Preservation of faecal continence during rises in intra-abdominal pressure: is there a role for the flap valve?

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SUMMARY Studies were carried out in 15 normal subjects and 14 patients with idiopathic faecal incontinence to test whether a rectoanal flap valve could be responsible for maintaining faecal continence in man. Intraluminal pressures were recorded from the rectum and from three sites in the anal canal during serial increases in intra-abdominal pressure, produced by forced expiration into a sphygmonanometer keeping the height of the column of mercury at prescribed levels. The anal pressures in the normal volunteers always remained higher than the intrarectal pressures even when these were as high as 230 cm H2O. This pressure gradient was the reverse of that which would be found if an anterior rectal flap valve maintained continence and suggests instead that continence is normally maintained by a reflex contraction of the external anal sphincter. The anal pressures in patients with idiopathic faecal incontinence, however, fell below the rectal pressure as the intra-abdominal pressure increased, creating the conditions for a flap valve. The valve was incompetent, however, because fluid infused into the rectum leaked from the anus whenever the rectal pressure exceeded the anal pressure.

Increases in abdominal pressure that occur during lifting, coughing, or rising from a chair are a major threat to faecal continence. Under these conditions, the acuteness of the anorectal angulation is thought by many to be crucial to continence. Patients who suffer from faecal incontinence usually have a more obtuse anorectal angle than normal subjects,1 while operations designed to restore the anorectal angulation often result in an improvement of continence.1−3

The acute anorectal angle is said to maintain continence by creating a flap valve, whereby a rise in intra-abdominal pressure forces the anterior rectal wall down on top of the anal sphincter, sealing the top of the anal canal, and preventing faeces from escaping from the rectum.4 Although there are several reasons why this mechanism seems unlikely, no previous studies have attempted to test its validity experimentally.

A valve only operates if it separates two compartments of different pressures. Thus, if a flap valve was responsible for maintaining continence during increases in intra-abdominal pressure, the anal pressures would have to be lower than the intrarectal pressures, as indicated by measurements of rectal pressure. The aim of these experiments was to test the validity of this mechanical theory of continence by measuring anal and rectal pressures as subjects increased their intra-abdominal pressure in a controlled manner.

Methods

SUBJECTS Studies were carried out in 15 normal volunteers (10 men, five women, aged 19–48 years, median=22 years), all of whom were healthy members of hospital staff or students; and 14 patients with idiopathic faecal incontinence (five men, nine women, aged 16–75 years, median=51 years).

Each subject gave his or her written informed consent for the study to be carried out and the protocol was approved by the Local Ethical Subcommittee of the Sheffield Area Health Authority (Teaching).
Role of flap valve in faecal continence

ANORECTAL MANOMETRY

Intraluminal pressures were recorded in the rectum and at three sites in the anal canal during controlled rise in intra-abdominal pressure. This was achieved by asking the subject to blow through a mouthpiece into a mercury manometer and maintain the height of the mercury column at levels corresponding to 20, 40, 60, 80, and 100 mm Hg. Subjects were instructed to use only their abdominal muscles and diaphragm to maintain the level of the mercury column and not to create a buccal seal by opposing the tongue and soft palate. The order in which the intra-abdominal pressure was raised to different levels was randomised.

Anal pressures were measured using a manometric assembly consisting of three water perfused catheters (od 1.5 mm) bonded together so that their side opening ports were situated 1-25 cm apart, the distal port being 2 mm from the tip of the cannula. The assembly was placed in the rectum and then withdrawn until the innermost port recorded a rise in pressure showing it was just within the upper anal canal. This left the other two ports recording from the mid and lower anal canal. The rectal pressure was measured using a separate soft narrow (od=2 mm) rectal catheter which had a side opening port situated 10 cm from the anal margin. This catheter was soft enough to retain its position without obstruction of the lumen after being bent at an angle of 90°.

Each of the manometric channels was continuously perfused with distilled water at the rate of 1 ml/min using a low compliance pneumohydraulic system (Mui, Mississauga, Ontario, Canada). Pressures were measured by means of transducers (Statham 230B Oxnard, California) situated in each perfusion line and linked to a chart recorder (Hewlett Packard 7758A, Waltham, Mass).

Studies were repeated in all 15 volunteers and 14 patients with idiopathic faecal incontinence after 200 ml fluid had been introduced into the rectum.

Fig. 1 Integrated electrical activity of the external anal sphincter and intrarectal pressure during graded increases in intra-abdominal pressure in a normal subject.

Fig. 2 Pressure recorded at three sites in the anal canal (1, 2, 3) (port 1 being the innermost) and rectum (R) during graded rises in intra-abdominal pressure in normal subjects. Results are shown as mean ± SEM.

Fig. 3 Plot of measurements of intrarectal pressure against simultaneous measurements of maximum anal pressure during graded rises in intra-abdominal pressure in 15 normal subjects.
allowed us to determine the relationship between anorectal pressures and leakage. Finally, the subjects were asked to carry out a maximal expiratory effort by blowing into the manometer and raising the column of mercury to as high a level as possible. The anal pressures, rectal pressure and the presence or absence of leakage were recorded.

