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Physiological studies in young women with chronic constipation

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Abstract. Manometric, radiological and neurophysiological investigations were performed on 34 women, aged between 14 and 53, who suffered with chronic constipation refractory to treatment, and on 27 age-matched normal female control subjects. The constipated patients had more difficulty in evacuating simulated stools than control subjects and 13 out of 19 patients tested obstructed defaecation by contracting the external sphincter during straining. The constipated group required a greater degree of rectal distension than control subjects to induce rectal contractions, anal relaxation and a desire to defaecate. Other modalities of rectal sensation were normal in the constipated subjects. Compared with controls, constipated patients had significantly lower anal pressures, an abnormal degree of perineal descent on straining and an obtuse anorectal angulation at rest. These results were compatible with weakness of the pelvic floor and neuropathic damage to the external sphincter. Mouth to anus transit time was abnormally prolonged in 60% of constipated patients, but was within the normal range in the remainder. Anorectal function in patients with slow transit was not significantly different from that in patients with a normal transit time. The mouth to caecum transit time of a standard meal was prolonged in constipated patients irrespective of the duration of the whole gut transit. Gastric emptying was not significantly prolonged.

Idiopathic constipation is common. Many patients are helped by dietary fibre, but some are resistant to treatment with diet or drugs. In these, management is difficult owing to poor understanding of the pathophysiological mechanisms. Previous physiological studies suggest that the aetiology of constipation is multifactorial [1–5] and it is probable that there are several distinct clinical sub-groups of constipated patients. Severe diet-resistant constipation in young to middle aged people is almost entirely confined to women. Young female patients may therefore form a distinct group with a common pathogenesis.

In a previous paper [6] we reported the results of several tests of anorectal function in 14 women with constipation so severe that they required saline cathartics or enemas to achieve any bowel action. We have now extended these studies to include young women with severe diet-resistant constipation who are, however, able to defaecate spontaneously or with the help of irritant laxatives. In this study anorectal function and intestinal transit in constipated women and age-matched controls have been compared.

Patients and methods

Studies were carried out on 34 women aged between 14 and 53 years (mean ± SEM; 32 ± 2) with severe constipation resistant to a high fibre diet. All had had difficulty in defaecation of at least 1 year's duration, necessitating straining at every stool and the passage of stool less than twice a week despite laxatives and a high fibre diet. The length of history varied from 1 to 37 years (median 20). The frequency of defaecation ranged from once every 3 to 4 days to once every 3–4 months. All patients were taking laxatives and 10 claimed that they would never pass a stool without the use of enemas. Seventeen (50%) patients said they never felt the desire to defaecate and the other 17 (50%) who experienced the desire to defaecate had great difficulty in evacuation. Twenty-two patients passed hard pellets despite a high intake of dietary fibre. Only 12 of the 34 subjects were noted to have stool in the rectum on initial examination, and none had faecal impaction.

One subject had had a hysterectomy but constipation had preceded this operation by 15 years. Two subjects had been sterilised, 2 had had a haemorrhoidectomy, 1 an operation for urinary incontinence and 1 an anal stretch. All of these operations took place after the onset of bowel symptoms.

One subject was taking tricyclic anti-depressants; none of the others were taking any medication known to predispose to constipation.

Most patients suffered with a variety of abdominal symptoms, including abdominal pain, bloatedness, nausea and indigestion (Table 1). A high proportion also complained of frequent headaches and feelings of lethargy. These symptoms tended to become more severe and debilitating the longer the period of constipation and were eased when the patient had achieved a satisfactory bowel action.

Table 1. Symptoms in patients with normal transit or slow transit constipation

Symptom ≧ 1/week	Normal transit ($n = 14$)	Slow transit ($n = 20$)	p
Upper abdominal pain	21%	25%	NS
Lower abdominal pain	79%	100%	NS
Bloated feeling	14%	40%	NS
Nausea/indigestion	71%	70%	NS
Lethargy	64%	80%	NS
Headaches	50%	75%	NS
Defecation less than once per month	21%	60%	NS

All patients had undergone routine clinical examination, and investigations including plasma electrolytes and calcium, thyroid function tests, sigmoidoscopy and barium enema; these revealed no abnormalities. Shortly after their first visit each patient was put on a trial of a minimum of 30 g coarse wheat bran for at least 1 month with no relief of constipation. Seventeen patients (50%) stated that their abdominal symptoms became worse on bran.

Studies were also carried out on 27 female volunteers, aged between 20 and 55 (mean age ± SEM; 34 ± 3 years), who had a normal bowel habit and had no gastrointestinal symptoms.

