Manometric evaluation of rectal prolapse and faecal incontinence

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SUMMARY Sixty-three patients with complete rectal prolapse and/or faecal incontinence have undergone anal manometry and the results have been compared with an equal number of age- and sex-matched controls. Maximal basal pressure (MBP) and maximum squeeze pressure (MSP) were measured before and at four months and a year after treatment. The anal pressures of normal subjects are presented. Patients with rectal prolapse alone had normal anal pressures, whereas patients with incontinence with or without prolapse had significantly lower basal and squeeze pressures than controls. Successful surgical treatment of prolapse or incontinence did not produce significant change in anal canal pressures, whereas the combination of pelvic floor exercises and a continence aid was associated with a significant rise in MSP.

Rectal prolapse is a common distressing disease of the elderly because it is frequently accompanied by faecal incontinence. Evidence of sphincter denervation has been found in such patients and it has been suggested that the incontinence of rectal prolapse is due to traction on the pudendal nerve, caused by perineal descent during long-standing straining at defacation.¹ If the theory of nerve damage is correct, one would expect a reduction in anal pressure in patients with rectal prolapse and incontinence. Furthermore, successful operative correction of the prolapse might prevent further damage and allow regeneration of the pudendal nerve. If the habit of straining were controlled, such events might be accompanied by a rise in anal pressure after operation.

Anorectal incontinence may be due to factors other than rectal prolapse, such as surgical, obstetric, and accidental trauma. Where therapy is aimed at restoring the sphincter mechanism a rise in anal pressures might occur after successful operation, whereas therapy aimed at restoring the anorectal angle is unlikely to influence anal pressures.

The aims of this study have been to estimate the manometric abnormalities in patients with rectal prolapse and incontinence and to assess the effects of successful treatment on anal pressure.

Methods

PATIENTS

Between January 1977 and September 1979, 63 patients were studied with complete rectal prolapse and/or faecal incontinence. Thirty-nine patients had rectal prolapse, of whom 31 (80%) had associated incontinence and another 24 patients had incontinence without any evidence of rectal prolapse. The group with incontinence alone consisted of seven patients who had had repeated treatment by anal dilatation and three who had had a sphincterotomy for haemorrhoids. In addition, there were three patients with previous obstetric tears, three patients with Crohn's disease, three patients who had had a fistula treated surgically, two who had trauma to the pelvis, one with an imperforate anus, one patient with a solitary rectal ulcer, and seven patients with idiopathic anorectal incontinence. Anal pressures in each group were compared with an equal number of age- and sex-matched controls who were patients admitted to the surgical wards for operation on conditions other than anorectal disorders.

TECHNIQUES

Patients were first evaluated clinically. Proctoscopy and sigmoidoscopy were performed on all patients and a digital assessment was made of the resting sphincter tone and of the influence on tone of voluntary sphincter contraction. The anorectal angle and the perineal body were also examined.

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Anal pressures were always measured after proctoscopy with the patient in the left lateral position. A closed water filled balloon probe mounted on a graduated hollow Perspex rod² was inserted into the anal canal at distances of 1, 2, 3, 4, and 5 cm from the anal verge. The probe was connected to a Statham strain gauge pressure transducer and thence to a multichannel recorder (Devices Ltd). Anal pressures were measured in centimetres. H₂O at rest (basal pressure) and after voluntary contraction of the sphincters (squeeze pressure). The highest basal and squeeze pressures were recorded as the maximal basal pressure (MBP) and maximal squeeze pressure (MSP).

Rectal prolapse was treated by abdominal rectopexy.³ Incontinence was treated by one of three methods used alone or in combination: external sphincter repair,⁴ postanal repair⁵ or pelvic floor exercises in conjunction with a continence aid.⁶ The continence aid delivered an intermittent stimulus to the external sphincters produced by an asymmetrical biphasic pulse of 0.8 volts at a rate of 80 Hz with a pulse width of 1 ms.

Anal manometry was performed before treatment and at four months and a year after treatment. At the same time the clinical effect of treatment for prolapse and incontinence was noted. Anal pressures were compared statistically using Student's *t* test.

