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Anismus in Chronic Constipation

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Among patients complaining of constipation, a group can be defined in which there is slow whole gut transit shown by retention of radiopaque markers but a rectum and colon of normal width judged by measurements of barium enema radiographs compared with control observations. It is not known whether their symptoms are due to an abnormality of colonic motility or to a failure of the defecatory mechanism. Defecation was simulated experimentally in a group of these patients by asking them to expel a water-filled rectal balloon. The constipated patients were not able to expel the balloon, whereas normal subjects could do so. Electromyography of the striated pelvic floor muscles during attempts at expulsion of the balloon in the constipated patients showed failure of the normal inhibition of resting activity. Failure of external anal sphincter relaxation on attempted defecation may contribute to the symptoms of some patients who complain of constipation.

In 1969 Hinton et al described a new method of measuring total intestinal transit time using radiopaque markers (1). Normal subjects were found to pass 80% or more of the ingested markers within five days. Among patients who complained of constipation, some had a normal transit time while in others transit was delayed. Patients with slow transit could be divided into those with a normal barium enema and those with megarectum and megacolon. The group with normal barium enema who retained more than 80% of the markers at five days have been described as having idiopathic slow-transit constipation (2). These patients have proved a difficult problem in management and the cause of their disorder is not known. In particular, we do not know whether their symptoms are due to a failure of colonic propulsion, to a disorder of defecation, or to both factors.

MATERIALS AND METHODS

Patients Studied. We have studied 15 women with a diagnosis of slow-transit constipation. Their ages, at the time of presentation, ranged from 16 to 36 years. Most were in their early 20s (Table 1) and had been symptomatic for at least 10 years. This severe form of constipation is unusual and during the period 1969–1979, 26 patients, all of whom were women, were seen with slow intestinal transit rate and normal barium enema, out of a series of 267 patients referred with a primary complaint of constipation to this specialist hospital.

Transit studies were performed using radiopaque markers (2). One patient recorded a bowel movement during the study and was found to have passed 10 of 20 markers, the other 14 patients still had all 20 markers present after five days.

Measurements were made of rectal area and of rectal and colonic width as described elsewhere (3) on radiographs obtained using a standard air-contrast barium enema technique. The measurements were compared with those obtained in 50 examinations reported as normal by the same technique of patients without a bowel disturbance, inflammatory bowel disease, or diverticular disease and performed for unexplained rectal bleeding or polyp follow-up. In every case, measurements made in the patients with constipation fell within the normal range and, in particular, the width of the colon at the pelvic brim fell within the range 3.2–6.0 cm, thus excluding megarectum or megacolon (3).

In every patient the resting pressure in the anal canal was measured by a stationary pull-through technique at 1-cm intervals using a water-filled microballoon, 4 mm in external diameter, connected by rigid tube to a pressure transducer. The high-pressure zone was of normal length (3.5 cm) and the maximal pressure was within the normal range (60–100 cm of water). The rectoanal inhibitory reflex was demonstrated as present in each patient by a

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TABLE 1. DETAILS OF PATIENTS STUDIED

Patient	Sex	Age	Length of history (yr)	Previous treatments	Transit study (shapes left at 5 days)	Reported interval between spontaneous bowel actions
1 DH	F	20	15	Laxatives, enemas, anal stretch	20	3 weeks
2 SM	F	36	30	Laxatives, enemas	20	10 days
3 DT	F	18	5	Laxatives, enemas, behavioral therapy	20	6 weeks
4 JP	F	16	2	Laxatives, enemas	20	10 days
5 MR	F	22	18	Laxatives	20	7 days
6 SS	F	20	4	Laxatives, enemas, sigmoid colectomy, anorectal myectomy, behavioral therapy	20	*
7 JD	F	21	10	Laxatives, enemas, anal stretch	20	10 days
8 PW	F	26	14	Laxatives, enemas, subtotal colectomy	20	7 days
9 HA	F	26	11	Laxatives, anal stretch	20	4 weeks
10 SB	F	24	7	Left hemicolectomy, laxatives, anal stretch	20	5 weeks
11 LS	F	24	7	Laxatives	20	7 days
12 MG	F	19	15	Laxatives, enemas, behavioral therapy	20	12 days
13 SG	F	20	5	Laxatives	10	7 days
14 PG	F	24	20	Laxatives, enemas, left hemicolectomy, anorectal myectomy	20	5 weeks
15 RW	F	24	10	Laxatives, anal stretch	20	8 days