The electrical activity of the external anal sphincter was recorded in four normal subjects during rises in intra-abdominal pressure by means of an anal plug electrode (Disa, Bristol) placed in the anal canal. The EMG signal was amplified (Disa uroamplifier 21C01), integrated and then relayed to the chart recorder.

**Results**

In normal subjects and incontinent patients, forced expiration into the sphygmomanometer produced a prompt increase in intrarectal pressure (Fig. 1) that was sustained until the subject stopped blowing, when it dropped abruptly. The pressure in the exhaled air, measured using the sphygmomanometer, was significantly correlated with intrarectal pressure \( r=0.85, p<0.001 \). These observations suggest that increases in rectal pressure, recorded during valsalva manoeuvres, were caused by transmitted increases in intra-abdominal pressure.

**Normal Subjects**

As the normal subjects raised their intra-abdominal pressures up to levels of 120 mm Hg the average pressures at each site in the anal canal always remained at least 5 cm water above those recorded in the rectum (Fig. 2) and no leakage was observed when saline was in the rectum. The highest pressures recorded in the anal canal were between 10 and 100 cm H_2O above rectal pressures whenever the subjects increased their intra-abdominal pressure (Fig. 3). Thus, we could find no evidence in any test in any volunteer to support the existence of a rectoanal flap valve.

The rises in anal pressure were probably related to increases in external sphincter contraction since electromyographic recordings of the external anal sphincter increased as the intra-abdominal pressure rose (Fig. 1).

**Incontinent Patients**

Patients with idiopathic faecal incontinence were unable to keep the anal pressure above the rectal pressure as the intra-abdominal pressure was increased (Fig. 4). Figure 5 shows that in most instances anal pressures remained above rectal pressures when the latter were below about 75 cm H_2O. Above this level, anal pressures were more often lower than rectal pressures. If saline was in the rectum, leakage occurred whenever the rectal pressure was higher than the anal pressure (Fig. 6). Only 8/14, however, incontinent patients leaked during the test when saline was in the rectum. The patients who did not leak were unable or unwilling to raise their rectal pressure as high as those who did leak \( (69±11 \text{ v } 115±18 \text{ cm (mean±SEM) water}) \) \( p<0.01 \).

**Discussion**

The results of this study failed to support the concept of an anorectal flap valve in normal subjects. As the
pressure in the rectum rose, the anal pressure was always maintained at a level at least 10 cm H₂O higher than that in the rectum, and if there was fluid in the rectum, continence was maintained. This pressure gradient is contrary to that which would permit the operation of a flap valve. Instead our results suggest that continence to fluids is normally maintained during increases in intra-abdominal pressure by a compensatory contraction of the external anal sphincter. Earlier studies documented increases in the electrical activity of the external anal sphincter in response to rises in intra-abdominal pressure caused by coughing, straining, and laughing, and more recently investigators have shown that the increases in external sphincter activity, that occur during graded Valsalva manoeuvres, are directly proportional to the intra-abdominal pressures.

In contrast with normal subjects, incontinent patients were unable to contract their anal canal sufficiently to keep the anal pressure above the rectal pressure as the intra-abdominal pressure rose. This may be because of the weakness of the external sphincter or it could be related to deferred recruitment of the muscle fibres of the external anal sphincter because of decreased sensory impulses from the pelvic floor. Whatever the mechanism, the poor contraction of the external sphincter created the conditions appropriate for the operation of a flap valve, but in spite of this, saline leaked from the anal canal whenever the rectal pressure exceeded the anal pressure. Thus, although a flap valve may have been present in incontinent patients, it did not prevent leakage occurring under the conditions of this test. It is unlikely that the presence of the rectal catheter could have interfered with the competence of a flap valve in the incontinent patients. This catheter was very narrow and was soft enough to retain its shape after being bent to 90° and to exert no leverage on the components of the anorectal angle. Moreover, any interference by the tube with a mucosal seal would have been expected to cause leakage in the normal subjects as well as the incontinent patients.

If the anorectal angulation does not normally preserve continence by creating a flap valve, does it have any role to play in the maintenance of continence? The angle must assist continence to solids, as considerable force would be required to mould a solid stool around such a sharp angle, but it is difficult to envisage how the angle per se could prevent leakage of fluid, unless the puborectalis compressed the anal canal against a relatively immovable object such as the cervix uteri in the female or the prostate in the male. Operations to restore the anorectal angle improve continence, but postoperative measurements indicate that in many the operation does not alter the anorectal angle but may increase anal pressures, and anal length or tighten the pelvic floor so that it becomes a more efficient sensor of increases in intra-abdominal pressure and may thus generate prompt increases in external sphincter activity in response to small increases in intra-abdominal pressure.

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References

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