Gut, 1978, **19**, 1163-1174

Progress report

Maintenance of anal continence: a review of pelvic floor physiology

This review is concerned with the physiology of the pelvic floor as it affects defaecation and the maintenance of anal continence. The reflex mechanisms involved are complex and the morphology of the anorectal region makes their study difficult since the separate components of the system are generally not accessible for study in isolation. A relatively small number of techniques are available for the observation of anorectal function in man. The literature on the pelvic floor reveals wide gaps in the understanding of basic mechanisms —for example, the reflex 'resting activity' of the external anal sphincter. This review attempts to bring together the results of the various studies of pelvic floor function, to describe the differences between recording techniques and how they influence the results obtained, and to compare the conclusions drawn.

The physiology of the pelvic floor has been investigated in man by making electromyographic (EMG) recordings of the activity of the puborectalis and external sphincter muscles; by recording the pressure in the anal canal and rectum; by attempting to separate the actions of the external and internal sphincters and by recording both electrical and mechanical activity of the latter; by *in vitro* muscle studies; by cineradiography of the anorectum; and by histological study of the external sphincter. Some animal studies (chiefly on the cat) have been made.

Pelvic floor in 'resting' state

The most prominent feature in lateral radiographic films of the resting pelvic floor[1,2] is the sharp 80° angulation between anal canal and rectum. The angulation is maintained by the puborectalis muscle which separates from the levator ani diaphragm to form a sling looping behind the anorectal junction. Continuous activity of the puborectalis and external anal sphincter has been reported in many EMG studies[2,3,4,5,6,7,8,9,10,11]. Using the criteria of firing frequency and motor unit potential amplitude and duration given by Tokizane[12] and Kawakami[13] to distinguish between tonic and phasic motor units, Kerremans[2] concluded that the pelvic floor consists mainly of tonic motor units. This has been confirmed by the results of histochemical studies[2,14]. The EMG activity varies with posture, though Taverner and Smiddy[8] report that the increase in activity associated with standing up is transient (lasting three to four seconds). The system seems to be very sensitive to changes in intra-abdominal pressure.

The activity of the external sphincter and puborectalis is maintained as part of a spinal reflex, as it is present (although slightly modified) in patients with complete transection of the spinal cord[3,5,15]. Patients with tabes dorsalis, in whom the posterior roots are destroyed but the motor pathway is left intact, show no tonic activity[5]. Sensory input from the pelvic floor is therefore required to maintain the reflex, but the source of this input is unknown.

The internal anal sphincter is also continuously active. Strips of this muscle show rhythmic spontaneous contractions *in vitro*. Kerremans[2] and Penninckx[16] found that strips from the distal portion of the sphincter contracted at a higher frequency than proximal strips, and this finding agrees with Kerremans' observations of motility *in vivo* where pressure waves were of a higher frequency in the distal canal. The amplitude of slow pressure waves (frequency between 8/min and 15/min) and ultra-slow pressure waves (frequency between 0·9/min and 6/min) and the ease with which they may be recorded in the anal canal seem to depend on the technique of pressure measurement used[17,18]. Continuous electrical activity has been recorded in the internal sphincter at rest[2,19,20]. Although slow potential changes have been seen, spike activity has not been reported.

The role of the autonomic nerve supply in maintaining internal sphincter activity is not clear. Frenckner and Ihre[21] concluded from the results of using high and low spinal anaesthesia in man that there was a tonic excitatory sympathetic discharge to the internal sphincter. Meunier and Mollard[22] found that meningocele subjects, who are theoretically devoid of parasympathetic pathways but have a normal sympathetic outflow, have a reduced anal canal resting pressure. Responses to electrical stimulation of the autonomic nerves in the cat were found to be variable[23]. Shepherd and Wright[24] using peroperative stimulation of the presacral nerves in man, found the sympathetic supply to be inhibitory to the internal sphincter. *In vitro*, however, noradrenaline causes contraction of the internal sphincter. α- and β-adrenergic receptors have been found in the internal sphincter of man and cat[2,25,26]. Various alternatives to noradrenaline as the sympathetic transmitter in the internal sphincter have been considered[27,28]. It has been reported that the normal parasympathetic transmitter, acetylcholine, has little or no effect in physiological concentrations on the internal sphincter[28]. Friedmann[25] and Parks[26] and colleagues found that upper and lower parts of the sphincter showed different responses to acetylcholine. Garrett and coworkers[23] showed that *in vivo* it was possible to obtain indirect cholinergic responses which operated through an adrenergic reflex.

Measurement of pressure within anal canal

Despite the difficulties of interpretation, anal canal pressure measurements are the commonest (presumably because the most convenient) means of assessment of sphincter function. In fact, the literature abounds with confusing, unexplained results and some doubtful conclusions about sphincter activity. Most workers have agreed on the existence of a high pressure zone or pressure barrier in the anal canal. However, they disagree about the most appropriate method of measuring this pressure, its source in terms of activity of the two anal sphincters and its importance in the maintenance of anal continence.

