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Anal manometry in patients with fissure-in-ano before and after internal sphincterotomy

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Abstract. The motility pattern of the internal anal sphincter was estimated manometrically in 42 patients with fissure-in-ano before and after left lateral internal sphincterotomy (LAS). Resting anal pressure and anal pressure during straining were significantly higher in patients than in controls (132 ± 21 SD cmH_2O vs 81 ± 14 SD cmH_2O $P < 0.0002$ and 46 ± 16 SD cmH_2O vs 13 ± 4 SD cmH_2O $P < 0.0005$), but were normal after LAS. Slow waves were more common in fissure patients (86 ± 6 SD % of total recording time vs 68 ± 11 SD % of total recording time, $P < 0.0002$), but also became normal after successful treatment. The presence of ultra slow waves was also more common in fissure patients ($P < 0.0001$), and although it was significantly reduced postoperatively ($P < 0.0001$), it did not return to normal. Sampling was less frequent in fissure patients ($P < 0.0001$) and improved significantly after successful treatment ($P < 0.0002$). Rectal distension produced significantly less reduction in anal pressure in fissure patients as compared to controls ($P < 0.01$), but successful treatment returned the response to normal. There were 2 patients with anal fissure who did not heal after left LAS. Those patients and a further 5 patients with non healed fissures after left LAS showed the same pathological manometric features as before surgery. Their fissures were successfully treated by additional right lateral internal sphincterotomy. In conclusion, increased internal sphincter activity is probably an aetiological factor in fissure-in-ano, while successful LAS improves anal sphincter function. Fissures which fail to heal after LAS, can be successfully treated by additional right internal sphincterotomy.

Résumé. L'aspect moteur du sphincter anal interne a été estimé manométriquement chez 42 patients qui avaient une fissure anale avant et après sphinctérotomie latérale interne gauche (LAS). Les pressions de repos et les pressions durant l'effort de retenue étaient significativement plus hautes chez les patients que chez les contrôles (132 ± 21 SD cmH_2O vs 81 ± 14 SD cmH_2O, $p < 0.0002$ and 46 ± 16 SD cmH_2O vs 13 ± 4 SD cmH_2O $p < 0.0005$), mais étaient normales après LAS. Des ondes lentes étaient plus communes chez les patients présentant une fissure (86 ± 6 SD % vs 68 ± 11 SD % du temps total d'enregistrement, $p < 0.0002$), mais se normalisaient aussi après traitement efficace. La présence d'ondes ultralentes étaient aussi plus commune chez les patients présentant une fissure ($p < 0.0001$) et de même étaient significativement réduites en post-opératoire ($p < 0.0001$), mais ne retournaient pas à la normale. Le réflexe d'échantillonage était moins fréquent chez les malades avec fissure ($p < 0.0001$) et s'améliorait significativement après traitement efficace ($p < 0.0002$). La distension rectale produisait significativement moins de réduction de pression anale chez les patients avec fissure comparée aux contrôles ($p < 0.01$), mais après traitement efficace la réponse retournait à la normale. Deux patients avec fissure anale n'ont pas cicatrisé après LAS gauche. Ces patients et 5 autres malades avec fissure non cicatrisée après LAS gauche montraient le même aspect manométrique pathologique qu'avant la chirurgie. Leurs fissures ont été traitées avec succès par une sphinctérotomie latérale interne droite. En conclusion, l'activité augmentée du sphincter anal interne est probablement un facteur étiologique de la fissure anale puisque la sphinctérotomie latérale interne améliore la fonction sphinctérienne anale. Des fissures qui ne cicatrisent pas, après LAS gauche, peuvent être successivement traitées par une sphinctérotomie interne droite additionnelle.