*No spontaneous bowel actions.

reduction of the resting anal pressure at its maximum point in the anal canal during distension of the rectum with an air-filled balloon at increments of 50 ml.

Biochemical tests did not reveal any cause for the constipation. Each patient had been instructed to take a high-fiber diet, usually with a bran supplement, but without benefit.

Control Subjects. Control subjects for the balloon expulsion studies were 15 women attending for colonoscopy. None had anorectal disease or a history of constipation. They were being investigated as part of another research project in which healthy subjects had been screened for occult blood in the stool. Their ages ranged from 19 to 45.

Methods. The balloon used for this study was a small child's party balloon with a deflated diameter of 3 cm. This balloon was mounted on a firm plastic catheter (internal diameter 0.25 cm) so as to produce an eliptical shape when distended. The plastic catheter terminated in a three-way tap for the introduction of water and was tied via a pulley system to a small tray holding weights.

When inflated with 50 ml of water, the dimensions of the balloon were 5.0 × 3.5 cm. This resulted in a firm bolus which was too large to pass through the normal anal canal unless compressed and elongated during straining efforts.

Thirty patients were studied by introducing the well-lubricated and deflated balloon into the lower rectum while the patient lay in the left lateral position. The balloon was then inflated with 50 ml of water and the patient asked to expel it as if during normal defecation. If this proved impossible, weights were applied via the pulley and raised by increments of 50 g during repeated straining efforts until expulsion occurred. The position of the pulley was adjusted so that the force was applied in the long axis of the anal canal. The weight needed to withdraw the balloon during straining was compared with that needed to withdraw the balloon when the patients were relaxed.

RESULTS

None of the 15 patients with slow-transit constipation was able to expel the balloon; all the control subjects were able to do so. The ability of the constipated patients to expel the balloon was not affected by posture, and they were unable to expel it either in the sitting or squatting position. In the normal subjects (Figure 1) an average weight of 700 g was needed to

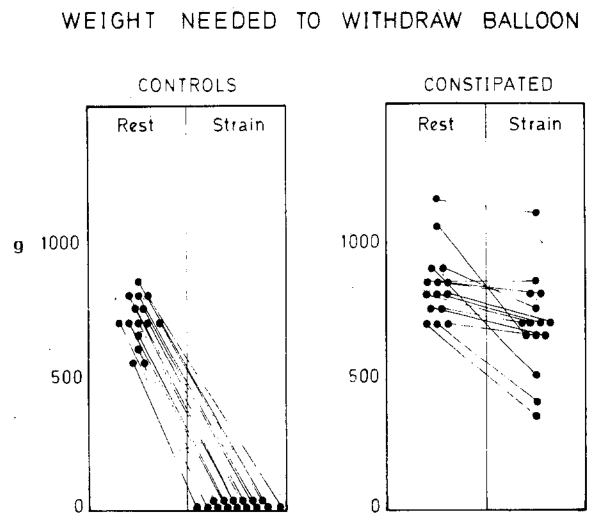

Fig 1. The weight applied via a pulley needed to withdraw a 50-cc water-filled balloon from the rectum at rest and during attempted defecation.