The two common methods of measurement are by balloon systems[1,2,9–11,18,29,30–33] and with open-tipped catheters[1,9,22,34–36]. In general, pressure values recorded with open-tipped tubes are lower than those obtained with balloon systems, but comparison is difficult because of the range of diameters of balloons and tubes used. There is a considerable list of 'odd' findings. Harris and Pope[36] found that the pressure profile depended on whether the tube had end- or side-openings and, in the latter case, on the distance of the opening from the end of the tube. One report states that negligible pressure was seen in the resting sphincter with an air-filled tube[1]. Harris and Pope[36] found that the pressure profile obtained as the tube was withdrawn through the anal canal from the rectum differs considerably from that obtained as the tube was inserted from the exterior. In a later discussion Harris[37] and colleagues conclude that the recording system 'traps the pressure recorded immediately before the tip enters the sphincter'.

Analysis of pressure in terms of forces acting within the anorectal region is difficult. The geometry of the system is complicated; to view the anal canal as a narrow tube with a regular, open lumen leading to the wider tube of the rectum is a clear oversimplification. Radiographic studies show that the anal canal is flattened from side to side rather than circular in cross-section, and that the anterior and posterior walls of the rectum are in close apposition at the anorectal junction[1,2]. The use of open-tipped catheters in such a small cavity as the anal canal might well present is suspect. Continuous perfusion of fluid in water-filled tubes to keep the tip open has been objected to on the grounds that perfusion causes irritation of the perianal skin which could cause reflex activity in the muscles being investigated[2]. Hancock[17] found that measured pressure increased with perfusion rate and that slow and ultra-slow pressure waves attributed to activity of the internal sphincter can be seen only with fairly high perfusion rates. Wheatley *et al.*[38,39] describe a continuously perfused sleeve device which records the maximum anal canal pressure rather than the pressure at a specific position, thus eliminating artefacts which might be obtained when an open-tipped catheter moves relative to the anal canal.

The use of balloons has been criticised because they act as a stimulus to the anal canal[3]. This must be true of any measuring device except perhaps those that are of very small diameter. The diameter of the balloons that are used varies from 3 mm to 15 mm, and the pressure measurements that are obtained vary with diameter and with the elastic properties of the balloon. The effect of the diameter of the measuring probe has been noted by several people[9,40,41]. Duthie and colleagues[42] found that the effect of probe diameter can be eliminated if sufficient time (around 15 minutes) is allowed for the anal canal muscles to adapt. A possible improvement on the use of an elastic ballon is the use of a Perspex or metal cylinder with an elastic diaphragm in its wall[11,17,43].

Harris and Pope[36] and Harris *et al.*[37] proposed that the pressure recorded in the anal canal depended not so much on the ability of the muscles to squeeze on to the balloon as on the resistance of the canal to opening. They recorded stepwise increases in pressure as successive small increments of fluid were injected through their open-tipped catheter, until the 'resting yield pressure' was reached. Beyond this, no increase was seen with further fluid increments. They found that voluntary contraction of the muscles of the pelvic floor did not cause an increase in pressure unless further fluid were injected. This result is at variance with that of Katz *et al.*[32] who used incremental increases in the volume of an air-filled balloon. In their experiments, voluntary contractions produced a pressure rise the magnitude of which increased as the volume of the balloon was increased, until a plateau at a fixed level above the resting yield pressure was reached.

It would seem that most of the pressure measurements that are made are attempts to record the force produced by the sphincter muscles. Wankling *et al.*[20] and Collins *et al.*[44,45] attempted to develop strain gauge systems to measure radial force in the anal canal. The method could not give absolute resting force values, but on voluntary contraction the multiple force gauge gave results which can be explained quite reasonably on the basis of the anatomical arrangement of the striated muscles. Collins and colleagues[44], comparing radial force measurements with balloon pressure measurements, found that the force profile along the anal canal in the resting state resembled the pattern of the pressure profile. However, pressure and force measurements do not always correspond. Since radial force measurements would seem to be a more reliable index of sphincter activity than pressure measurements, it is unfortunate that the multiple force gauge is difficult to construct and prone to mechanical failure.

Because of the concentric arrangement of the internal and external anal sphincters, the attribution of a recorded pressure or pressure change to activity in one or other of the sphincters can be particularly hazardous. Ideally, only pressure measurements supported by simultaneous observations of electrical activity should be interpreted in this way. Some researchers have believed that, by suitable positioning of recording devices in upper and lower parts of the anal canal, it is possible to measure pressure due to activity in internal and external sphincters separately[29,46]. Others do not accept that this is feasible, although it does seem that pressure recordings from the upper part of the canal are frequently interpreted in terms of internal sphincter activity without consideration of puborectalis function. In some studies, separation of the effects of internal and external sphincters has been achieved by abolishing external sphincter activity. Frenckner and v. Euler[11] applied a bilateral pudendal nerve block, while Duthie and Watts[9] and Varma and Stephens[47] used general anaesthetics and muscle relaxants. Varma and Stephens found a reduction of 69% of the resting pressure, while Frenckner and v. Euler[11] found a reduction of only 16% (both used balloon systems). Duthie and Watts[9] found that the percentage reduction depended on the measuring system used, being only 29% for open-tipped catheter measurements and 40% when a 5 mm diameter balloon was used. An intermediate reduction was found for a 3 mm diameter balloon. They interpreted these results as indicating that the external sphincter made a significant contribution to anal canal pressure only when a bolus was present. Bennett and Duthie[34] found, using open-tipped catheters, that complete section of the internal sphincter caused a reduction of resting pressure of almost 50%.