Results

Table 1 shows the MBP and MSP in male and female controls according to age in decades. The MBP remained constant with a median of 105 cm H₂O in both sexes until the eighth decade, when it fell. In women in the eighth and ninth decades the MBP fell to a mean of 43 cm and 38 cm H₂O respectively and in men it fell to a mean of 78 cm and 65 cm H₂O in the eighth and ninth decades respectively. The normal MSP remained constant with a median of 230 cm H₂O in women and 300 cm in men. The MSP fell in women to a mean of 101 and 102 cm H₂O in the eighth and ninth decades respectively. In men, the MSP was maintained until the ninth decade when it fell to a mean of 119 cm H₂O.

Table 1 Anal manometry in controls (cm H_2O)

Age	Female			Male		
(yr)	No.	MBP	MSP	No.	MBP	MSP
20-30	8	91±35	270±122	6	113 ± 17	284±90
30-40	10	102 ± 28	197 ± 66	10	103 ± 25	262 ± 81
40-50	7	109 ± 39	221 ± 94	14	114 ± 24	319 ± 56
5060	8	109 ± 30	236 ± 86	15	106 ± 26	129 ± 75
6070	11	101 ± 25	232 ± 95	8	104 ± 29	209 ± 52
70-80	9	43 25	101 + 37	6	78 ± 14	259 ± 85
80-90	8	38 + 8	102 - 25	4	65 + 17	119 +10

MBP: Maximum basal pressure. MSP: Maximum squeeze pressure.

Table 2 Anal manometry before treatment $(cm H_2 O)^*$

	No.	MBP		MSP	
		Controls	Patients	Controls	Patients
Prolapse Prolapse and	8	57 ± 34	48 ± 35	128 ± 102	115 ± 56
incontinence Incontinence	31 24	${}^{60\pm33}_{102\pm40}$	${}^{31\pm24+}_{56\pm34+}$	$\begin{array}{c} 148 \pm 97 \\ 246 \pm 103 \end{array}$	$\begin{array}{r} 88 \pm 53 \\ 112 \pm 69 \\ \end{array}$

*Patients in each group were compared with an equal number of ageand sex-matched controls. Age matching was by decades. +p < 0.001.

For the purpose of analysis patients were divided into three groups: (1) those with a rectal prolapse and no incontinence, (2) those with incontinence associated with a rectal prolapse, and (3) those with faecal incontinence without a rectal prolapse. The pretreatment pressures are shown in Table 2. The MBP and MSP in patients with rectal prolapse alone did not differ significantly from age- and sexmatched controls. On the other hand both groups of patients with faecal incontinence, irrespective of whether or not they had a rectal prolapse, had a significantly lower MBP (P < 0.001) and MSP (P < 0.001) than the control group.

The clinical results of treatment were as follows: rectopexy was successful in controlling the rectal prolapse in all patients, but in only 12 of 19 patients (63%) was rectopexy completely successful in controlling incontinence. Postanal repair was performed in 16 patients either for persistent incontinence after rectopexy or for patients with idiopathic anorectal incontinence. Continence was completely restored in nine patients (56%) and four more were improved (81%). External sphincter repair was performed in 12 patients with damaged external sphincters; continence was fully restored in eight (67%) and one more was improved (75%). Pelvic floor exercises with the continence aid were used in 27 patients, often while awaiting operation. In 12 patients the improvement in continence was so good that operation was deferred (44%); four more patients were improved (59%). Using a combination of therapy 50 of the 55 patients with faecal incontinence have been improved (91%) and 47 are completely continent (85%).

Table 3 shows the pretreatment pressures together with the pressures at four months and a year after each of the four methods of treatment. Rectopexy, external sphincter repair, and postanal repair failed to have any significant effect on the MBP or MSP, even though a large proportion were cured of their incontinence. Pelvic floor exercises produced a significant rise in MSP at four months (t=2.444, P < 0.05), although there was no change in MBP. However, if we subtract those patients whose in-

	No.	MBP	MSP
Rectopexy			
Pretreatment	24	44 ± 31	117 ± 60
4 months	24	50 ± 28	125 ± 87
1 year	11	44 ± 31	$93\!\pm\!42$
Postanal repair			
Pretreatment	14	47 ± 28	107 ± 62
4 months	14	56 ± 25	122 ± 57
1 year	6	35 ± 30	90 ± 53
External sphincter repair			
Pretreatment	11	35 ± 26	63 ± 51
4 months	11	39 ± 36	77 ± 59
1 year	6	27 ± 14	83 ± 52
Faradism			
Pretreatment	21	40 + 26	78+49
4 months	21	42 + 27	100 + 43*
1 year	5	68 ± 25	181 ± 132

Table 3 Anal manometry after treatment (cm H_2O)

*****P<0.05.

continence was not significantly improved by any of the four methods of treatment and look only at those whose incontinence was completely alleviated (Table 4) we find an overall rise in MSP at four months (t=2.299, P<0.05) but no significant change at 12 months, probably because of small numbers. There was no change in MBP.