All subjects gave written informed consent for investigations, and the protocol was approved by the Ethical Subcommittee of the Sheffield Area Health Authority in October 1981.

Anal sphincter pressure

Maximum basal and maximal squeeze pressures in the anal canal were measured by a station pull-through technique using three low compliance water perfused catheters (O.D. 1.5 mm) bonded together so that the distal side openings were situated 2.5 cm apart and orientated to sample points 120° apart [7].

Anal responses to rectal distension

Rectal distension with a balloon usually causes a transient contraction of the external anal sphincter and a relaxation of the internal anal sphincter [8]. To record these responses, pressures were measured at 6 sites in the anal canal during distension of a rectal balloon with increasing volumes of air [6].

Changes in anal pressure were noted for each distending volume. The pressures in each channel prior to rectal distension (basal pressure) and the lowest pressure during each rectal distension (residual pressure) were recorded. At a certain degree of rectal distension, residual pressure fell to a level which was not reduced by further distension (minimum residual pressure). The lowest rectal distending volumes required to elicit relaxation and to prevent recovery of the anal pressure to the baseline within 1 min were recorded.

Distension of a rectal balloon with air

Rectal pressure was measured and subjective sensations were recorded during distension of a rectal balloon with air as previously described [9].

Tests of defaecation of simulated stools

These tests were carried out in 19 constipated patients and 13 controls and were designed to evaluate the ability of subjects to pass water filled balloons or small plastic spheres which were placed in the rectum to simulate stools. Balloons containing 25 and 50 ml water simulated two sizes of soft stool. The large balloon was made from an unstretched condom, the smaller from an unstretched rubber finger stall. Each balloon was tied around a nylon catheter of external diameter 3 mm, fitted to a 3-way tap. When filled with fluid, each balloon was firm and cylindrical in shape. In addition, each subject was asked to pass a hard plastic solid sphere of diameter 18 mm which resembled the small faecal pellets that many constipated patients produce. This was attached to a metal beaded chain, which lay along the anal canal. With the subject in the left lateral position each simulated stool was inserted digitally into the rectum, using KY jelly (Johnson and Johnson Ltd., Slough) as a lubricant. After insertion the balloons were filled with 50 ml or 25 ml or warm water (37 °C) using a syringe. Each object was pulled down to bring the lower pole against the innermost aspect of the sphincter.

The subject then sat on a commode and was given a stopcock. She was instructed to start it on beginning to strain and to stop it when the balloon or sphere was passed. The subject was left alone whilst she tried to pass the simulated stool, although a technician was always within speaking range behind a screen. Each subject was allowed 5 min to pass the object. If she was unable to do so within that time, the simulated stool was withdrawn through the anus.

The electrical activity of the external sphincter was recorded in 19 randomly selected constipated patients and 13 controls while they attempted to pass the 50-ml balloon. The subject lay on her left side and a bipolar electrode, consisting of two very fine (0.025 mm) trimel coated wires [10] with the ends bared, hooked, and offset to avoid electrical contact, was inserted into the subcutaneous part of the external sphincter using a fine gauge hypodermic needle. The needle was then withdrawn, leaving the hooked ends of the wire in the muscle. Subjects were unable to feel the wires when in situ. They were connected to an amplifier (DISA, Type 14A 11 Electromyograph), connected in turn via an integrator to the chart recorder.

Anorectal radiology

The anorectal angle and the degree of perineal descent below the pubococcygeal line were measured on lateral radiographs of the anorectal region [11] carried out during the first 10 days of the subject's menstrual cycle.

Measurements of gastrointestinal transit time

All subjects were fasted for at least 9 h before the study, which was performed within the first 10 days of the menstrual cycle [12]. A meal of sausage, baked beans and mashed potato, the latter containing 25 μCi (0.93 MBq) ^{99m}Tc-sulphur colloid and 50 segments (2 mm × 3 mm) of radio-opaque plastic tubing, was given between 9 and 10 a.m. [13].

Immediately after, the subject lay supine and gastric emptying was monitored by measuring the decline in radioactivity over the surface of the stomach using a collimated crystal scintillation detector (Type DM-1 Nuclear Enterprises Ltd., Edinburgh) positioned over the gastric fundus [14, 15]. Mouth to caecum transit time (MCTT) of the head of the meal was determined by measuring the hydrogen concentration in samples of expired air, collected at 10-min intervals throughout the study [14]. The hydrogen profiles showed an early transient peak and a later rise, which was correlated with the entry of the test meal into the colon [16]. The MCTT was taken as the time from ingestion to a secondary increase in breath hydrogen concentrations of at least 3 ppm, sustained but usually increasing over three consecutive ten minute readings.