To add a final note of confusion to the subject of the source of the anal canal pressure, Varma and Stephens[47] report four cases of children who had had operations to correct congenital anorectal deformities in which the rectum was directed to the perineum through the puborectalis sling. Neither the internal nor the external sphincter was present in these cases. However, the resting pressure in this group ranged from 14 to 34 mmHg compared with a range of 13 to 50 mmHg for normal children. This would apparently indicate that the puborectalis alone can maintain a 'pressure barrier' in the anal canal.

Reflex activity of pelvic floor

A number of reflex responses of the system have been described, but little is known about the nervous pathways or the receptors involved. It is often difficult to define an effective stimulus, since the morphology of the system prevents the application of precisely defined, controlled stimuli. Several of the questions arising from consideration of reflex mechanisms have yet to be answered by anatomical and histological studies.

Information about muscle receptors in the anorectal musculature is somewhat scarce. Muscle spindles have been found both in the external sphincter[14,48] and the levator ani[49], but there has been no survey of the frequency and distribution of these receptors. Spindles have been examined histologically and electrophysiologically in the external sphincter of the cat[50]. Apart from a mention of Pacinian corpuscles observed in the region of the external sphincter[51], no other muscle receptors have been reported.

No organised sensory endings have been found within the wall of the rectum, despite the search for a 'rectal stretch receptor'. The rectal mucosa is relatively insensitive to touch, pressure, and temperature. The lining of the anal canal contains a wealth of receptors ranging from bare nerve endings to laminated corpuscular structures[51] and is very sensitive to light touch, pressure, pain, and temperature. Encapsulated endings of the forms found in the anal canal have been found to function as rapidly-adapting mechanoreceptors when studied in other sites[52,53], though this, of course, is no guarantee of the function of the anal canal receptors.

Effects of rectal distension

The effects on the anal sphincters of inflating a balloon in the rectum to simulate the progressive filling of the rectum with faeces have been extensively investigated. Descriptions of the responses observed in various studies differ in detail. Pressure recorded in the upper anal canal falls when the rectum is distended, the amplitude and duration of the response depending on rectal volume and/or pressure. For prolonged distension with small volumes (20 to 50 ml), the pressure fall is transient (10 to 15 seconds reported by Ihre[10]), but, as the rectal volume is progressively increased, the pressure takes longer to return to the baseline. At volumes above about 150 ml the pressure may remain at its lowest level for the duration of the distension[2,10], or stabilise at a level between this minimal level and the resting level[11,29,44]. The effectiveness of the balloon inflation in producing the pressure fall has been reported to increase as the balloon is moved distally from sigmoid colon to rectal ampulla[54,55], though Schuster *et al.*[29] found no difference in the response to balloons situated at 25 cm (in the rectosigmoid) and 15 cm (in the rectum) from the anal verge. Ihre[10] maintains that it is the value of intrarectal pressure rather than volume which is the major factor determining the anal response. The pressure response is sometimes biphasic, with an increase preceding the fall[2,11,15,47]. It is generally accepted that the drop in pressure represents relaxation of the internal sphincter. The pressure changes in the distal anal canal (where the internal sphincter does not act directly) may be the same as in the proximal canal[47], less pronounced but of the same form as in the proximal canal[2,9], or consist of a pressure increase only[2,30,31].

The increase in pressure, when observed, is accompanied by increased EMG activity in the external sphincter. This increased activity is transient at low rectal volumes, lasting only two to three seconds. The pattern of response as rectal volume is progressively increased is more variable. Melzak and Porter[5] found that relaxation followed the increase in activity, with this relaxation becoming more marked as the intrarectal volume increased until finally the external sphincter and whole pelvic floor relaxed. They and Frenckner (1975) found a similar pattern in paraplegics[15]. In these cases, relaxation was followed by automatic expulsion of the balloon. Other studies have shown the inhibition at high rectal volumes to be present in a varying proportion of normal subjects, indicating different amounts of cerebral interference with the spinal reflex pattern. Ihre[10] found inhibition occurred in approximately one half of all subjects by the time maximal tolerable volume of the rectal ampulla was reached. Frenckner[15], while finding inhibition in seven out of eight paraplegic subjects, found it in only two out of eight normal controls. Duthie and Watts[9] reported transient inhibition only at degrees of rectal distension sufficient to cause pain.

