Pressure and prolapse – the cause of solitary rectal ulceration

N R WOMACK, N S WILLIAMS, J H M HOLMFIELD, AND
J F B MORRISON

From the Surgical Unit, the London Hospital, Whitechapel, London, the University Department of Surgery, the General Infirmary at Leeds, Leeds, and the Department of Physiology, the University of Leeds, Leeds

SUMMARY The cause of solitary rectal ulceration has been investigated using a method that radiologically visualises rectal voiding whilst simultaneously measuring intrarectal pressure and external anal sphincter electromyographic activity. Control subjects and patients with the solitary rectal ulcer syndrome, both with and without mucosal ulceration, have been studied. A high incidence of rectal prolapse (94%) was present in the patients who voided. Overactivity of the anal sphincter during evacuation contributed to the fact that patients with mucosal ulceration required higher intrarectal pressures to void than the controls and the patients without mucosal ulceration. The results indicate that a combination of rectal prolapse and a high voiding pressure may act to cause the mucosal ulceration in this syndrome by exposing the rectal wall to a high transmural pressure gradient.

It is suggested that the solitary rectal ulcer syndrome (SRUS) forms part of a spectrum of disorders (the 'mucosal prolapse syndrome') that is characterised by histological changes in the rectal mucosa,¹ and is caused by prolapse of the rectal mucosa.² A relationship between solitary rectal ulceration and rectal prolapse, which is often clinically occult, has been well demonstrated³⁻⁶ but if the mucosal prolapse theory of the aetiology of SRUS is to be accepted it is necessary to be able to explain why only a small percentage of patients with rectal prolapse ulcerate, and the majority do not. It may be that other factors act in combination with the prolapse to cause the mucosal damage seen in patients with ulceration.

The objectives of this study were, therefore, to test the mucosal prolapse theory of the aetiology of the SRUS, and should it prove correct, to identify the additional factors responsible for the mucosal ulceration. To do this we have investigated the anorectal function of control subjects and patients with a diagnosis of SRUS confirmed by rectal biopsy, using a technique that radiologically visualises the

anorectum during voiding, whilst simultaneously measuring intrarectal pressure and anal sphincter EMG activity.

Methods

SRUS PATIENTS AND CONTROL SUBJECTS

Eighteen patients with SRUS (12 women, six men, age 30 years (19–64) (median and range)) confirmed by the presence of the characteristic changes of the syndrome on rectal biopsy and nine control subjects (six women, three men, age 44 years (35–52)) were investigated. Eleven of the patients (SRUS-U, seven female, four male, 27 years (19–59)) had ulceration, seen on sigmoidoscopy done in the week before the investigation, in addition to the biopsy changes. Seven patients (SRUS-NU, five women, two men, 46 years (22–64)) had an abnormal biopsy but no ulceration on sigmoidoscopy. The patients with ulcers were significantly younger than those without ulceration ($p < 0.01$) and the control subjects ($p < 0.01$). The latter, because of a restriction placed upon the selection of control subjects of child bearing age by the Ethical Committee of The General Infirmary at Leeds on approval of this study in March 1985.

Address for correspondence: Professor N S Williams, FRCS, Surgical Unit, London Hospital, London E1 1BB.

Received for publication 19 March 1987.

Table 1 Clinical features of the SRUS patients

	SRUS-U	SRUS-NU	p (Fisher)
Rectal bleeding	100%	71%	NS
Rectal pain	91%	28%	<0.02
Repeated straining at stool	82%	43%	NS
Tenesmus	73%	57%	NS
Difficulty initiating defecation	82%	28%	<0.05
Self digitation	73%	43%	NS
Aware of prolapse at presentation	45%	14%	NS

The patients' symptoms are detailed in Table 1. Rectal bleeding was a common symptom and was the reason for presentation in five of the SRUS-NU patients and six of the SRUS-U patients. Pain in the perineum or lower abdomen was more common in the SRUS-U patients and was the reason for presentation in two of them. Repeated straining at stool was also common in both groups. It was related to tenesmus in the SRUS-NU patients as they were able to initiate defecation without difficulty at the first call to stool. This was different from the SRUS-U patients who commonly could not initiate defecation, without difficulty, on any occasion. Nearly half of the SRUS-U patients admitted being aware of some degree of rectal prolapse on direct questioning, although it was the reason for presentation in only one patient from each group.

The control subjects were volunteers free from anorectal symptoms who had been admitted to the infirmary for minor surgical procedures.