Subjects were asked to collect each bowel movement in individual polyethylene bags, labelled with the time and date. The collection was continued for a minimum of 72 h after the test meal and usually until at least 50% of the markers had been passed. The stools were X-rayed to determine the number of plastic markers in each sample.

Statistics

Data that appeared to be normally distributed were analysed by Student's t-test, whereas data that were obviously not normally distributed were assessed by the Wilcoxon rank sum test.

The probability of data which could be set out in a "2×2 contingency table" (or "fourfold table") was read from standard probability tables [17] based on the exact probability test devised by R. A. Fisher, J. P. Irwin and F. Yates [18].

Results

Anal manometry

Both the mean highest basal and the mean highest squeeze pressures in constipated patients were lower than in control subjects (Table 2) ($p<0.05$).

Anal responses to rectal distension

All subjects demonstrated a rectoanal inhibitory reflex upon balloon distension of the rectum. Values for the basal anal pressure at any point in the anal canal before balloon distension, the minimum residual anal pressure after balloon distension of the rectum and the minimum rectal volume which reduced the anal sphincter pressure by at least 5 cm of water, were similar in controls and constipated patients (Fig. 1).

The minimum rectal volume which caused sustained relaxation of the anal sphincter for 1 min was significantly higher in constipated patients than in controls ($p<0.05$) (Fig. 1). There was no significant difference between constipated and control subjects in the minimum rectal volumes required to stimulate anal contraction.

Rectal responses to rectal distension

The initial perception of the rectal balloon occurred at similar volumes (Fig. 2) in constipated patients and in controls. All controls and all constipated patients except one felt the presence of the balloon at 200 ml or less. The exception was a patient with an increased whole gut transit time who did not feel the balloon even when it was distended with 500 ml of air.

Constipated patients required higher rectal distending volumes to stimulate the desire to defaecate than the control subjects (Fig. 2). This difference between the percentages of patients and controls experiencing the desire to defaecate reached statistical significance at a distending volume of 150 ml. The percentages of controls and patients experiencing pain at different rectal distending volumes were not significantly different (Fig. 2), and there was no significant difference in the maximum rectal volumes that could be tolerated by patients and controls (Table 2).

Table 2. Physiological measurements in constipated women and age-matched controls

	Constipated ($n=34$)	Controls ($n=27$)	p
Age (years)	32 ± 2	34 ± 3	NS
Transit measurements			
Gastric emptying ($t\frac{1}{2}$) min	100 ± 5	93 ± 8	NS
Mouth to caecum transit time (min)	331 ± 12	242 ± 21	$p<0.001$
Whole gut transit time			
First marker (h)	72 (29 to > 130)	24 (11– 59)	$p<0.01$
50% markers (h)	100 (72 to > 400)	48 (26–105)	$p<0.01$
Anal pressures (cm water)			
Mean highest basal pressure	84 ± 5	103 ± 8	$p<0.05$
Mean highest squeeze pressure	175 ± 11	217 ± 16	$p<0.05$
Rectal compliance (ml/cm H_2O)	10.6 ± 1.0	8.5 ± 1.2	NS
Maximum tolerated rectal volume (ml)	321 ± 21	290 ± 18	NS
Anorectal angle (degrees)			
At rest	106 (70–127)	89 (78–96)	$p<0.05$
Position of anorectal angle above pubococcygeal line (cm)			
At rest	0 (+1.6 to −6.5)	−0.1 (+1.0 to −0.7)	NS
Straining	−2.6 (+0.2 to −7.0)	−1.7 (−0.9 to −2.7)	$p<0.05$

All results except radiology and whole gut transit time appeared normally distributed and are expressed as mean ±SEM. Radiology and whole gut times were not normally distributed and are expressed as median and range