The influence of the external sphincter activity on the pressure recorded in the anal canal is difficult to determine. While the initial part of the biphasic pressure response (when observed) is attributed to external sphincter activity, EMG activity frequently continues during the ensuing pressure fall. Most studies seem to indicate that external sphincter activity has little influence on the pressure measurements when the internal sphincter is relaxed. Frenckner and v. Euler[11] tried to assess the contribution of the internal and external sphincters to pressure measured during rectal distension by comparing pressure before and after an anaesthetic block of the pudendal nerve to the external sphincter. They found the extent of the relaxation was greater after the block, and that the pressure fell to a lower level. They calculated that the internal sphincter activity accounts for 85% of anal pressure at rest, but only 40% during the pressure fall following sudden substantial rectal distension. It was assumed that the pressure recorded after the block was due entirely to the internal sphincter and no allowance was made for any mechanical component in the pressure measurements. These results do not agree with those of Duthie and Watts[9] who compared pressure in conscious subjects and in anaesthetised subjects in whom the external sphincter had been paralysed. Although the resting pressure was reduced in the latter group, the level to which the pressure fell on rectal distension was the same in both groups. These rather unexpected results seem to suggest that the external sphincter activity seen in the conscious subject during the pressure fall makes no contribution to measured pressure, and also that the drop in pressure after rectal distension is greater when the external sphincter is active than when it is paralysed. Experiments of this type demonstrate further the difficulty of interpretation of pressure measurements in terms of the separate activity of the two anal sphincters. Collins *et al.*[44] compared pressure measurements with measurements of radial force in the anal canal during rectal distension. They found the usual pressure fall, but there was no significant force response to rectal distension. The authors suggested that the pressure fall is due to passive mechanical effects.

It is generally accepted that the response of the internal sphincter is due to an intramural reflex, since Denny-Brown and Robertson[56] showed that the response is present in the absence of any spinal pathway. More recent results[21,22] have shown that, when autonomic nerve supply is blocked or absent, the pressure drop is still observed. The response is absent in children with Hirschprung's disease in which there is a lack of intramural ganglion cells.

Since rectal distension leads both to a reflex response of the external sphincter and a sensation of rectal fullness and the urge to defaecate, it has been postulated that there are receptors in the rectal wall responsive to stretch. It is not clear whether the nervous pathways subserving rectal sensation are the same as those in the afferent limb of the external sphincter spinal reflex. The existence of stretch receptors in the rectal wall is suggested by the results of some studies on patients in whom part of the rectum has been removed and the colon joined to the remaining rectal stump. Porter[3,4] and Parks *et al.*[4] found that distension below the anastomosis produced a normal external sphincter response, while distension above the anastomosis (at a distance from the anal verge which would normally produce a response) did not. However, there have been reports of low level anastomoses in which the normal external sphincter response can be produced by distension above the anastomosis[57]. The suggested alternative stimulus for the urge to defaecate (and the external sphincter response) is stretch of the puborectalis[47,55,57], but there are indications that this cannot be the only effective stimulus. Rectal sensation is unaffected by block of the nerve supply to the external sphincter and puborectalis[11], but is lost after section of the parasympathetic pelvic nerves[58]. It seems probable, therefore, that more than one pathway subserves rectal sensation in the normal subject.

Response to increased intra-abdominal pressure

Coughing produces a transient rise in intra-abdominal pressure, and an increase in the EMG activity of the external sphincter and puborectalis which correlates well with a rise in pressure in the anal canal[1,2,3,59]. This reflex response is seen in paraplegics[5]. An increase in anal canal pressure is also seen when a more prolonged rise in intra-abdominal pressure is produced during the Valsalva manoeuvre or straight leg raising[1,44]. A 'moderate' increase in EMG activity was seen[44]. Increases in radial force were much smaller than the corresponding pressure increases, particularly in the upper part of the anal canal[45].

Intra-abdominal pressures recorded during straining as if to defaecate vary from 10 to 180 cm water[1]. The EMG studies of Parks *et al.*[4] showed that, at the start of straining, activity in the external sphincter increased markedly, but this was followed by complete inhibition of the tonic activity. Kerremans[2] found patterns of EMG activity during straining that could be classified into four types: (1) a sudden abolition of all activity, (2) a pattern similar to that described by Parks *et al.*[4] consisting of an initial burst of activity followed by a moderate or reduced activity level with a final burst of activity at the end of the manoeuvre, (3) highly increased activity for the duration of straining, (4) a modified type of activity consisting of grouped potentials at high frequency. He does not say what proportion of the total number of cases each type represented, but notes that 80% of subjects showed increased activity for part or all of the time. Simultaneous anal canal pressure measurements showed that when the initial burst of EMG activity was present it was always accompanied by a rise in pressure, but this could be followed by no further change, by prolonged increase, or by decrease in pressure. Thus no correlation was found between EMG activity and pressure after the initial stage of straining. Rutter[60] also failed to find the clear-cut pattern of inhibition described by Parks *et al.*

The sequence of a burst of activity followed by inhibition during straining was also seen in paraplegic patients[5] in whom the level of cord transection permitted limited voluntary control of intra-abdominal pressure. This demonstrates that the reflex response to prolonged increases in intra-abdominal pressure is inhibition of the pelvic floor muscles, and might suggest that in normal subjects any prolonged excitatory response reflects cerebral interference. Since raised intra-abdominal pressure can present a threat to continence and is also used to assist defaecation, the complex reflex response of excitation followed by inhibition is interesting. EMG studies combined with simultaneous measurement of intra-abdominal pressure are required to determine under exactly what conditions the response changes from being one of excitation to one of inhibition. Porter[3] describes a similar pattern of response in the paraplegic subject to digital stretch of the puborectalis and to inflation of a balloon within the anal canal.

