators have had similar results and have shown an improvement in resting and squeeze pressures after surgical repair, which was associated with an improvement in fecal continence.[1,7] Others have shown no significant improvement in sphincter function as measured by manometry after repair of rectal prolapse, although most of these studies showed improvement in continence.[3,5,8–11] Long-term follow-up of patients with rectal prolapse and incontinence has shown no improvement in resting or squeeze pressures for up to one year after repair.[9]

Internal anal sphincter recovery has been reported as one factor that may contribute to improved continence after correction of rectal prolapse.[2,12,13] Continued internal anal sphincter relaxation, a result of prolapsing rectum eliciting the rectoanal inhibitory response, may contribute to leakage of stool before surgical repair. The internal anal sphincter provides approximately 80 percent of normal resting pressure but does not contribute significantly to active squeeze function.[14] Correction of rectal prolapse may improve bowel function by allowing the internal sphincter to resume its normally contracted state at rest and would be seen as an increase in postoperative resting pressure. Our results do not support this theory, and we found no alteration or improvement in resting pressures after surgical repair of rectal prolapse.

Sphincter length is determined by measuring the high-pressure zone in the resting state and is often not reported in manometric studies of rectal prolapse. The double-balloon manometric method used may not accurately reflect sphincter length, and very low resting pressures may make it difficult for precise determination of actual sphincter length by capillary perfusion. Presence of a prolapsing rectum may alter manometric reading by lengthening the high-pressure zone. It is difficult to explain how an abdominal procedure could alter sphincter length in any way, but this phenomenon has been found by others.[15] It is possible that improved internal anal sphincter function occurring after rectal prolapse repair allows for an increased length of the high-pressure zone. Significantly shorter sphincter lengths have been found in patients who remain incontinent after rectal prolapse repair.[15] Other groups have found that improved internal anal sphincter function does not correspond to increased sphincter length.[13] Patients in our series who became or remained continent postoperatively showed no change in sphincter length. However, patients who were incontinent after surgery had a significant decrease in sphincter length and no improvement in internal sphincter function (Table 3). Because few studies report sphincter length and the

number of patients in the reported studies is low, it is difficult to draw conclusions about the role of sphincter length in continence after surgery.

Complete prolapse of the rectum has been thought to cause incontinence by stretch injury of pudendal nerves.[10, 16] The pudendal nerve arises from the anterior rami of S2-4 and primarily supplies the external anal sphincter.[17] It enters the perineum through the lesser sciatic foramen and passes through the sacrotuberous and sacrospinous ligaments. The nerve is relatively fixed at this point, and injury (stretch or ischemic) is thought to occur here with chronic straining. Branches of sacral nerves that innervate the puborectalis lie above the pelvic floor.[17] Patients with neurogenic anal incontinence have electromyographic and histologic evidence of damage to the innervation of the puborectalis and external sphincter.[16, 18] Conduction delay in patients with idiopathic fecal incontinence has been shown to occur distally, suggesting a distal stretch injury to the pudendal nerve.[19] One would, therefore, expect to see abnormal PNTML and an associated abnormal function of the external anal sphincter (low squeeze pressures) in patients with fecal incontinence associated with rectal prolapse.

There have been few studies using electromyography to assess external sphincter function in patients with rectal prolapse and even fewer studies after surgical repair. Preoperative evaluation has demonstrated abnormal EMG in patients with rectal prolapse and incontinence, whereas patients with rectal prolapse who are continent demonstrated normal EMGs.[20] Follow-up EMGs were not done in that study; thus, it is unknown whether these changes (EMG and incontinence) persisted postoperatively. In the current study, abnormal postoperative PNTML correlated with a decrease in external sphincter function (maximum squeeze pressure) and persistent fecal incontinence. Increased postoperative PNTML was seen in 7 of 12 patients and is a finding difficult to explain because abdominal operations should not affect the pudendal nerve. It may be attributable to progressive ischemic neurogenic injury, which is not reversed by simple anatomic correction of the prolapse. Many patients continue to strain to evacuate, despite repair; poor rectal emptying was shown to occur by defecography, thus injury to the pudendal nerve may continue.

Multiple abdominal operations have been used for repair of rectal prolapse with varying results. We found that there does not appear to be a functional difference between LAR and RSF in treatment of complete rectal prolapse (Table 2). Our findings are similar to those of a prior randomized study that showed no significant difference in clinical outcome between sigmoidectomy compared with mesh rectopexy for repair of rectal prolapse.[3] We have shown that control of rectal prolapse by surgical repair may correct the anatomic defect, but sphincter function does not improve if there is bilateral pudendal nerve injury. Resting pressure, an indication of internal anal sphincter function, is not affected by rectal prolapse repair. Anal continence after repair of rectal prolapse correlates with postoperative external sphincter and pudendal nerve function. Mesh rectopexy and low anterior resections give similar functional results and physiologic repair. Further studies of anorectal function after repair of rectal prolapse should include evaluation of innervation of the anal sphincter.

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