Our technique for the dynamic assessment of anorectal function has been described in detail elsewhere.⁷ Briefly it entails delineation of the rectum by 300 ml of a semisolid contrast medium made from barium sulphate, porridge oats and water. The contrast medium also simulated a soft stool to be voided by the subject. The intrarectal pressure was measured *via* a pressure sensitive radiotelemetry capsule placed within the rectum. The electromyographic activity of the puborectalis and superficial parts of the external anal sphincter was measured by the insertion of fine stainless steel wire electrodes. A graphic display of the pressure and EMG data was produced by a BBC B microcomputer. This image was mixed with the video output of the image intensifier and the composite picture, containing the radiological, pressure and EMG data was stored on videotape. Lateral fluoroscopy of the anorectum was done with the patient seated on a commode. The patient first maximally contracted the sphincter muscles, and then attempted to void the contrast medium.

Measurements of radiation exposure were made using thermoluminescent dosimeters. With expo-

sure factors for fluoroscopy of 90–110 kVp, 2–3 mA and screening times of between one and two minutes skin exposure was measured at 1–4 rads. From these data the total body dose was calculated to be 30–120 mrem, and the dose to the gonads 50–200 mrem.

STATISTICAL ANALYSIS

Fisher's exact test has been used for the analysis of differences in the patients' symptomatology. The other data were not normally distributed and values have been quoted as median and range. The Mann Whitney U test has been used for the analysis of differences between groups.

Results

Voiding was achieved in all control subjects and in 16 (89%) of the SRUS patients. The radiological abnormalities revealed on proctography are listed in Table 2. The incidence of lesions involving prolapse of the rectal mucosa into the anal canal was 94% in the SRUS patients who voided. The incidence of the types of lesion shown was similar in the two subgroups of SRUS patients. Figure 1 shows an example of an intra-anal intussusception of the rectum, this was the most common lesion seen in both of the groups of SRUS patients.

During voiding in both SRUS and control subjects the puborectalis and superficial parts of the external anal sphincter always behaved in a similar manner, either increasing or decreasing in activity together, if not by exactly the same degree. Expressing sphincter activity as the mean of the puborectalis and superficialis activities voiding in the control subjects was associated with inhibition of the sphincters to median value of 25% of the resting level of activity (Fig. 2). Inhibition occurred with the onset of straining in four of the subjects and after a period of increased sphincter activity in the others (Fig. 3a). In the SRUS patients voiding was accompanied by increased

Table 2 Radiological abnormalities on proctography

	Controls	SRUS	SRUS-U	SRUS-NU
n	9	18	11	7
Voided	9	16	9	7
Radiological abnormalities				
Anterior rectal wall prolapse	0	2	1	1
Intra-anal intussusception	0	8	3	5
Full thickness rectal prolapse	0	5	4	1
Rectocele	1	0	0	0
With prolapse %	0	94	89	100

Fig. 1 Intra-anal intussusception, the head of the intussusception (➤) has entered the anal canal (—) during voiding, there has been some recruitment of anal sphincter EMG activity. (Upper trace, intrarectal pressure, bar = 75 mmHg; middle trace, puborectalis EMG activity; lower trace, superficial external anal sphincter EMG activity.)

activity in the sphincters that persisted throughout voiding (Fig. 3b). This increased activity, with a median value of 24% of the activity of a maximal voluntary contraction, was significantly different to the activity in the control subjects ($p < 0.02$).

In the SRUS-U patients voiding was accompanied by overactivity of the sphincters to a median level of 30% of the activity of a maximal voluntary contraction. This was significantly different from the behaviour of the sphincters in the controls ($p < 0.01$) and the SRUS-NU patients ($p < 0.05$), in whom the median level of activity during voiding, was unchanged from the resting level of activity (Fig. 2).

The rise in intrarectal pressure measured during voiding, or maximal attempts to void, was significantly higher in the SRUS patients (70 mmHg (45–155)) than in the controls (44 (37–103), $p < 0.02$). The highest voiding pressures were recorded in the

SRUS-U patients (74 (53–155)), their voiding pressures being significantly greater than the controls ($p < 0.01$) and the SRUS-NU patients (48 (45–70), $p < 0.01$). In the 13 SRUS patients (nine SRUS-U, four SRUS-NU) in whom sphincter activity increased or remained unchanged during voiding (Fig. 2) there was a significant inverse correlation ($R_s = 0.63$, $p < 0.05$) between the age of the patients and the pressure required to void contrast and a significant direct correlation ($R_s = 0.68$, $p < 0.01$) between the pressure required for voiding and the activity of the anal sphincter.