How closely the pattern of muscle activity seen during straining resembles the situation during actual defaecation is difficult to determine. Ihre[10] made pressure and EMG measurements during evacuation of a partially filled rectal balloon. Eleven out of 16 subjects managed to expel the balloon, and in eight of these 11 activity in the external sphincter and puborectalis disappeared during the straining effort. The remainder expelled the balloon despite continued sphincter activity. The cineradiographic studies[2] of the defaecation of radio-opaque faeces indicate co-ordinated, sequential relaxation of the levator ani and external sphincter. As a result of the relaxation of the levator ani and puborectalis, the anorectal junction moves downwards, its angulation is decreased, and the walls of the rectum become funnel-shaped. The anal canal becomes shorter, and the funnel shape of the rectum progressively lengthens to include the anorectal junction and the upper part of the anal canal. However, the distal part of the canal remains firmly closed until the upper canal and lower rectum are considerably stretched by the downward moving faecal mass. This would seem to contradict the widely held assumption that the pelvic floor muscles act as a unit. Shafik[61,62,63], as a result of detailed anatomical studies of the pelvic floor, has suggested that the mechanism of defaecation involves contraction of the levator ani diaphragm which acts to open the upper part of the anal canal, while relaxation of the external sphincter allows the passage of faeces through the canal. This proposed mechanism, however, would not account for the descent of the pelvic floor seen in cineradiographs of straining.

Theories about maintenance of continence

The physiological studies of the pelvic floor have so far failed to give a clear overall picture of the ways in which the actions of the puborectalis, external and internal anal sphincters, and rectum combine to maintain anal continence. In the literature, different views have been expressed about the importance of such things as the maintenance of an anal canal 'pressure barrier', the variety of reflex responses of the anal sphincters and the mechanical effects which result from the anatomical arrangement of the anorectum. Some of the 'theories of continence' were reviewed by Duthie[64].

It is generally agreed that the most important element in the system is the puborectalis sling. Any damage to this muscle results in major defects of continence. In children with congenital abnormalities of the anorectum, quite a high degree of continence can be achieved in the absence of internal and external sphincters provided that a functional puborectalis sling is present[47]. Several authors emphasise the importance of the angulation at the anorectal junction, which is maintained chiefly by the puborectalis. Parks[57] describes cases of idiopathic incontinence where the puborectalis is 'lengthened backwards' and the anorectal angulation abolished. Kerremans[2] points out that the more acute the angle between anal canal and rectum, the lower the squeeze force required for the retention of a faecal mass in the rectum. Parks *et al.*[27] (see also Parks[57] and Shafik[63]) suggest that the mucosa of the anterior rectal wall just above the anal canal can, because of the anorectal angle, act as a flap valve. When intra-abdominal pressure rises, they propose that the anterior wall of the rectum is forced down more firmly to occlude the anal canal. The puborectalis has also been credited with an important sensory function. Some believe that stretch of the puborectalis is an important stimulus leading to the urge to defaecate, and question the existence or significance of rectal stretch receptors in this regard[55,57,65].

It has been widely accepted that the overall squeeze action of the tonically active, concentrically arranged internal and external sphincters present a barrier to the passage of material through the anal canal. There is, however, quite a wide gap between the simple qualitative observation of some form of pressure zone in the anal canal and the interpretation of pressure changes in quantitative or functional terms. Certain discrepancies between force and pressure measurements[44] cast doubt on the interpretation of pressure changes in terms of sphincter muscle activity. Conventional resting pressure measurements have been rejected by some[32,36,37], as an index of sphincter competence. These authors maintain that the concept of 'resistance to opening' provides a more useful index. While Harris *et al.*[37] found a significantly lower resting yield pressure in incontinent than in continent subjects, Katz *et al.*[32] found no such correlation using their slightly different technique. A more obvious difference between continent and incontinent subjects in both studies lay in the maximum pressure recorded during voluntary contraction of the pelvic floor muscles. This finding seems to bring the emphasis back toward the contractile capacity of the sphincter muscles and the concept of squeeze.

The tonic or resting activity of the external sphincter (as distinct from the puborectalis) is likely to make little contribution to the maintenance of continence. Since maximal voluntary contraction of the external sphincter can be maintained for only very short periods[2,59], this can serve only as an emergency measure preventing entry of material into the anal canal. The major contribution of the external sphincter to continence would seem to lie in its reflex activity—for example, the excitatory response to transient rises in intra-abdominal pressure which present a threat to continence.

