INTERNAL ANAL SPHINCTER DERANGEMENT WITH ANAL FISSURES

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Rectosphincteric manometric studies in patients with anal fissures demonstrated an abnormal internal anal sphincter reflex in response to rectal distention. This abnormal reflex response, characterized by an overshoot contraction immediately after a normal relaxation, was present in all 7 patients with fissures, but not in 17 subjects without anal disease. In 3 patients studied after treatment, the response returned to normal. These studies suggest that reflexly stimulated internal sphincter spasm plays a role in the pathogenesis of anal fissures, and that surgical approaches should be directed to the internal, rather than the external anal sphincter.

Fissure in ano is a common proctological problem about which little physiological information is available. Although a variety of operations has been devised empirically to treat chronic anal fissures, more understanding of basic pathophysiology is needed for rational treatment. To investigate the possible motor disorders, the specific roles of the internal and external anal sphincters in this motor disorder, and the physiological effects of sphincterotomy, rectosphincteric manometric studies were performed on patients with proved fissure in ano and compared with normal subjects.

Materials and Methods

Seven patients with proved fissure in ano were studied prior to medical or surgical therapy, and results were compared with those of 10 normal subjects and 7 patients with "irritable bowel syndrome." Five patients had the diagnosis of fissure in ano established by anoscopy prior to the study. In 2 cases, the patients underwent rectosphincteric evaluation as participants in a drug study and had an abnormal response indicating the presence of fissures. Anoscopy subsequently demonstrated fissures in both. Three patients were restudied after successful treatment: 1 surgically and 2 medically. One patient had paralysis of the external sphincter due to a spinal cord lesion, and another patient was studied with 10 mg intravenously of diazepam (Valium) which abolished measurable external sphincter reflexes, but did not interfere with internal sphincter responses.

Simultaneous pressure recordings were obtained from the internal and external anal sphincters using a double balloon device with two separate compartments formed by a molded latex balloon tied around a hollow steel cylinder. This device was placed in the anal canal and each balloon was distended with 10 cc of air so that the internal balloon was surrounded by the internal sphincter and the external balloon by the subcutaneous bundle of the external sphincter. A third balloon was inserted into the rectum through a hollow core in the recording device (fig. 1). All three compartments communicated by polyethylene tubing with pressure transducers (Sanborn Model 267B, Sanborn, Cambridge, Mass.). The pressure tracings were recorded at a speed of 1 mm per sec on three channels of a direct writing recorder (Sanborn Model 964).

Each subject was studied in the left lateral decubitus position. The rectal balloon was transiently distended with air to stimulate sphincteric reflexes. Beginning with 50 cc of air,
distending volumes were decreased in 10 cc steps in order to establish a threshold for the reflex response. Threshold is defined as the smallest volume of air for rectal distention that will elicit a detectable sphincteric response.

Results

In the patients with fissure in ano, the “resting” pressure within the internal sphincter balloon ranged from 75 to 100 mm Hg as compared with 25 to 75 mm Hg in the control group.

The normal response to rectal distention with 50 cc of air was an abrupt relaxation of the internal sphincter (fig. 2). This is manifested by a precipitous decrease in pressure within the internal sphincter balloon. The average pressure decrease was 25 mm Hg and the average duration of relaxation was 15 sec, during which time there was a slow return to the base line pressure. In the external sphincter balloon, a brief, rapid contraction manifested by a rise in pressure coincided with the relaxation phase in the internal sphincter. This type of response was obtained in all subjects without anal disease (normal subjects and patients with irritable bowel syndrome).

In all patients with fissure in ano, rectal distention produced the expected initial internal sphincter relaxation. However, in contrast to the normal pattern, this relaxation was followed by a marked and often prolonged contraction above the initial base line (fig. 3). This characteristic overshoot response was most prominent with the largest volumes (50 cc) of rectal distention employed and decreased progressively with smaller distending volumes, eventually disappearing at 20- to 30-cc volumes. The table compares responses of the patients with fissures and the subjects without anorectal disease. Since the response of normal volunteers and patients with irritable bowel syndrome were similar, these two groups are combined in a single group without anal disease and will be referred to as the control group. Although the overshoot phenomenon sometimes appeared in the control group, patients with fissures differed from controls in the following ways (table 1): (1) Resting pressure in the internal sphincter balloon was higher in patients with fissures. (2) Ninety per cent of suprathreshold distentions evoked the overshoot pattern in the fissure group as compared with 26% in the control group. Five patients with fissures demonstrated

![Fig. 1. Schematic diagram of pressure-sensing device. The internal sphincter surrounds the internal sphincter balloon. The external sphincter balloon is surrounded by the subcutaneous bundle of the external sphincter and helps to anchor the device in place. Rectosphincteric reflexes are initiated by transient distention of the rectal balloon. (Printed with permission of the Bulletin of the Johns Hopkins Hospital.)](image)

![Fig. 2. Normal internal sphincter response. Internal sphincter relaxation induced by transient rectal distention (arrow) is recorded as a pressure decrease in the internal balloon. External sphincter contraction is recorded as a pressure rise in the external balloon.](image)
an overshoot response 100% of the time with suprathreshold distention, the other 2 patients 66% of the time. (3) Amplitude of overshoot contraction was higher in the fissure group (fig. 4). (4) Threshold for stimulation of overshoot was lower in the fissure group. In all 3 patients studied after successful treatment of fissures, the overshoot phenomenon was no longer present, and the reflex responses had returned to normal.