Discussion

Before discussing the results of these investigations the definition of rectal prolapse needs to be considered. The generally accepted definition is a con-

Fig. 2 Anal sphincter activity during voiding. (■ patient voided; □ patient failed to void.)

dition where part or all of the rectal wall protrudes through the anal orifice.⁸ The introduction of techniques involving radiological visualisation of rectal voiding, as used in this study, however, has necessitated a reappraisal of the definition of rectal prolapse, since degrees of prolapse which cannot be diagnosed clinically become apparent with these techniques. We have, therefore, considered any condition in which rectal mucosa enters the anal canal to be one of rectal prolapse because, for reasons explained below, these apparently minor abnormalities may have detrimental effects on the prolapsing mucosa and be the cause of much discomfort to the patient. Using this definition of rectal prolapse the results of this study support the 'mucosal prolapse syndrome' hypothesis of the aetiology of the solitary rectal ulcer syndrome as defecography revealed rectal prolapse in 94% of the SRUS patients who voided.

The presence of rectal prolapse in all the SRUS-NU patients studied implies that rectal prolapse, at least when acting alone, is not the cause of mucosal ulceration in this syndrome. The results suggest that it is the combination of rectal prolapse and a high voiding pressure, caused in most cases by overactivity of the external anal sphincter during voiding, that leads to mucosal ulceration. It would seem, therefore, that a mechanical explanation for the ulceration is most likely. Previous authors (reporting overactivity of the puborectalis muscle during straining,

in the SRUS) have explained the ulceration in terms of abrasion of the prolapsed mucosa by the unyielding puborectalis muscle bar,⁹ or as a result of ischaemia in the prolapse caused by its compression within the anal canal.¹⁰ Both of these hypotheses propound that the damage to the prolapsed mucosa occurs from without, as a result of muscle overactivity, and, therefore, they cannot satisfactorily explain the presence of ulceration in those patients where the sphincters relax during voiding. We suggest an alternative mechanism whereby the disruptive force acts from within the prolapsed mucosa and is not entirely dependent upon the activity of the sphincters during voiding.

To consider the normal situation first, during voiding the rectal mucosa is supported by the rectal contents which are at a similar pressure to the intra-abdominal pressure (Fig. 4a). The pressure gradient across the rectal wall will be small, being the intra-abdominal pressure – intrarectal pressure. The direction of the gradient may vary according to whether or not there is contraction of the rectal wall muscle. In the patient with a rectal prolapse the situation is different, here the prolapsed mucosa loses the support of the rectal contents, and as the prolapse reaches the outer end of the anal canal it comes in contact with air at atmospheric pressure. Under these circumstances the pressure gradient across the prolapsed rectal wall will be great (Fig. 4). This high pressure may lead to distension of the submucosal

Fig. 3 Intrarectal pressure and anal sphincter activity during voiding. (a) normal subject, the sphincter is inhibited during voiding. (b) SRUS subject, activity in the sphincter recruits with straining and persists throughout voiding. (IRP=intrarectal pressure, PUBO=puborectalis EMG activity, SEAS=superficial external anal sphincter EMG activity, MVC=maximal voluntary contraction of the sphincters, 100 cm water=75 mmHg.)

blood vessels resulting in rupture with rectal bleeding and devitalisation of the rectal mucosa. As this hypothesis depends upon the voiding pressure, in combination with rectal prolapse, to explain the mucosal ulceration it does not require the sphincters to be overactive during voiding, although this may be the most frequent cause of a high voiding pressure in these patients. Other causes of a high voiding pressure such as hard constipated stool, or obstructed defecation due to the presence of the head of an intussusception in the anal canal may be sufficient to create the correct conditions for ulceration to occur.

This proposed aetiological mechanism for solitary ulceration may explain the non-random situation of these lesions within the rectum.¹ Solitary ulcers characteristically occur at a distance of 7–10 cm from the anal margin. This was the usual level of origin of the investigations of the rectal wall that prolapsed into the anal canal as an anterior wall prolapse,

intussusception or full thickness rectal prolapse. The usual position of solitary ulceration, therefore, coincides with that part of the rectal mucosa subjected to a high transmural pressure gradient during voiding. The characteristic anterior position of solitary ulcers may result from selective exposure of the anterior rectal wall to a high transmural pressure gradient occurring as a result of the way the rectum prolapses. Prolapse usually starts with invagination of the anterior rectal wall at the initiation of voiding. In some patients the lesion progresses no further and only the anterior rectal wall is exposed to the high pressure gradient. In other patients the prolapsing mucosa becomes circumferential as voiding proceeds. The anterior component, however, is often the largest (Fig. 1) and it forms the leading part of the intussusception as it descends the anal canal. In such an intra-anal intussusception the anterior rectal mucosa would be exposed to the greatest pressure gradient and, therefore, would be the preferential site for ulceration. In a full thickness rectal prolapse the prolapsed mucosa would be exposed to a high pressure gradient circumferentially and ulceration could occur at any point. In this study the occult forms of prolapse, that would favour anterior ulceration, were more common (Table 2) and it would be expected, therefore, that the most frequent site for ulceration would be anterior.