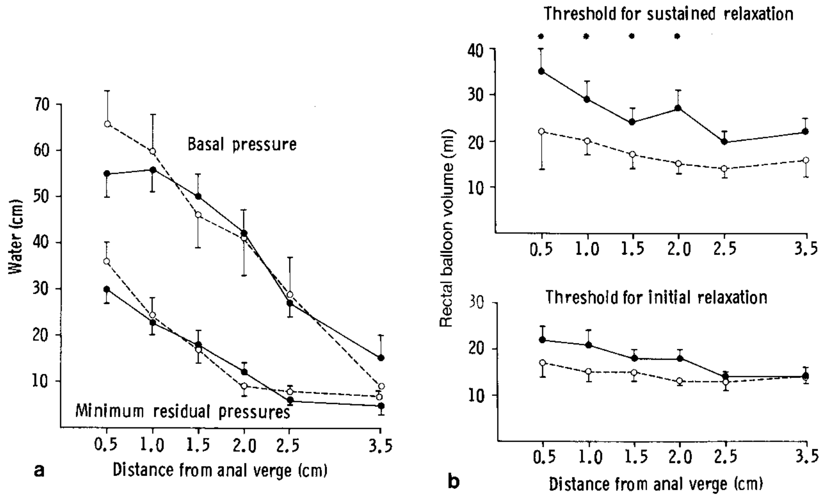

Fig. 1. a Basal pressure and minimum residual pressure during distension of a rectal balloon; **b** minimum rectal volume required to inhibit anal tone (bottom), and to prevent recovery of anal inhibition for 1 min (top). Results (mean ± SEM) for control subjects (○---○) and constipated patients (●——●) are plotted against distance of the recording port from the anal verge. * $p < 0.05$

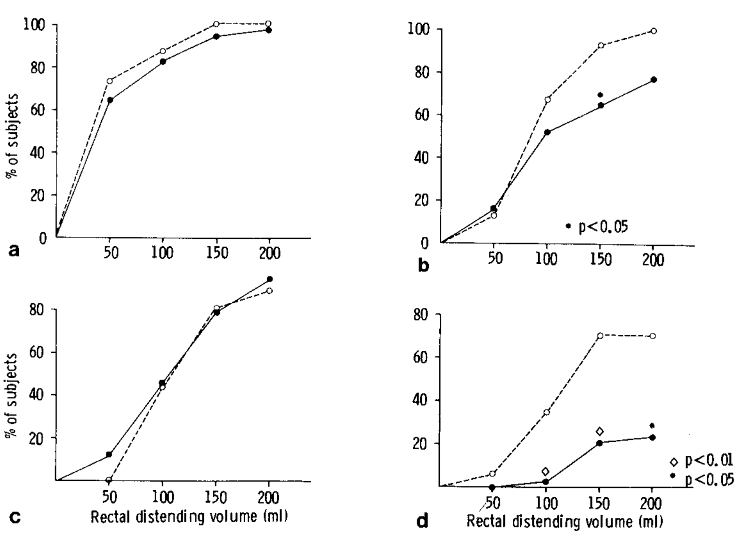

Fig. 2a–d. Rectal distension volumes and cumulative percentages of constipated patients (●——●) and control subjects (○ --- ○) who perceived **a** the presence of a rectal balloon, **b** the desire to defaecate, **c** abdominal pain and **d** demonstrated regular rectal contractions at each rectal distending volume

There was no significant difference in the basal rectal pressures at rest and during distension of a rectal balloon between constipated patients and controls. Values for rectal compliance were likewise similar in the two groups (Table 2).

Phasic contractions in response to rectal distension were recorded in 60% of constipated patients and in 90% of control subjects ($p > 0.1$). Seventy-one percent of controls but only 24% of constipated patients ($p < 0.05$) demonstrated regular rectal contractions upon rectal distension, and the stimulation of regular rectal contractions occurred at lower distending volumes in controls compared with constipated patients ($p < 0.01$) (Fig. 2).

Radiology

The ano-rectal angle at rest was significantly more obtuse in the constipated patients than controls ($p < 0.05$) (Table 2).

The distance of the anorectal angle from the pubococcygeal line was similar in the constipated and control groups at rest (Table 2), but on straining the constipated subjects demonstrated significantly more pelvic floor descent ($p < 0.05$).

Simulated defaecation

All control subjects passed the 50-ml balloon, 93% passed the 25-ml balloon and 76% passed the 18-mm sphere within 5 min, and most of these passed the 50-ml balloon (63%), the 25-ml balloon (87%) and the sphere (53%) within 20 s (Fig. 3). Only 25% of constipated patients were able to pass the 18-mm sphere within 5 min (cf. controls $p < 0.01$) and only 11% within 20 s (cf. controls $p < 0.05$), and a significantly lower proportion of constipated patients than controls were able to pass the 25-ml balloon within 20 s (cf. controls $p < 0.01$) (Fig. 3).