TABLE 2. MEAN ANAL CANAL PRESSURES (± SEM) IN CONSTIPATED PATIENTS AT REST AND ON ATTEMPTED EXPULSION OF AN INTRARECTAL BALLOON

Distance from anal verge (cm)	Pressure (cm H_2O)	
	Resting	Straining
1	51.6 ± 12.5	136.6 ± 22.2
2	89.5 ± 12.8	142.0 ± 18.1
3	53.5 ± 11.7	94.0 ± 10.7
4	23.2 ± 6.2	72.2 ± 11.8
Rectum	0	70.1 ± 8.6

withdraw the balloon when relaxed. When they strained, no weight was required and the balloon was expelled spontaneously. In the constipated patients, the average weight needed to withdraw the balloon at rest was 830 g, but when they strained an average force of 690 g was still required to withdraw the balloon. This value approximated to that required to withdraw the balloon in control subjects at rest.

The findings in the constipated group, particularly the tension needed to withdraw the balloon during straining, suggested that there was a failure of relaxation of either the internal or external anal sphincters, or that inadequate straining efforts were being made so that the intrarectal pressure was not high enough to expel the balloon. Further observations were therefore made in the constipated patients to investigate the problem.

Measurement of Intrarectal and Anal Canal Pressures During Straining. To determine whether the constipated subjects were making genuine straining efforts, a pressure probe was placed within the rectal balloon to record the rise in intraabdominal pressure during attempts at its expulsion without the weights being applied. The mean rise in intrarectal pressure during straining in the constipated subjects was 70.0 ± 8.6 cm H_2O (SEM), confirming that intraabdominal pressure had increased and genuine straining efforts were being made. The range of intrarectal pressure rise was 40–130 cm H_2O. Pressure recordings were also made at rest in the anal canal at 1, 2, 3, and 4 cm from the anal verge in the anal canal using a miniature water-filled balloon connected to a pressure transducer. These readings were compared with those during maximal voluntary contraction of the anal sphincters and on attempted expulsion of the balloon. Comparison of the results during resting and attempted defecation showed that there was a marked rise in anal canal pressures on attempted defecation and that the mean maximum anal canal pressure was twice that of the intrarectal pressure. This pressure gradient would prevent the movement of a balloon into the anal canal (Table 2).

Electromyography of Pelvic Floor. In normal subjects there is continuous tonic activity of the striated muscle of the pelvic floor at rest and, in the majority, inhibition of this activity can be demonstrated during straining (4). Because pressure recordings had demonstrated an abnormal rise in anal canal pressure on straining in the constipated patients, we carried out electromyography of both the puborectalis and external sphincter muscles. A concentric needle electrode was introduced through the skin behind the anus and the activity of the striated muscle recorded at rest, during voluntary contraction, and during attempts to expel the rectal balloon.

Figure 2A shows a recording of anal canal pressure at 1.5 cm from the anal verge and electromyography of the external anal sphincter muscle in a normal subject. On coughing there is a sharp rise in anal canal pressure accompanied by an increase in electrical activity in the muscle. On straining however, anal canal pressure falls, and electrical activity is reduced.

Figure 2B shows a similar recording in one of the patients with slow-transit constipation. There was a normal response to coughing, but a marked and sustained rise in anal canal pressure occurred on straining associated with increased activity of the external sphincter. The results of electromyography in the 15 constipated patients are summarized in Table 3. No inhibition of electrical activity was seen in either the puborectalis or external sphincter muscles, but rather there was increased activity in one or both of these muscles associated with the rise in anal canal pressure. In a few subjects abnormal electrical activity persisted after the straining effort had ceased and in one patient continued for 2 min (Table 3).