Assessment of the importance of the functioning of the internal sphincter presents perhaps even greater problems, since there is no really reliable indicator of its activity. Like the external sphincter, it is not essential to continence. Internal sphincterotomy (in which a varying proportion of the fibres are sectioned) can lead to minor defects of continence, chiefly of flatus and mucus (see Hardy[66], for the results of several studies of the effects of sphincterotomy). Most people do not regard the internal sphincter as being concerned with gross continence—for example, Hardy refers to it as a 'modifier' of continence. However, Frenckner and v. Euler[11] conclude that it is 'of major importance for continence at rest' as a result of their study of its contribution to anal canal pressure. Kerremans suggested a possible anti-peristaltic action of the internal sphincter as a consequence of an observed gradient in the frequency of pressure waves recorded from the anal canal (see also Gutierrez *et al.*[41]; Hancock[17]). He also suggested that the internal sphincter may influence the mechanical closure of the canal by regulating the balance of filling and emptying of the internal haemorrhoidal plexus.

Of clear importance to continence are the sensory pathways from the anorectal region which give awareness of rectal filling and the need to defaecate and also form the afferent side of the numerous complex reflexes of the pelvic floor. The importance of the reflex and voluntary responses to distension of the rectum has long been recognised. Gaston[54] emphasised the need to preserve rectal sensation after rectal resection. The voluntary contraction of puborectalis and external sphincter in response to the urge to defaecate is seen as a temporary measure required during rectal contraction or until the rectum adapts passively to a new volume and the level of intrarectal 'pressure is reduced[3]. Ihre[10], Todd[67] and Meunier[68] have stressed the importance of the level of rectal filling at which the urge to defaecate is experienced. It is necessary to have sufficient margin between the level of filling at which there is awareness of rectal content and the level at which both internal and external sphincters are reflexly inhibited.

Phillips and Edwards (1965) found that the behaviour of the anal sphincters was inadequate to explain the maintenance of continence, and suggested that some mechanical valve was necessary. They proposed a flutter valve mechanism acting at the level of the anal canal just after its passage through the pelvic diaphragm. The internal sphincter is seen as 'priming' the valve and keeping the walls of the slit-shaped canal in apposition, while positive intra-abdominal pressure serves to close the valve more tightly. This suggestion, however, has been criticised by Kerremans (1969) on anatomical grounds. It is true, though, that passive mechanical effects resulting from the anatomical arrangement of the anorectum should not be ignored in considering the mechanisms of continence.

The author wishes to thank Mr N. H. Porter of the New Sussex Hospital, and Dr N. B. Jones of Sussex University for their help in this work, and the South East Metropolitan Hospital Board for their financial support.

V. A. DICKINSON
The Graduate Division of Biomedical Engineering
School of Engineering and Applied Sciences
University of Sussex
Falmer
Brighton BN1 9QT

Received for publication 19 June 1978

References

[1]Phillips, S. F., and Edwards, D. A. W. (1965). Some aspects of anal continence and defaecation. *Gut*, **6**, 396-406.

[2]Kerremans, R. (1969). *Morphological and Physiological Aspects of Anal Continence and Defaecation.* Editions Arscia S.A.: Brussels.