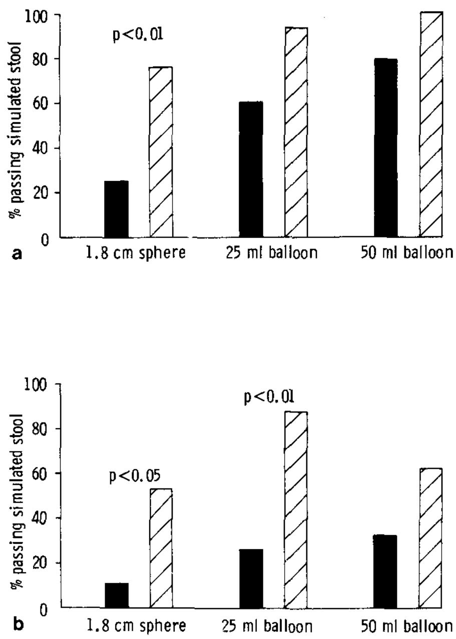

Fig. 3a and b. Percentage of constipated patients (■) and control subjects (▨) able to expel an 18-mm plastic sphere and balloon filled with 25 ml and 50 ml water from the rectum in **a** 5 min,

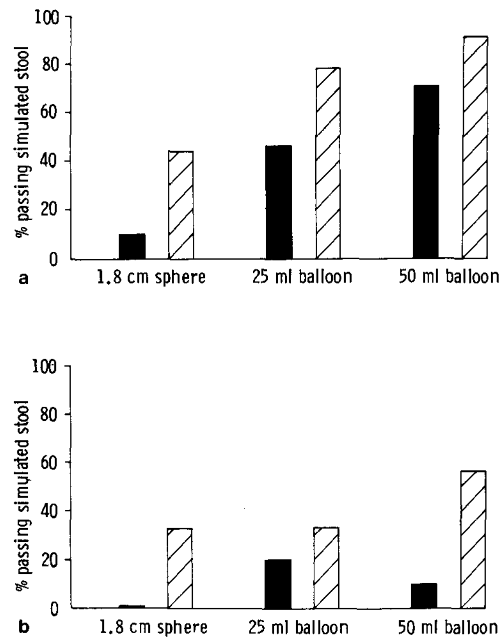

Fig. 4a and b. Percentages of patients with slow transit constipation (■) and normal transit constipation (▨), who could pass simulated stools from the rectum within **a** 5 min and **b** 20 s

Eight out of 13 controls but only 2/19 constipated patients inhibited the electrical activity of their external anal sphincter (EAS) when they attempted to expel the 50-ml balloon ($p<0.01$). Thirteen out of 19 constipated patients but only 2/13 control subjects ($p<0.01$) actually increased external sphincter electrical activity during passage of the balloon.

Measurement of gastrointestinal transit

Gastric emptying. The half times for the emptying of the solid test meal from the stomach in constipated and control subjects (Table 2) were not significantly different.

Mouth to caecum transit time. The MCTT was significantly prolonged in the constipated subjects ($p<0.001$) (Table 2).

Whole gut transit time. Constipated patients took a significantly longer time than controls to pass the first faecal marker and 50% of markers (Table 2). Twenty constipated patients failed to pass any markers within 72 h of ingestion (range 72 to >130 h); these were designated patients with slow transit constipation (STC). The remaining 14 patients passed markers within 72 h (range 29–54 h); these were designated patients with normal transit constipation (NTC).

Differences in results between patients with slow and normal transit times

The frequency of defaecation and range of symptoms were similar in patients with slow transit and normal transit constipation (Table 1). Both groups reported a similar frequency of bowel action (range 0–8 per month; median = 4 in each group).

There were no significant differences in anal manometry, anorectal radiology or in the responses to rectal distension between constipated patients with normal or slow whole gut transit times (Table 3). Measurements of gastric emptying and small bowel transit time in the two groups were also not significantly different (Table 3).

More patients with normal whole bowel transit times passed each of the simulated stools from the rectum within the prescribed time limit than those with slow whole bowel transit time (Fig. 4). These differences, however, failed to reach statistical significance. Eight out of a total of 10 patients with slow transit times and 5 out of 9 patients with normal transit times increased the electrical activity of the external sphincter on attempting to pass a 50-ml balloon. None of the 10 patients with slow whole gut transit times inhibited external sphincter electrical activity on attempting to expel the 50-ml balloon. Two out of 9 patients with normal transit times showed some external sphincter inhibition and passed the balloon with 16 and 12 s respectively.