Despite the fact that several theories have attempted to explain the pathogenesis of anal fissure, the origin of the condition has not been fully elucidated. All those theories consider trauma of the anoderm as the common aetiological factor. In addition most reports have shown anal hypertonia [1–5], although there are three studies in which anal pressure has been found to be normal in anal fissure [6–8]. It has been also speculated, that any hypertonia encountered is an epiphenomenon either because of anal dilatation at manometry or defaecation [7], or because of rectal stimulation such as that occuring, when stools enter the rectum [1].

Whether cause or effect, increased anal resting pressure is reduced by almost 50% after either anal dilatation or internal sphincterotomy and that reduction is sufficient to allow fissures to heal in more than 95% of cases

[1–3, 9, 10]. Other authors however claim, that reduction in anal pressure is not a prerequisite for healing, and although lysis of spasm is of benefit because it relieves pain, widening of the anal canal is considered to be the principal reason for healing after anal dilatation or internal sphincterotomy [1]. On the other hand no clear data on anal pressure profiles after unsuccessful therapy are available, and future management is controversial. We prospectively studied 42 patients with fissure-in-ano to elucidate the pathogenesis of the condition and analyze the effects of lateral sphincterotomy on fissure healing and anal motility.

Patients and methods

Patients. Of a series of 51 consecutive patients with anal fissure, 42 (10 nulliparous females) completed the pre- and postoperative tests, and participated in the study. Their age ranged between 26 and 63 years (mean age: 40.5 ± 11.4 SD years). History of anal fissure was present for more than 6 weeks. Digital examination and proctoscopy, using a paediatric proctoscope, were well tolerated and confirmed the diagnosis of fissure-in-ano. Anal fissures were posterior in all but 3 patients, in whom the fissure was located anteriorly. Local applications and laxatives were not allowed for 24 hours prior to anal manometry, which was performed on one occasion preoperatively (approximately 1 week before operation) and on two occasions (1 and 6 months) postoperatively. Manometry was also performed 1 and 6 months after the second operation in those patients, who required reoperation.

All 42 patients underwent subcutaneous left lateral internal sphincterotomy (LAS). The 2 patients with fissure which did not heal after left LAS, along with another 5 patients with recurrent fissure-in-ano after left LAS performed elsewhere, underwent a subcutaneous right LAS. Follow-up ranged between 9 and 36 months (mean: 15 months). Anal manometry was also performed on one occasion in 25 healthy normal subjects (6 nulliparous females) with a mean age of 36.2 ± 10.7 SD years (range: between 20 and 59 years).

Manometric recordings. Two types of flexible polyvinyl probes were used for anorectal manometry. The first one, 3.4 mm in external diametre, consisted of 4 blindly ended tubes (1 mm in internal diameter). Each tube carried a side opening at 5 cm from the tip of the probe. All 4 side openings were located at the same level and arranged radially at 90° around the periphery of the probe. The second probe, 4.2 mm in external diametre, consisted of a central tube (1.5 mm internal diametre) and 4 tubes (1 mm internal diametre) arranged radially at the periphery of the central tube. The central tube ended in an inflatable balloon at the tip of the probe. Each of the 4 peripheral tubes ended blindly and possessed a side opening. The 4 opening were arranged at 0.5 cm intervals along the probe. The dital opening was placed 5 cm central to the tip of the probe. Each tube was continuously perfused with distilled water at 0.6 ml/min by a low compliance perfusion system (Arndorfer, Medical Specialities, Greendale, Wisconsin, USA). A pressure transducer, incorporated in each perfusion line, was connected to an amplifier and a chart recorder (Beckman, Dynograph R-611, Beckman Instruments Inc., Calif., USA).

No bowel preparation was performed, although subjects were allowed to defaecate prior to manometry, if they wished to do so. With the patient in the left lateral position, the first probe was inserted into the rectum after lubrication, so that the level of the side holes was initially located at the lower rectum. The probe was then withdrawn in 30 sec intervals at 0.5 cm stations. Only resting pressure values at the end of the 30 sec plateaux of each side hole at any station were measured. The mean values of the 4 radial openings for each station were calculated. The mean maximal value at rest was considered as the maximal mean resting pressure (MMRP). From the readings during withdrawal of the probe, the length of the anal sphincter was calculated. The same probe was reinserted and the subjects were asked to contract the anus maximally on three occasions at each station. Similarly, the maximal mean squeeze pressure (MMSP) of the anus was calculated. Care was taken that the side openings remained at the anterior, posterior and both lateral positions respectively on insertions and withdrawals of the probe.

