Hypertrophy of the external anal sphincter in haemorrhoids: a histometric study

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SUMMARY Biopsies of the external anal sphincter taken from 24 male patients with haemorrhoids were subjected to examination using histochemical techniques. Fibre size (type 1 mean: 33 μm ± 11·0 SD and type 2 mean: 47·9 μm ± 15·0 SD) was increased when compared with control subjects. The distribution of muscle fibre types in these patients showed markedly greater type 1 fibre predominance (92%) than in control subjects. These abnormalities of the external sphincter did not show any clear relationship with age, degree of haemorrhoidal change, length of history of haemorrhoids, history of straining or of constipation, or perineal descent; however, increasing length of history up to 10 years was associated with increasing type 1 fibre predominance. We suggest that the external anal sphincter in patients with haemorrhoids is in a state of increased tonic contraction, which causes work induced muscle hypertrophy and may contribute to increased resting pressure in the anal canal in patients with haemorrhoids.

The histology of the external anal sphincter muscle and of the pelvic floor musculature has been investigated, using enzyme histochemical techniques, in normal subjects,1 in patients with anorectal incontinence and in rectal prolapse.2-3 A history of straining during defaecation is a common feature of patients with idiopathic anorectal incontinence and in patients with rectal prolapse, and it therefore seems of interest to extend these investigations to patients with haemorrhoids. In previous investigations of this disorder attention has been paid to changes in anal canal pressure, and to abnormalities in the internal anal sphincter,4-6 but little attention has been paid to the external anal sphincter muscle. In this paper we describe histological abnormalities in the anal sphincter muscle in a series of patients with haemorrhoids.

Methods

Patients
Twenty-five consecutive patients undergoing haemorrhoidectomy at St Mark's Hospital were studied. Twenty-four of these 25 patients were male; the single female patient was excluded, because the histological differences in the anal sphincter musculature found in normal men and women1 seemed likely to lead to difficulties in the statistical analysis. The 24 men ranged in age from 27 to 75 years (mean: 47 years) and their haemorrhoids had been symptomatic from one to 25 years (mean: 10 years). The degree of haemorrhoids was classified into three groups; first (no prolapse), second (prolapse with spontaneous reduction), and third (prolapse requiring replacement). Two patients had first degree haemorrhoids, nine had second degree haemorrhoids, and 13 had third degree haemorrhoids. Twenty-two (92%) had experienced bleeding, and 12 (50%) gave a history of pain. There was a history of constipation, as defined by a bowel frequency of less than once a day, in 12 (50%) and of excessive straining at stool for many years in 19 patients (79%). Seven patients (29%) showed perineal descent—that is, descent of the perineum below the ischiorectal tuberosity during voluntary straining.

In each of these patients muscle biopsies were obtained from the left posterior portion of the external anal sphincter muscle during haemorrhoidectomy.

Similar biopsies of the external sphincter muscle, taken shortly after death, and examined by an identical technique in the same laboratory were available from eight normal subjects, aged 40 to 76 years (mean: 54 years) and these were used as

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controls. The morphometric and histological features of these normal external anal sphincter muscle biopsies have been reported previously. As far as could be ascertained from the medical records these patients had been free of anorectal disorders.

**Techniques**

Biopsies of the external anal sphincter obtained during haemorrhoidectomy were snap-frozen in isopentane cooled in liquid nitrogen and a series of consecutive transverse sections 8 μm thick was cut from each biopsy in a cryostat. These sections were prepared for light-microscopy using haematoxylin and eosin, and modified Gomori trichrome stains. In addition, a standard series of enzyme histochemical methods was used, including nicotine adenine dinucleotide tetrazolium reductase (NADH tr) and myosin adenosine triphosphatase (ATPase) preincubated at pH 9.4 and 4.3 (7). Fibre typing (Table 1) was performed using the ATPase preparations, and the proportion of type 1 and type 2 fibres, calculated as a percentage of the total number of fibres in each biopsy, was expressed as the dominant fibre type. The ATPase preparation, preincubated at pH 4.3, was used for fibre typing because it gives a more definite and more constant fibre type differentiation than the more alkaline preincubations.

The histochemical classification of muscle fibres in striated muscle can be correlated with the physiological characteristics of these fibres. Type 1 fibres, dependent on oxidative metabolic pathways are adapted to maintain tonic contraction, and type 2 fibres, dependent on non-oxidative metabolism, are adapted to brief, phasic contraction (Table 1). The external anal sphincter, as would be expected in a muscle which is maintained in a state of constant reflex contraction showed marked type 1 fibre predominance (74% in men).

The lesser diameters of all the fibres of each histochemical type were measured in sections of each biopsy stained with ATPase preincubated at pH 4.3 using an eye-piece micrometer. The lesser diameter (the maximum diameter across the lesser aspect of each muscle fibre) was chosen because it overcomes the difficulty of error introduced by obliquity in the plane of section of individual fibres. The means and standard deviations of these measurements of type 1 and type 2 fibre diameters were calculated, for each muscle, and histograms of type 1 and type 2 fibre diameter were plotted. In addition, changes in morphology of individual fibres and other histological abnormalities were noted.