Table 3. Physiological measurements in constipated women with slow and normal whole gut transit times

	Slow whole bowel transit ($n=20$)	Normal whole bowel transit ($n=14$)	p
Age (years)	32 ± 3	33 ± 3	NS
Transit measurements			
Gastric emptying ($t_{\frac{1}{2}}$) min	108 ± 7	88 ± 8	NS
Mouth to caecum transit time (min)	333 ±17	327 ±20	NS
Anal pressures (cm water)			
Mean highest basal pressure	80 ± 7	89 ± 6	NS
Mean highest squeeze pressure	184 ±17	162 ±14	NS
Rectal compliance (ml/cm H_2O)	11.1± 1.3	9.9± 1.6	NS
Maximum tolerated rectal volume (ml)	295 ±29	336 ±29	NS
Minimum rectal volume to elicit anal contraction (ml)	28 ± 3	40 ±11	NS
Anorectal angle (degrees)			
At rest	108 (73–120)	103 (70–127)	NS
Position of anorectal angle above pubococcygeal line (cm)			
At rest	0 (+1.6 to −2.5)	0 (+1.6 to −6.5)	NS
Straining	−1.6 (+0.2 to −6.0)	−3.5 (−0.8 to −7.0)	NS

All results except radiology are expressed as mean ± SEM. Radiology expressed as median and range

Differences in results between parous and nulliparous patients

Twenty patients had undergone vaginal delivery and 14 were nulliparous. There were no significant differences between the nulliparous and parous patients for the anorectal angles at rest (nulliparous 100 ± 5°; parous 103 ± 3° [mean ± SEM]) or the basal (nulliparous 95 ± 7; parous 78 ± 6 cm H_2O), or squeeze sphincter pressures (nulliparous 180 ± 15; parous 169 ± 13 cm H_2O), though perineal descent on straining was significantly greater in parous women [2.8 (0.2–7.0) cm vs. 1.5 (0–4.5) cm below the pubococcygeal line; median (range) ($p < 0.01$)]. The parous group were, however, significantly older [37 ± 2 years vs. 22 ± 2 years (mean ± SEM) ($p < 0.02$)] than the nulliparous and their history of constipation and straining at stool was also significantly longer [15 ± 3 years vs. 6 ± 2 years (mean ± SEM) ($p < 0.02$)].

Discussion

Defaecation comprises visceral and somatic muscular events, often initiated by stimuli from the gut, but probably also regulated by the central nervous system. The arrival of faeces in the rectum is thought to give rise to a desire to defaecate and to elicit rectal contractions and relaxation of the circular smooth muscle of the internal anal sphincter. Rectal contraction causes the faecal bolus to be squeezed towards the anal sphincter, the smooth muscle fibres of which relax to admit it. If conditions are appropriate for defaecation, the abdominal muscles contract, raising intra-abdominal pressure, and anal resistance is lowered by inhibition of puborectalis and external anal sphincter [19]. Defaecation can, however, be prevented when conditions are inappropriate by phasic contraction of the external anal sphincter and the puborectalis. Patients who cannot defaecate properly may exhibit abnormalities in any of these components of the defaecation mechanism.

Previous studies have shown that anal pressures may be abnormally elevated in some constipated patients [1–5], implying that the resistance to defaecation may be increased. Our data contradict those findings since both basal and squeeze sphincter pressures were lower in young constipated women than in age-matched female controls. The differences between the results of the present and previous studies may be explained in part by differences in the patients studied. In one previous study the subjects were children [2], in another the patient group was not defined [3]. In the two largest studies [1, 5], the large age range and the inclusion of males makes comparison difficult. Only one of these studies [4] investigated young constipated women and this showed that while 11 women with painless constipation had a mean maximum resting anal pressure within the normal range, patients with painful constipation showed abnormally high resting pressures. The latter group, however, included both males and females and the severity of the constipation was not specified.

Our group of young constipated women had a wider anorectal angle than normal when lying at rest in the left lateral position and when they strained, they exhibited a greater degree of perineal descent. It has been suggested that these abnormalities and the low sphincter pressures may occur as a result of chronic straining at stool, causing the pelvic floor to descend, with stretching of the pudendal nerve as it winds around the ischial spine [20, 21]. Women may be particularly susceptible to these changes since increased perineal descent and reduced power of the external sphincter are commonly found after vaginal delivery [22] and may be exacerbated by excessive straining at stool [21]. In support of this hypothesis, we found that straining produced a greater degree of perineal descent in parous compared with nulliparous patients but the parous patients were significantly older and had a significantly longer history of constipation.

Both control subjects and patients found it more difficult to expel the small hard plastic spheres than the larger soft sausage-shaped balloons. This observation may be important since 22 out of the 34 (66%) of the young constipated women that we studied said that they always produced small hard pellets similar in shape and consistency to the plastic sphere used. However the difficulty in defaecation experienced by constipated patients is not simply related to the type of stool since constipated patients found it more difficult to pass any of the simulated stools than normal controls. Electromyography showed that all except two of the constipated patients failed to inhibit the external anal sphincter on attempting defaecation and most actually contracted the muscle. It is unlikely that this was due to embarrassment since most age-matched controls relaxed the sphincter under the same experimental conditions. While these results support the findings originally reported by Preston and Lennard-Jones [23], it is unlikely that this is the only pathophysiological mechanism of constipation since none of our constipated patients had faecal loading in the rectum and indeed most had a completely empty rectum. This observation suggests the possibility of other factors, for example impaired delivery of faeces from the colon to the rectum or retropulsion of faeces in the rectum back into the sigmoid colon.