Intravenous administration of 10 mg of diazepam in 1 patient with fissures completely abolished the external sphincter response. The internal sphincter response remained unchanged and continued to demonstrate the overshoot pattern. A 2nd patient with an anal fissure had, in addition, a spinal cord lesion which resulted in complete paralysis of the external anal sphincter. In this patient also, the internal sphincter responded with an initial normal relaxation followed by a rebound overshoot.

**Discussion**

Fissure in ano is a common proctological problem resulting in bleeding, pruritus, constipation, and anal pain typically induced or aggravated by defecation. It was originally thought that anal fissures were situated over the subcutaneous portion of the external sphincter and that the symptoms of fissures were due mainly to spasm of the external sphincter. Division of this muscle was considered essential to the therapy of fissures. 2-7 Eisenhammer, 8, 9 and Goligher et al. 10 suggested that the internal, not the external sphincter, was the

**TABLE 1. Average values (bold numbers) and ranges (parentheses) of motility data for patients with fissure in ano and control subjects**

<table>
<thead>
<tr>
<th></th>
<th>FISSURE</th>
<th>CONTROL</th>
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<tbody>
<tr>
<td>Internal Sphincter</td>
<td>85</td>
<td>50</td>
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<tr>
<td>&quot;Resting&quot; pressure (mm Hg)</td>
<td>(75-100)</td>
<td>(55-75)</td>
</tr>
<tr>
<td>Percent of suprathreshold distensions overshoot</td>
<td>90.5</td>
<td>26.4</td>
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<tr>
<td>Amplitude of overshoot with 50 cc rectal dist (mm Hg)</td>
<td>27</td>
<td>11</td>
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<tr>
<td>Threshold for overshoot (mm Hg)</td>
<td>(20-30)</td>
<td>(9.15)</td>
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<tr>
<td>Duration overshoot with 50 cc distention (sec.)</td>
<td>50</td>
<td>48.5</td>
</tr>
<tr>
<td>Threshold for relaxation (mm Hg)</td>
<td>12</td>
<td>13.7</td>
</tr>
<tr>
<td>Amplitude external sphincter contraction (mm Hg)</td>
<td>23</td>
<td>24</td>
</tr>
<tr>
<td>Duration external sphincter contraction (sec.)</td>
<td>8</td>
<td>5</td>
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**Fig. 4.** Scattergram depicting average overshoot contraction in each subject with 50-cc rectal distention.

**Fig. 3.** Rebound internal sphincter contraction in 2 patients (a and b) with anal fissures. Rectal distention (arrow) induces a normal internal sphincter relaxation which is followed by a prolonged overshoot contraction of high amplitude. The external sphincter contracts during the early phase of internal sphincter relaxation.
important muscle, and that fissures lay over the lower third of this muscle. They contended that inadvertent internal sphincterotomy probably accounted for the success of earlier surgical procedures.

Duthie and Bennett,\textsuperscript{11} using a manometric pull through technique with open end water-filled tubes, found no difference in resting sphincter pressure between preoperative patients and controls. They concluded that anal sphincter spasm was not a consistent accompaniment of fissure in ano when the anal canal was undisturbed by distention, and that spasm might be important only during and immediately after defecation. In our studies, the resting pressure within the internal sphincter balloon was about twice that of the control subjects. The higher pressures in anal fissures demonstrated by our technique may be attributed to the fact that anal distention by our balloon system was greater than distention by the catheters employed by these workers.

That the external anal sphincter is not significantly involved in the pathophysiology of anal fissures is suggested by several of our findings. In the first place, there was no significant difference between controls and patients with fissures in the duration, amplitude, or configuration of the external sphincter response to rectal distention. Secondly, in normal subjects, as in patients with fissures, the external sphincter contraction takes place during the early phase of internal sphincter relaxation (fig. 3). At the time of the overshoot contraction, no pressure changes occur in the external sphincter balloon. It might be argued that the deeper external sphincter bundles which surround the internal sphincter are responsible for the rebound overshoot. However, the overshoot was present in the face of complete absence of external sphincter responses in 2 cases.\textsuperscript{12} Since patients with fissures have a normal internal sphincter relaxation preceding the overshoot, there is probably no loss of ability of this sphincter to "harmoniously dilate" as had been recently suggested by Arnous and Denis.\textsuperscript{13}

Recently, a new surgical approach to the treatment of anal fissures has been described: lateral subcutaneous internal sphincterotomy.\textsuperscript{14-16} This procedure, which can be performed under local anesthesia, involves transecting only the internal sphincter peripherally without touching the anal mucosa or the fissure itself. After internal sphincterotomy, the fissure usually heals within 3 weeks with little postoperative discomfort or complication, and a reported recurrence rate of only 3%.\textsuperscript{4} Medical treatment as well as other operative procedures, such as routine internal sphincterotomy or brusk dilation, also may induce healing of fissures by abolishing sphincter spasm.

The technique clearly distinguishes between patients with fissures and normal subjects or patients with irritable bowel syndrome, since a clean separation without overlap existed on the basis of internal sphincter resting pressure and amplitude of overshoot contraction (table 1, fig. 4).

These studies coupled with the work of Duthie and Bennett\textsuperscript{11} suggest that resting pressure in the unstimulated sphincter is normal, but that sphincter spasm may result from rectal stimulation such as may occur when stool enters the rectum or during defecation.

REFERENCES

9. Eisenhammer S: The internal anal sphincter: its