Thereafter the second probe was inserted into the anorectum, with the proximal side hole placed at 0.5 cm from the anal verge. Stationary recordings were obtained at rest for 1 hour. From those recordings the following variables were measured: a) MMRP, as the mean value of the 3 highest peaks of the recording site which showed the highest overall amplitude, b) presence, frequency and amplitude of any slow and ultra slow waves and c) frequency duration and extent of transient drops in anal sphincter pressure (sampling phenomenon) [12].

After 1 hour of resting recording, the rectoanal inhibitory reflex (RAIR) was tested by inflating the balloon with air in increments of 10 ml for periods of 60 s and up to 150 ml. In between inflation periods, intervals of 60 s each with the balloon deflated were allowed. The following variables of the RAIR were measured: a) minimal volume at which the reflex was first elicited, b) percentage and absolute value of anal pressure drop during inflation of rectal balloon at 100 cc and c) volume of air in the rectal balloon at which anal pressure failed to recover within the minute the balloon remained inflated [13].

At the end of the test the subjects were asked to attempt a maximal effort to strain on three occasions 30 s apart, and the anal pressures were calculated.

Operations. The patients were operated on under general anaesthesia and placed in the Lloyd-Davis position. Open left LAS was performed according to the technique discribed by Parks [14], modified in that the skin incision was 1 cm long and not sutured after the sphincterotomy. Right LAS was performed through a circumferential incision 1.5 cm long at the level of the intersphincteric groove on the patient's right. The internal sphincter was dissected free from the anoderm and the external sphincter from hour 7 to hour 11 and then divided up to the level of the dentate line. The skin incision was not sutured and a 1 cm thick pack was left for haemostasis within the anal canal, for 5 to 6 hours postoperatively.

Statistical analysis. For statistical analysis the following tests were applied: Fisher's exact test for 2×2 contingency analysis and Wilcoxon test for paired (data pairs from pre- versus postoperative values) and Mann-Whitney U test for unpaired (patients versus controls) values. P values of less than 0.05 were considered to be statistically significant.

Results

After left LAS anal fissure healed in all but 2 patients, within 5 weeks postoperatively. Four patients complained of occasional mucous leak, but none of them presented with frank incontinence to liquid stools. Pain relief was immediate after the operation, although it gradually recovered in those 2 patients with unhealed fissures. Right LAS cured all fissures, and only 1 out of the 7 patients complained of occasional mucous leak per anum after operation.

Patients with fissure-in-ano demonstrated a significantly higher anal resting pressure as compared to controls ($P < 0.0002$). Successful left LAS reduced anal resting pressure to significantly lower levels, which were also lower than those of the controls ($P < 0.001$). Patients who failed to have their fissure healed after left LAS, continued to present a significantly higher resting anal pressure ($P < 0.0008$), which however significantly dropped after successful right LAS as compared to both the preoperative state ($P < 0.0005$) and the controls ($P < 0.0008$). Anal squeeze pressure in fissure patients did not differ from

Table 1. Anal pressures and sphincter activity variables (mean ± SD) in controls and all groups of patients