Young constipated women required larger rectal volumes to stimulate regular rectal contractions or to induce sustained relaxation of the internal anal sphincter than age-matched controls. Other investigators have shown that patients with severe constipation exhibit disturbances in colonic motility [24, 25], and histological abnormalities in the myenteric plexus [26, 27] though it is uncertain whether this is secondary to prolonged intake of laxatives [28].

The constipated patients also required larger rectal volumes than controls to elicit a desire to defaecate. This cannot be part of a generalised sensory disturbance since rectal perception and pain were unimpaired. Perhaps the normal desire to defaecate is a necessary sensation to facilitate rectal contraction and anal relaxation. Without it reflex contraction of the anal sphincter as occurs normally in response to raised abdominal pressure [29] may then become the predominant reaction.

Whole bowel transit time estimations have been used by others to categorise constipated patients [1, 30]. The separation of patients into those with normal and those with slow transit times may however be specious under certain circumstances. Transit times in the same person can vary considerably depending on whether the measurement coincides with a period of bowel activity or not. Moreover, patients with slow whole gut transit are not necessarily in a different pathophysiological category than those with normal transit; they may simply have more severe disease. In our study, young constipated women with normal whole gut transit times exhibited symptoms and physiological abnormalities that were no different from those found in patients with slow transit times (Tables 1 and 3). Patients with slow transit had, however, consistently greater difficulty in passing each of the simulated stools (Fig. 4), though this difference did not reach statistical significance for any individual simulated stool. The fact that the patients with normal whole gut transit times passed some stool within 3 days is at variance with the history of 0–8 bowel actions per month reported by this group, and probably reflects the unreliability of the history of bowel frequency given by patients [31, 32].

The slower small bowel transit found in patients with constipation could be either due to a primary disturbance in the small bowel or secondary to retarded colonic transit. Slow small bowel transit is associated with the passage of smaller amounts of ileal effluent [33] producing a weaker stimulus to colonic propulsion [34] but it can also be induced in normals by intermittent distension of the rectum [35]. While it is possible that colonic distension with faeces may have a similar effect in constipated patients, we have been unable to demonstrate any accelerated small intestinal transit in constipated subjects after emptying the colon with saline cathartics and enemas (Youle and Read, unpublished observations).

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References

1. Martelli H, Devroede G, Arhan P, Duguay C (1978) Mechanisms of idiopathic constipation: outlet obstruction. Gastroenterology 75:623–631