Clinical Observations. Unlike patients with idiopathic megacolon, those with slow-transit consti-

TABLE 3. EFFECT OF ATTEMPTED DEFECATION OF INTRARECTAL BALLOON ON ELECTRICAL ACTIVITY OF STRIATED MUSCLES OF PELVIC FLOOR IN 15 PATIENTS WITH SLOW-TRANSIT CONSTIPATION

	EMG activity		
	Reduced	Unchanged	Increased
Puborectalis	0	0	15
External sphincter	0	3	12

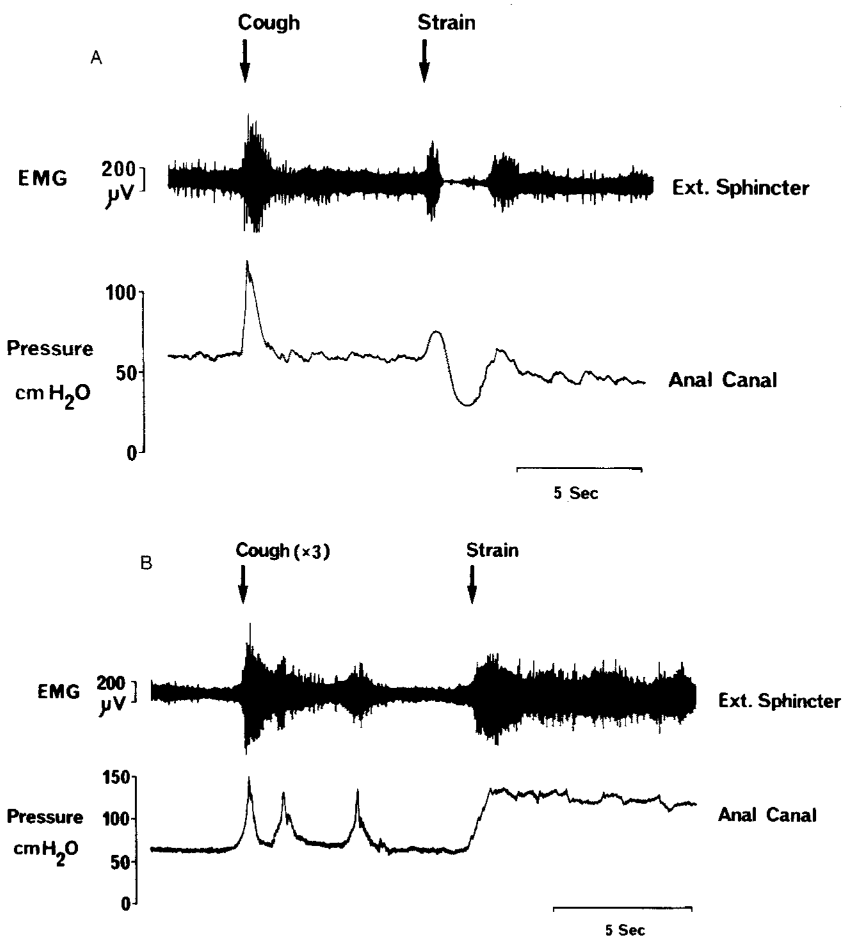

Fig 2. Anal canal pressure and external sphincter activity in a normal subject (A) and a patient with slow-transit constipation (B) showing the response to coughing and attempted defecation.

pation do not have fecal soiling. The anus in the patients studied appeared normal on external examination, and there was no perineal descent when they attempted balloon expulsion. Observation as the balloon was pulled out by the weights showed that the sphincters did not relax as judged by a constant position of the anal margin. The balloon was pulled lengthways, in some cases until it was over 15 cm long, before withdrawal. In contrast, in normal subjects the anus could be seen to dilate and the balloon was passed as a bolus with only minimal distortion.

When given large doses of an osmotic laxative sufficient to liquefy the stool, some patients with slow-transit constipation are still unable to defecate. When a proctoscope was passed on these patients and its obdurator removed, there was an immediate flood of liquid stool. This finding suggested a distal obstruction to the passage of feces.

Six further patients were also studied who had undergone subtotal colectomy with ileorectal or cecorectal anastomosis—yet had remained constipated. They showed the same electromyographic abnormality, and none could expel the rectal balloon. The mean weight applied via the pulley to withdraw the balloon during straining in these patients was 682 g. This finding gives further support to the idea that there might be a lesion in the rectum or anus contributing to the symptom of constipation in these patients.