	Controls	Preop.	Postop.	Failures	Reoperation
Anal pressures (cm H_2O)					
MMRP	81 ± 14	132 ± 21	69 ± 11	127 ± 22	60 ± 9
MMSP	194 ± 43	178 ± 52	182 ± 40	173 ± 31	190 ± 35
MMiStP	13 ± 4	46 ± 16	9 ± 9	61 ± 28	10 ± 8
Slow waves					
Presence (% of time)	68 ± 11	86 ± 6	59 ± 24	91 ± 5	50 ± 18
Presence (*n* of pts)	23	12	17	2	3
Frequency (c/min)	10.9 ± 1.4	9.1 ± 2.3	11.1 ± 3.3	12	11.3 ± 0.3
Ultra slow waves					
Presence (% of time)	6 ± 2	72 ± 9	30 ± 6	79 ± 4	43 ± 5
Presence (*n* of pts)	6	33	19	6	3
Frequency (c/min)	1.8 ± 0.4	1.6 ± 0.3	1.8 ± 0.2	1.7 ± 0.2	1.8 ± 0.1
Amplitude (cm H_2O)	31 ± 9	43 ± 8	28 ± 8	42 ± 3	27 ± 2
Sampling					
n of events/hour	10 ± 2	2 ± 1	5 ± 3	2 ± 1	4 ± 2
% reduction of MMRP	54 ± 12	32 ± 16	59 ± 21	26 ± 4	49 ± 5

MMRP Mean Maximal Resting Pressure; *MMSP* Mean Maximal Squeeze Pressure; *MMiStP* Mean Minimal Pressure during Straining

Table 2. Rectoanal inhibitory reflex in controls and all groups of patients (mean values ± SD)

	Controls	Preop.	Postop.	Failures	Reoperation
Minimal rectal Vol for transient IAS relaxation (ml)	30	33 ± 6	33 ± 5	30	32 ± 4
Minimal rectal Vol for sustained IAS relaxation (ml)	113 ± 16	140 ± 12	100 ± 35	146 ± 14	93 ± 21
Residual IAS pressure at 100 ml of rectal air (%)	43 ± 5	58 ± 8	46 ± 5	64 ± 7	40 ± 6
Residual IAS pressure at 100 ml of rectal air (cm H_2O)	35 ± 8	77 ± 23	32 ± 16	71 ± 18	39 ± 14

normals and remained unchanged after left or even right LAS. Anal pressure on straining was less than 15 cm H_2O in controls. In contrast, although fissure patients presented a significant drop in resting anal pressure during straining, the reduction was significantly less than that observed in controls ($P < 0.0005$). Anal pressure during straining became normal after successful sphincterotomy (Table 1).

Slow waves were present for significantly longer periods in fissure patients as compared to controls ($P < 0.02$). Successful sphincterotomy reduced the presence of slow waves to normal levels ($P < 0.01$). Ultra slow waves were present in significantly more patients (patients: 33 of 42 or 79%, healthy subjects: 6 of 25 or 24%, $P < 0.01$) and for longer periods than in controls ($P < 0.0001$). Amplitude of ultra slow waves was also higher in fissure patients ($P < 0.01$). Successful sphincterotomy significantly reduced: a) the number of patients with ultra slow waves (19 of 40 or 48% $P < 0.02$), b) the percentage of time ultra slow waves were observed ($P < 0.005$) and c) the amplitude of ultra slow waves ($P < 0.001$). However even after successful sphincterotomy, both the number of patients with ultra slow waves and the percentage of time, those waves were present, were still significantly higher than in controls ($P < 0.01$ and $P < 0.0001$ respectively) (Table 1).

Sampling was significantly less common and associated with lower pressure reductions as compared to controls ($P < 0.001$ and $P < 0.0008$ respectively) and was improved after successful sphincterotomy although the frequency did not reach that seen in the controls (Table 1). Duration of transient sphincter relaxations did not differ between the groups (1.4 ± 0.9 SD min at an average).

All patients and controls showed a drop in anal pressure after rectal distension with approximately 30 ml of air. However, the drop was significantly less (absolute value and percentage) after rectal distension with 100 ml of air in fissure patients and those with non healed fissure after sphincterotomy as compared to controls ($P < 0.0004$ and $P < 0.001$ respectively). Successful sphincterotomy normalized the anal response to rectal balloon distension with 100 ml of air. Furthermore, the rectal volume at which the anal sphincter failed to recover, while the balloon remained inflated, was significantly higher in fissure patients as compared to controls ($P < 0.005$), but was normalized after successful sphincterotomy (Table 2).