2. Meunier P, Marechal JM, de Beaujeu MJ (1979) Rectoanal pressures and rectal sensitivity studies in chronic childhood constipation. Gastroenterology 77:330–336
3. Behar J, Biancani P (1984) Rectal function in patients with idiopathic chronic constipation. In: Roman C (ed) Gastrointestinal motility. MTP Press, Lancaster, pp 459–466
4. Lanfranchi GA, Bazzocchi G, Brignola C, Campieri M, Labo G (1984) Different patterns of intestinal transit time and anorectal motility in painful and painless chronic constipation. Gut 25:1352–1357
5. Ducrotte P, Denis P, Galmiche J-P, Hellot M-F, Desechalliers J-P, Colin R, Pasquis P, Hecketsweiler P (1985) Motricite anorectale dans la constipation idiopathique. Etude de 200 patients consecutifs. Gastroenterol Clin Biol 9:10–15
6. Read NW, Timms JM, Barfield LJ, Donnelly TC, Bannister JJ (1986) Impairment of defecation in young women with severe constipation. Gastroenterology 90:53–60
7. Read NW, Harford MV, Schmulen AC, Read MG, Santa Ana C, Fordtran JS (1979) A clinical study of patients with fecal incontinence and diarrhea. Gastroenterology 76:747–756
8. Schuster MM, Hookman P, Hendrix TR, Mendeloff AI (1965) Simultaneous manometric recording of internal and external anal sphincteric reflexes. Bull John Hopkins Hosp 116:79–88
9. Read M, Read NW, Barber DC, Duthie HL (1982) Effects of loperamide on anal sphincter function in patients complaining of chronic diarrhea with fecal incontinence and urgency. Dig Dis Sci 27:807–814
10. Basmajian JV, Stecko G (1962) A new bipolar electrode for electromyography. J Appl Physiol 17:849
11. Bartolo DCC, Read NW, Jarratt JA, Read MG, Donnelly TC, Johnson AG (1983) Differences in anal sphincter function and clinical presentation in patients with pelvic floor descent. Gastroenterology 85:68–75
12. Wald A, Van Thiel DH, Hoechstetter L, Gavaler JS, Egler KM, Verm R, Scott L, Lester R (1981) Gastrointestinal transit: the effect of the menstrual cycle. Gastroenterology 80: 1497–1500
13. Read NW, Miles CA, Fisher D, Holgate AM, Kime ND, Mitchell MA, Reeves AM, Roche TB, Walker M (1980) Transit of a meal through the stomach, small intestine and colon in normal subjects and its role in the pathogenesis of diarrhea. Gastroenterology 79:1276–1282
14. Cann PA, Read NW, Brown C, Hobson N, Holdsworth CD (1983) Irritable bowel syndrome: relationship of disorders in the transit of a single solid meal to symptom patterns. Gut 24:405–411
15. Ostick DG, Green G, Howe K, Dymock IW, Cowley DJ (1976) Simple clinical method of measuring gastric emptying of solid meals. Gut 17:189–191
16. Read NW, Al-Janabi MN, Bates TE, Holgate AM, Cann PA, Kinsman RI, McFarlane A, Brown C (1985) Interpretation of the breath hydrogen profile obtained after ingesting a solid meal containing unabsorbable carbohydrate. Gut 26:834–842
17. Diem K, Lentner C (1970) Scientific tables, 7th edn. Geigy Pharmaceuticals, Macclesfield
18. Armitage P (1971) Statistical methods in medical research, 1st edn. Blackwell Scientific, Oxford
19. Ihre T (1974) Studies on anal function in continent and incontinent patients. Scand J Gastroenterology 9 [Suppl 25] 1–64
20. Parks AG, Swash M, Urich H (1977) Sphincter denervation in anorectal incontinence and rectal prolapse. Gut 18:656–665
21. Snooks SJ, Barnes PRH, Swash M, Henry MM (1985) Damage to the innervation of the pelvic floor musculature in chronic constipation. Gastroenterology 89:977–981
22. Snooks SJ, Swash M, Setchell M, Henry MM (1984) Injury to the innervation of pelvic floor sphincter musculature in childbirth. Lancet 2:546–550
23. Preston DM, Lennard-Jones JE (1985) Anismus in chronic constipation. Dig Dis Sci 30:413–418
24. Preston DM, Lennard-Jones JE (1985) Pelvic motility and response to intraluminal bisacodyl in slow transit constipation. Dig Dis Sci 30:289–294
25. Schang J-C, Devroede G, Duguay C, Hemond M, Hebert M (1985) Constipation par inertie colique et obstruction distale; etude electromyographique. Gastroenterol Clin Biol 9: 480–485
26. Preston DM, Butler MG, Smith B, Lennard-Jones JE (1983) Neuropathology of slow transit constipation. Gut 24:A997
27. Krishnamurthy S, Schuffler MD, Rohrmann CA, Pope CE (1985) Severe, idiopathic constipation is associated with a distinct abnormality of the colonic myenteric plexus. Gastroenterology 88:26–34
28. Smith B (1968) Effect of irritant purgatives on the myenteric plexus in man and the mouse. Gut 9:139–143
29. Parks AG, Porter NH, Melzak J (1962) Experimental study of the reflex mechanism controlling the muscles of the pelvic floor. Dis Col Rect 5:407–414
30. Hinton JM, Lennard-Jones JE (1968) Constipation: definition and classification. Postgrad Med J 44:720–723
31. Manning AP, Wyman JB, Heaton KW (1976) How trustworthy are bowel histories? Comparison of recalled and recorded information. Br Med J 2:213–214
32. Read NW, Krejs GJ, Read MG, Santa Ana CA, Morawski SG, Fordtran JS (1980) Chronic diarrhea of unknown origin. Gastroenterology 78:264–271
33. Holgate AM, Read NW (1983) Relationship between small bowel transit time and absorption of a solid meal. Dig Dis Sci 28:812–819
34. Chauve A, Devroede G, Bastin E (1976) Intraluminal pressures during perfusion of the human colon in situ. Gastroenterology 70:336–340
35. Youle MS, Read NW (1984) Effect of painless rectal distension on gastrointestinal transit of solid meal. Dig Dis Sci 29:902–906

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