Discussion

The maximal basal pressure represents the tone of the anal sphincter at rest. This is a combination of the internal sphincter, which is continuously active⁷ except when inhibited by rectal distension,⁵ and the external sphincter, which is under voluntary control but is nonetheless active while at rest from spindles in the pelvic muscle.⁸ The MSP, however, represents the tone of only the external sphincter and puborectalis at maximum activity superimposed upon the basal pressure.

Our recordings of anal pressure represent single measurements from each individual. We know from other studies, however, that there is considerable individual variation in both the MBP and the MSP, although reproducibility in the same patients on a single day is satisfactory. We have attempted to standardise the conditions of measurement and, in each instance, manometry was performed in the left lateral position after digital examination and

Table 4 Anal manometry after cure of incontinence

	No.	MBP	MSP
Pretreatment	22	44+31	101 + 64
4 months	22	47 + 29	129+108*
1 year	8	47 ± 30	135 ± 142

*****P <0.05.

proctoscopy, and using the same 7 mm probe. Variations still occur, however, as patients learn to co-operate during successive visits and there appeared to be a learning phase for squeeze pressures. It is possible that a finer probe producing less distortion of the anal canal might have produced less variation. On the other hand, in the patulous anus a fine bore catheter would not have been gripped by the sphincters. In our experience digital assessment of pressure was an unreliable indicator of MBP or MSP.

The controls were patients with a variety of surgical conditions. There was no reason to suppose that their anal pressures differed from the population at large. The mean MBP and the mean MSP in males and females were remarkably constant up to the end of the seventh decade, despite considerable variation between individuals. The decline in pressure after this age was probably due to muscle and nerve degeneration, which is known to affect continence in the elderly.⁹

Patients with faecal incontinence, with or without rectal prolapse, were shown to have a significantly lower MBP and MSP than normal. On the other hand patients with rectal prolapse alone did not have significantly lower pressures. Sphincter denervation may have accounted for the reduction in pressures in incontinent patients, but this denervation has also been demonstrated in patients with prolapse alone.¹ There is clearly a difference between the two groups of patients with rectal prolapse, but our patients did not differ in other respects, either in age or in duration of prolapse. The difference may have been due to loss of the anorectal angle on defaecation, which has been demonstrated cineradiographically in patients with prolapse.¹⁰

Control of rectal prolapse by rectopexy might prevent further denervation of the anal sphincter, thus allowing reinervation to occur with time. Although prolapse was cured by rectopexy in all patients, there was no rise in MBP or MSP within two years of operation. This contrasts with Kirkman's¹¹ findings, where no change in pressure was recorded at nine months but where there was a return to normal anal canal pressure after three years.

Operative treatment of faecal incontinence was not associated with any rise in pressures. Postanal repair aimed at restoring the anorectal angle without influencing the external sphincter and would not therefore have been expected to cause a rise in pressure. We might have anticipated an increase in the length of the high pressure anal zone after postanal repair, but none was recorded in our patients. It is noteworthy, however, that the pressures were not reduced after postanal repair as one might expect if the operative technique had damaged the nerve supply of the sphincters.⁵ A rise in pressure after repair of the external sphincter might have been suspected but was not found in the small number of patients who were studied.

Pelvic floor exercises and the continence aid produced a rise in MSP but no appreciable change in MBP, although this method of treatment was the least effective. The rise in MSP was maintained well beyond the end of treatment, suggesting that a learning process is involved. There is no doubt from our own clinical and repeated anal manometry findings that elderly patients often misinterpret the request to squeeze and that the MSP is increased if time is taken to allow the patient to practice. It is likely that the principal benefit of electrical therapy is in teaching the patient to use the external sphincters. It has been proposed that the extent of damage to the external sphincter in incontinent patients would render attempts at treatment by electrical stimulation valueless.1 Our results, however, do not support this hypothesis.

Patients whose incontinence was cured, irrespective of the cause or the method of treatment, had a significant rise in MSP from pretreatment levels, thus indicating that the return of continence was associated with an improvement in tone of the external sphincter.

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