**Results**

**Histometric Findings**

In patients with haemorrhoids the mean diameters of type 1 and type 2 fibres were 33 μm (SD. 11.0) and 47.9 μm (SD. 15.0), respectively. This represents hypertrophy of type 1 and type 2 fibres of 57% and 60% respectively (Table 2), compared with controls (p < 0.01). Type 2 fibres were significantly larger (Fig. 1) than type 1 fibres (p < 0.001). No difference in fibre diameter was found with increasing age, with degree of haemorrhoids, with a history of constipation, or of straining at stool, or with perineal descent. Further, there was no increase in muscle fibre diameter with increasing length of history of haemorrhoids.

**Table 2  Muscle fibre diameter and % type 1 fibre predominance**

<table>
<thead>
<tr>
<th>Fibre type</th>
<th>Diameter (μm)</th>
<th>% difference</th>
<th>Significance of difference</th>
<th>% Type 1 fibre predominance</th>
<th>Significance of difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean±SD</td>
<td></td>
<td></td>
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<tr>
<td>Type 1</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Control</td>
<td>21±10.8</td>
<td>57</td>
<td>p&lt;0.01</td>
<td>74</td>
<td></td>
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<tr>
<td>(1407)*</td>
<td></td>
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<tr>
<td>Haemorrhoids</td>
<td>33±11.0</td>
<td></td>
<td></td>
<td>92</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>(23644)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Type 2</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>30±15.6</td>
<td>60</td>
<td>p&lt;0.01</td>
<td></td>
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<tr>
<td>(1174)</td>
<td></td>
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</tr>
<tr>
<td>Haemorrhoids</td>
<td>47.9±15.0</td>
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<td>(2181)</td>
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*Numbers in parentheses indicate total number of fibres measured.*
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Type 1 fibre predominance (Fig. 1, Table 2) was significantly greater (92%) in patients with haemorrhoids than in the control subjects (74%) (p < 0.001). In the group of patients who had a history of haemorrhoids for less than 10 years, type 1 fibre predominance increased with increasing length of history of haemorrhoids (r: 0.81, t: 5.43, p < 0.001); however, in those patients whose haemorrhoids had been present for longer than 10 years, type 1 fibre predominance showed no additional increase with increasing length of history (Fig. 2).

There was no difference in the degree of type 1 predominance with increasing age, with degree of haemorrhoids, with a history of constipation, or of straining at stool, or with perineal descent. Histograms of type 1 fibre diameter showed a normal distribution in all cases, but histograms of type 2 fibre diameter showed multiple peaks with increased range of diameter in three of the 24 patients.

HISTOLOGICAL FINDINGS

Central nucleation was seen in more than 3% of the fibres in 19 of 24 biopsies, but only two biopsies showed more than 10% of such fibres. In normal muscle central nucleation is found in less than 3% of fibres. Necrotic fibres, granular fibres, and split fibres were rare and no features of denervation or reinnervation were seen. Increased fibrous tissue was found in only two of the 24 cases, the same cases in which there was more than 10% central nucleation.

Discussion

The external anal sphincter is in a state of continuous tonic contraction in normal subjects. Skeletal muscles which have a predominantly tonic postural function—for example, soleus and tibialis anterior—show a high proportion of type 1 fibres. Beersiek et al. found that the external anal sphincter muscles of normal subjects showed type 1 fibre predominance (74%), as would be expected in a muscle which was in a state of continuous partial contraction. The external anal sphincter muscle biopsies in our patients with haemorrhoids showed increased type 1 fibre predominance and hypertrophy of
both type 1 and type 2 muscle fibres (Table 2). The degree of type 1 fibre predominance increased with increasing length of history of haemorrhoids, up to 10 years. These observations suggest that the external anal sphincter muscle in patients with haemorrhoids may be in a state of more marked tonic contraction than normal. Increased type 1 fibre predominance occurs in skeletal muscles in a number of neuromuscular disorders,7 but these were excluded on clinical and histological grounds in our patients.

The degree of fibre hypertrophy found in our patients' external anal sphincter muscles was less than we have reported in anorectal incontinence,1,8 and none of the extensive fibrosis and loss of muscle fibres found in the incontinent patients was noted in these patients with haemorrhoids.

Muscle hypertrophy is due to increased work load. This can be caused either by partial loss of muscle fibres, as occurs in idiopathic anorectal incontinence,1 those fibres which remain carrying a heavier burden, or by an actual increase in work required of the muscle as a whole. Our results strongly suggest that the second of these alternatives is important in haemorrhoids.

It has been shown that patients with haemorrhoids have a high resting pressure in the anal canal, and an abnormal pattern of motility of the internal anal sphincter muscle,4,5 and it has been suggested that this increase in pressure is due to increased activity of the internal anal sphincter muscle complex.6 However, the resting pressure in the anal canal is not produced by the internal anal sphincter muscle alone. Frenckner and von Euler12 have suggested that 85% of the resting pressure in the anal canal in normal subjects is contributed by the internal sphincter, but even this figure may be an underestimate of the role of the internal sphincter, as sudden rectal distension results in a decrease in the anal canal pressure of as much as 60%. Duthie et al.13 have suggested that the external sphincter contributes more to the pressure in the anal canal when a bolus is present within the canal.

The cause of an increased work load of the external anal sphincter in patients with haemorrhoids could be increased reflex stimulation due to the presence of the haemorrhoids within the anal canal or to the effort required to prevent haemorrhoidal prolapse. Either of these phenomena could thus explain the association of a high anal pressure in patients with haemorrhoids with hypertrophy in the external sphincter muscle.

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References
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