Discussion

The results of the present study confirmed that patients with anal fissure have anal sphincter hypertonia. This is in agreement with previous reports [1–5]. We also studied other aspects of internal sphincter function and confirmed further abnormalities in anal fissure. For example, increased presence and amplitude of ultra slow waves, reduced frequency of sampling and a defective response to rectal distension were observed. The above manometric abnormalities are unlikely to be the result of spasm secondary to anal distension [1], because the probe diameter was small (~4 mm) and the findings were present for approximately 60 min after the probe introduction. A similar view has been expressed by Gibbons and Read [4], who in addition, attribute the normal anal resting pressure observed in anal fissure by other authors [6, 7, 15] to

methodology related factors. Gibbons and Read [4] make the assumption that increased anal resting pressure is a prerequisite for anal fissure not to self heal, because such anal hypertonia results in low perfusion and ischaemia of the overlying anoderm. In addition, for anatomical reasons, posterior and anterior positions of anoderm are less well supplied by arterial blood, and this may explain the predisposition of fissures to develop either posteriorly or anteriorly. Furthermore, they speculate that pain in anal fissure is of ischaemic origin, while lack of epidermal regrowth and of basal granulation in this condition may also be the result of ischaemia. Contradictory to the speculation that anal hypertonia is secondary to spasm induced by defaecation [1, 7], is the observation of the present study, according to which fissure patients show a significant drop in anal pressure during straining, although the pressure reduction is less than in controls. Furthermore, distention of the rectum stimulates the inhibitory rectoanal reflex and anal resting pressure is reduced in fissure patients, although that reduction is not as deep as that observed in normals. Therefore, speculation that anal hypertonia in fissure-in-ano may be a pathological response to stools entering and distending the rectum [1] is rather unsound. The increased internal anal sphincter activity, as expressed by the reduced frequency of sampling and by the lower percentage of pressure reduction at sampling, may add a further explanation, apart from that of pain, for constipation and hard stools observed in patients with fissure-in-ano.

Subcutaneous left lateral internal sphincterotomy heals the anal fissures in percentages higher than 90 percent [5, 9, 16–19]. This is attributed to the drop in anal resting pressure, which according to some authors relieves spasm and improves drainage [19], or according to others abolishes ischaemia of the anoderm [4].

Successful sphincterotomy, not only significantly reduced anal resting pressure, but also normalized the anal sphincter relaxation during straining and the anal sphincter response to rectal distension. Although some variables, such as the time of presence of ultra slow waves and the frequency of sampling, improved significantly after sphincterotomy, they did not reach the control levels. Those observations signify that increased activity is a primary disorder of internal sphincter function in anal fissure and not an epiphenomenon of the condition. Anal dilatation by the haemostatic pack is rather unlikely to contribute to the reduction in anal resting tone, mainly because probes of 1 cm in diametre do not significantly affect the anal resting pressure [4].

Failure of the anal fissure to heal after left LAS was associated with manometric findings similar to those observed preoperatively. Presumably, sphincterotomy to those patients was inadequate and failed to improve anal sphincter function to a level that would allow anal fissure to heal. Inadequacy of sphincterotomy might have been the result of apposition of the cut edges of the sphincter and healing back to continuity. Right LAS with minimal mobilization of the internal sphincter from adjacent anoderm and external sphincter healed all recurrent fissures and was associated with manometric findings similar to those observed after successful left LAS. No frank incontinence in liquid stools was observed after bilateral LAS.

In conclusion, increased internal anal sphincter activity is a major factor in anal fissure pathogenesis. Successful left internal sphincterotomy heals the fissure and improves the manometric performance of the anal sphincter. Recurrent fissures after surgery can be successfully treated by additional right internal sphincterotomy.

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