To analyse the data from this study we have divided the SRUS patients into two groups according to whether ulceration was present or absent on sigmoidoscopy at the time of the study. Such a division might be considered to be artificial as there are a few patients with this condition in whom the ulcer may heal and recur. Whilst we accept that there may be some overlap between the two groups, the few longitudinal studies of this condition that have been published^{1–10} suggest that the ulceration is indolent – that is, once the ulcer has formed it remains. None of our patients showed any evidence of relapsing ulceration during the period of management before the study. It may be that the factors which we believe are the cause of ulceration act intermittently and this may explain the relapsing nature of the disease in some patients.

Overactivity of the sphincters during voiding was a common factor contributing to a high voiding pressure, occurring in approximately 70% of the patients in this study. The cause of the overactivity is unknown but the correlation between the intrarectal pressure and the degree of sphincter overactivity implies that it is because of a failure to inhibit the myogenic stretch reflex that normally modulates the activity of the pelvic floor muscles.¹¹ It results in excessive straining at stool, which, over a period of time, is thought to cause neurogenic damage to the

Fig. 4 Diagrammatic representation of the pressures involved in rectal voiding. Δp = pressure gradient across the rectal wall. (a) normal. (b) with intra-anal intussusception.

sphincters resulting in weakness.¹² This in turn would be expected to result in lower voiding pressures and indeed an inverse correlation between age and voiding pressure was shown in this study. This may explain why the peak incidence of solitary rectal ulceration occurs in younger subjects, where voiding pressures are highest, and decreases with increasing age.¹⁰

The findings of our study have implications for the management of this condition. If the dual factor hypothesis of the aetiology of solitary rectal ulceration is correct removal of one of the factors should be sufficient to cure the ulceration. There is at present no successful treatment for sphincter over-activity during voiding and therapy, therefore, should be directed at the rectal prolapse. Such treatment has been reported¹³ to improve symptoms that can be related to the presence of ulceration or the prolapse itself. Such treatment, however, would not be expected to remove all obstructive symptoms as some of these will be due to the pelvic floor muscle abnormality which will be unaffected by rectopexy. These residual symptoms are probably best dealt with by explanation of the problem and advice to resist the urge to excessive straining as much as possible.

This work was supported by a grant from the Medical Research Council. The authors wish to thank Professor D Johnston, Department of Surgery, The General Infirmary at Leeds, for allowing us to study his patients and Janice Womack for secretarial assistance.

References

- Madigan MR, Morson BC. Solitary ulcer of the rectum. *Gut* 1969; **10**: 871–81.
- Du Boulay CE, Fairbrother J, Isaacson PG. Mucosal prolapse syndrome – a unifying concept for solitary ulcer and related disorders. *J Clin Pathol* 1983; **36**: 1264–8.
- Schweiger M, Alexander-Williams J. Solitary rectal ulcer syndrome of the rectum – its association with occult rectal prolapse. *Lancet* 1977; **i**: 170–1.
- White CM, Findlay JM, Price JJ. The occult rectal prolapse syndrome. *Br J Surg* 1980; **67**: 528–30.
- Womack NR, Holmfeld JHM, Morrison JFB, Williams NS. Anorectal function in the solitary rectal ulcer syndrome (SRUS). *Br J Surg* 1986; **73**: 491.
- Mahieu P, Pringot J, Vanheuverzwijn R, Gancette L. Les prolapsus du rectum. *Acta Gastro-Enterolog Belg* 1981; **44**: 502–12.
- Womack NR, Williams NS, Holmfeld JHM, Morrison JFB and Simpkins KC. New method for the dynamic assessment of anorectal function in constipation. *Br J Surg* 1985; **72**: 994–8.
- Goligher J. Rectal prolapse. In: *Surgery of the anus, rectum and colon*. London: Baillière-Tindall, 1984: 246.
- Rutter KRP. Electromyographic changes in certain pelvic floor abnormalities. *Proc R Soc Med* 1974; **67**: 53–6.
- Rutter KRP, Riddel RH. The solitary rectal ulcer syndrome. *Clin Gastroenterol* 1975; **4**: 505–30.
- Parks AG, Porter NH, Melzak J. Experimental study of the reflex mechanism controlling the muscles of the pelvic floor. *Dis Colon Rectum* 1962; **5**: 407–14.
- Kiff ES, Barnes P, Swash M. Evidence of pudendal neuropathy in patients with perineal descent and straining at stool. *Gut* 1984; **25**: 1279–82.
- Nicholls RJ, Simson JNL. Anteroposterior rectopexy in the treatment of solitary rectal ulcer syndrome without overt rectal prolapse. *Br J Surg* 1986; **73**: 222–4.