[3]Porter, N. H. (1962). A physiological study of the pelvic floor in rectal prolapse. *Annals of the Royal College of Surgeons of England*, **31**, 379-404.
[4]Parks, A. G., Porter, N. H., and Melzak, J. (1962). Experimental study of the reflex mechanism controlling the muscles of the pelvic floor. *Diseases of the Colon and Rectum*, **5**, 407-414.
[5]Melzak, J., and Porter, N. H. (1964). Studies of the reflex activity of the external sphincter ani in spinal man. *Paraplegia*, **1**, 277-296.
[6]Parks, A. G., Porter, N. H., and Hardcastle, J. (1966). The syndrome of the descending perineum. *Proceedings of the Royal Society of Medicine*, **59**, 477-482.
[7]Floyd, W. F., and Walls, E. W. (1953). Electromyography of the sphincter ani externus in man. *Journal of Physiology*, **122**, 599-609.
[8]Taverner, D., and Smiddy, F. G. (1959). An electromyographic study of the normal function of the external anal sphincter and pelvic diaphragm. *Diseases of the Colon and Rectum*, **2**, 153-160.
[9]Duthie, H. L., and Watts, J. M. (1965). Contribution of the external anal sphincter to the pressure zone in the anal canal. *Gut*, **6**, 64-68.
[10]Ihre, T. (1974). Studies on anal function in continent and incontinent patients. *Scandinavian Journal of Gastroenterology*, **9**, suppl., 25.
[11]Frenckner, B., and von Euler, C. (1975). Influence of pudendal block on the function of the anal sphincters. *Gut*, **16**, 482-489.
[12]Tokizane, T., Kawamata, K., and Tokizane, H. (1952). Electromyographic studies on the human respiratory muscles. *Japanese Journal of Physiology*, **2**, 232-247.
[13]Kawakami, M. (1954). Electromyographic investigation on the human external sphincter muscle of the anus. *Japanese Journal of Physiology*, **4**, 196-204.
[14]Parks, A. G., Swash, M., and Urich, H. (1977). Sphincter denervation in anorectal incontinence and rectal prolapse. *Gut*, **18**, 656-665.
[15]Frenckner, B. (1975). Function of the anal sphincters in spinal man. *Gut*, **16**, 638-644.
[16]Penninckx, F., Kerremans, R., and Beckers, J. (1973). Pharmacological characteristics of the non-striated anorectal musculature in cats. *Gut*, **14**, 393-398.
[17]Hancock, B. D. (1976). Measurement of anal pressure and motility. *Gut*, **17**, 645-651.
[18]Aaronson, I., and Nixon, H. H. (1972). A clinical evaluation of anorectal pressure studies in the diagnosis of Hirschprung's disease. *Gut*, **13**, 138-146.
[19]Ustach, T. J., Tobon, F., Hambrecht, T., Bass, D. D., and Schuster, M. M. (1970). Electrophysiological aspects of human sphincter function. *Journal of Clinical Investigation*, **49**, 41-48.
[20]Wankling, W. J., Brown, B. H., Collins, C. D., and Duthie, H. L. (1968). Basal electrical activity in the anal canal in man. *Gut*, **9**, 457-460.
[21]Frenckner, B., and Ihre, T. (1976). Influence of autonomic nerves on the internal anal sphincter in man. *Gut*, **17**, 306-312.
[22]Meunier, P., and Mollard, P. (1977). Control of the internal anal sphincter (manometric study with human subjects). *Pflugers Archiv*, **370**, 233-239.
[23]Garrett, J. R., Howard, E. R., and Jones, W. (1974). The internal anal sphincter in the cat: a study of nervous mechanisms affecting tone and reflex activity. *Journal of Physiology*, **243**, 153-166.
[24]Shepherd, J. J., and Wright, P. G. (1968). The response of the internal anal sphincter in man to stimulation of the presacral nerve. *American Journal of Digestive Diseases*, **13**, 421-427.
[25]Friedmann, C. A. (1968). The action of nicotine and catecholamines on the human internal anal sphincter. *American Journal of Digestive Diseases*, **13**, 428-431.
[26]Parks, A. G., Fishlock, D. J., Cameron, J. D. H., and May, H. (1969). Preliminary investigation of the pharmacology of the human internal anal sphincter. *Gut*, **10**, 674-677.
[27]Parks, A. G., Fishlock, D. J., Cameron, J. D. H., and May, H. (1966). Catecholamine release in the lower gastro-intestinal tract (Abstract). *Gut*, **7**, 104.
[28]Shepherd, J. J. (1972). The nerve supply of the internal anal sphincter. *Australian and New Zealand Journal of Surgery*, **42**, 50-52.
[29]Schuster, M. M., Hendrix, T. R., and Mendeloff, A. I. (1963). The internal anal sphincter response: manometric studies on its normal physiology, neural pathways and alteration in bowel disorders. *Journal of Clinical Investigation*, **42**, 196-207.
[30]Schuster, M. M., Hookman, P., Hendrix, T. R., and Mendeloff, A. I. (1965). Simultaneous manometric recording of the internal and external anal sphincteric reflexes. *Bulletin of the Johns Hopkins Hospital*, **116**, 79-88.
[31]Alva, J., Mendeloff, A. I., and Schuster, M. M. (1967). Reflex and electromyographic abnormalities associated with fecal incontinence. *Gastroenterology*. **53**, 101-106.
[32]Katz, L. A., Kaufmann, H. J., and Spiro, H. M. (1967). Anal sphincter pressure characteristics. *Gastroenterology*, **52**, 513-518.
[33]Long, W. J., and Nixon, H. H. (1971). Continence and the yield pressure of the anus. *Proceedings of the Royal Society of Medicine*, **64**, 1189-1190.
[34]Bennett, R. C., and Duthie, H. L. (1964). The functional importance of the internal anal sphincter. *British Journal of Surgery*, **51**, 355-357.
[35]Duthie, H. L., and Bennett, R. C. (1963). The relation of sensation in the anal canal to the functional anal sphincter; a possible factor in anal continence. *Gut*, **4**, 179-182.
[36]Harris, L. D., and Pope, C. E. (1964). "Squeeze" vs resistance: an evaluation of the mechanism of sphincter competence. *Journal of Clinical Investigation*, **43**, 2272-2278.
4 [37]Harris, L. D., Winnans, C. S., and Pope, C. E. (1966). Determination of yield pressures: a

