Internal sphincter and haemorrhoids: a pathological study

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SUMMARY Histological examination of the lower margin of the internal sphincter has demonstrated an increased amount of fibrous tissue in patients with haemorrhoids compared with controls. This finding may be of relevance to the treatment of haemorrhoids by maximal anal dilatation.

Interest in the internal sphincter in patients with haemorrhoids has been stimulated by the observation that maximal anal dilatation will relieve symptoms. It seems possible that there is a degree of stenosis in the anal canal which is relieved by dilatation. Recent studies (Hancock and Smith, 1975; Hancock, 1977) have demonstrated a motor abnormality of the sphincter which was abolished by dilatation, but this was not present in every case so there may be other factors that produce a stenosis. Lord (1969), who popularised the dilatation treatment, suggested that there was a pecten band consisting of fibrous tissue in the anal submucosa that may be a primary cause of haemorrhoids.

Since the first detailed description of the pecten band (Miles, 1919) its existence has been disputed and it is now generally accepted as a result of studies at operation that the pecten band is in fact the internal sphincter.

Because there have been no pathological studies of the internal sphincter in patients with haemorrhoids, we examined biopsies of the internal anal sphincter from presumed normal subjects and patients with haemorrhoids.

Material and methods

Specimens of internal sphincter were taken from 14 patients (11 males, 3 females) during the operation of haemorrhoidectomy. A small piece, about 3 mm³, was removed from the lower left lateral margin of the sphincter. Only large vascular haemorrhoids were included in the study.

A similar sized sample was taken in the same way from 'normal' internal sphincter obtained from postmortem subjects (20 males, 5 females).

The specimens were stained by haematoxylin and eosin, van Gieson, and Masson trichrome techniques and were classified into one of three grades according to the amount of fibrous tissue associated with the smooth muscle as follows:

Grade I: Fibrous tissue either absent or present as thin strands running parallel to the muscle fibres (Fig. 1).

Grade II: Fibrous septa abundant and more conspicuous, occupying the entire interstitial spaces (Fig. 2).

Grade III: Dense fibrous tissue in two planes, running parallel to and intersecting groups of muscle fibres (Fig. 3).

The mean age of the patients was 50 years and all had large prolapsing haemorrhoids. In 10 patients a preoperative anal pressure profile was measured (Hancock, 1976). The mean age of the controls was 62.5 years and it was not known if any of these subjects had suffered with haemorrhoids during life.

Results


<table>
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<tr>
<th>Distribution of fibrous tissue</th>
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<tr>
<td>Grade I</td>
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<td>Controls (n = 25)</td>
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<td>Haemorrhoids (n = 14)</td>
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χ² = 14.6 p < 0.001
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Fig. 1  Empty interstitial spaces or thin strands of fibrous tissue. (van Gieson × 55)

Fig. 2  Abundant fibrous tissue encircling groups of muscle fibres. (van Gieson × 148)
There was no apparent association between the amount of fibrous tissue and the size or duration of haemorrhoids nor with sphincter activity as estimated by measurement of the preoperative anal pressure.

Discussion

There appears to be an increased amount of fibrous tissue in the internal sphincter of some patients with haemorrhoids. This is unlikely to be due to an ageing process because the mean age of the controls was greater than that of the patients with haemorrhoids. A possible cause of fibrosis is long-standing vascular congestion, or it could be as a result of repeated traction on the mucosal suspensory ligament which passes as fibromuscular bundles through the internal sphincter.

It might be thought that disruption of fibrous tissue by anal sphincter stretching would lead to increased fibrous tissue formation but the longer term studies after dilatation do not suggest that this happens (Hancock, 1977), so the motor abnormality of the sphincter may be the more important cause of stenosis in some patients with haemorrhoids. This finding of increased fibrous tissue in the internal sphincter, however, does lend support to the idea of treating some patients by dilatation.

We thank all consultant surgeons for allowing us to study many of their patients, and the technicians of the histopathology laboratory of the University Hospital of South Manchester for their help and co-operation.

References