DISCUSSION

Continence in normal subjects is thought to be the result of sustained contraction of the internal anal sphincter and the striated pelvic floor muscles. The central portion of the levator ani muscle, the puborectalis, acts as a sling pulling the anorectal junction forward to create an angle of about 90 degrees between the rectum and anal canal. This angulation is probably an important factor in the maintenance of normal continence (5). Reflex contraction of the pelvic floor occurs in response to raised intraabdominal pressure—for example during coughing (4). Temporary voluntary contraction of the pelvic floor and superficial external sphincter can augment the reflex mechanism if required. During normal defecation both the internal and external sphincters relax, and the anorectal angle straightens with relaxation of the puborectalis (6).

In the constipated subjects studied, reflex inhibition of the internal sphincter in response to rectal distension was normal. However, these patients were unable to expel a water-filled balloon used as a model of defecation. Relaxation of the puborectalis and external sphincter did not occur on straining as in normal subjects (4). Furthermore, the tension required to pull the balloon through the anal canal approximated that required in normal subjects at rest. It therefore appeared that the constipated subjects might have a disorder of the defecatory mechanism associated with failure of relaxation of the striated muscle of the pelvic floor.

The reflex mechanism by which the striated muscle of the anus relaxes on defecation has not been elucidated. It has been suggested that downward pressure on the puborectalis muscle results in an urge to defecate with a wave of rectal contraction that may lead to external sphincter relaxation (7). It seems from our studies that if this is the case, this reflex may be abnormal in patients with slow-transit constipation. Pulling the balloon down against the puborectalis muscle did not appear to result in sphincter relaxation, and the balloon was considerably distorted by withdrawal through a tightly closed sphincter.

This present study might be regarded in some ways as unphysiological, in that the patients were asked to "defecate" lying on their side. To overcome this problem we developed, in a parallel study, another model of defecation in which a barium-filled balloon was observed during attempted defecation in the normal position (8). In that study similar results were obtained in patients with slow-transit constipation. None were able to expel the rectal balloon and the anal canal, outlined by barium, was not seen to relax.

It seems likely that a disorder of the pelvic floor muscles contributes, at least in part, to the symptoms of some patients complaining of constipation. Other observations have shown a similar abnormality to that described here in patients with idiopathic megacolon and some patients who complain of constipation but with a normal whole gut transit rate and barium enema (9). However, abnormalities of colonic function may also be important (10) and anatomical abnormalities of the myenteric plexus of the colon are demonstrable in some patients (11, 12).

Three of our patients were treated by anorectal myectomy, which removes a segment of the internal anal sphincter. These operations were not successful, and they were still unable to expel a rectal balloon afterwards, suggesting that the internal sphincter was not responsible for their problem. This operation has been performed with success in some patients with constipation, not only those with short-segment aganglionosis but also others with normal ganglia (13). On this evidence, Martelli and colleagues (13) have suggested that a group of patients exists with organic constipation due to outlet obstruction of anorectal junction origin. Our observations support their hypothesis and indicate that the outlet obstruction is due, in some patients, to contraction of the striated muscles of the pelvic floor.

The sustained contraction of the striated muscles of the pelvic floor on attempted defecation in our patients could be due to an involuntary reflex or to voluntary suppression of the normal inhibition. The disorder may be analogous to the spasm of the pelvic floor muscles that occurs in vaginismus and perhaps could be called "anismus." If, as seems possible, the muscle dysfunction is an acquired disorder, then some form of retraining might help patients to relax these muscles during defecation. Normal subjects can learn to inhibit the contraction of the external sphincter which occurs when the rectum is distended (14). Adaptation of this technique is indicated, rather than the training successfully used in children with idiopathic megarectum who can be taught to increase their intraabdominal pressure in order to expel a stool (15).

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