method for measuring anal sphincter competence. *Gastroenterology*, **50**, 754-760.
[38]Wheatley, I. C., Hardy, K. J., and Dent, J. (1977). Anal pressure studies in spinal patients. *Gut*, **18**, 488-490.
[39]Dent, J. (1976). A new technique for continuous sphincter pressure measurement. *Gastroenterology*, **71**, 263-267.
[40]Hill, J. R., Kelley, M. L., Jr., Schlegel, J. F., and Code, C. F. (1960). Pressure profile of the rectum and anus of healthy persons. *Diseases of the Colon and Rectum*, **3**, 203-209.
[41]Gutierrez, J. G., Oliai, A., and Chey, W. Y. (1975). Manometric profile of the internal anal sphincter in man (Abstract). *Gastroenterology*, **68**, 907.
[42]Duthie, H. L., Kwong, N. K., and Brown, B. (1970). Adaptability of the anal canal to distension (Abstract). *British Journal of Surgery*, **57**, 388.
[43]Yanchev, V. G., (1963). Anal tonometry. *Federation Proceedings*, suppl. **22**, 110-113.
[44]Collins, C. D., Duthie, H. L., Shelley, T., and Whittaker, G. E. (1967). Force in the anal canal and anal continence. *Gut*, **8**, 354-360.
[45]Collins, C. D., Brown, B. H., Whittaker, G. E., and Duthie, H. L. (1969). New method of measuring forces in the anal canal. *Gut*, **10**, 160-163.
[46]Meunier, P., Mollard, P., and de Beaujeu, M. J. (1976a). Manometric studies of anorectal disorders in infancy and childhood: an investigation of the physiopathology of continence and defaecation. *British Journal of Surgery*, **63**, 402-407.
[47]Varma, K. K. and Stephens, D. (1972). Neuromuscular reflexes of rectal continence. *Australian and New Zealand Journal of Surgery*, **41**, 263-272.
[48]Walls, E. W. (1959). Recent observations on the anatomy of the anal canal. *Proceedings of the Royal Society of Medicine*, **52**, suppl. 85-87.
[49]Winckler, G. (1958). Morphologie et innervation du releveur de l'anus. *Archives d'Anatomie, d'Histologie, et d'Embryologie*, **41**, 79-95.
[50]Chennells, M. W. F., Floyd,. and Gould, R. P. (1960). Muscle spindles in the external anal sphincter of the cat. *Journal of Physiology*, **151**, 23P-24P.
[51]Duthie, H. L., and Gairns, F. W. (1960). Sensory nerve-endings in the anal region in man. *British Journal of Surgery*, **47**, 585-595.
[52]Iggo, A. (ed.) (1973). *Handbook of Sensory Physiology*, vol. 2. *Somatosensory System*. Springer: Berlin.
[53]Iggo, A., and Ogawa, H. (1977). Correlative physiological and morphological studies of rapidly adapting mechanoreceptors in cat glabrous skin. *Journal of Physiology*, **266**, 275-296.
[54]Gaston, E. A. (1948). Fecal continence following resections of various portions of the rectum with preservation of the anal sphincters. *Surgery, Gynecology, and Obstetrics*, **87**, 669-678.
[55]Scharli, A. F., and Kiesewetter, W. B. (1970). Defaecation and continence: some new concepts. *Diseases of the Colon and Rectum*, **13**, 81-107.
[56]Denny-Brown, D. E., and Robertson, E. G. (1935). An investigation of the nervous control of defaecation. *Brain*, **58**, 256-310.
[57]Parks, A. G. (1975). Anorectal incontinence. *Proceedings of the Royal Society of Medicine*, **68**, 681-690.
[58]Goligher, J. C., and Hughes, E. S. R. (1951). Sensibility of the rectum and colon: its role in the mechanism of anal continence. *Lancet*, **1**, 543-548.
[59]Fay, D. F. (1974). *Data Acquisition for the Modelling and Analysis of Neuromuscular Systems.* Thesis: University of Sussex.
[60]Rutter, K. R. P. (1974). Electromyographic changes in certain pelvic floor abnormalities. *Proceedings of the Royal Society of Medicine*, **67**, 53-56.
[61]Shafik, A. (1975). A new concept of the anatomy of the anal sphincter mechanism and the physiology of defaecation. II Anatomy of the levator ani muscle with special reference to puborectalis. *Investigative Urology*, **13**, 175-182.
[62]Shafik, A. (1976). A new concept of the anatomy of the anal sphincter mechanism and the physiology of defaecation. III The longitudinal anal muscle: Anatomy and role in anal sphincter mechanism *Investigative Urology*, **13**, 271-277
[63]Shafik, A. (1976). A new concept of the anatomy of the anal sphincter mechanism and the physiology of defaecation. V. The rectal neck. Anatomy and function. *Medical Journal of Cairo University*, **44**, 117-140.
[64]Duthie, H. L. (1971). Anal continence. *Gut*, **12**, 844-852.
[65]Varma, K. K. (1972). The role of the voluntary anal sphincter in the maintenance of faecal continence in normal and abnormal states. *Australian and New Zealand Journal of Surgery*, **42**, 52-55.
[66]Hardy, K. J. (1972). nvoluntary sphincter tone in the maintenance of continence. *Australian and New Zealand Journal of Surgery*, **42**, 48-50.
[67]Todd, I. P. (1959). Discussion on rectal incontinence. *Proceedings of the Royal Society of Medicine*, **52**, 91-93.
[68]Meunier, P., Mollard, P., and Marechal, J. M. (1976b). Physiopathology of megarectum: the association of megarectum with encopresis. *Gut*, **17**, 224-227.